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FALSE AND MISLEADING ADVERTISING

(FILTER-TIP CIGARETTES)

HEARINGS

BEFORE A

SUBCOMMITTEE OF THE COMMITTEE ON GOVERNMENT OPERATIONS HOUSE OF REPRESENTATIVES

EIGHTY-FIFTH CONGRESS

FIRST SESSION

JULY 18, 19, 23, 24, 25, AND 26, 1957

Printed for the use of the
Committee on Government Operations

STORE/30590



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(FILTER-TIP CIGARETTES)

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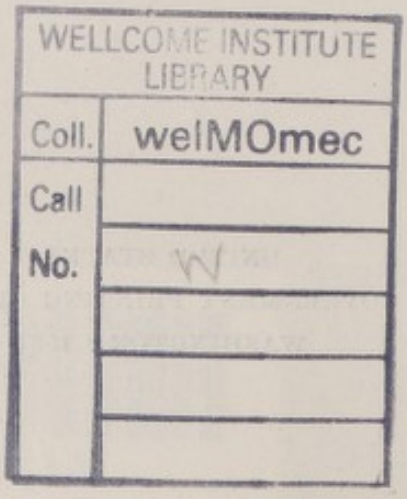
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FALSE AND MISLEADING ADVERTISING

(Filter-Tip Cigarettes)

THURSDAY, JULY 18, 1957

HOUSE OF REPRESENTATIVES,
LEGAL AND MONETARY AFFAIRS SUBCOMMITTEE OF THE
COMMITTEE ON GOVERNMENT OPERATIONS,
Washington, D. C.

The subcommittee met, pursuant to notice, at 10:15 a. m., in room 100, George Washington Inn, Hon. John A. Blatnik (chairman), presiding.

Present: Representatives Kilgore, Griffiths, Brooks, Hardy, Meader, Minshall, and Smith.

Also present: Jerome S. Plapinger, counsel; Curtis E. Johnson, staff director; and Elizabeth D. Heater, clerk.

Mr. BLATNIK. The Legal and Monetary Affairs Subcommittee of the House Government Operations Committee will please come into session for hearings on matters pertaining to fraudulent and misleading advertising.

I have a brief opening statement. This is a meeting of the Legal and Monetary Affairs Subcommittee, opening today what we hope will be the first of a series inquiring into the effectiveness of Federal agencies charged with protecting the public against advertising claims which may be false or misleading.

Congress has assigned this responsibility to the Federal Trade Commission, the Post Office Department, and the Food and Drug Administration.

As the art of advertising has been refined and as its influence has grown through newspapers, periodicals, radio, and television, the responsibilities of these agencies have grown accordingly.

Our hearings today are concerned with the advertising of cigarettes—particularly filter-tip cigarettes. This year Americans will smoke over 400 billion cigarettes. The cigarette industry has spent enormous sums to promote its products. On a number of occasions, the Federal Trade Commission has found it necessary to proceed against some of this advertising—principally with respect to claims that a particular brand of cigarette was less irritating to the human respiratory tract, or that it had less nicotine and tars than other brands.

Cigarette smoking and its effects on health have been a matter of major public concern now for over 5 years. This concern has grown principally from reports that cigarette smoking is a contributing cause to lung cancer. Other reports have suggested that it may also contribute to cardiovascular diseases.

May I state at this point that the committee does not and cannot go into the medical aspects of any possible relationship between any serious diseases and smoking as a subject in itself, or make any medical determination in this connection. But we feel it is necessary to know or be informed by the best possible sources from a professional level representing all points of view, in order to have a better backdrop upon which we superimpose this problem of the claims which are being made through advertising for the effectiveness of the filters in cigarettes.

These medical reports were first highly publicized about 5 years ago, in 1952 and 1953, and they had a very adverse effect on cigarette sales. The industry then countered by introducing filter cigarettes promoted by a tremendous advertising campaign, some of it featuring health protection or less nicotine and tars. The fears of the public were thus quieted, sales climbed again and are now at an alltime high.

About 5 years ago, of the total cigarette sales, only about 1.2 percent of all cigarettes sold and consumed were of the filter-type variety. Today it is estimated—and we shall verify this—that over 40 percent of all cigarettes sold are of the filter-tip variety.

The subcommittee has arranged for the appearance of scientific and medical witnesses who will present various points of view on the relationship of cigarette smoking to health in the opening phase of these hearings.

This testimony will be followed by representatives of testing laboratories, the tobacco industry, and the Federal Trade Commission.

May I state here—I know I speak for the committee—our intent is a positive and a constructive one. We are not here, either directly or indirectly, to cast any reflection or do any harm to any economic, professional, or social group or to any individual in the study of something which affects and concerns, and certainly is of interest to millions of American people. We hope to be informed and to learn from men who have spent years of study and research in this field all that is possible to learn as laymen, and then to check and to inform ourselves on the effectiveness and the efficiencies of governmental agencies that are directed by laws passed by Congress in protecting people from fraudulent and misleading advertising.

As I stated, this is the first of a series. We hope later on to get into medical products. The first one suggested as the next subject is the reducing field in which it is estimated that hundreds of millions of dollars are extracted from the consumers of America for products that are worthless and in some cases even harmful.

We hope to get into the so-called tranquilizing drugs that are merely old common drugs sold often not only in drugstores but over the soda fountains—again extracting hundreds of millions of dollars from the consumers of America for purposes that are questionable.

This briefly is the nature of the series of hearings. We pick on no one commodity. We pick on no one group or no one individual. We may now proceed.

We have as our first witness Dr. E. Cuyler Hammond, of the American Cancer Society, who will present his testimony on the observations and the findings of his organization here on the influence of smoking on the death rate.

Dr. Hammond, will you please take the chair.

Dr. Hammond, would you give your full name, your official capacity, and a brief summary of your professional background and major field of interest?

STATEMENT OF DR. E. CUYLER HAMMOND, DIRECTOR OF STATISTICAL RESEARCH, AMERICAN CANCER SOCIETY

Dr. HAMMOND. My name is Edward Cuyler Hammond. I am director of statistical research of the American Cancer Society and I am professor of science at Yale University at the present time.

I obtained my doctor's degree in the field of science in the year 1938. From there I went to the Public Health Service in industrial hygiene. During the war I was in the Air Force, part of the time in Washington and part of the time head of the Statistics Department, School of Aviation Medicine, at Randolph Field.

At the end of the war I worked on medical aspects of the atomic bomb and I got out of the Air Force and went to the American Cancer Society. Since that time I have been largely engaged in research on cancer.

Mr. BLATNIK. Will you please proceed with you statement, Doctor?

Dr. HAMMOND. Mr. Chairman, I want to apologize. I think I had better read this because there are so many pages in it. If I do it from memory I might make an error.

Mr. BLATNIK. Please proceed.

Dr. HAMMOND. Evidence that smoking is a serious health hazard has been accumulating slowly since about 1915. However, research on the subject has increased enormously during the last 8 years. The impetus for this was the alarming trend in the death rate from lung cancer. The reported number of deaths from this disease rose from 2,500 in 1930 to 18,000 in 1950 to an estimated 29,000 in 1956.

I should like to interject here that we are not certain that all of that rise is really a true rise. A part of it is probably due to better medical diagnosis, and a part of it is due to the aging of the population.

Nevertheless, it was alarming.

I would first like to give a brief description of the state of knowledge in 1950; then describe the results of a study we started in 1951 and just completed last month; and finally discuss the meaning of our findings in the light of other evidence.

As early as 1915, it was reported that cancer of the mouth occurs most frequently among people who smoke or chew tobacco. This has since been confirmed by several independent investigators. In 1928, Lombard and Doering of the Massachusetts Health Department found smoking to be more common among patients with various sorts of cancer than among people free of cancer.

By the end of 1950, nine independent groups of investigators in this country and abroad had reported a much higher percentage of smokers among men with lung cancer than among other groups of men. In 1936, an English scientist found an increased number of lung tumors among mice exposed to many different inhalants, including cigarette smoke. Three years later, an Argentinian scientist produced cancer on the skin of rabbits with tar distilled from tobacco. The tobacco wasn't smoked. It was distilled tar.

In 1938, Raymond Pearl, of the Johns Hopkins University, published the results of a study on smoking in relation to the life expec-

tancy of men. Among men in the middle age groups, the death rate of heavy smokers was found to be about double that of nonsmokers. Pearl made no mention of the causes of death.

However, such a large difference could hardly have occurred unless the death rate from cariovascular disease was higher among smokers than among nonsmokers. That is so because cardiovascular diseases make up such a large proportion of the total death rate.

In 1940, a group of doctors at the Mayo Clinic reported smoking to be more common among patients with coronary artery disease than among people in a suitable control group. This was again found in a study reported from a veterans' hospital in 1950. It has also been reported by at least two or three other investigators.

Investigators had reported that smoking has an acute effect on the circulatory system including (*a*) an increase in heart rate, (*b*) an increase in blood pressure, and (*c*) a constriction in peripheral blood vessels.

Clinical studies had shown that smoking has an extremely serious effect on patients with Buerger's disease, a rather rare circulatory disease which causes gangrene of the extremities.

Clinical studies on patients with peptic ulcers indicated that it is very difficult to cure this disease unless the patient gives up smoking.

In spite of all the evidence collected up to 1951, a number of investigators, myself included, were not convinced that smoking was an important factor in lung cancer. I was smoking two to four packs of cigarettes a day myself at that time, and I didn't like to believe it.

The major reason for my skepticism was doubt as to the validity of the method which had been used in studies showing an association between smoking and this disease. In all of the studies made up to that time, patients with lung cancer were not questioned about their smoking habits until after they developed the disease. For this reason, there was a theoretical possibility that illness biased the responses of the patients.

By 1951, the mounting evidence on smoking, together with the great increase in lung cancer, made it imperative to determine beyond reasonable doubt whether the total death rate as well as the lung cancer death rate was higher among smokers than among nonsmokers.

For this reason, two large-scale followup studies were started that year. One was conducted, and is still being carried on, in England by Doll and Hill under the auspices of the Medical Research Council. The other was conducted in this country by Hammond and Horn under the auspices of the American Cancer Society.

(See appendix, exhibit 1, p. 310.)

Dr. HAMMOND. I will now describe our study.

After designing and pretesting a smoking questionnaire, we trained over 22,000 American Cancer Society volunteers as researchers for the study. Each researcher was asked to get a smoking questionnaire filled out by about 10 white men between the ages of 50 and 69 whom she knew well and would be able to trace.

The researchers were told not to enroll a man if he was seriously ill or if they knew he had lung cancer. Once a year thereafter they reported on each man as "alive," "dead," or "don't know," and recorded all changes of address. A copy or abstract of the death certificate was obtained on each death reported.

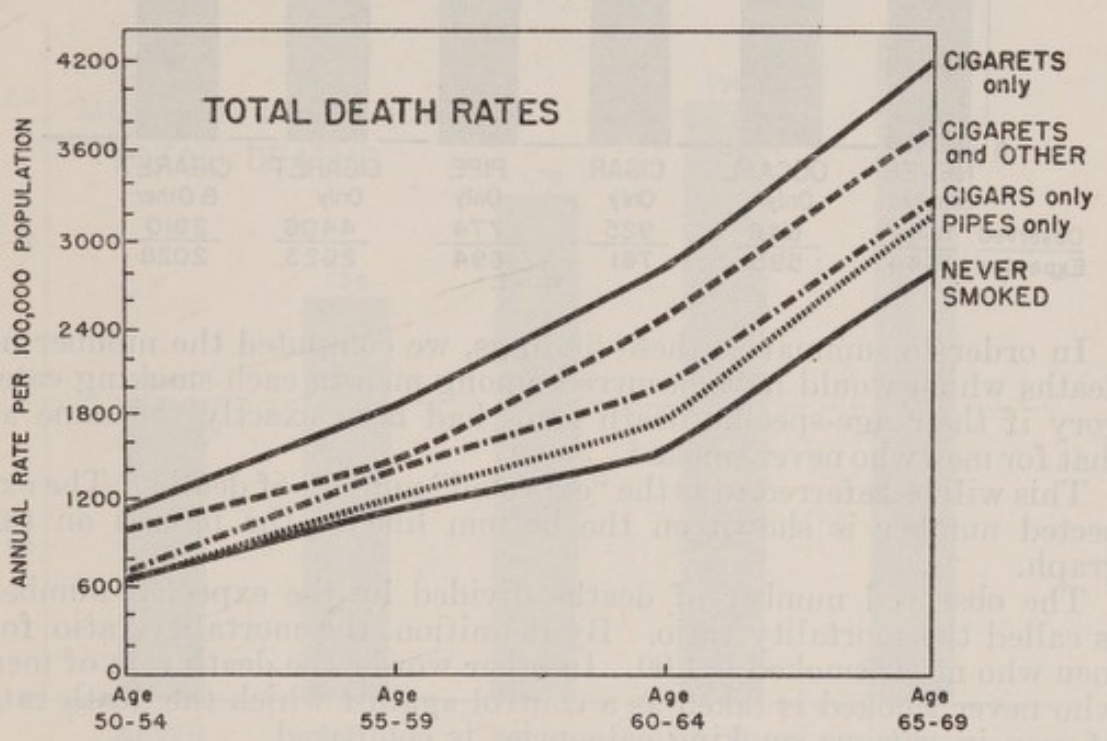
Whenever cancer was mentioned on a death certificate, further information was sought from the doctor, hospital, or tumor registry. The study area included 394 counties in 9 States: California, Illinois, Iowa, Michigan, Minnesota, New Jersey, New York, Pennsylvania, and Wisconsin.

A total of 187,783 men who filled out smoking questionnaires between January and June of 1952 were traced through October 31, 1955. Eleven thousand, eight hundred and seventy deaths were reported during this period of time and the total experience covered 667,753 man-years.

I have some lantern slides and with your permission I would like to show them.

Mr. BLATNIK. Doctor, for the record, these slides which you are to show us now are the same slides you also have reproduced and attached to the copy of your report?

Dr. HAMMOND. That is correct, sir. May I say, sir, what I am about to show is an abridged version of a longer report I read at the American Medical Association. I have cut it in length so as not to take up too much of your time.

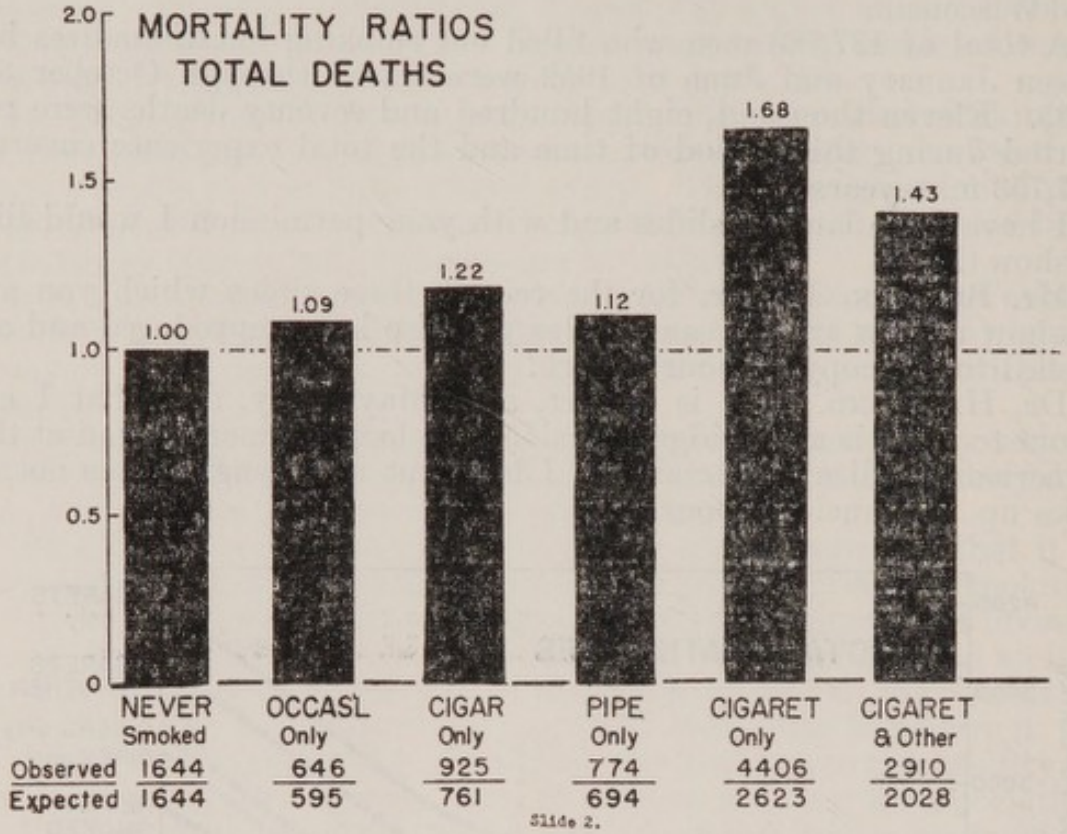


Slide 1. Total Death Rates by Type of Smoking (Lifetime History) and by Age at Start of Study

This slide (1) shows death rates per 100,000 man-years by type of smoking for each of four age groups. Age, as shown here and elsewhere in this report, refers to the ages of the men at the time they were questioned in 1952. Of course the men were growing older.

Note that in all four age groups, by far the highest death rate was that for men with a history of regular cigarette smoking only. Men who never smoked had the lowest death rate. Men with a history of regular cigarette smoking who also smoked cigars and pipes—that is the dotted line labeled cigarettes and other—had death rates somewhat lower than the death rates of men who smoked cigarettes only. The death rates of men who had only smoked pipes

were just slightly above the rates for men who never smoked. The death rates of cigar smokers were slightly higher than those for pipe smokers.



In order to summarize these findings, we computed the number of deaths which would have occurred among men in each smoking category if their age-specific death rates had been exactly the same as that for men who never smoked.

This will be referred to as the "expected" number of deaths. The expected number is shown on the bottom line of the figures on the graph.

The observed number of deaths divided by the expected number is called the mortality ratio. By definition, the mortality ratio for men who never smoked is 1.00. In other words, the death rate of men who never smoked is taken as a control against which the death rate of men in various smoking categories is compared.

Four thousand, four hundred and six deaths occurred among men with a history of regular cigarette smoking only. Just 2,623 of these men would have died between January 1952 and October 1955 had their age-specific death rates been the same as for men who never smoked. The mortality ratio is 1.68. In other words, the death rate of these cigarette smokers was 68 percent higher than the death rate of a comparable group of men who never smoked, age being taken into consideration.

The mortality ratio was 1.43 for men with a history of regular cigarette smoking who also smoked cigars or pipes. It is interesting that these men with mixed smoking habits had somewhat lower death rates than men who smoked cigarettes only. This is partly due to the fact that there were fewer heavy cigarette smokers among those with

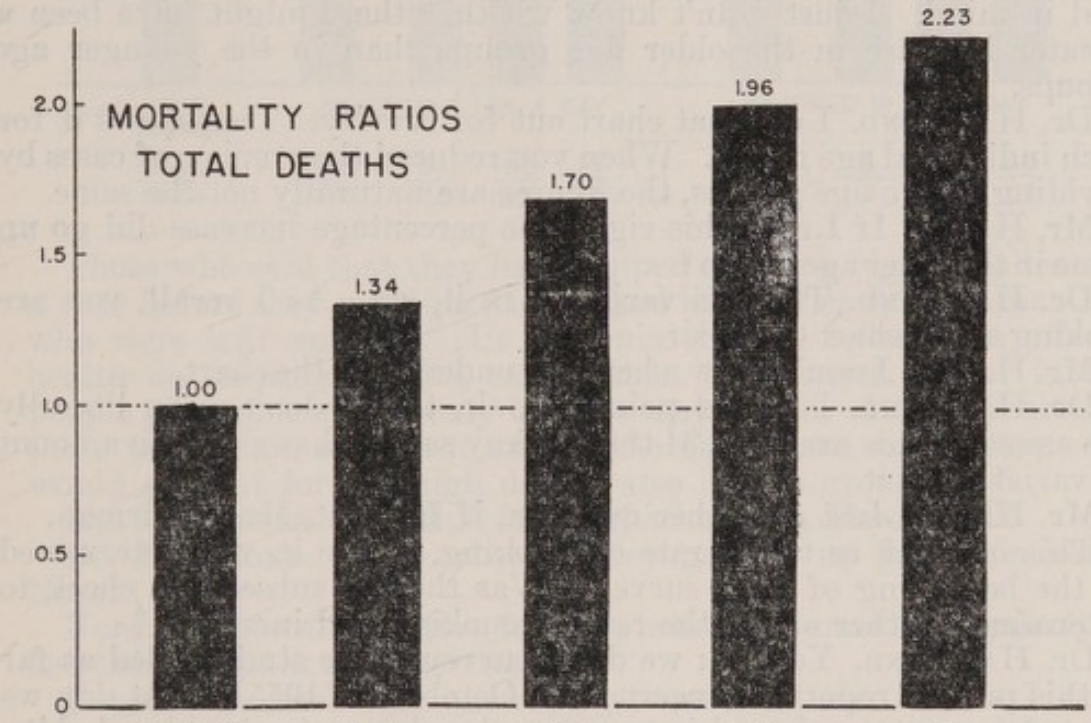
mixed habits than among those who smoked cigarettes only. However, this does not entirely account for the difference.

The mortality ratios were 1.22 and 1.12 respectively for men with a history of cigar smoking only and for men with a history of pipe smoking only, as shown by the blue bar and the yellow bar on the chart.

In both instances, the difference between the observed and expected number of deaths is statistically significant. However, the effect of pipe smoking seems to be small as compared with the effect of cigarette smoking.

Six hundred and forty-eight deaths occurred among men with a history of occasional smoking only as compared with 595 expected. This difference is not statistically significant. Therefore, it appears that occasional smoking has little or no effect on death rates.

The men with a history of regular cigarette smoking only were classified by their current amount of cigarette smoking at the time of questioning in 1952. In all four age groups death rates increased markedly with amount of cigarette smoking.



Slide 3. Mortality Ratios by Number of Cigarettes Smoked per Day

This slide (3) shows the same data summarized in the form of mortality ratios. The mortality ratio rose from (*a*) 1.00 for men who never smoked, (*b*) to 1.34 for under one-half-a-pack-a-day cigarette smokers, (*c*) 1.70 for one-half to 1 pack a day, (*d*) 1.96 for 1 to 2 packs a day, and (*e*) 2.23 for 2 pack or more a day.

In other words, the death rate of men smoking regularly at a rate of less than one-half a pack of cigarettes a day was 34 percent higher than the death rate of men who never smoked. The death rate of men smoking 1 to 2 packs of cigarettes a day was 96 percent higher than the death

rate of men who never smoked. The death rate of men smoking 2 packs or more of cigarettes a day was 123 percent higher than the death rate of men who never smoked. That is, more than double.

Mr. BLATNIK. Doctor, excuse me, Congressman Hardy has a question.

Mr. HARDY. Before you leave that chart, I would just like to know whether those figures relate to all age groups or whether there was any distinction made as to age groups with respect to the quantity of smoking?

Dr. HAMMOND. Sir, I left out one slide which I will be glad to show you here. Those relationships were true in the four age groups studied. We divided the men into age groups 50 to 54, 55 to 59, 60 to 64, and 65 to 69. This type of relationship, not the exact figures but this relationship, is true in all four age groups.

Mr. HARDY. The relationship was practically the same in all.

Mr. BLATNIK. We will recess for 15 seconds for a picture.

(A short recess was taken.)

Mr. BLATNIK. The committee is back in session.

Mr. HARDY. Mr. Chairman, that response answered the question I had in mind. I just didn't know whether there might have been a greater increase in the older age groups than in the younger age groups.

Dr. HAMMOND. I cut that chart out for brevity. This shows it for each individual age group. When you reduced the number of cases by dividing it into age groups, the figures are naturally not the same.

Mr. HARDY. If I read this right, the percentage increase did go up some in the older age group?

Dr. HAMMOND. There is variation in it, sir. As I recall, you are looking at the chart there, sir.

Mr. HARDY. I don't know whether I understand the chart.

Dr. HAMMOND. I should point out, sir, that I don't mean literally the association is exactly 1.34 there. Any sample has a certain amount of variation in it.

Mr. HARDY. Just one other question, if I might, Mr. Chairman.

This question as to the rate of smoking, I take it, was determined in the beginning of your survey. Was there a subsequent check to determine whether or not the rate of smoking had increased?

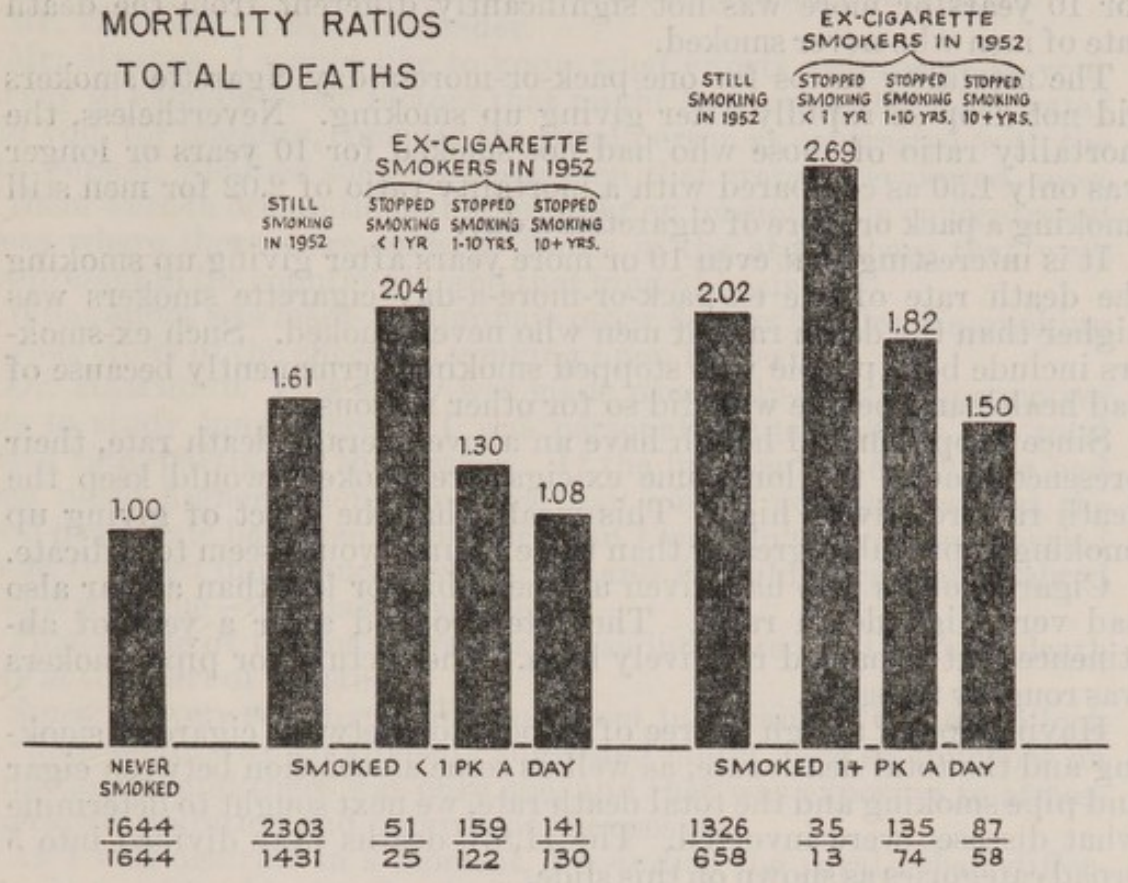
Dr. HAMMOND. Yes, sir; we did a survey. The study ended as far as this present report is concerned on October 31, 1955. That day we started requesting the subjects to see the change in smoking habits. We can't do it very often because people don't like to answer questionnaires very often.

Mr. BLATNIK. Please proceed, Doctor.

Dr. HAMMOND. I should point out that the corresponding rates for men with mixed smoking habits were somewhat lower than those shown on this slide.

Ten thousand and ninety-five men with a history of regular cigarette smoking only said that they had stopped smoking, that is, they had stopped at the time they were questioned in 1952. These men were classified by the length of time since last smoking and by their maximum previous amount of cigarette smoking.

Slide 6.



Those who said that they had stopped smoking cigarettes less than a year before they were questioned had higher death rates than those who were still smoking. In our opinion, this reflects the effect of health on smoking habits, rather than the reverse. That is, some people give up smoking only because they are ill. It is probable that there were some such men among the short-time ex-smokers. This would account for the high death rates in this group.

Mr. PLAPINGER. Was this factor checked, Dr. Hammond?

Dr. HAMMOND. As to whether they gave it up for their health? No, sir.

Note the mortality ratios for men who once smoked regularly but less than one pack of cigarettes a day.

Could I go back and answer that question, sir. What I said a moment ago was an interpretation of ours—that is, that it resulted from some people giving it up because they were sick. The alternative explanation which I doubt is that there is a severe withdrawal effect from giving up smoking.

It is true that in some very serious habit-forming drugs when a person is taken off of it, it has serious effects. That may be so with smoking, but I have no information or no guess that it is so. Perhaps someone else will have something to say on that.

Note the mortality ratios for men who once smoked regularly but less than one pack of cigarettes a day. The mortality ratio for those who had given up smoking 1 to 10 years before questioning was 1.30 as compared with a mortality ratio of 1.61 for men who were still smoking at this level. The death rate of those who had not smoked

for 10 years or more was not significantly different from the death rate of men who never smoked.

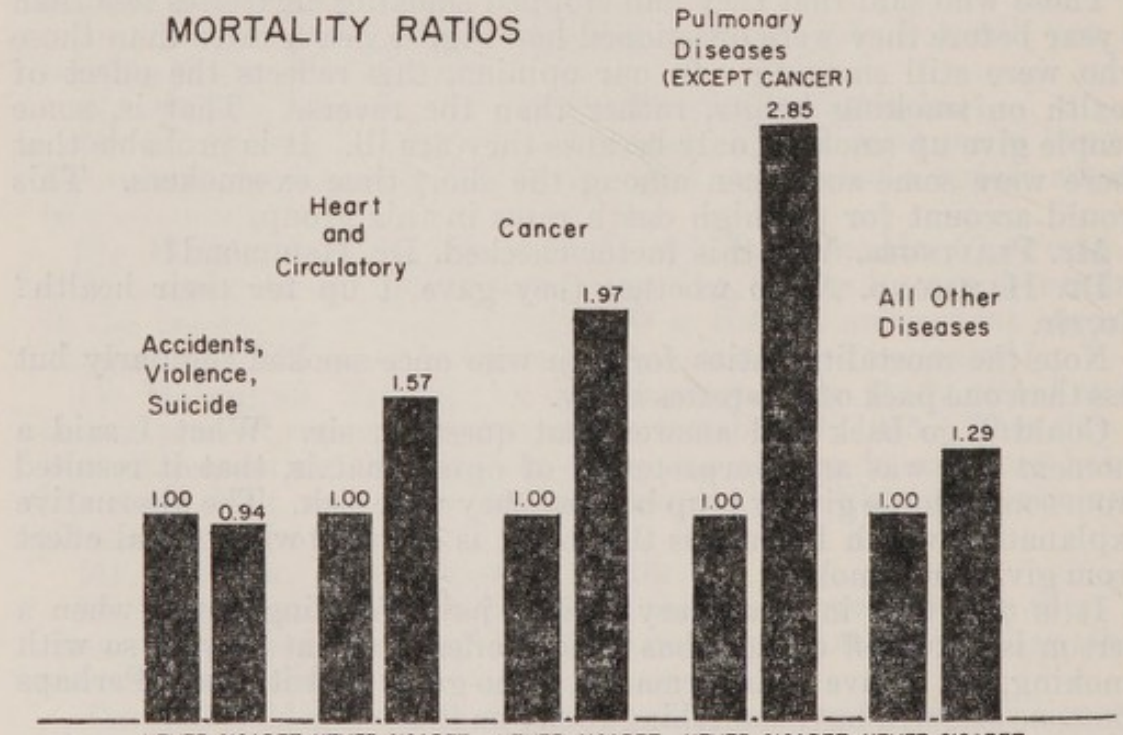
The mortality ratios for one-pack-or-more-a-day cigarette smokers did not drop so rapidly after giving up smoking. Nevertheless, the mortality ratio of those who had not smoked for 10 years or longer was only 1.50 as compared with a mortality ratio of 2.02 for men still smoking a pack or more of cigarettes a day.

It is interesting that even 10 or more years after giving up smoking the death rate of the ex-pack-or-more-a-day cigarette smokers was higher than the death rate of men who never smoked. Such ex-smokers include both people who stopped smoking permanently because of bad health and people who did so for other reasons.

Since people in bad health have an above-average death rate, their presence among the long-time ex-cigarette-smokers would keep the death rate relatively high. This meant that the effect of giving up smoking is probably greater than these figures would seem to indicate.

Cigar smokers who had given up the habit for less than a year also had very high death rates. The rate dropped after a year of abstinence, but remained relatively high. The picture for pipe smokers was roughly the same.

Having found a high degree of association between cigarette smoking and the total death rate, as well as some association between cigar and pipe smoking and the total death rate, we next sought to determine what diseases were involved. The 11,870 deaths were divided into 5 broad categories as shown on this slide.



Slide 5. Mortality Ratios by Major Causes of Death. Cigarette Smokers Compared with Men who Never Smoked.

Mr. MEADER. Mr. Chairman, might I ask a question at this point?

Mr. BLATNIK. Yes, Mr. Meader.

Mr. MEADER. I would like to know what efforts were made in your survey to exclude other factors that might bear upon the death rate? Did you, for instance, consider the rural person as contrasted with the city person, whether some of these men that were interviewed, most of them were in occupations that brought on tension or in concentrated areas where there were perhaps fumes in the atmosphere that were not present in the country air, and that kind of business?

What effort was made to eliminate other factors besides the smoking in your study of the effects of smoking upon longevity?

Dr. HAMMOND. This study was made primarily—our major motive was to study lung cancer. I was personally interested in the total death rate, partly for this reason. If the lung-cancer death rate had been much higher among smokers than among nonsmokers, but the total death rate had been no higher, then I wouldn't have been worried about the high lung-cancer death rate, and I would have even doubted that it was true—if I make myself clear.

That is one of the main reasons I was interested in the total death rate at the start of the study.

Since we were seeking to find out about lung cancer, we had to look at those factors which anybody suggested might have an influence on lung cancer, and there was only one, and that was exposure to atmospheric air pollution and occupational exposure.

As I will describe in a moment, we studied the rural-urban difference in considerable detail. As far as occupation is concerned, we did not ask it on the original smoking questionnaire primarily because studies of that are very hard to do and if you ask too many questions, people refuse to cooperate.

However, we did get the occupation of men after they had died from the death certificate, and the distribution by occupation of smokers and the nonsmokers, there was hardly any difference at all.

The survey made by the Census Bureau shows there is very little occupational variation in smokers and nonsmokers.

Mr. MEADER. Do you have any way of telling of the 187,000 men interviewed, how many were city dwellers and how many were rural?

Dr. HAMMOND. Yes, sir. I will give those figures in a moment.

Mr. MEADER. That is all, thank you.

Mr. BLATNIK. Please proceed, Doctor.

Dr. HAMMOND. If you would like, sir, I have some slides showing this urban and rural effect, which I don't have in the record. But I have them here to answer just those questions. I can do that later, if you wish, sir.

The death rate from accidents, violence, and suicide was almost exactly the same for men with a history of regular cigarette smoking as for men who never smoked. That is, accidents and violence and suicide grouped together, heart and circulatory, cancer, pulmonary diseases other than cancer, and all other causes.

In contrast, 1,460 cigarette smokers died of cancer compared with an expected of only 741 deaths had their age-specific cancer death rates been the same as for men who never smoked. The mortality ratio was 1.97.

The deaths of 4,593 cigarette smokers were attributed to diseases of the heart and circulatory system as compared with 2,924 expected; a difference of 1,669 deaths. The mortality ratio was 1.57.

Only 338 of the 11,870 deaths were attributed to pulmonary diseases other than lung cancer. They showed a very high degree of association with cigarette smoking. Two hundred thirty-one deaths of cigarette smokers were attributed to these pulmonary diseases as compared with only 81 expected. The mortality ratio is 2.85.

The 338 deaths included in this category consisted of 124 attributed to pneumonia or influenza, 41 to pulmonary tuberculosis, 76 to asthma, and 97 to other pulmonary diseases, including bronchitis, abscess of lung, pneumoconiosis, and bronchiectasis.

Seventy-eight men with a history of regular cigarette smoking died of pneumonia or influenza compared with 20 expected, the mortality ratio being 3.90, as shown by the red and green bar on the left of the chart. I should say to make it clear, the green bars are the relative death rate of the men who never smoked and the red are the cigarette smokers. The green is an index figure.

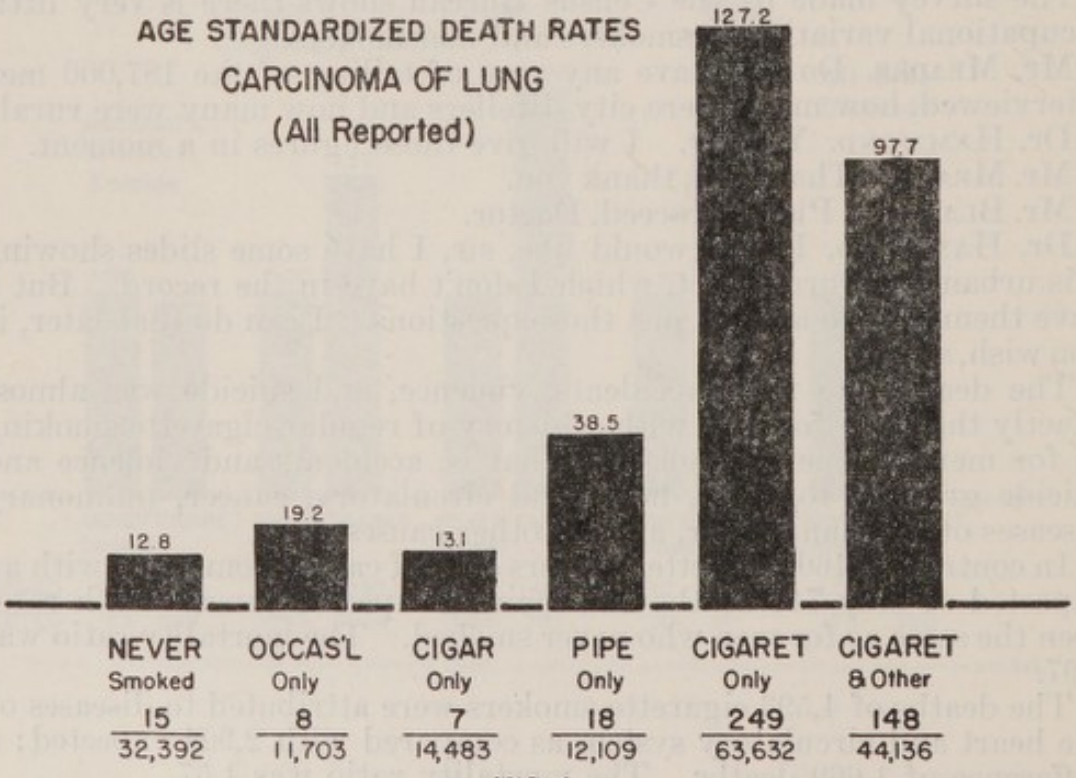
Twenty-six cigarette smokers died of pulmonary tuberculosis against 12 expected giving a mortality ratio of 2.17, based on a rather small number of cases.

Fifty-one cigarette smokers died of asthma against 29 expected, giving a mortality ratio of 1.76.

Other pulmonary diseases accounted for the death of 76 cigarette smokers compared with 21 expected, the mortality ratio being 3.62.

Deaths attributed to all other causes combined—including cause of death uncertain or unknown—accounted for less than 10 percent of the 11,870 deaths. Taken together, this group showed some association with cigarette smoking as shown by the mortality ratio of 1.29. As will be shown later, a few diseases in this category account for most of this relationship.

Four hundred and forty-eight deaths were attributed to primary cancer of the lung. Only 15 of these were men who never smoked. Including these 15, only 51 had never smoked cigarettes regularly, whereas 397 had a history of regular cigarette smoking.



Slide 6.

This slide (6) shows the age standardized lung-cancer death rates by type of smoking. The three lung-cancer deaths of men with a history of both pipe and cigar smoking are not shown on this lantern slide.

The figures at the bottom of the slide indicate the number of men enrolled in the study and the number of lung cancer deaths. The rates were very low indeed for men who never smoked, occasional smokers, and cigar smokers.

Pipe smokers had an appreciably higher rate. The rate for men with a history of regular cigarette smoking only was nearly 10 times as high as the rate for men who never smoked.

I should add that was not a statistically significant difference.

Of the 448 deaths, 32 were microscopically proved adenocarcinomas of the bronchus.

Mr. PLAPINGER. Dr. Hammond, on page 4 you said that the general mortality rate of cigar smokers is higher than that of pipe smokers. Is that right?

Dr. HAMMOND. That is right, in general. There is a slight difference.

Mr. PLAPINGER. I just was curious as to whether there was any conflict there, whether you could draw any conclusions from these figures.

Dr. HAMMOND. As I can show later, sir, these figures on pipe and cigar smokers are not dramatic at all. It isn't enough difference to get me to smoke a pipe or a cigar, but I have to present the findings. As you will see a little later, pipe smoking does some harm as far as lung cancer is concerned—not a great deal, but some. However, that seems to have no relation practically speaking, with coronary or heart diseases. With cigar smoking, it is the reverse.

It seems to have very little—no effect as far as I can see—on lung cancer, but it does seem to have some effect on heart diseases. Heart disease is so much more common than lung cancer that the overall effect of cigar smoking is a little bit worse. Frankly I wouldn't worry about it, but the figures are shown here.

Of the 448 deaths, 32 were microscopically proved adenocarcinomas of the bronchus. I will have to explain a little bit on this. The only way to diagnose cancer to any certainty is to look at a piece of tissue under the microscope. In a lung there are two principal types, and they look at it in a microscope and they see there are differences between them. There are two principal types. One type which is called adenocarcinoma has something of the appearance of blood.

The other type called epidermoid carcinoma, to put it in the simplest terms, looks more like the skin. It is a flattened cell. That is called epidermoid. The epidermoid is much the more common type, particularly in men.

The adenocarcinoma is a rarer type. A good many pathologists think the only form of lung cancer that has increased much is the epidermoid type. That is not proved but it is the opinion of a good many pathologists.

Back some years ago Dr. Evarts Graham, who performed the first operation that cured lung cancer, gave as his opinion that adenocarcinoma of the lung and epidermoid were two different diseases. They had two different causes. He thought one might be related to

smoking and not the other. It was for this reason that we separated them according to what pathologists and certain surgeons said.

Mr. PLAPINGER. Is this then the procedure in all of these studies?

Dr. HAMMOND. To separate the two?

Mr. PLAPINGER. Yes.

Dr. HAMMOND. I think most workers have done so wherever they have had the opportunity. I abridged this report in order not to take up too much of your time. I should have explained more fully that whenever cancer is approved on a death certificate, we then wrote to the doctor who signed the death certificate to get all the details we could, and wherever possible, we got the report from the pathologist. We classified these according to what the pathologist said.

As I will say in a moment, there are few cases in which there were an appreciable number in which it wasn't observed microscopically until we analyzed it separately. We made the separation only because many pathologists thought it important and we wanted to get the data for them.

I have shown it here both ways and everybody can draw their conclusions.

Mr. MEADER. Mr. Chairman, may I ask a question on that?

Mr. BLATNIK. Yes.

Mr. MEADER. Dr. Hammond, in these cases where there was a pathological examination, was the causative relationship found or was it merely assumed that because the person died and he had lung cancer, that lung cancer was the cause of death? Were other causes of death excluded?

Dr. HAMMOND. That is what the doctor gave us as his opinion of what caused the death. It was a professional opinion on the part of the doctor. There are some instances in which the doctor is called into a hotel room after the man has died, and he has never seen him before. In those cases it is nothing more than a guess.

Where a man has gone to a hospital, been treated, and particularly where they have done an autopsy afterwards, they are pretty certain of it.

There are instances in which there is a lot of doubt. There were quite a large number of people who died with cerebral hemorrhage—that is, stroke—and also with a heart attack. Either one of them would have probably killed the man. Both are written on the death certificate and we reported both. We have analyzed the figures. The doctor usually expresses an opinion of which killed the man.

For cancer, however, to be sure of it you have to look at the other end of the microscope. In 79 percent of the cases of cancer reported on the death certificate, it was proved microscopically—79 percent. The remaining 21 were not proved microscopically since some of them probably were not cancer. But the evidence is pretty good in most of the cases. There are few in which it was not good.

Mr. MEADER. And in these cases where lung cancer existed, the relationship between the existence of lung cancer and a death was determined by the pathologist, was it?

Dr. HAMMOND. I can't give you the exact number. About 10 percent of these were verified by autopsy, which is as good evidence as you can get. In the other cases it was the opinion of the doctors and in 79 percent of the cases it was also the opinion of the pathologists.

Cause of death is not always certain. We had about 10 deaths of people who died in automobile accidents and also had coronary attacks. On some of them we got quite a report from an inquiry as to whether the man first had a coronary and that made him run into a ditch, or whether he ran into a ditch and that brought on the coronary attack.

That is a very small percentage of the total, but such cases do exist.

I think I see what you are driving at, sir. Let me give you a case where I think there is a very real doubt.

I showed on that first slide a high degree of association between smoking and deaths attributed to pneumonia and influenza. Pneumonia is often a terminal cause of death. It happens simply when the person is dying, the lungs fill up. Unless they have done an autopsy, it is quite uncertain as to whether that was the real cause or whether it was simply the last straw.

It is perfectly possible that that association was due to a misdiagnosis of lung cancer—that is, some of those people who were not autopsied, where they didn't do an exploratory operation, they might have been put down as deaths due to pneumonia, whereas actually it was lung cancer.

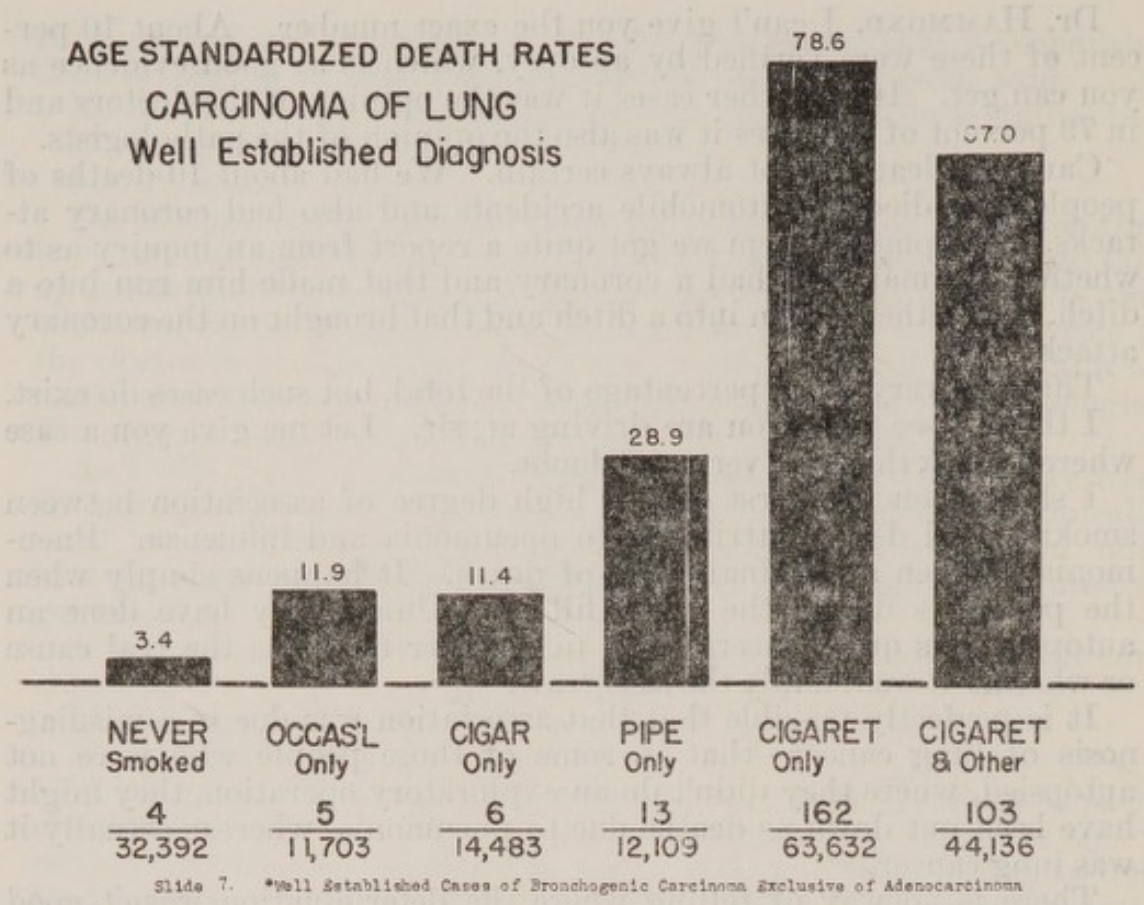
There is no way of telling where the determination wasn't good enough. Therefore I think there is really no effect of smoking on pneumonia at all, but that apparent effect was a further misdiagnosis of lung cancer.

Mr. MEADER. Thank you.

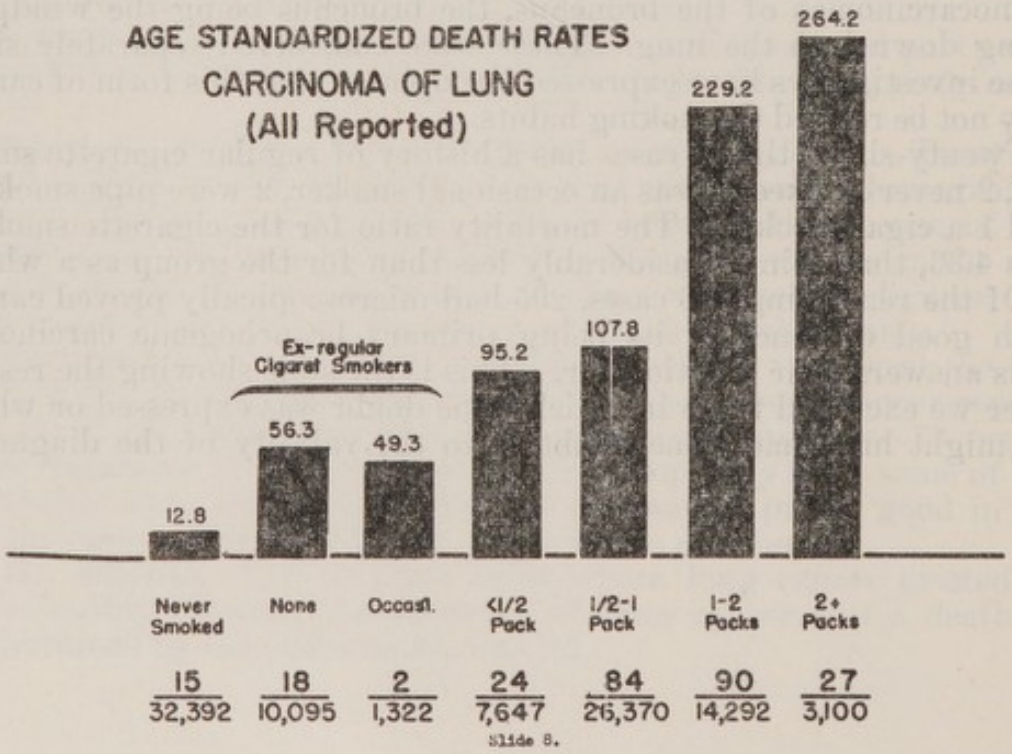
Dr. HAMMOND. Of the 448 deaths, 32 were microscopically proved adenocarcinomas of the bronchus, the bronchus being the windpipe going down into the lung. These were considered separately since some investigators have expressed the opinion that this form of cancer may not be related to smoking habits.

Twenty-six of the 32 cases has a history of regular cigarette smoking, 2 never smoked, 1 was an occasional smoker, 2 were pipe smokers, and 1 a cigar smoker. The mortality ratio for the cigarette smokers was 4.33, that being considerably less than for the group as a whole.

Of the remaining 416 cases, 295 had microscopically proved cancer with good evidence of its being primary bronchogenic carcinoma. This answers your question, sir. This is the slide showing the results after we excluded those in which some doubt was expressed or which we might have had some doubt as to the validity of the diagnosis.

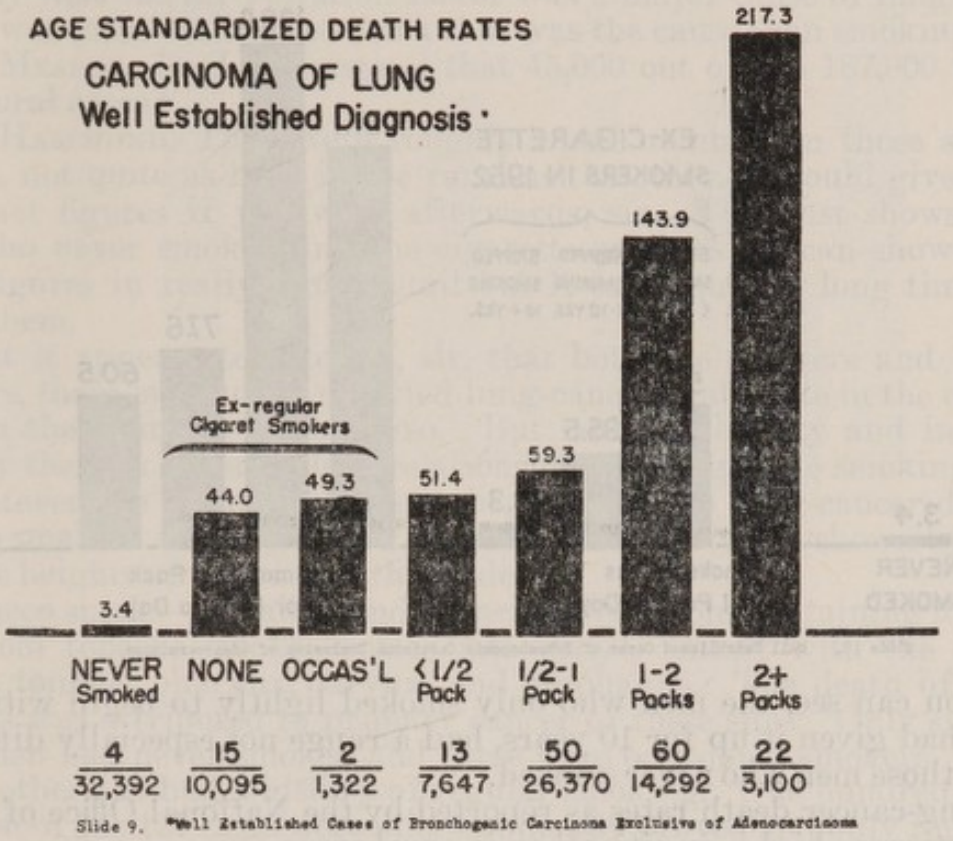


This slide (7) shows age-standardized death rates for these 295 cases. The picture is much the same as in the previous slide except that for these well-established cases the association with smoking habits is even more pronounced.



This slide (8) shows the age standardized death rates by amount of cigarette smoking, that is, the amount they were smoking in 1952,

for men with history of regular cigarette smoking only. Only 3,100 men who entered the study said that they smoked cigarettes only and were currently smoking 2 packs or more a day. Within the ensuing 44 months, 27 of these men had died of lung cancer. On the other hand, out of 32,392 men who never smoked, only 15 died with this diagnosis.



Slide 9. *Well Established Cases of Bronchogenic Carcinoma Exclusive of Adenocarcinoma

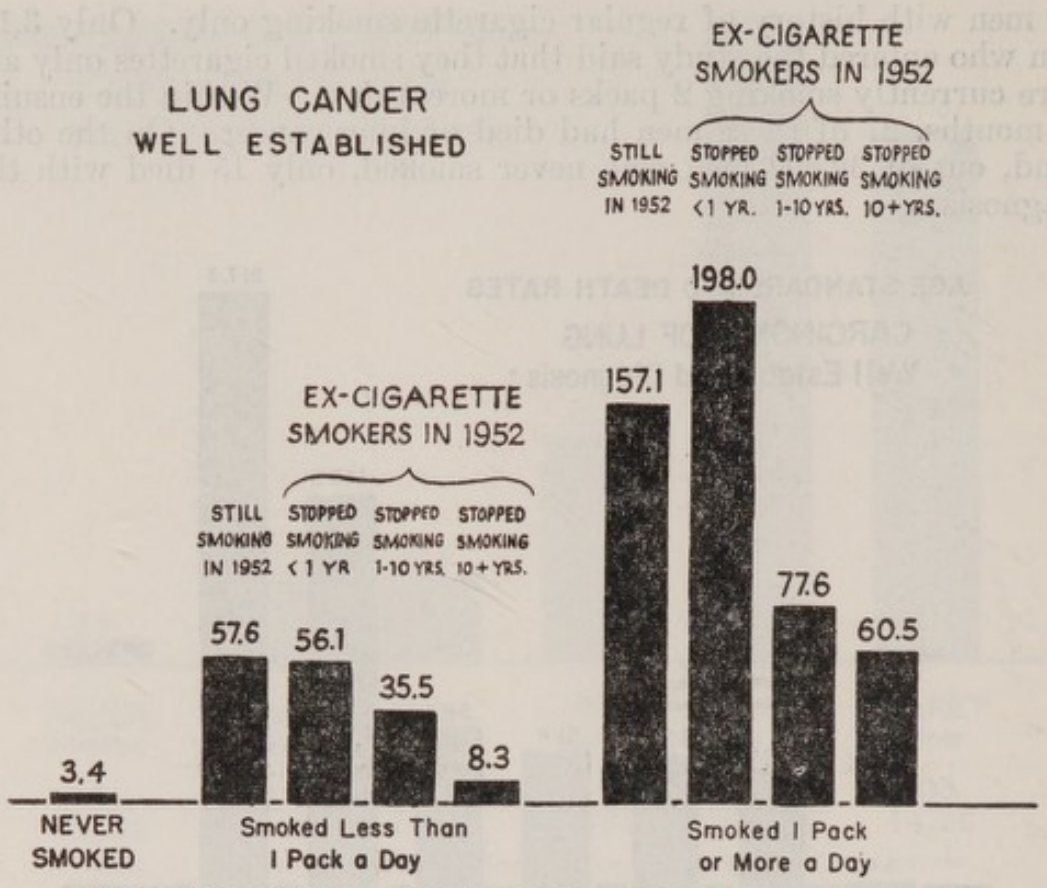
The slide (9) shows the figures for well-established cases of bronchogenic carcinoma other than adenocarcinoma. In each case we first looked at the total group and then we looked at those that are more serious. The age standardized death rate for the 2-pack-or-more-a-day smokers with this diagnosis was 217.3 per 100,000 per year.

In contrast, the age standardized death rate for the 2-pack-or-more-a-day smokers with this diagnosis was 217.3 per 100,000 per year. In contrast, the age standardized death rate from microscopically proved cancer of all sites combined was only 177.4 per 100,000 per year for men who never smoked. In other words, among two-pack-a-day cigarette smokers, the death rate from bronchogenic carcinoma alone is higher than the total cancer death rate of men who never smoked.

I mention that because no amount of confusion between primary sites can account for that finding, they being microscopically proved cases. There is uncertainty as to site sometimes.

Those of us who are ex-very-heavy-cigarette-smokers have something of a personal interest in the lung-cancer death rate of men who stopped smoking cigarettes.

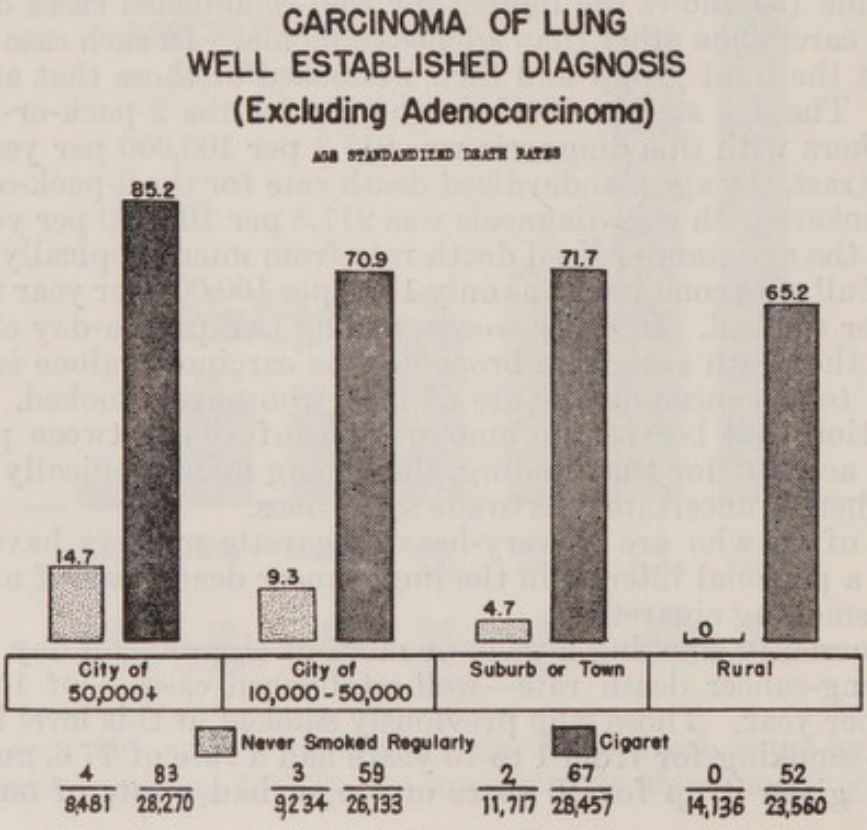
Men currently smoking a pack or more of cigarettes a day in 1952 had a lung-cancer death rate—well-established cases— of 157.1 per 100,000 per year. Those who previously smoked at this level but had given up smoking for from 1 to 10 years had a rate of 77.6, and those who had given it up for 10 years or longer had a rate of only 60.5.



Slide 10. Well Established Cases of Bronchogenic Carcinoma Exclusive of Adenocarcinoma

As you can see, the men who only smoked lightly to begin with and then had given it up for 10 years, had a range not especially different from those men who never smoked.

Lung-cancer death rates as reported by the National Office of Vital Statistics are higher in cities than in rural areas.



Slide 11.

However that may be, the lung-cancer death rate was low among men who never smoked cigarettes regularly and high among cigarette smokers in large cities, small cities, suburbs and towns, and rural areas. There were between 40,000 and 45,000 men in each of those areas. We deliberately went to rural areas in order to get an answer to this question. At the time the study was started, I thought it just as likely that the air-pollution factor was a major cause of lung cancer—I was more inclined to think that was the cause than smoking

Mr. MEADER. Do I understand that 45,000 out of the 187,000 were from rural areas?

Dr. HAMMOND. They were roughly equal numbers in those small groups, not quite as high in the rural as the other. I could give you the exact figures if you wish afterwards, sir. This just shows the men who never smoked and the cigarette smokers. I can show you these figures in really more detail but it takes quite a long time to study them.

What it appears to show is, sir, that both the smokers and nonsmokers, there is a higher reported lung-cancer death rate in the cities than in the country. That is so. But both in the city and in the country there is a tremendous relationship with cigarette smoking.

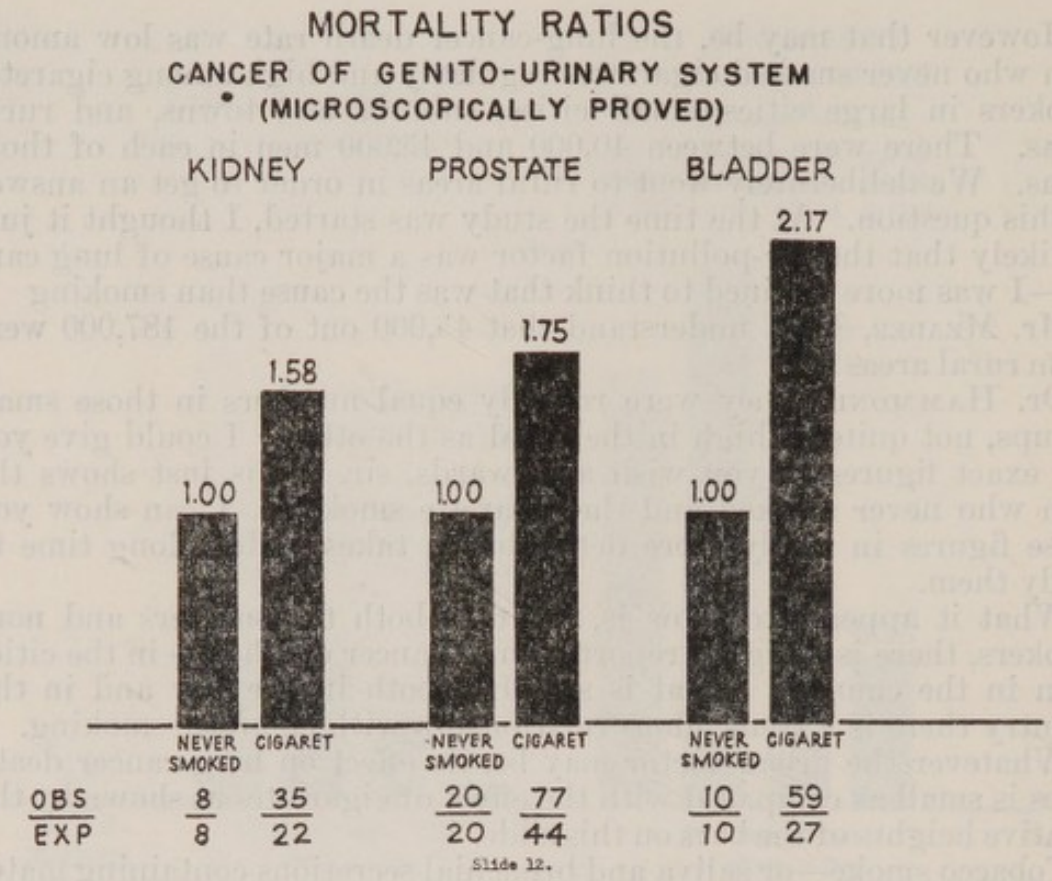
Whatever the urban factor may be, its effect on lung-cancer death rates is small as compared with the effect of cigarettes as shown by the relative heights of the bars on this slide.

Tobacco smoke—or saliva and bronchial secretions containing material from tobacco smoke—comes into direct contact with the lips, mouth, tongue, pharynx, larynx, and esophagus. The death of 127 subjects was attributed to primary cancer of these sites. Only six of these men had never smoked and three were occasional smokers.

The other 118 had a history of regular smoking. One hundred and fourteen of the 127 cases were microscopically proved and only four of these were men who never smoked. Considering microscopically proved cases only, the mortality ratio was 7.00 for men with a history of regular cigarette smoking; 5.00 for men who smoked only cigars; and 3.50 for men who smoked only pipes.

Still considering microscopically proved cases: Out of 34 deaths from cancer of the esophagus, only 1 was a man who had never smoked; of 25 pharynx cases 2 had never smoked; and of 16 tongue cases, 1 had never smoked. There were no men who never smoked among 24 larynx cases, 14 mouth cases, and 1 lip case.

I should say that the numbers are too small to be certain of this, but that it appears that for these sites of cancer, at least some of them pipe and cigar smoking and chewing of tobacco are probably as bad and probably may be worse than cigarette smoking for these particular sites.

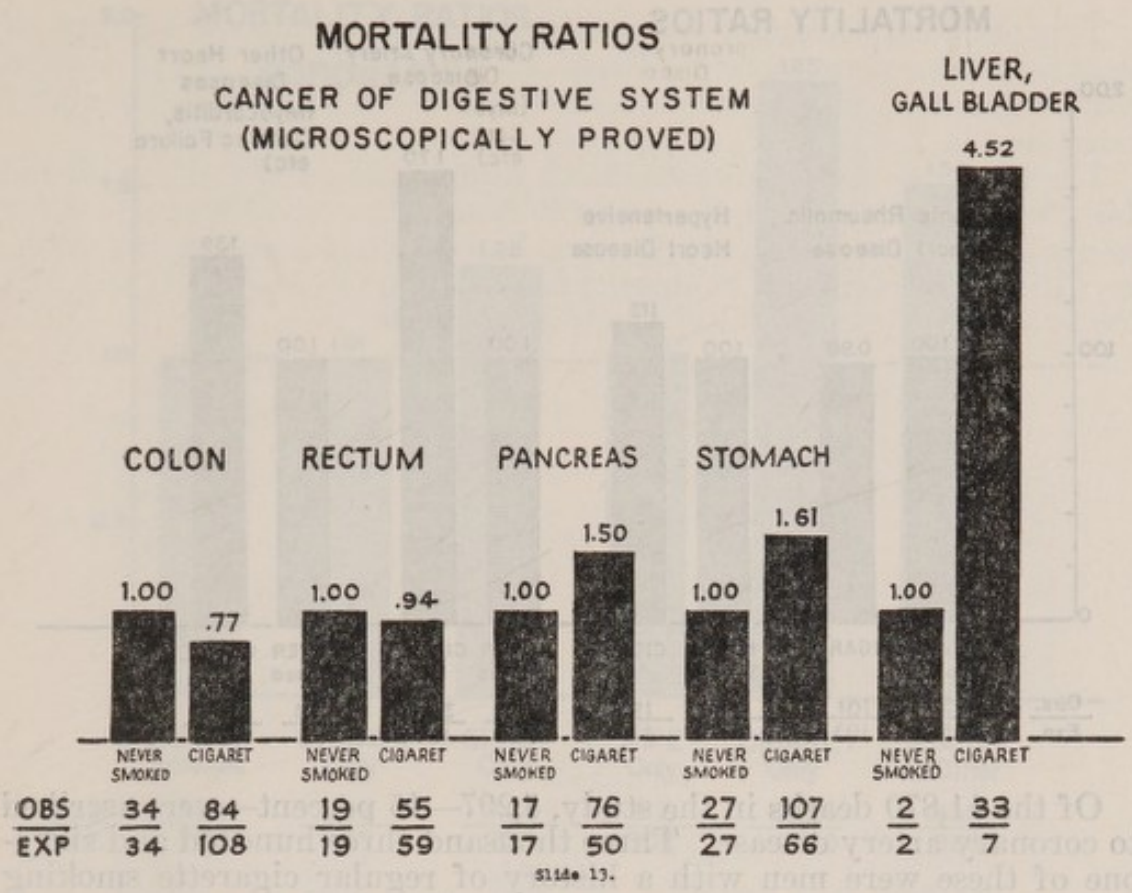


This slide (12) shows mortality ratios for microscopically proved cancer diagnosed as primary in the genito-urinary organs. The mortality ratio of cigarette smokers was 2.17 for cancer of the bladder, 1.75 for cancer of the prostate, and 1.58 for cancer of the kidney. In most of these cases, cancer was present at death in two or more of these sites as well as in other parts of the body. That is when a person gets cancer of the bladder, by the time he has died, it has spread to the surrounding areas into the kidneys and the prostate and in those cases it is not always easy to tell which of the particular sites it started in.

While cancer was proved microscopically, the evidence as to the exact primary site was far from conclusive in many instances.

I should interpose here that may mean that all of this association is due to one of the sites, perhaps cancer of the bladder, and the others are spread out.

There was no association between cigarette smoking and microscopically proved cancer of the rectum.



S1146 13.

The mortality ratio of cigarette smokers for cancer of the colon was 0.77. In other words, less than that for men who never smoked. This negative association for cancer of the colon, based on 84 observed against 108 expected deaths, is not statistically significant.

The mortality ratio of cigarette smokers was 1.61 for cancer of the stomach and 1.50 for cancer of the pancreas. In neither case was the difference between observed and expected deaths statistically significant.

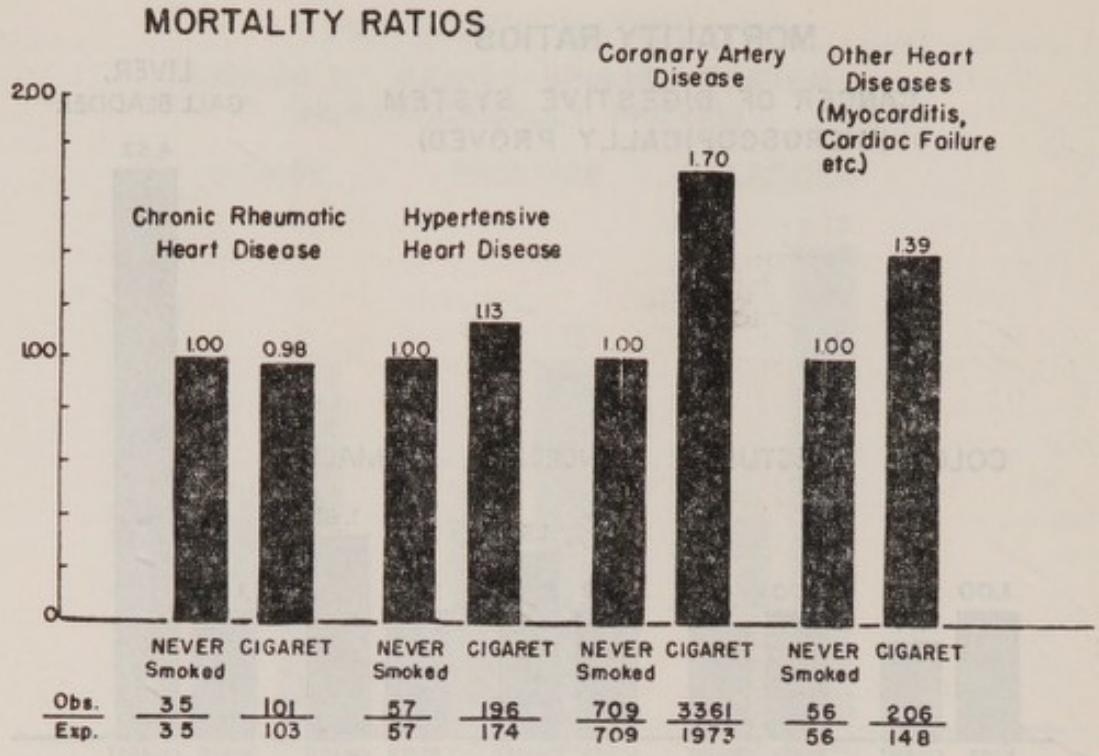
Deaths from cancer of the liver, gall bladder, and biliary passages appeared to be highly associated with cigarette smoking. However, there was a reasonable doubt as to the primary site in many of these cases. I should have said "most." I shall explain.

Cancer kills most often by spreading to some other part of the body. The liver is the place to which it most frequently spreads. In a very large proportion of all people who have cancer any place, it spreads to the liver and I make no assertion to what that means.

Personally I think it is very likely metastatic from some place else rather than actually liver cancer, but of that we don't know enough.

Leukemia showed no indication of an association with cigarette smoking.

Hodgkin's disease as well as lymphosarcoma and reticulosarcoma appeared to be associated with cigarette smoking, but not to a statistically significant degree.



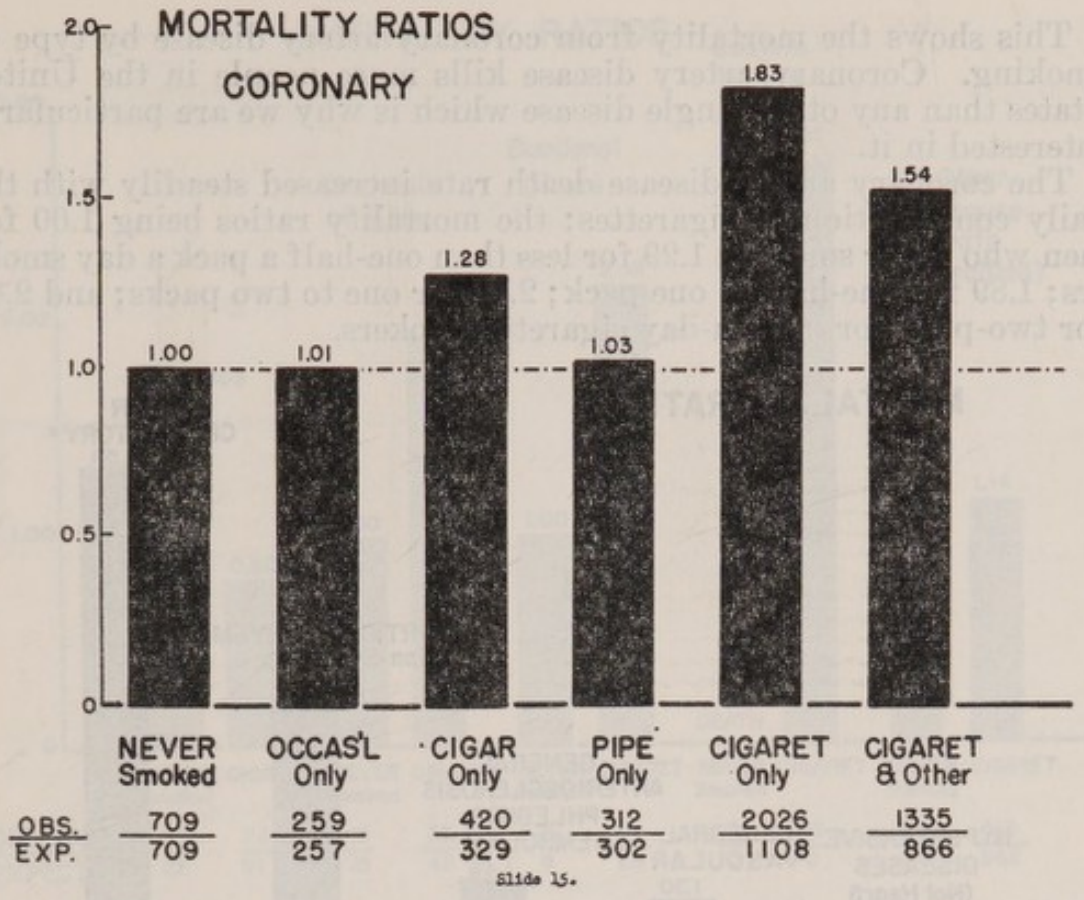
Of the 11,870 deaths in the study, 5,297—45 percent—were ascribed to coronary artery disease. Three thousand three hundred and sixty-one of these were men with a history of regular cigarette smoking whereas the expected number was only 1,973. This is a difference of 1,388 deaths and a mortality ratio of 1.70.

That is the high bar there for the coronary diseases.

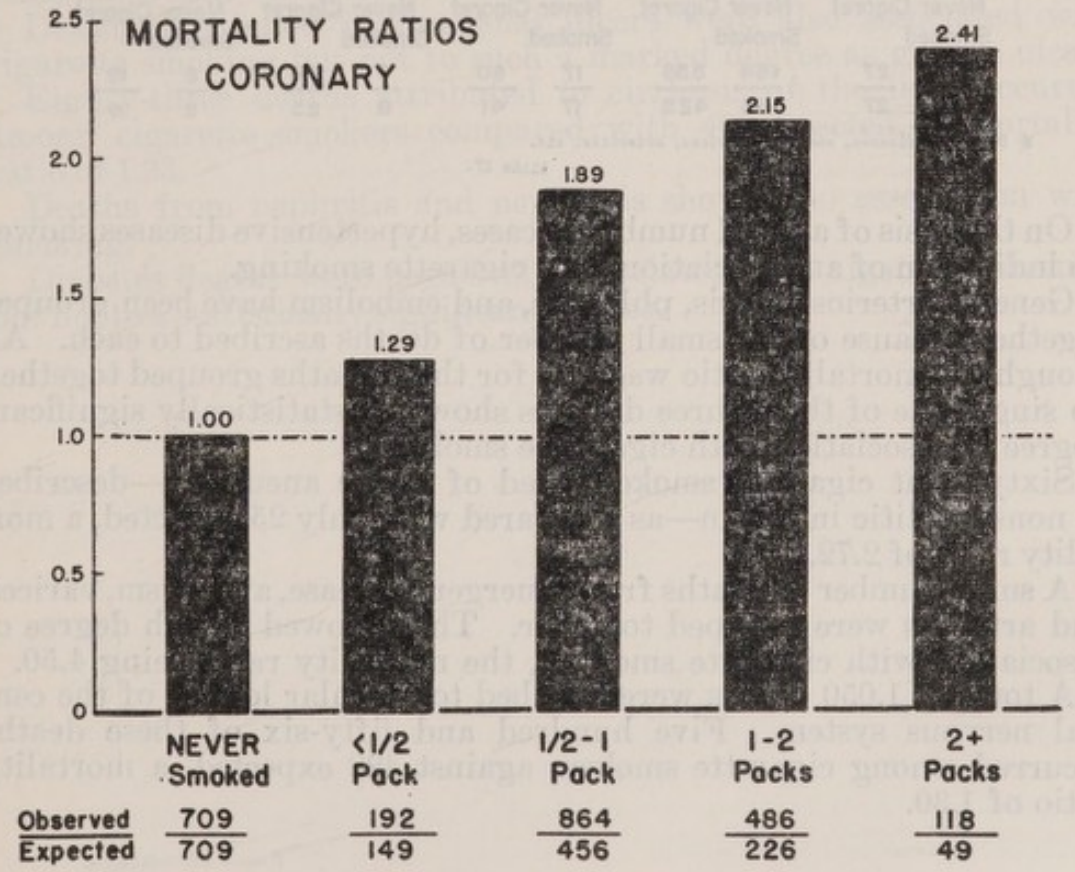
The death rate from chronic rheumatic disease was almost exactly the same for cigarette smokers as for men who never smoked.

The mortality ratio for deaths ascribed to hypertensive heart disease was 1.13, this being based on 196 observed against 174 expected deaths. This difference is not statistically significant.

Deaths described as due to myocarditis, cardiac failure, cardiac degeneration, and similar terms showed a positive association with cigarette smoking.

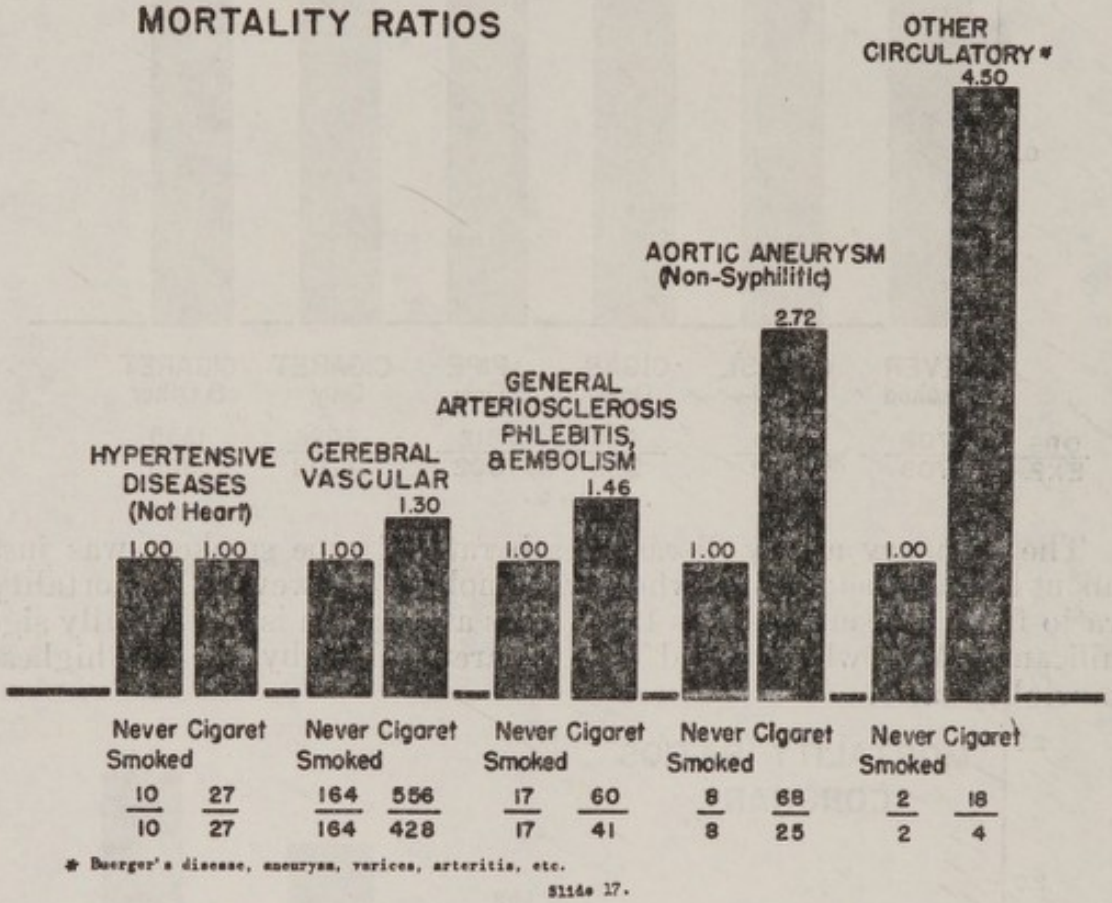


The coronary artery disease death rate of pipe smokers was just about the same as for men who never smoked. However, the mortality ratio for cigar smokers was 1.28. This association is statistically significant. Men who smoked only cigarettes had by far the highest mortality ratio.



This shows the mortality from coronary artery disease by type of smoking. Coronary artery disease kills more people in the United States than any other single disease which is why we are particularly interested in it.

The coronary artery disease death rate increased steadily with the daily consumption of cigarettes; the mortality ratios being 1.00 for men who never smoked; 1.29 for less than one-half a pack a day smokers; 1.89 for one-half to one pack; 2.15 for one to two packs; and 2.41 for two-packs-or-more-a-day cigarette smokers.



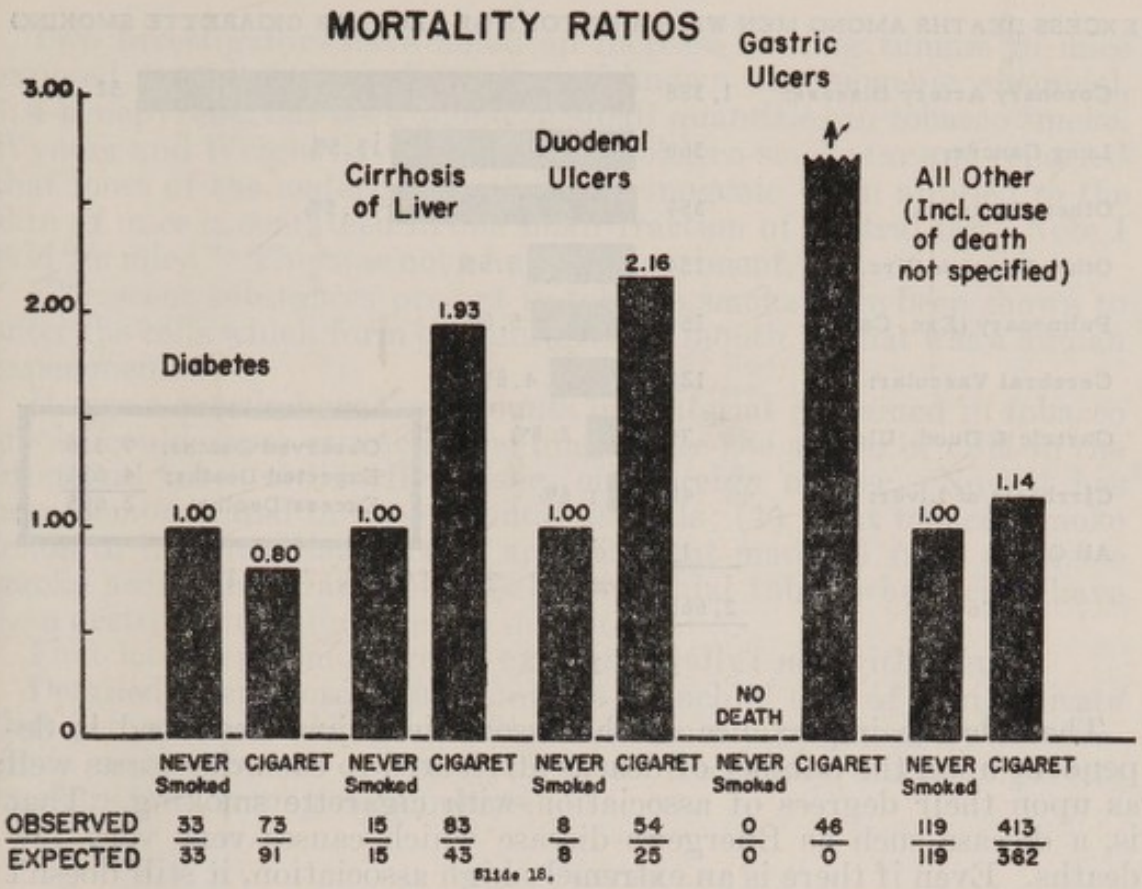
On the basis of a small number of cases, hypertensive diseases showed no indication of an association with cigarette smoking.

General arteriosclerosis, phlebitis, and embolism have been grouped together because of the small number of deaths ascribed to each. Although the mortality ratio was 1.46 for these deaths grouped together, no single one of these three diseases showed a statistically significant degree of association with cigarettee smoking.

Sixty-eight cigarette smokers died of aortic aneurysm—described as nonsyphilitic in origin—as compared with only 25 expected, a mortality ratio of 2.72.

A small number of deaths from Buerger's disease, aneurysm, varices, and arteritis were grouped together. They showed a high degree of association with cigarette smoking, the mortality ratio being 4.50.

A total of 1,050 deaths were ascribed to vascular lesions of the central nervous system. Five hundred and fifty-six of these deaths occurred among cigarette smokers against 428 expected, a mortality ratio of 1.30.



Fifty-one deaths were attributed to gastric ulcers. Forty-six of these were men with a history of regular cigarette smoking, 2 had a history of pipe smoking only, and 2 had a history of cigar smoking only, and 1 smoked both pipes and cigars. Not a single one of these cases was a man who never smoked.

Deaths attributed to duodenal ulcers were also associated with cigarette smoking but not to such a marked degree as gastric ulcers.

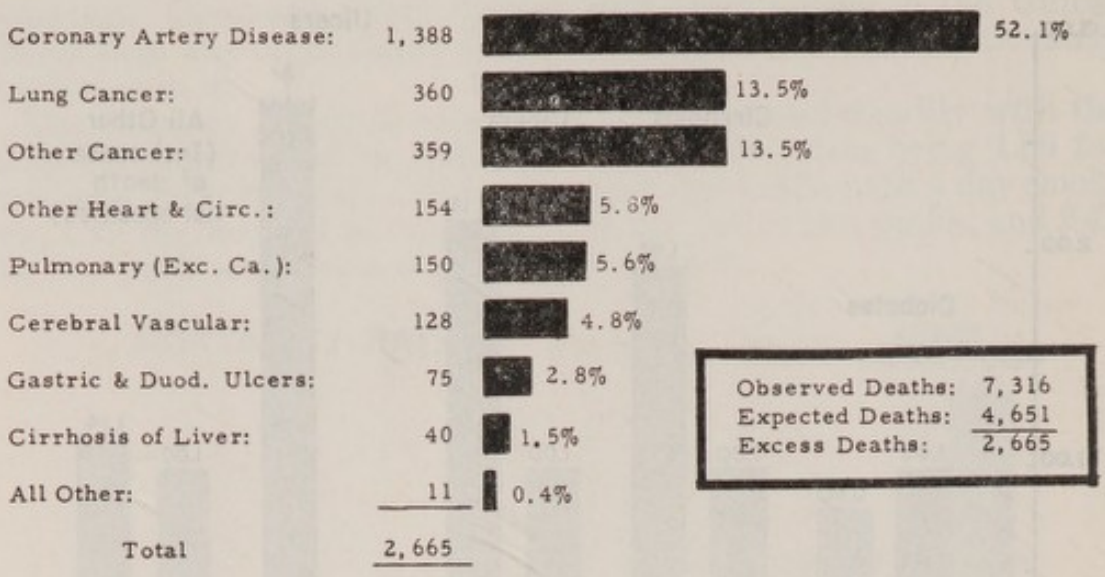
Eighty-three deaths attributed to cirrhosis of the liver occurred among cigarette smokers compared with 43 expected, a mortality ratio of 1.93.

Deaths from nephritis and nephrosis showed no association with smoking.

Diabetes deaths were negatively associated with cigarette smoking but not to a statistically significant degree.

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EXCESS DEATHS AMONG MEN WITH A HISTORY OF REGULAR CIGARETTE SMOKING



Slide 19

The relative importance of the associations just described is dependent upon the number of deaths attributed to each disease as well as upon their degrees of association with cigarette smoking. That is, a disease such as Buerger's disease which causes very very few deaths. Even if there is an extremely high association, it still doesn't make any appreciable effect on the overall death rate.

Coronary-artery disease accounted for 52.1 percent of the excess deaths among cigarette smokers; lung cancer accounted for 13.5 percent; cancer of other sites accounted for 13.5 percent; other heart and circulatory diseases, 5.8 percent; pulmonary diseases—other than lung cancer—5.6 percent; cerebral vascular lesions, 4.8 percent; gastric and duodenal ulcer, 2.8 percent; cirrhosis of the liver, 1.5 percent; and all other diseases combined, 0.4 percent.

The data just described were checked in many ways to assure accuracy. For example, we independently traced a sample of 38,583 of our subjects to make sure that failure of the volunteers to report some deaths had not biased the results.

We requestioned a large number of the men about their cigarette smoking. We studied the time trend in the death rates and found that the results in the last 2 years of the study fully confirmed the early findings which we had reported in 1954. Since these and other checks have been described elsewhere, and will be described in much more detail in the report we are now publishing, I will not take your time to discuss them in detail again here.

I would now like to discuss our findings in the light of other evidence.

EXPERIMENTAL AND PATHOLOGICAL EVIDENCE ON CANCER

No less than six independent investigators have now produced cancer experimentally with tar condensed from tobacco smoke. Cancer has also been produced with tar distilled from tobacco. Another investigator has shown that tobacco-smoke condensate acts as a cocarcinogenic agent when applied together with a known carcinogen to the skin of mice. I should say none of us know whether a mouse is anything like a man in this respect.

Two investigators have noted an increase in lung tumors in mice exposed to tobacco smoke. A well-known carcinogenic chemical, 3, 4-benzpyrene, has been found in small quantities in tobacco smoke. Wynder and Wright have fractionated tobacco-smoke tar and reported that most of the material which is carcinogenic when applied to the skin of mice is contained in one small fraction of neutral tar. Note I said "in mice." This was not a human experiment.

Florescent substances present in tobacco smoke have been shown to enter the cells which form the lining of the mouth. That was a human experiment.

It was a puzzle how the amounts of material contained in tobacco smoke could cause cancer in the lungs since the action of cilia in the bronchial tubes ordinarily washes out foreign matter. Now it has been demonstrated in experimental animals: (1) That tobacco smoke tends to inhibit ciliary action and (2) that material from cigarette smoke accumulates at spots in the bronchial tubes where cilia have been destroyed or ciliary action inhibited.

That has been demonstrated experimentally; not with men.

Detailed microscopic studies of the bronchial tree of men indicate that a number of changes, such as are usually produced by carcinogenic materials, are encountered more frequently among cigarette smokers than among nonsmokers.

LUNG CANCER

We found lung cancer death rates to be extremely low among nonsmokers and high among heavy cigarette smokers, the rates increasing with the amount of cigarette smoking. Doll and Hill have reported similar findings in their followup study of British physicians. No less than 19 independent studies of the smoking habits of lung-cancer patients have been carried out in the United States, England, Germany, France, the Netherlands, Finland, and Norway.

In every instance a higher proportion of smokers was found among lung-cancer patients than among people free of this disease. The experimental and pathological evidence cited above supports these epidemiological findings.

In my opinion, the evidence is overwhelming that cigarette smoking is a causative factor of great importance in the occurrence of lung cancer. This does not imply that cigarette smoking is the only cause of lung cancer.

CANCER OF THE BUCCAL CAVITY, LARYNX, AND ESOPHAGUS

Cigarette smoke or material condensed or dissolved from cigarette smoke comes in direct contact with the mouth, tongue, and other parts of the buccal cavity as well as the larynx and esophagus. A number of investigators have found an association between the smoking and chewing of tobacco and cancer of the buccal cavity.

Pipe and cigar smoking and the chewing of tobacco may be more important than cigarette smoking in this respect. An association has also been reported between smoking and cancer of the larynx.

We found the death rate from cancer of sites just named, as well as cancer of the esophagus, to be very much higher among smokers

than among nonsmokers. The experimental evidence is consistent with these findings.

In my opinion, smoking is a causative factor in the occurrence of cancer of these sites.

CANCER OF OTHER SITES

We found death rates from cancer of a number of sites which are not directly exposed to tobacco smoke to be higher among smokers than among nonsmokers. This evidence is so recent that there has not yet been time for proper evaluation. More research on the subject is indicated.

EXPERIMENTAL AND CLINICAL EVIDENCE ON HEART AND CIRCULATORY DISEASES

Experimental studies on human beings have shown that smoking produces a number of acute effects on the circulatory system, including: (*a*) A constriction of peripheral blood vessels, (*b*) an increase in blood pressure, and (*c*) an increase in heart rate.

It has been shown experimentally that cigarette smoking produces changes in the electrocardiograms and ballistocardiograms of some patients with coronary artery disease. There is some evidence that smoking has an effect on the flow of blood through the coronary arteries. Eisen and Hammond showed that cigarette smoking produces an increase in the red blood cell count and in packed cell volume.

There is strong clinical evidence that smoking has a severe effect on patients with Buerger's disease.

CORONARY ARTERY DISEASE

We found that death rates increase markedly with the amount of cigarette smoking. Doll and Hill have reported similar findings. This is in agreement with the results of at least three studies on the smoking habits of patients with coronary artery disease.

Considering the acute effects of smoking on the heart, circulation, and blood, I am of the opinion that cigarette smoking causes an increase in the death rate from coronary artery disease. This does not necessarily mean that smoking is a cause of atherosclerosis of the coronary arteries, a disease in which diet apparently plays an important role. It may be that smoking only increases the risk of death of those who are already suffering from this disease.

OTHER CIRCULATORY DISEASES

Our evidence suggests that cigarette smoking increase death rates from aortic aneurysm and may increase death rates from cerebral vascular lesions.

The clinical evidence leaves little doubt that smoking has a severe effect on patients with Buerger's disease. Our data coincides with that. We had too few cases for it to have any great meaning.

OTHER DISEASES

The clinical evidence, taken together with our findings, strongly indicates that smoking has a severe effect on patients with peptic ulcers.

There is evidence that smoking has an effect on death rates from respiratory diseases other than lung cancer.

The data suggest that cigarette smoking may be a factor in cirrhosis of the liver, but there is insufficient evidence on which to base definite conclusions.

THE EFFECT OF GIVING UP SMOKING

Both our study and the study of Doll and Hill showed that lung cancer death rates were lower among men who had given up smoking for a year or more prior to questioning than among men who continued to smoke. Our study also indicated that giving up cigarette smoking results in a reduction of the overall death rate.

Mr. BLATNIK. Thank you very much, Dr. Hammond. We will have questions. May I merely announce that we have another witness speaking for research—Dr. Clarence Little, appearing on behalf of the Tobacco Industry Research Committee. We would like very much to hear him today and not to inconvenience him by holding him over. With mutual aid coming up at 12 o'clock, I know all members are anxious to get to the floor as promptly as possible. So if we keep our questions to major points, other details that will be required can be furnished later.

Are there any questions on my right?

Mrs. GRIFFITHS. Have these experiments been conducted only with men, or have women been included?

Dr. HAMMOND. The two major studies are ours and the Doll-Hill survey, which were conducted by first questioning people and following them for a number of years. Both of these were confined to men. The Doll-Hill study was confined to physicians; all the subjects were doctors.

The reasons we both took men—and in our study we took men in the older age group—is that because that is where the largest number of cancer deaths occur. We would like to study women, but we would have to study between 500,000 and 600,000, because fewer older women—and by old in this case we mean 50 and above—die from lung cancer, and that is shown by the Bureau of the Census.

Two studies have been made on lung cancer in women—1 of them in Norway and 1 in this country. Both of them showed the same sort of relationship as in men, but both were rather small, and it is hard to get enough lung cancer cases among women to study.

Mrs. GRIFFITHS. Thank you very much.

Mr. HARDY. I know with respect to some of these findings you discuss smoking from a causative point of view. In lung cancer, for instance, you listed smoking as a causative factor, and you said, "It doesn't imply that it is the only cause." With respect to some of the other cancer sites it is given as a causative factor.

But then when you get into the heart and circulatory diseases, you speak of it as having a severe effect on patients who already have the disease, rather than as a causative factor.

Dr. HAMMOND. The reason for the distinction is this. If it had been possible, I would have liked to study the onset of the disease as well as the deaths. But that would have required that we examine each man periodically by medical examination—which was not feasible.

Therefore, the data from the study is confined to deaths. Lung cancer unfortunately has such a high death rate—95 percent of the people who get it die from it—that, practically speaking, what causes the disease is what causes death, since they all die.

For heart disease, that is not so. Therefore, we cannot distinguish between something which causes death in a person who has the disease and what causes the disease. I want to make it clear that we did not have the knowledge.

Mr. HARDY. That is the thing I was trying to understand. With respect to Buerger's disease, you said there is strong clinical evidence that smoking has an effect on patients with the disease.

Dr. HAMMOND. That is right.

Mr. HARDY. Is there anything to indicate that smoking may be the cause of Buerger's disease?

Dr. HAMMOND. That is disputed, sir. Almost all the evidence on the matter is concerned with the people who have Buerger's disease. We don't know the other answers.

Mr. HARDY. If we might pursue it, then, with respect to circulatory diseases, you have the same observation to make with respect to peptic ulcer. I thought you had indicated earlier in your statement that there might be some connection from the standpoint of cause—smoking causing peptic ulcers. You don't make that statement in your conclusions.

Dr. HAMMOND. I wonder if I can make myself clear, sir, on this. From our study, the only thing we have is association between smoking habits and death. That is the only evidence we have.

For lung cancer, the occurrence of the disease and deaths from the disease are practically the same thing, since they all die—not all, but practically all.

With heart disease, this is not so; and with peptic ulcers, the majority don't die of it. Therefore, when it came to peptic ulcers, I based my summary statement not just on our findings but on clinical studies published in the Mayo Clinic and others.

In many cases it is reported they have had great difficulty in curing a person with gastric ulcers. They often can't cure him. We found they had higher death rates with those two tied together. Therefore, I was willing to draw a conclusion.

But many people have gastric ulcers who don't die of them. I have no evidence as to whether smoking causes gastric ulcers. Maybe it does; maybe it doesn't. I didn't want to go beyond that.

Mr. HARDY. That is the thing I wanted to clear up.

Dr. HAMMOND. I say that smoking may cause heart disease, but I don't know. Not knowing, I wanted to make it clear that I didn't know.

Mr. BLATNIK. Questions on my left?

Mr. MINSHALL. I have one question, Mr. Chairman.

Mr. BLATNIK. Mr. Minshall?

Mr. MINSHALL. Projecting your statistics, what percentage of the overall population can be classed as smokers and what percent nonsmokers? Have you got those figures?

Dr. HAMMOND. I do have them in my briefcase here, sir. I can give you the whole breakdown on them.

Mr. BLATNIK. Can he furnish that for the record?

Dr. HAMMOND. I would hate to do it from memory, sir, because it depends on how you define them. I would rather put it in the record.

Mr. MINSHALL. As long as you are doing it, would you be kind enough to do it as it relates to men and women?

Dr. HAMMOND. May I say, sir, the Bureau of the Census, at the request of the Public Health Service 2 years ago, made a very good survey on this for the total population of the United States. They covered both males and females. The age, I think, was 20 up. So that survey answers your question with a high degree of precision.

What they found was almost exactly what we found, but we only covered a narrow age group. So, concerning women and men in other age groups, I would have to give you the Bureau of the Census figures. They are just the same as ours.

(These are as follows:)

Smoking habits of men and women 18 years and over in the civilian population outside institutions, United States, as of February 1955

	Percent of total men	Percent of total women
Never smoked during lifetime	22.9	67.5
Smoked during lifetime	77.1	32.5
Smoked regularly during lifetime	71.9	28.2
Smoked occasionally during lifetime	5.2	4.3
Never smoked cigarettes during lifetime	32.0	67.6
Smoked cigarettes during lifetime	68.0	32.4
Smoked cigarettes regularly during lifetime	63.4	28.1
Smoked cigarettes occasionally during lifetime	4.6	4.3
Currently not smoking cigarettes	43.1	71.6
Currently smoking cigarettes	56.9	28.4
Currently smoking cigarettes regularly	52.6	24.5
Currently smoking cigarettes occasionally	4.3	3.9

Source: Haenszel, W.; Shimkin, M. B.; Miller, H. P. Tobacco Smoking Patterns in the United States, Public Health Monograph No. 45, U. S. Government Printing Office, Washington, D. C., 1956.

Mr. MINSHALL. In response to Mr. Hardy's question you said there was some doubt, in your mind at least, as to whether or not smoking was the cause of cancer in certain parts of the body.

However, on page 17 of your report in the next-to-the-last paragraph on that page you say:

> In my opinion the evidence is overwhelming that cigarette smoking is a causative factor of great importance in the occurrence of lung cancer.

Dr. HAMMOND. Yes, sir.

Mr. MINSHALL. That is beyond any reasonable doubt in your mind?

Dr. HAMMOND. That is my personal opinion, sir.

Mr. MINSHALL. Is that disputed by any of your colleagues in the medical profession?

Dr. HAMMOND. I think there are some people who do dispute it, sir.

Mr. BLATNIK. Doctor, who finances this statistical research work and other research of the American Cancer Society?

Dr. HAMMOND. The American Cancer Society solicits contributions from the public, and those contributions are used for this and other research.

In this particular study, the volunteers did work on it that would have cost, I estimate, up in the millions of dollars. Before using

volunteers, I went to some of the public-opinion-survey people and asked what they would charge to question the subjects for us. They wanted $10 to $15 per subject. Now they would want more. That would have been $2 million right there. So I think, in this case, what was essentialy the cost of the study was contributed in volunteer work rather than in money.

Mr. BLATNIK. Doctor, is your society carrying on any other research in addition to the statistical analyses? Are you conducting any biological or laboratory medical experiments?

Dr. HAMMOND. The American Cancer Society is supporting a very great deal of research in the field in lung cancer; that is, laboratory research, clinical research, pathological research.

Mr. BLATNIK. Can we have an example—not necessarily all, but the major types—of the research which you are supporting and where they are conducted, later on for the record?

Dr. HAMMOND. I can send it in.

(See appendix, exhibit 2, p. 333.)

Mr. BLATNIK. My last question: You had quite a long series of bar graphs showing very significant differentiations between nonsmokers and smokers. But in the smokers of cigarettes, you never differentiated between those that used filter cigarettes and those that used nonfilter cigarettes.

Did you have any information on that?

Dr. HAMMOND. At the time the study started in 1952, according to all reports, very, very few filter-tip cigarettes were sold—so few that we didn't even ask the question. I think one brand accounted for almost all the sales.

Later, we did question the men again in 1955; right at the end of the study. I do have the information. It was that about 28 percent of those who were then smoking cigarettes regularly were using filter tips—of these particular men, of course.

Mr. BLATNIK. Is there any indication of any significance in your death rates between non-filter-tip smokers and filter-tip smokers? In short, is there anything to attract the interest of your organization to continue further statistical gathering of evidence and research?

Dr. HAMMOND. I frankly doubt that this method of study could be applied to the study of filter-tip cigarettes—at least in the near future. Certainly, from the information we got in requestioning our own subjects, and from everything I have heard, I think it is in a state of flux right now, with people switching from one to another and back and forth.

I don't know quite how one would study it by this method. If people settle down to one particular type of filter tip and smoke it for the next 15 years, then maybe we could answer it by a study of this sort. But, otherwise, I believe it would be impossible to get human information on the effect of filter tips, as far as cancer is concerned. I don't think there would be any way to do it.

Mr. HARDY. Just one other question. Doctor, have you attempted to make any determination as to the basis on which cigarette smoking has the effect that these statistics have pointed out? For instance, related to tar, nicotine, or anything else that could be removed by filters or otherwise?

Dr. HAMMOND. Sir, our own study showed nothing on it. If you would like, I can briefly review what is known on the subject. I can

speak only from having reviewed the literature and from the very little bit of experimental work.

Mr. HARDY. That will be all right. We can get that from another source.

Dr. HAMMOND. May I mention one thing, since you asked me, sir. Almost all of the attention has been centered in the experimental work on the neutral tar—that is, the tar in the cigarette smoke that is neither acid nor base. One of the components has practically not been studied at all except by Cornell, in England, and that is carbon monoxide.

I have some reason to believe, from statistical studies and from at least one experimental study, that that should be investigated before definite conclusions are reached on the subject.

I hope I have made myself sufficiently vague. I am not asserting that it had anything to do with cancer.

Mr. HARDY. That is quite all right. I think that probably is a phase that we may discuss with some other individuals. But your study has been primarily in gathering statistics and evaluating statistics. Is that right?

Dr. HAMMOND. I made one study—experimental study—of the effect on the blood; and I have preliminary data from another study on it.

Mr. HARDY. But still it was a statistical study rather than a medical study?

Dr. HAMMOND. No, sir; it was strictly an experimental study on human beings.

Mr. HARDY. Was that based on medical determination?

Dr. HAMMOND. It was both experimental and medical. The study I referred to was done jointly by Dr. Eisen and myself. Dr. Eisen is a physician and did the actual handling of the patients.

Mr. HARDY. What I was trying to get at, you haven't been making medical analyses yourself, have you, thinking in terms of this statistical study?

Dr. HAMMOND. On the study I described here?

Mr. HARDY. Yes.

Dr. HAMMOND. No. On this other study, on the effect of smoking on the blood, with a collaborator I did. And on this statistical study, I didn't do all this calculating-machine work.

Mr. HARDY. The reason I asked the question was that, in your personal information which you gave, you didn't indicate that you were a medical doctor.

Dr. HAMMOND. Oh, no; I am not a physician.

Mr. HARDY. So your major work in any event would be the compilation and evaluation of statistics and results of various tests that might be run and medically evaluated by others?

Dr. HAMMOND. Well, sir, I am not sure that I say that. A very large proportion of the advances in medical science have been brought about by people who didn't have to be physicians. Dr. Hill, as far as I know, is not a physician.

Mr. HARDY. I don't question that at all, but I was just thinking of the medical evaluation that has been made, not necessarily made by a medical doctor, either.

Dr. HAMMOND. As a matter of fact, the clinical trials which are being carried on in England, for example, for vaccines, for drugs, to see that they are effective, are being carried on by Dr. Hill, who is the associate also in this other study. He is one of the most prominent

persons in the whole clinical trials procedures and he is a statistician. He evaluates the medical work of the doctors for the British Medical Research Council. He is the most prominent person in the world in that field, I would say.

Mr. HARDY. To clear up my point, what I was trying to establish isn't too clear, but I was thinking primarily of all of these determinations as to the cause of death, which were based on statistics that you gathered, rather than in the determination which you and your associates did yourself.

Dr. HAMMOND. I am sorry. No; I didn't look at a single one of these men who died—if that is what you mean. I got reports from doctors and pathologists who had examined them.

I am sorry, I didn't understand your question.

Mr. BLATNIK. Thank you very much, Dr. Hammond, for your very comprehensive and detailed report.

Our next witness is Dr. Clarence Cook Little.

STATEMENT OF DR. CLARENCE COOK LITTLE, REPRESENTING THE TOBACCO INDUSTRY RESEARCH COMMITTEE

Dr. LITTLE. I will try to make the best time I can.

Mr. BLATNIK. Doctor, you have a prepared statement?

Dr. LITTLE. Yes, sir. That is in the hands of the committee, I believe.

Mr. BLATNIK. Doctor, would you please proceed; give your full name and your title and a brief summary of your professional background.

Dr. LITTLE. My name is Clarence Cook Little. I am director emeritus of the Jackson Laboratory at Bar Harbor, Maine. For 16 years I was managing director of the American Society for the Control of Cancer, which is now the American Cancer Society.

In 1954 I became chairman of the scientific advisory board of the tobacco industry research committee and scientific director of the tobacco industry research committee. I am not a doctor of medicine. I earned a doctor of science degree in my graduate work at Harvard. I am a biologist interested in the origin and nature and future prevention of cancer.

Mr. MEADER. Mr. Chairman, might I say that Dr. Little was president of the University of Michigan when I graduated from that institution in 1927.

Mr. HARDY. Dr. Little, can I have a little private conversation with you after the session?

Dr. LITTLE. Yes, sir. I might say that the experimental mice went along with me, both to Maine and Michigan, and I spent my evenings studying and doing research on them, so it was unbroken, even though I did commit the sin of being the university president.

Mr. BLATNIK. Please proceed, Doctor.

Dr. LITTLE. My chief interests in the problem of tobacco and health are two: First, to help the pioneer coordinated efforts of a very great industry to allow a group of scientists to plan and support research. This is pioneer work, and if those of us on the scientific advisory board of the TIRC can do a good job, can be patient, intelligent, and work without bias, we may be able to contribute to setting

up a precedent of support of basic research that will have tremendous influence on the country later on.

This will be because in a democracy it is very desirable to have an intelligent attitude in industry and the citizenry as a whole toward basic research.

The announced purposes and objectives of the Tobacco Industry Research Committee are to aid and assist research in tobacco use and health and to make available to the public factual information on this subject.

Without at all feeling the need of being defensive, I think it will be well to outline my own relationship. My appointment is annual, and it is clearly understood with the Tobacco Industry Research Committee that if, as, and when the slightest pressure as to what type of direction we should take in research or what the publication of the research should be, is evident, that my resignation takes effect immediately. I can say truthfully and honestly that, during the period that I have worked on this problem, there has not been the slightest effort to "pull punches," to select evidence, or to limit objectives for research.

I want to make that clear, because when one is supported in his activities by an industry, it is quite natural for some people to ask how much is this man dependent on the industry and what is his attitude toward it.

Secondly, after 50 years of research on the cause and nature of cancer, I have a tremendous respect for this disease and for its complexity. Most people don't realize that an early cancer is a terrifically vigorous, virile, healthy biological unit, more healthy and more vigorous than the body in which it originates. That is why it "outeats" it, outlasts it, and eventually kills it. If you remove a cancer from the body and culture it in a test tube, or if you transplant it from animal to animal, it is essentially immortal. There are mouse cancers alive today 30 years after they were discovered, and the Methuselah among mice isn't more than 3 years old.

Therefore, this original mouse tissue, this cancerous tissus, has lived 10 times as long as the oldest living mouse, yet it was a part of a mouse and is a part of a mouse.

I mention that because the origin of cancer is a biological phenomenon. It is fascinating. It is natural. It is going to occur, no matter how long mankind exists, because it is the opposite side of a sheet of paper that means "control of growth."

Therefore, from this point of view, I feel that it is very important that openmindedness and interest in further study—a willingness to be convinced too fast by any line of evidence as to causation—is an extremely important thing. We have got to have an open road. We have got to have scientists and people realizing that this problem is nowhere near solved—whether it is the etiology of lung cancer or any other type of cancer, we are a long, long way from home.

During each of these 50 years I have thought I knew a little more at the end of each year. But I am certain of what I don't know, and what I don't know has increased, I regret to say, much faster than what I know, and therefore, perhaps, it is just as well that I have reached the retirement age and can let somebody else do that kind of worrying.

The scientific advisory board has been given a completely free hand. I should like to talk, if I might, for a moment on some of the complexities of lung cancer causation to show why I feel that it is so necessary to keep openminded and not to go off on the trail of the first "fox" that you come on. It is very desirable to keep a lot of the pack of research workers looking for other "foxes" that may be causing the trouble.

Genetic constitution: There is a great difference in the reaction of different individuals of the same age, sex, and environment to the formation of lung cancer. According to four independent investigations on the probability of lung cancer, the vast majority of even excessive smokers do not develop the disease.

Why does a small minority develop it, while the great majority does not? What type of person—physiologically, psychologically, and emotionally—is a bad cancer and health risk? Preliminary studies indicate that there are significant differences in these respects, and the taking up of excessive smoking may well prove to be an indication of such differences, rather than the cause of them.

I mention this to show how little we really know about what starts cancer in this tremendous and rather inspiring population of living units that form the human body, and we need to recognize that there are different risks, continuing risks as long as we live, and that to emphasize any one risk is legitimate and proper, but it is only a part of the whole picture.

Sex differences: It has already been pointed out that lung cancer is 6 to 8 times more prevalent in men than in women of the same ages. Why is this? There is a hormonal factor involved here which needs explanation. I might incidentally mention that spontaneous lung cancer in mice, although it is of the type that occurs less frequently in humans, also has a sex difference, and the males form more of it than the females.

Why is this? This must have to be explained if we are to know the whole causation of the disease.

Environment: A number of research workers have pointed out a clear relationship between industrial hazards of different types and the incidence of lung cancer. A number of investigators have pointed out the increased incidence of lung cancer among urban dwellers as compared with rural people.

Why are these things? These facts are affecting the problem in which we are all interested, and these things must not be lost sight of by following any one lead.

Inconsistencies evident concerning the claimed cause and effect relationship: Nonsmokers may and do develop lung cancer; the great majority of heavy smokers do not.

There is lack of consistent correlation between various nations and among cities in the——

Mr. BLATNIK. I hate to interrupt you, Doctor. But, you are using two scales: When referring to nonsmokers you say they may develop; when you refer to smokers you say they did not develop.

Dr. LITTLE. The vast majority do not, but some do.

Mr. BLATNIK. Would you also say, then, that the great majority of nonsmokers do not develop cancer?

Dr. LITTLE. You mean cancer of any and all types?

Mr. BLATNIK. You say nonsmokers may develop lung cancer. Is it true that a great majority of nonsmokers do not develop cancer?

Dr. LITTLE. Do not develop lung cancer; that is true.

Mr. BLATNIK. Then you say that a great majority of heavy smokers do not develop cancer.

Dr. LITTLE. Yes; you can say it for both groups. In other words, lung cancer is a relatively rare disease, and there is no necessarily simple cause and effect relationship with smoking habits. If there was, you would get, instead of perhaps 1 out of 20, 1 out of 40, 1 out of 50 heavy smokers developing it, as is often the case or perhaps 1 out of 10 where it is at its very worst, then you would find a very much higher correlation than that.

It is not a simple single cause-and-effect proposition. The point is that it is not a perfect correlation. It is not a thing on which you can rely. It is a serious problem as to what causes lung cancer, but it still remains true that it is not a simple cause-and-effect relationship.

Mr. BLATNIK. Would it be correct to say, then, to follow up further, Doctor, of two equal groups, equal numerically and in other characteristics, of nonsmokers compared to smokers, that the percentage of those getting cancer is higher in the smokers?

Dr. LITTLE. That would be correct, according to the present statistics.

Mr. BLATNIK. Would it be correct to say it would be considerably higher, the percentage would be considerably higher?

Dr. LITTLE. That would vary entirely on your methods of selecting your group and what you were looking for. If you are looking for the single relationship between the smoking, that represents one type of data. If you are taking an overall picture and considering other variables, some of which were mentioned earlier in this hearing, the results might not be anywhere nearly as clear cut.

To establish a cause-and-effect relationship on statistical association without experimental evidence is not safe. It cannot be done. You may get an indication of something to look for, but to say that the case is finished, the evidence is all in, and that you can satisfy experimental scientists, all of them; that is not possible, because too many of us have seen too many statistical relationships which have not meant the "cause and effect" relationship.

For example, the per-capita consumption of tobacco in England is much less than in the United States, while the lung cancer incidence is much greater. Similarly, in cities of the same size in the United States, where no local difference in tobacco use is known, there may be very different rates of incidence of lung cancer. These, I will admit, are statistical.

The average life span has increased strikingly hand in hand with an increased consumption of cigarettes. Such general——

Mr. BLATNIK. I apologize for interrupting, but the increase in the use of cigarettes—is that a per capita increase?

Dr. LITTLE. I am talking about a gross increase at the present moment. I would have to look up the statistics to find out how that could be related to per capita, because the actual data on the use per capita of cigarettes is very interesting. If I have time, I will try——

Mr. BLATNIK. The life span is measured in per capita terms; isn't it? It is so many years added per person?

Dr. LITTLE. That is right; the average lifetime.

Mr. BLATNIK. So I was wondering if the smoke consumption per person per capita increased. That is why I asked the question.

Dr. LITTLE. It has obviously increased.

Such findings, I would say, definitely disprove a single cause-and-effect relationship between cigarette smoking and lung cancer, and this, I think, is admitted by everybody. I think Dr. Hammond himself said that he considered it an important cause, but that he also admitted that it was only a part of the story.

The methods of assay of cancer-forming effects of tobacco products has been attempted by animal experimentation. The painting of known chemical cancer-forming substances on the skin of mice and other laboratory animals is one of the favorite methods. Dr. Hammond referred to this. It is a well-known, well-established method.

In some experiments negative results have been obtained. In other experiments, various percentages of benign and malignant tumors have been obtained. There are marked differences in susceptibility between different strains of mice and rats and other animals, and a very large number of chemical agents not related to tobacco can produce the same or even more striking results.

It is very interesting that there are, I believe, some 1,000 substances that can produce cancer on animals by painting. This means that we are using a method of evaluation which is subject to a lot of value as a temporary test, but in which, to extrapolate wholesale to man is a very dangerous procedure. To do this it gives us a false sense of security, and we may waste a lot of time following up leads on this basis which may prove sterile.

Although certain chemists reported that they detected in tobacco smoke certain of the chemical substances which produce cancer on mouse skin, there is no evidence that these are present in tobacco in any form in sufficient quantity to produce this reaction even in mice.

Dr. Wynder, whom I understand you are going to hear later, has made the statement that:

> There exists, therefore, so far, no evidence that a single known carcinogen in condensed tobacco tar can account for the established carcinogenic activity in mice and rabbits.

I simply mention this because there is a great tendency and a very human one to grab at this animal evidence and swallow it whole and transfer it to man in the hope that we have found a solution.

I would be the first one to welcome it if that were true, but I have worked long enough with animals to know the need of very great caution in generalizing from them, or even in quantitatively taking the detailed individual experimentation. Very great danger exists in both of these activities. Conservatism is the wiser course when you are dealing with a disease like cancer.

Painting of the skin of monkeys—primates, these are—with known cancer-forming chemicals which do produce cancer easily in mice has been uniformly negative, although the exposure may have continued for as much as 8½ years.

It is therefore probable that the cancer-forming reaction of mice does not accurately measure the cancer-forming reaction of primates, including man, because I am afraid that you would have to admit that the monkey, physically at least, and chemically, resembles man more

closely than the mouse does, although I regret to say I have seen some men who acted more like mice.

A large number of experiments involving inhalation of cigarette smoke by a total of thousands of animals have failed to produce a single lung cancer of the type most common in man. This is very important to me, because you have heard this morning of the 2 types of cancer, the 2 commonest types of the lungs of men. The type that is commonest in humans has not been produced by smoke-inhalation experiments in animals, as far as I know. The type which is relatively rare in man is not only hastened by inhalation in animals, but it is hastened by any process that you use, practically speaking, to ill treat these animals.

In other words, if you age these animals fast enough artificially, or if you can age them naturally and observe them, the percentage of cancer increases, which is a very common phenomenon of almost all types of cancer.

But as far as I know there is no evidence that repeated and intensive smoke-inhalation experiments with animals have produced the type of lung cancer commonest in man. This I think is very important if you are going to study cause-and-effect relationship.

On the educational side of this work—in which I was very much interested, naturally, because of my past contact with the Cancer Society—for 16 years my associates and I worked to allay irrational fears and develop judicial attitudes by the public toward cancer.

After 1945 there was a tremendous expansion of the program of education, service, and support of research by the American Cancer Society. At that time there was also initiated the development of its own statistical and investigative staff and program.

The first major scale effort that division of the society was this collection of data on lung cancer. There were, as I understand, some 20,000 lay workers trained and instructed to collect these data, and their purpose was to collect data for studying of the reported association between cigarette smoking and lung cancer.

Drs. Graham and Wynder, in 1949, had definitely accused smoking as being of great importance in the causation of lung cancer; and the Cancer Society in this great big statistical study took that as an objective—to see whether this was or was not true.

This is important to remember. The specific definiteness of this objective, I think, was shown by the relatively small amount of information collected on other habits or previous health records of the population, or any information whatever, except smoke habit, place of current residence, and cause of death.

A positive association is claimed now by the American Cancer Society between excessive smoking and some of the diseases of which you have heard today. I will go through them very rapidly. Mind you, this is not claimed by them as significant, because often the members are so small that a larger number would be needed in order to make the difference observed statistically significant. But these are all on the positive correlation side: Death from cancer of the lung, both bronchogenic and adenocarcinoma; larynx, pharynx; esophagus; tongue; mouth; lip; bladder; prostate; stomach; pancreas; liver; gall bladder; Hodgkin's disease; lymphosarcoma; retinosarcoma kidney; also with pneumonia; influenza; other pulmonary diseases; aortic aneurysm; duodenal ulcer; cirrhosis of the liver; asthma; coronary

disease; myocarditis; cerebrovascular disease; hypertensive heart; nephritis; pulmonary TB; arteriosclerosis; and phlebitis embolism.

In fact, I believe that the only diseases listed by this study in which no positive association of some degree is observed were cancer of the colon, rectum, and brain; leukemia; chronic rheumatic heart disease, and diabetes.

There may have been 1 or 2 others, but, generally speaking, it is a difficult, if not impossible, scientific gymnastic feat to imagine a cause-and-effect relationship in this overall mixture of human ailments, especially with a strikingly increased life span of our population and parallel increased national tobacco consumption staring us in the face.

This is a hard thing to imagine on a cause-and-effect basis, I think, really. It is almost, not quite, reductio ad absurdum. It certainly makes one look for other factors in this situation than the statistical associations as meaning cause and effect.

Narrowly focused emphasis on a causal relationship between smoking and lung cancer with powerful and continuous propaganda pressure has far-reaching results.

1. It limits research objectives by setting off a large amount of intensive activity on but a single trail which may well prove a false one, or at best provide only a partial answer.

2. It creates fear, mental and emotional disturbances, and a false sense of proportionate values in considering the whole complex problem of lung cancer prevention and control.

3. The definition of "smoking habits" is vague and is subject to the possibility of radical change when accurate information is obtained.

4. Since a long—approximately 20-plus years—latent exposure period before cancer formation is required to explain the present unfavorable statistical results, supposedly preventive measures, even if practical, could not be safely evaluated for 2 decades.

5. Should further knowledge force reversal, abandonment, or even radical modification of definite claims of the seriousness of the possible cause-and-effect relationship, research efforts not only in this particular field but in many others may be discredited and progress toward eventual victory over cancer be seriously delayed.

I should like, in closing, to make this one thing clear again. My interest in the fight against cancer is a very long one and a very intense one. I have given most of my active life to it. I want the public to realize the seriousness of the problem.

I don't want them to be misled by "apparent" victories, even partial victories, that might block continued research. An openminded attitude is terrifically important in order to hasten the day—and I am afraid the day is still far off—when we can claim victory over the different types of cancer.

They are very much at home in us. As I have said, they are a part of us. They grow faster than we do. They outlive us. They are a much more effective biological unit, a much more effective living cell. The tragedy is when they occur in a limited human body and create the crisis.

With all these factors, those of you who are in a position to maintain sanity and balance and openmindedness and the zeal to get this knowledge as quickly as possible are, I think, in a terribly important position, perhaps much more important than the fellows who are in

the laboratories or even the people who are on the fringe of education in this work.

To keep an openminded attitude is the only way to conquer this disease. This doesn't mean that we can't let everybody who feels that he has got evidence give his evidence. By all means, yes. Let him interpret it in any way he wants. But remember that, in as complicated a subject as this, an open mind and great courage and great tenacity and great wisdom and great inventiveness are going to be needed for a great deal longer, I am sorry to say, than probably anybody sitting here in this room will live.

I would be very glad to try to answer any questions. I have tried to hurry through this rather rapidly to keep you under your deadline of time.

Mr. BLATNIK. Questions on my right?

Mr. BROOKS. I would like to ask the doctor one thing. You refer to painting animals, and all of that. Have you ever tried the process of the application of heat continued for a time?

Dr. LITTLE. Yes. In some animals there has been evidence that around burns or around areas stimulated mainly by heat there is apt to be overgrowth of skin and cancer—an example is the forming of cancer of the lip following the excessive smoking of the old TD pipe, the old clay pipe that used to practically cook the lip.

Chronic irritation, I think, is a bad risk in any form of cancer. Heat is one type of chronic irritation if it is kept up long enough.

Mr. HARDY. I take it, Doctor, that essentially your feeling about the statistics that were gathered and presented earlier is that it is premature to make a final adjudication of what they mean?

Dr. LITTLE. I think that is true not only of these statistics but of all the experimental work of persons like myself and others who have tried to find the answer. I think we don't know the whole picture by a long shot yet, and that an open mind and all kinds of courage and effort are the things to have rather than a precrystallization and a prejudgment which may turn out to be fallacious and block progress for years to come.

Mr. HARDY. Then you would disagree with Dr. Hammond's conclusion that there is a causative relationship between smoking and lung cancer?

Dr. LITTLE. I would say that no evidence has yet been produced that has convinced me of that. I admit that he has an entire right to his own opinion and he has worked with these data and collected them. But from the point of view of somebody who has worked experimentally with the disease in animals, I am not convinced that this relationship is a real one yet; or, if real, is anything like as important as it is now being made to appear.

I think that it has become fashionable only because if you find a possible cause-and-effect "agent," humanity is hungry for something to prevent cancer. It is a cruel, miserable disease, and, of course, it is a wonderful encouragement if you think you can put your finger on it.

But it is an awfully tough disease, and it is ingrained right in the nature of the person himself. It belongs there. It is quite at home: just as much at home as the rest of his body. And I have great respect for it.

Therefore, I cannot be driven to accepting the relationship with smoking, and especially with cigarette smoking, as being as significant as it is taken to be.

Mr. HARDY. I would like to ask you a question comparable to one I asked Dr. Hammond. If there should be a relationship between smoking and lung cancer from a causative point of view, would that be due to some specific element in smoking that could be removed in your opinion?

Dr. LITTLE. I wouldn't want to estimate it—not because I wouldn't like to be able to, but because I don't know enough. I have too much respect for the different triggers that can set off cancer to be willing to evaluate any one until I had a better overall picture of the whole terrain in which this battle was being fought.

I don't want to pick out one battleground and say that this is the whole campaign, and give a misleading effect that, if you solved the problem in that battle, you have won the war—because I don't think that you have.

I think that it is very doubtful whether the chemical carcinogens which have so far been accused of being bad in this picture—they have been accused by some and abandoned by others—have a very great significance. I have a very great desire to get something definite, but I have very great doubt whether the problem is anywhere near as simple as that.

Mr. HARDY. Maybe I am getting ahead of the discussion that we are trying to develop, but it leads me to wonder whether I am properly interpreting your statement there to mean that any idea that filters would remove the cause of cancer is fallacious or is certainly not justified.

Dr. LITTLE. I wouldn't want to go as far as to say that. I don't know. First, I don't know whether there is any cause in it. Second, I don't know whether this cause is a chemical or a physical one.

I don't know whether minute particles of material that we inhale are bad—and we inhale much, much more of them from motor exhaust fumes than we do from tobacco smoke, and much more of the suspected chemicals.

I would like to see the efforts which the TIRC is trying to make progress on succeed, and see us get human lung cancer outside of the body where we can match what happens to it when we challenge it. It is hidden.

Mr. HARDY. The reason I sort of got a little bit out of the line of the discussion we have had up to now was that actually the subject this committee is concerned with is possible misleading or false advertising that has been going on. That is why I was interested in your reaction to the possibility that if cigarette smoking does have a causative effect on lung cancer, then whether or not a filter would reduce that cause or the extent to which it would reduce it is the subject that we are immediately concerned with, and claims that it reduces it, if such claims are made.

That is the thing that seems to me is really the key to what we are concerned with, and I gather from your testimony that you don't know of any reason to suppose that it makes any difference.

Dr. LITTLE. It is very hard for me, when I don't know the cause of a disease, to have opinions about what is supposed to prevent it.

Mr. HARDY. I think that is a very reasonable reply.

Mrs. GRIFFITHS. Mr. Chairman, may I ask a question?

Mr. BLATNIK. Certainly.

Mrs. GRIFFITHS. If, in your judgment, smoking didn't cause cancer, do you know why the cigarette companies came out with the filters?

Dr. LITTLE. I don't. Very fortunately, I couldn't pass an examination as to which company makes which kind of cigarette. And if I may say so, it is a matter of complete and enthusiastic indifference to me. I am very much more interested in studying cancer than I am in cigarette companies.

Mrs. GRIFFITHS. Are you interested in studying the connection between cigarettes and cancer?

Dr. LITTLE. I am interested in studying either claimed or real associations between anything and cancer.

Mrs. GRIFFITHS. Have you ever studied whether or not the filters actually keep you from inhaling smoke?

Dr. LITTLE. No; I never have. I have been very much more interested in trying to study the nature of cancer itself. I am not at all interested in the economic or practical side of the problem. I am much more interested in the human being, the boy who is going to pay the bills if he has cancer.

Mrs. GRIFFITHS. Perhaps it isn't a fair question to ask you; perhaps you don't know and couldn't answer it anyhow. But if it were definitely true that there is a real connection between cigarettes and lung cancer to the satisfaction of everybody, are the cigarette companies going to admit this? Are they going to stop selling cigarettes? Or are they going to advise the public?

Dr. LITTLE. I couldn't answer that. I suppose when you say association between tobacco and cancer, you mean causative association?

Mrs. GRIFFITHS. Yes.

Dr. LITTLE. Just a parallel habit or parallel incidence of the two may mean nothing. You mean if a causative effect is proved?

I would say that from what I have known in my brief contact with the committee of tobacco executives, they would do everything humanly possible to find out the truth about this. I don't believe there is one of those men that wants to bluff for a minute. I don't think there is one of them that is stupid enough to want to bluff for a minute.

It is now essential that they find out the truth. They want to find it out as much as anybody else. They don't want to take a risk of producing a lethal or a semilethal agent. That is why they have come right up with the complete freedom they have given my associates and me, and some of the evidence that is coming from experiments may not be what they would hope. It may raise questions. If it does, then they are prepared to continue to study in the face of those questions, because this accusation has been made and they are at grips with a very serious enemy.

Mrs. GRIFFITHS. But you don't know whether the filters were put there for a bluff?

Dr. LITTLE. I have no conception of that at all. I really don't. To tell you the honest truth, it would seem to me, from my point of view, to be more of a "sporting event" to try to find out the motives for something like that than it would really to have anything to do

with the seriousness of the situation; for if they were put up for a bluff, nobody except the fellow who put them up that way is being as heavily fooled as he is, because he is building on an unknown situation out of ignorance.

I hope that they are too good citizens and too good businessmen to try to bluff the thing. I am not a cigarette smoker any more than I can help being, because my nose is so long that when they are halfway down it begins to be uncomfortable.

Those that I have smoked with filters delivered less that I had to remove afterward from my mouth than those without filters, and I found them cleaner and more comfortable to smoke.

Mrs. GRIFFITHS. Do you know whether in England more people who are heavy smokers die of lung cancer than those who do not smoke? I noticed you had some statistics.

Dr. LITTLE. I think that the Doll and Hill investigation was just a population of doctors and tended to show that association. I know that the per capita consumption of tobacco in England is far below this country, and that the lung cancer death rate is almost twice as high per capita.

Mrs. GRIFFITHS. That would not necesasrily mean that there were not just as many heavy smokers in England as there are in this country. Would that be right? If you are dividing cigarette sales by the people who live in England, I presume that is meant per capita.

Dr. LITTLE. I would rather see the statistics themselves in order to try to evaluate them, because sometimes you can set up a thing that looks perfectly reasonable and then by failure to include the borderline variables get a misleading result.

All I get the impression of is, that there is no definite correlation that one can really arrive at between the amount of individual smoking and lung cancer death rate between nations. Whether this is the biological differences of the people who smoke, I wouldn't know. But certainly there is no foolproof cause and effect correlation that goes through the whole gamut of variables and leaves you confident of it at the end.

Mr. HARDY. May I ask just one question, Mr. Chairman? I believe he has already answered it, but just so I can have a clear answer to it: Doctor, based on your own research and on your knowledge of the studies of others, do you know whether there is any health advantage in smoking a filter-tip cigarette over one that doesn't have a filter tip?

Dr. LITTLE. I don't know.

Mr. MEADER. Dr. Little, as I appraise your statement, it is one of caution against jumping to conclusions from statistical associations. I don't find too much in here about the origin of the committee of which you are the director, nor the progress that your committee has made in its efforts, and precisely what the objective of the committee is.

Dr. LITTLE. The list of the scientists who are on my board, I think, should be or is appended there.

(See appendix, exhibit 3, p. 337.)

Mr. MEADER. When was the committee created?

Dr. LITTLE. The committee was created in 1954.

Mr. MEADER. Was it a result of the public interest in the subject of the possible relationship between cigarette smoking and cancer?

Dr. LITTLE. And also cigarette smoking and coronary and heart disease. In general it was, I think, a fine and honest recognition of the industry that the time had come when it would have to study these claimed cause-and-effect relationships and do its best to find out whether they were true or not. It realized that if they were true something would have to be done to correct them, and if they weren't true, the industry would like to know that so that they could go ahead and be about their business.

Mr. MEADER. Let me ask you whether the financial support of your undertaking is derived solely from the manufacturers of cigarettes, or are the growers themselves involved in this and do they contribute to the financial support?

Dr. LITTLE. Subject to correction, it is my impression that the growers and the various phases of the industry are involved. But I couldn't possibly give you the list of the names because I don't remember them.

Mr. MEADER. Could you give us some idea of the magnitude of your research undertaking in terms of finances and employees and facilities and so on?

Dr. LITTLE. Yes. We do no research of our own at all. We give two kinds of support—grants-in-aid to existing research centers, and to individuals on problems which they have submitted to us; and we also give very small summer scholarships to help medical students in any phase of research that they want.

These fellowships are not at all confined to the field of tobacco and health. The grants we give have also been very, very broad, because we have some question about reactions of tissues that may not have any immediate obvious relationship to the problem.

Since we are left alone to decide whom we shall support, and there is no industry pressure whatever, we have tried to look at it from a longtime point of view.

Mr. MEADER. Could you submit for our record the budgets of the committee by years?

Dr. LITTLE. I will be very glad to do so.

Mr. MEADER. With some breakdown as to the grants made and the purposes of the grants and the individuals to whom the grants were made for research purposes in this field.

Dr. LITTLE. Yes. The committee has made available about $2,200,000; and my impression is that we have appropriated $1,800,000 of that.

Mr. MEADER. Since the beginning?

Dr. LITTLE. Since the start of it; yes. Also, I have a very definite feeling that the industry would increase its support if those of us who were responsible for the program asked it to do so.

But I have believed that it pays to proceed with caution and soundness rather than to try to make a splurge and to try to get results rapidly but superficially. I think that the problem of cancer is one that brings you right up on your haunches with a warning to you—"Don't fool with me. If you mean business, you take hold of me and stay with me and don't try to hurry things, because I am the boy that is going to fool you if you try to do that with me."

Mr. BLATNIK. Not to interrupt you, but on one point, Doctor, I don't quite agree on "hurrying"—not that I am a scientist. I grant you my undergraduate work was all in chemistry and mathe-

matics. The only way we solved the atom bomb was by a crash program. We solved it in about 2½ years. That is a very complex problem. We stepped up the second stage in the race for the hydrogen bomb.

The only reason we were successful in solving that problem was by an all-out crash program. I don't quite follow you when you say we should not increase and intensify and broaden this all-out accelerated research work.

Dr. LITTLE. I don't blame you for questioning me on that. I would be very dumb if that is only what I really meant. What I mean is, you should not proceed any faster than genuine opportunity offers itself. You should not go through the motions of asking for a lot of money that you can't wisely spend, and then having to explain how you spent it afterward.

To take the atomic bomb problem—without going into the technical side of it—the groundwork by which the all-out drive was possible had been laid over a long period of years by a lot of men who had gone through——

Mr. BLATNIK. It was all theoretical on three sheets of paper. There wasn't even a laboratory assembled of the items we are talking about.

Dr. LITTLE. You probably know much more about that than I do, but I had the general impression—I still do—that great discoveries had previously been made. Certainly in biology—which I must confess if I know anything at all, I know more than I do about the atomic-energy program, about which I know nothing—in biology you are dealing with an awfully tough proposition. You are dealing with all the delicacies and balances of life processes. You are dealing with a population of living units, and a human being probably has more living units than there are people in any census anywhere today.

All of these units are coordinated into a perfectly wonderful balanced system. The wonder is that more errors like cancer don't happen, that more cells don't break loose, that the control is so perfect; and because the control is perfect, there are any one of a great number of agents that may break it or that may threaten it.

So I would say that great caution was necessary, and I think that just spending money for the sake of spending money oftentimes boomerangs because the people who have been giving it say, "What have you got?"

Mr. MEADER. That was a question I was leading up to, Dr. Little. I have one preliminary question. Are your research grants in the field of cancer confined to the effect of the use of tobacco upon various types?

Dr. LITTLE. Some of them are in the field of cancer generally. Some of them are in the field of heart or other circulatory disturbances. Some of them are in the field of stomach ulcers—things of that kind. They are not confined to the effects of tobacco or any tobacco product. Some of them are, but some of them are not. And there is no fixed policy.

Mr. MEADER. Now I would like to ask the question you just asked: What progress have you made?

Dr. LITTLE. We have found some interesting leads, and these are all being published by the investigators who found them in regular scientific journals when and how they want. We have no influence on,

and never shall attempt to exert even the tiniest influence on, publication or interpretation of results.

They are coming out in the journals now. I don't know whether my associate, Dr. Hockett, here would know how the publication is progressing, but obviously there was a timelag after we began supporting research because you don't just press the button and get scientific results. But there is an encouraging growth in first-rate scientific communications coming out.

If you knew how exciting that is to me—realizing that a big industry is willing to get back of that, and what that may mean—before so many years we may be able to go to a lot of industries and say, "Come on now, boys, you get back of free research. Go at things, and we may be able to speed up discoveries in biological sciences and chemical sciences a great deal."

Mr. MEADER. Are there any discoveries which a layman could understand which you could mention as an example of the progress of your research program?

Dr. LITTLE. Well, that is pretty hard for me to do. I would much rather give you the abstracts of them, give the whole committee the abstracts and let you form your own judgment, because if I tried to tell them to you in a condensed way, I am very much more likely to run the chance of overemphasizing something that is of particular interest to me.

I hope you will give me the time to let you have the abstracts on them and let you skim over them. Some of the things that do interest me are the relationship of stomach ulcers and cancer, things of that kind, the discovery that it was not a simple cause-and-effect relationship, that smoking doesn't change the chemical content of the material which is in contact with the ulcer, so that it doesn't do any good, it doesn't do any harm apparently.

Smoking is a neutral factor as far as the analysis of the gastric secretion of stomach ulcer patients are concerned.

We are just getting some very interesting results in the field of the types of people who are health risks. This factor of psychological strain and stress and the person's reaction to it—I don't know that Dr. Hockett has a list of materials here, but Dr. Brosek's work is interesting in that it shows a correlation between smoking and certain health behavior which may lead to more enlightenment and more knowledge as to what is good and what is bad in this picture.

That there are some at least supposedly beneficial effects of a habit that has been long used I suppose would have to be taken for granted. The question is, are these effects real?

Mr. MEADER. I understand you can supply the committee with a list of the grants that have been made and a list of the publications?

Dr. LITTLE. Yes; we will be glad to supply you with a list of the grants and the names, and these will be supplied you also with a résumé—an author's summary—of what he thinks he has found.

(See appendix exhibits, pp. 361, 364.)

Mr. PLAPINGER. Is Dr. Brosek included in this list?

Dr. LITTLE. Yes; he is.

Mr. MINSHALL. Doctor, you have no staff, then, of medical men who are actually conducting experiments or research along this line?

Dr. LITTLE. That's right. We don't hire a single person to do research, and we have no control over his research or the conclusions coming from it.

We give him grants-in-aid exactly the way the Rockefeller Foundation does or the Carnegie Foundation. We kiss the money goodby when we give it to an investigator and hope that he will find something for the good of humanity.

Mr. MINSHALL. The Roscoe B. Jackson Laboratory in Maine: Is that where you are headquartered? Does that have anything to do with the Tobacco Institute in any way?

Dr. LITTLE. We have a grant from that group—the laboratory does. I am no longer active with the laboratory. But we have a grant by which we try to produce animals which can be used by other grantees.

You see, the Jackson Laboratory, in addition to its own, is the one supply research center of the standardized animals which are so uniform due to inbreeding that a person can buy 5 or 5,000 or 50,000 of them and be working on a population that is essentially as alike as identical twins are in humans.

In other words, it is the standardization and production of uniform biological material. That is besides our own research, but we are the service station for the whole country and really, practically speaking, for the world in that type of material.

This grant is to enable the laboratory to have readily available for people doing research on the effect of tobacco on health to get this material as quickly and effectively as possible. We are not doing any experiments to test the efficiency of tobacco there at all.

Mr. MINSHALL. In other words, you are just a breeding station?

Dr. LITTLE. We are suppliers; yes. That is practically what we are.

Mr. MINSHALL. You sell these animals to anyone——

Dr. LITTLE. Any legitimate person. I mean by that, any legitimate person who has a connection with an established laboratory, institution, or university. We do not sell them to individuals who appear to be irresponsible and are likely to be crackpots and who might produce a false sense of scientific achievement.

We would do that—not about the tobacco thing, but about the use of the mice in general. We just wouldn't let them have them if they wanted to see whether they got indigestion from eating grapefruit or something.

We just wouldn't let them have them, period.

Mr. MINSHALL. Doctor, based on your association with this institute for the past 3 years, and the wealth of information that has certainly come to your attention, do you still believe that there is no connection between smoking and cancer?

Dr. LITTLE. No; I have never stated a belief of any kind on it. I am openminded.

Mr. MINSHALL. What is your belief?

Dr. LITTLE. My belief is that I would like to get more facts before I am able to say whether there is any relationship; and if any relationship, where and how that occurs. Whether it is a cause-and-effect relationship, or a correlation that doesn't mean anything, I don't know.

I am very, very much from Missouri about this, because the greatest mistakes in the world that can be made when you are dealing with an enemy like cancer is to play your cards before you know the kind of a fellow you have to deal with.

Mr. MINSHALL. Based on your progress so far, or lack of progress, when do you think you will know? Ten years? Twenty years? Fifty years?

Dr. LITTLE. I will say that I hope we will know more each succeeding year. But when a man will be able to sit up and say, "Yes" or "No" about a type of cancer, when he will be able to do that, I wish I did know.

Mr. MINSHALL. You know more now than you did at the end of the first year; do you not?

Dr. LITTLE. Yes; we know some of the complexities that exist and some of the dangers in oversimplification.

Mr. MINSHALL. Based on that experience, when do you think it might be? Do you think it will be in 5 years; 10 years? When do you think you will be able to definitely formulate some conclusions?

Dr. LITTLE. I think we will be forming tentative conclusions fairly rapidly for specific questions. But for an overall sweeping condemnation of any human habit or any human structure in the cancer problem, I doubt whether we will be unwise enough to make a statement that gives a false sense of values.

Mr. MINSHALL. What kind of tentative conclusions do you think will be formed and when will they be formed?

Dr. LITTLE. I couldn't answer that. I wish I could. I honestly do. How can a fellow tell what new trails are going to come to him? Not even the prophets were too good at that, and certainly I am not a prophet.

All one can do is to keep plugging honestly and courageously and without fear and without favor, and that is what this particular group is trying to do. The vast majority of people everywhere are trying to do this about this cancer problem. When somebody crystallizes or somebody states a belief, that doesn't mean that those who don't share that belief think little of him.

If Dr. Hammond, whom I respect tremendously, has a belief, he has a perfect right to that belief; and it may be right and mine may be wrong. But to keep an openminded attitude is the one way of being sure that the best final judgments will be reached.

People get convinced at different stages along the line. Some people will take evidence as final that other people are skeptical about. Maybe it is just because I am a "damn Yankee" that I am very skeptical about things. Maybe it is just because I have worked with cancer for so long that I respect it terrifically, and know my own shortcomings.

Mr. MINSHALL. In other words, then, as far as you are concerned there is no goal in sight when you might reach some definite conclusions?

Dr. LITTLE. Yes; I think there is. I think there will be methods of testing. I think there will be more methods of doing this.

Mr. MINSHALL. You have me confused now. First you said you didn't know, but now you say there is a goal.

Dr. LITTLE. If I may just take a second and single out one sort of problem. You can't see what happens in lung tissue when it is in the body, and you can't reach it very successfully in any controlled way. It is beautifully hidden and protected because that is what it is there for—to do a very delicate job.

Now, supposing we can get this lung tissue out into tissue cultures, and by giving it different nutritive medium and by challenging with

different agents—including the products of tobacco—we can see what the changes are in relation to what the lung is doing in aging by itself without the other agents.

Supposing we can take lungs that have had tuberculosis and compare them with lungs that haven't; or lungs that have inhaled asbestos with those that haven't. That is the kind of thing which isn't visionary; it isn't impossible; and one of the things that we are doing on the TIRC is that we have got 3 young tissue-culture people learning all they can about lung-tissue culture under 3 of the very best men in the country, so that we can get the technique by which we can pull the lung tissue out of the dark and make it play the game where we want to play it instead of having to go and play on its terrain.

I think that is a cardinal principle of scientific research—get your enemy out into the open. That is the kind of thing I mean. All I can tell you is that we are making progress toward it.

I wish I could tell you just when we are going to turn the corner and begin to be able to do something, but it isn't unhappy. I would say give us 3 years, perhaps, for that, on a guess.

Mr. MINSHALL. Three years is your best guess now. That is all I wanted.

Dr. LITTLE. On that particular problem; yes. I would say that by 3 years we ought to be able to be culturing lung tissue in a very much better way than at present, and making it obey, jump through some of the hoops that we want it to jump through instead of having to go to it. To get your enemy out in the open is a good principle, if you can do it.

We may fail, but it won't be because of lack of effort. And I am sure that if my scientific advisory board finds other places where trainees can be trained, we can go to the Tobacco Research Committee and say, "O. K., instead of 3 of these fellows working on this, we want 5; we want 6."

But at the present moment it looks as though there were only three good centers of tissue-culture work where enough progress could be made on that to justify taking it.

Mr. MEADER. In connection with the material you are going to furnish the committee on the grants and the research and projects that have resulted from them, can you also furnish the committee with whatever charter there is for the committee?

Dr. LITTLE. Yes; I can.

Mr. MEADER. And also any regular reports that you may have issued.

(See appendix, exhibit 3, p. 348.)

Dr. LITTLE. We will be very glad to do that. This year's annual report isn't ready yet, but we can at least send you all the other material and, let's say, a typed preview of the introductory part of this year's report, which will talk about the reasons for our programs and the philosophy of it, provided, of course, that you will take that not as a final documnet. We are very anxious to have you see our attitude while we are thinking about things and while we are in the formative stage as well as after we have come to a decision.

Mr. BLATNIK. Mr. Kilgore?

Mr. KILGORE. Doctor, there is one thing that I am not quite clear on with respect to the functions of your committee. As I understood

your testimony, you allocate grants to existing research foundations. But then in connection with published findings of these organizations, do you maintain any evaluation procedure for the findings that are developed by independent research organizations? Do you, within the committee, maintain any means for evaluating or correlating that?

Dr. LITTLE. We review them, but the evaluation is purely personal. There is nothing official. We don't rate them. We don't put an official stamp of approval or disapproval on them. These men are just as free as they can be to find out anything they can. I think they have enjoyed that. We have had some scientific conferences. It has been very amusing. Some of the tissue-culture gentlemen are quite temperamental. We did have them come in to get their ideas as to how practical lung culture was. First it was largely a matter of their avoiding that rather than getting together. After that they got together beautifully and they gave us some criticisms and ideas.

Mr. KILGORE. Then the function of your committee would not envision or anticipate the publishing by the committee itself of any evaluation or compilation or comments?

Dr. LITTLE. No comments on them other than a general introductory comment which I, as the director, the chairman, would make trying to survey the whole field and point out the areas of interest and their relative development.

But as far as singling out a piece of research and putting a stamp of approval on it; no. If a piece of research could be used as an example of encouragement or an example of something that we thought might be further considered, I think that would be quite right—the way the Carnegie Institute or the Rockefeller Foundation do. They give a report each year in which they pick out things that seem perhaps interesting to them.

Mr. KILGORE. Then I have a specific question I want to verify my thinking on. If I remember your comment accurately, in response to a query made by Mr. Brooks with respect to the relationship of pipe smoking to lip cancer and the possible correlation of heat factor, I think you made a general comment to the effect that you would consider any chronic irritation as a bad risk insofar as cancer is concerned.

Dr. LITTLE. Any chronic irritation—I take it you can say as a general principle—sets up an unbalance of some kind in the body and the body has to correct that.

The body is amazingly efficient in correcting not 999 out of 1,000, but nearer 6 figures. It is a beautifully adjusted mechanism. Once in a while it cannot do that. Once in a while for some reason in the origin of cancer a cell becomes independent.

It no longer will go back as it should and join the community and mind its own business. But it becomes independent with a terrific vigor, and with an increased vitality compared with the cells that go back and obey. This is the awe-inspiring thing about it—the tremendous vigor of a cancer cell and the fact that all the pain, suffering, disaster, and death we have are the secondary and tertiary results of an overgrowth that the body can't take care of, or of sepsis coming to a breakdown of tissue to blockage of a vital path.

Mr. KILGORE. Doctor, that, of course, naturally raises the next question of whether or not the regular or irregular inhalation of smoke wouldn't properly be classified as an irritant to the lung tissue and to the larynx.

Dr. LITTLE. I should say that any substance other than the actual material of the body itself in place has potential value as an irritant. For instance, the salt you eat has. For some people, milk might be. In other words, anything that you bring in contact with human tissue has a potential value that may differ entirely for the type of individual where this experience occurs.

The old statement that what is one man's meat is another man's poison is pretty true biologically, not only of agents that you may single out and put a finger on but a whole lot of things, including too much water intake for certain people of certain chemical types, and so forth.

Mr. KILGORE. Would it be a fair evaluation of your answer, then, Doctor, to say that such an inhalation of smoke would be an irritant, as there are many other irritants?

Dr. LITTLE. Yes. Anything inhaled into the lung is a potential irritant; and I think that I would say that there is nothing unique that I can see in tobacco smoke that may not very well be possessed by hundreds of things that get into our lungs.

It is visible and you can smell it, and that is a good kind of an enemy to chase because it gives you an immense sense of satisfaction. "Here is a guy I can knock out."

Mr. KILGORE. To some extent you can control it. That gives it another factor—in contrast to carbon monoxide from automobiles.

Dr. LITTLE. I would be much more scared of the carbon monoxide from automobiles or from the colorless fumes that you are getting from diesel exhaust. Let's say I would be just as suspicious of them, and perhaps more so, because they aren't so obvious or so easy to detect and to blame.

Mr. KILGORE. There was one other portion of your testimony with relation to this particular area that I thought was interesting, and perhaps related to the same thing. In discussing the failure of test animals to develop the type of lung cancer that man develops when exposed to the inhalation of smoke in smoking, I think you made a statement to the effect that any ill treatment of an animal is calculated perhaps to reduce the life span.

I wondered if the use of that term "ill treatment" would indicate to you that the laboratory testing of animals by requiring them to inhale smoke would necessarily be ill treatment?

Dr. LITTLE. It wouldn't necessarily, but the ones that I have seen are—I think we have all done experimentally something we wouldn't like to experience that had not very much resemblance to what we do when we smoke.

These poor beasts are usually put in practically an airtight container, and smoke is pumped in and pumped in in such quantities that a white rat becomes yellow or yellowish brown, and their food is all covered with material and they have to inhale this perfectly ghastly stuff which—not because it is tobacco but because it is smoke—would choke almost anything.

They give them this terrific beating and still the animal is not obliging enough to develop the type of lung cancer that they rather hoped that it would, because everybody hopes for positive results. Negative results are almost immediately buried.

Mr. KILGORE. This is a ridiculous thing for a layman to conclude, I guess, but the prior differentiation you made between the biological

structure of mice compared to man might be the factor which would cause the mouse to react differently to something induced into——

Dr. LITTLE. Yes; it might be. It isn't only between mice and men. It is very, very difficult to induce any kind of a skin cancer on a guinea pig. I don't mean by this that guinea pigs are any more like man, but in that respect they are. There are these literally hundreds of materials that you can paint on the skin of mice and you can get skin cancer in some mice.

There, again, I could go back to the Jackson Laboratory and ask them to pick strain X and give it to an experimenter, and he would get perhaps only half of 1 percent, or 1 percent, skin cancer. I can say, "All right, give the boy a break and give him strain Y," and he will get as high as 90 to 100 percent skin cancer with the same material and the same dosage.

Mr. KILGORE. Doctor, in commenting on the increase in the life span of man during the same period in which there has been a marked increase that there has been in smoking, would it be fair also to say—using the information presented earlier with respect to this controlled group of smokers and nonsmokers—that the increase in the life span of nonsmokers during this same period of time has been greater than has been the increased life span of smokers?

Dr. LITTLE. I am not sure about those data. I am honestly not. I could look them up. I don't know whether any comparison has been made where the other factors of the environment have been equalized.

You see, if one takes just the age and the smoking habit alone, he forgets a lot of other things. I shouldn't be at all surprised if the type of person who was an excessive smoker is a bad health risk, not alone for smoking but for automboile accidents and violent death of different types.

In other words, a person who reacts to life by excessive habits is apt to be a person off the normal and is not apt to be a good risk as far as well-adjusted participation in life is concerned.

But when one gets to the cause-and-effect basis, then I think it is a great mistake to let these relationships have any lasting influence at all, because the cause-and-effect relationship is a tough thing to prove and has not been proven.

Mr. KILGORE. Just one last question. Somewhat in summary of your position, it would be accurate to say, then, would it not, that you do not challenge the existence of a higher incidence of death among smokers in comparison to nonsmokers insofar as many causes of death are concerned, but you do not see sufficient evidence to establish to your thinking the causative relationship to death and smoking?

Dr. LITTLE. I have no reason to challenge the death rate figures in the statistics. I would say that I see no reason whatever to single out a single factor and look upon that as the key to this situation. I see no evidence yet that convinces me that, per se, smoking is a causative factor in this situation.

Mr. PLAPINGER. Is the reverse true, Doctor?

Dr. LITTLE. No; I don't think it is. There are some figures in lung cancer incidence that are very, very interesting. As far as that is concerned, practically the age situation alone affects it importantly.

There was actually a bigger percentage increase—percentage increase—in increasing age groups among the nonsmokers than there was in the smokers. In other words, if you take the death rate or

death quantity at this age limit and take that as a unit and compare it with the next one, and then take this as a unit and compare it with the next one, you actually find a decreasing rate of death in the smokers from what you do in the nonsmokers.

What this means I am not prepared to say, but I will go this far, that we do need to know an awful lot more than we do about this whole problem, in my opinion, before we become fixed or dogmatic about it.

I am very much humbled by the power of cancer, by how much it feels at home in our body, and the fact that it can outlive us and outlast us. I think we have got hold of an enemy that is well worth a great deal of caution on our part before we attempt to say we know about it.

Mr. BLATNIK. Doctor, to wind up the hearings, we appreciate your sincere testimony here. I am still not clear now, but am I correct in saying that, as far as you know, smoking does not induce cancer of the lung or any other cancer?

Dr. LITTLE. As far as I know, in human beings the cause-and-effect relationship between smoking and lung cancer has not been established beyond question. This does not mean that I do not think it is an open question, because I think it is.

I think it is one of the things that we ought to know more about. But if you were to ask me, am I satisfied with the evidence and am I ready to accept it as cause-and-effect relationship and begin to build for the future on that, I would say "No."

Mr. BLATNIK. But as far as you know now, it cannot be proven that smoking causes cancer. Is that correct?

Dr. LITTLE. I think that is right. I also urge, please, if I may, that the study be continued, because I don't say that it can't.

Mr. BLATNIK. I understand that, and I agree with you. Then, Doctor, are there any other harmful effects that you can ascribe to smoking or heavy smoking?

Dr. LITTLE. I can't ascribe these. I have seen the correlations in Dr. Hammond's paper. I would say that the list of diseases, which is pretty impressive—which I read earlier—could, if the relationship continues and the figures get significant, some day be accused in the same way that it is now being accused for lung cancer.

I very much doubt personally whether it would show the cause and effect any more than I think the present figures do. An association, yes; but an association is quite different from a proven cause-and-effect relationship. A lot of things may happen that don't come from the same cause.

Mr. BLATNIK. So a general conclusion would be that, as far as you know, findings available to date indicate there are no other harmful effects that can be ascribed to smoking, either?

Dr. LITTLE. I couldn't go so far as to say that. I think that I might say that abuse of almost any human habit could lead to harmful effects. I would include asceticism as one of the harmful human habits because I think that often kills more people than——

Mr. BLATNIK. Would you have any idea, then, Doctor, why the industry which has created your most worthy research committee must have facts that impelled them to go to quite some special effort and additional expense to themselves and to consumers to produce filters in such a rapid increase in the past 4 years?

Dr. LITTLE. I wouldn't know about the filters, but the attack on tobacco was so well organized, so well propagandized, and so professional, that when it came first I think it stunned the industry wide awake. I think it suddenly realized, "The accusations that are being made now, we don't intend to ignore any more. We are going to stay with this problem until we find out, if we can, what the facts are, because if they are true they have got to be corrected, if possible; and if they are not true, we have got to find out. We can't go along any more subject to violent attacks by people without taking steps to do the best we can to find out whether these attacks are well founded or not."

Then at that stage of the game, when they came to me, I said, "All right, if you want me to do this, I would love to try this. There are probably better people. But this you remember, the important thing you said was we are going to try to find out the effects, whether they are good or bad," and that is going to be true.

The industry is prepared for that, because it is no longer going to be willing to go on with uncertainty and accusations or even comments without knowing if it can what the basis for those really is.

I think maybe the power of the attack on smoking may have been such that a very normal reaction might have been, "Well, is there anything that can be done or should be done while we are trying to find out what the fact is?"

I have no opinion about, as I say, the filter at all. I don't know why it was done, and I frankly—if you don't think I am in contempt—care very little. I care less, really.

Mr. BLATNIK. I am curious now. You are conducting research. Have you asked your own people, your tobacco industry, why did they promote filter tips at such great expense?

Dr. LITTLE. No.

Mr. BLATNIK. You haven't?

Dr. LITTLE. No.

Mr. BLATNIK. Are you interested in knowing why they do it? They are spending considerably more money for filter tips than they are for your research work.

Dr. LITTLE. Personally I am not awfully interested in why they are doing it. I wish they would spend more money for the research work than they do for filter tips. Maybe someday they will.

Mr. BLATNIK. Do they have any other research facilities perhaps unknown to you in their own laboratories or any other financing of other foundations or any other sources?

Dr. LITTLE. I think it is generally known that some of the companies at least have had research laboratories working for years. The scientific advisory board visited at least two of these laboratories, but they have done absolutely nothing to suggest research to them; nor have they commented on or advised as to the types of research being done.

It has simply gone in there to observe, and it would seem to me, frankly, that the question, about filters, Mr. Chairman, that that should be of much more interest to the industrial laboratory than it is to my committee or to me.

It isn't that we are pure. It is just that we have got hold of a lion and we can't let him go.

Mr. BLATNIK. May I ask, do these tobacco companies then consult you on the effectiveness of filter tips?

Dr. LITTLE. They don't consult us at all.

Mr. BLATNIK. They have not consulted you, not made any recommendation?

Dr. LITTLE. Not in the least. I think they are very wise because all I would say is I don't know.

Mr. BLATNIK. You don't know?

This book was sent to the committee, I believe, either from your office—Science Looks at Smoking. Are you familiar with this book?

Dr. LITTLE. It wasn't sent you by the scientific advisory board. Whether it was sent from anybody connected with the industry or not, I don't know. But it was not sent to you by the scientific advisory board.

Mr. BLATNIK. Somebody connected with the industry, then, sent this to the committee. This book was sent to the subcommittee by a public-relations firm known as Hill & Knowlton, which I am informed represents the Tobacco Industry Research Committee, which is your committee. Is that correct?

Dr. LITTLE. Yes; it represents the public-relations phase of that.

Mr. BLATNIK. Would that include your research committee, or is this information——

Dr. LITTLE. It would definitely include the scientific committee as a subsidiary, you see. But that Hill & Knowlton has any effect on the scientific program would be absolutely negative. They not only don't have any effect on it but they are not consulted about it and I don't think they would have any advice to give about it. If they did, it frankly would not be of interest to my associates or to me. They are not scientists.

Mr. BLATNIK. Are they to release information prepared or compiled by your research committee or by the tobacco industry?

Dr. LITTLE. I don't know what they are doing about the book. I don't know what they are releasing about the book or what they aren't. This book is not sponsored by the committee of which I am the chairman. We have no part in the preparation of this book beyond the fact that I was shown Dr. Greene's introduction to it, which is a purely scientific evaluation, and Greene is doing research himself and I was interested in it as to his ideas about research.

Mr. HARDY. Are you familiar with the book, Dr. Little?

Dr. LITTLE. With this book? I haven't read it through. I am familiar with the fact that it exists; yes.

Mr. BLATNIK. The reason I ask, Doctor, here is a book and I wanted to check this, I am informed and I may be in error, I don't want to present this as the final—this book was prepared by Hill & Knowlton, a public-relations firm which represents the tobacco industry, including your Tobacco Industry Research Committee.

They have released a book called Science Looks at Smoking. Is this a scientific agency?

Dr. LITTLE. Hill & Knowlton?

Mr. BLATNIK. Yes, Hill & Knowlton.

Dr. LITTLE. I don't believe anybody would call it a scientific agency. I think it is a firm of public relations advisers and counsel. But perfectly frankly——

Mr. HARDY. Let me ask a direct question. Is that propaganda or is it scientific?

Dr. LITTLE. As far as I know it is a fair statement of this man's belief—what is his name—Northrup?

I believe that these are his honest——

Mr. HARDY. I haven't seen it. I don't known anything about it.

Dr. LITTLE. There have been a number of pro and con statements as you know, that have come out on this problem and there will probably continue to be. I don't know that this could be called a pro, or whether it is just in the nature of an attempt at a review.

Mr. BLATNIK. The reason I ask, here is a book distributed by Hill & Knowlton which is a public-relations firm for the tobacco industry and your committee and they are releasing a book "Science Looks at Smoking," and on page 183 referring to filter tips, of which you have no opinion, they state:

> On the other hand, there is no scientific evidence that filter tips provide a special safeguard in smoking. They merely reduce the smoking particle intake but not particularly selectively.

This is not either the findings of your committee or the tobacco industry?

Dr. LITTLE. No.

Mr. BLATNIK. But it is a book which was released by the public-relations firm of Hill & Knowlton——

Dr. LITTLE. Which represents the industry, yes.

Mr. MINSHALL. Doctor, it is difficult for me to believe you don't know anything about the effectiveness of filter tips on cigarettes.

Dr. LITTLE. I don't.

Mr. MINSHALL. A man in your high position visiting these various scientific laboratories operated by the tobacco companies, haven't they ever shown you any of the results of the abilities of these filters to remove tars or nicotine?

Dr. LITTLE. No, they really haven't.

Mr. MINSHALL. Have you ever inquired about it?

Dr. LITTLE. No, I haven't been in the least interested.

Mr. MINSHALL. Why aren't you interested?

Dr. LITTLE. Because I don't believe they are the answer to the problem. The first thing to do is to find out what substance in tobacco, if any, causes the trouble. The question of removing them or possible manipulation of them comes after their identification, and they haven't been identified.

Mr. MINSHALL. You have never seen any records or any data of any kind about the ability of certain kinds of filters to remove tars and nicotines from cigarettes?

Dr. LITTLE. Only what I have read in the press.

Mr. MINSHALL. You have never seen any publications or any data supplied by the tobacco people?

Dr. LITTLE. I haven't read a single paper on it. Very honestly, I am not interested. I have got too many other things of real interest in connection with the origin and nature of cancer, and any possible relationship that there may be, to get into the technical problems. I am not interested.

Mr. MINSHALL. I thought you said your job was just to raise mice up there in Maine to distribute to these people, and all the scientific data was done by these grants and these fellowships.

Dr. LITTLE. I happen to be chairman of the advisory committee to the industry. I am the ex-director of the laboratory. I have these associates who are listed in this material that has been given you. My job is to do everything I can to spend the tobacco industry money for good, sound, constructive research leading to the truth.

I retired as director of the Jackson Laboratory last year, because I reached an age limit when I felt that was wise in the whole picture. But I am vitally interested in the laboratory because I helped found it and it is the place I wanted to work in.

Mr. MINSHALL. Your job right now then is—all you do is hand out the money to these various organizations?

Dr. LITTLE. That is all the job that I have in connection with the tobacco industry, yes, is to hand out the money, confer with the scientific associates that I have as to possible new leads that we should be investigating.

Mr. MINSHALL. Don't some of these people that you grant money to check the effectiveness of filters as part of their study?

Dr. LITTLE. Are any of them working on filters, Bob?

Dr. HOCKETT. Not to my knowledge. They are trying to find out whether the typical cigarette of the type that has been smoked for the last 25 or 30 years does or does not do certain effects. That question isn't answered yet.

Dr. LITTLE. If you mean have we a program to try to justify filters or evaluate them; no. Fortunately we have been left absolutely clear of commercial impact of a single blessed thing we are studying. They are letting us study anything we want and you would be surprised if you saw some of the things that are being studied. They are about as far removed from tobacco and its possible effect on cancer as you can possibly go. When you see them, you will see the list and see what you think of it.

Mr. BLATNIK. Doctor, back to the filters and the cigarettes, you state as far as you know there is nothing in the cigarette that would induce cancer or be carcinogenic in nature. On page 16 of the testimony by Dr. Hammond, he refers to 3.4 benzpyrene. Is that substance or hydrocarbon in tobacco smoke?

Dr. LITTLE. That is carcinogenic on the skin of mice, if you give it to them in quantity. But the quantity in which it appears most radical in analysis by the biochemists, as I understand it, is such that a person would have to smoke 250,000 packages of cigarettes in a month or something of that kind. It is well known that benzpyrene also exists in the exhaust of any diesel engine or any of the common polycyclic hydrocarbon producing combustion products.

Mr. BLATNIK. It is carcinogenic?

Dr. LITTLE. It is for mice.

Mr. BLATNIK It exists in extremely small quantities?

Dr. LITTLE. Yes.

Mr. BLATNIK. Is it cumulative?

Dr. LITTLE. There is no evidence of that. That is a hard question for me to answer because I am thinking hard now of the types of tests of accumulation that experimental people have made of it. I would say, not to a sufficient degree to expect a local effect from it.

Mr. BLATNIK. Has any research work been done on benzpyrene?

Dr. LITTLE. It is being worked on quite hard at the present moment. The British are working on it, and there is a team over in Switzerland

working on it. The moment a suspected thing like that comes up, the usual thing is to just give it everything you have got—drive it, find out what concentration will produce a cancer, find out what concentration it exists in.

Mr. BLATNIK. You mentioned British and Swiss teams working on it. Is anyone in America working on it?

Dr. LITTLE. They are working on it independently of tobacco. It is a very common chemical used in most of the cancer laboratories to induce cancer in animals.

Mr. BLATNIK. But it is the only substance that has been identified to be present, although in minute quantities, in tobacco smoke that is carcinogenic?

Dr. LITTLE. I think that there have been claims by some that another polycyclic hydrocarbon related to benzpyrene——

Mr. BLATNIK. So there may be more than one?

Dr. LITTLE. There may be.

Mr. BLATNIK. But this definitely is carcinogenic?

Dr. LITTLE. If you get enough of it at the right place; yes, sir.

Mr. BLATNIK. And you have run no tests on whether it is cumulative?

Dr. LITTLE. I don't know of any evidence that would bear that out.

Mr. BLATNIK. The reason I ask that question, Doctor—along about in the early thirties, if I recall the date correctly, when lead gasoline, high-test gasoline, was first coming out on the market, the amounts of lead were so minute that it was impossible, it was felt, to get lead poisoning. Yet there developed severe cases of lead poisoning in the industry, and the medical people felt it was incredible. They could not get this poisoning in the work they were doing with this lead gas, until they discovered it was cumulative.

Over a period of years, like in smoking 10 or 20 years, here was this accumulation of lead poisoning, which was serious and in some cases fatal. Could that be possible with this carcinogenic?

Dr. LITTLE. I am not a good enough chemist to answer your question, but just from the point of view of trying to reason toward it, I wonder why, if this was retained by an organ like the lung, that any evidence which might be attributed to it occurs in such a minute location? It would seem to me more that the importance of the reaction was in the nature of the soil affected than it was the agent affecting it, because we know that cancer can be triggered by hundreds of different chemicals.

The question is, why isn't it triggered all the time by almost everything?

Mr. MINSHALL. Mr. Chairman, at this point I would like to put in the record an article that appeared in the Journal du Dimanche of Paris on Sunday, June 30, 1957, that relates to this benzpyrene research that the French scientists have been doing.

Mr. BLATNIK. Are you asking to put this in the record?

Mr. MINSHALL. Yes.

Mr. BLATNIK. These are French scientists?

Mr. MINSHALL. Yes, sir.

Mr. BLATNIK. I thought we were talking about England and Switzerland where they have teams. Also in France? Is that correct?

Mr. MINSHALL. Yes.

Dr. LITTLE. I think this is a different benzpyrene from the one you spoke of; I am sorry.

Mr. BLATNIK. This refers to 3,4-benzpyrene. Without objection, the article will be inserted in the record at this point.

(The article is as follows:)

[Translation of an article published in Le Journal du Dimanche, Paris, Sunday, June 30, 1957]

THE NONCANCERIGENOUS CIGARETTE IS BORN—FIVE FRENCH SCIENTISTS HAVE GIVEN IT THE FINAL TOUCH—TWO SMOKING ROBOTS USED FOR TESTS

French tobacco and cigarette paper made in France are the first in the world to be treated with a view to reducing considerably the risks of 2 types of smokers' cancers—that of the lung and that of the larynx—2 ills which are becoming increasingly current, according to statistics published in various countries.

Two laboratories are from now onward bound to cooperate closely: that of the famous cancer specialist Dr. Raymond Latarjet of the Radium Institute, and that of the chemist Jean-Louis Cusin, attached to the Tobacco Government Administration. They are in fact working on two chemical processes, one applied to tobacco and the other to cigarette paper, intended to protect the health of heavy smokers.

The various substances used in those processes, on an industrial scale, have already been selected. In order to prevent the formation of dangerous paper tars, ammonium sulfamate will be used and for tobacco certain nitrogenous compounds.

A SMOKING MACHINE

Two robot smokers have been used for the experiments. The first has been put up at Quai Branly by Cusin and the other by Latarjet, Rue Pierre-Curie. The robot consists of a glass appliance, where combustion speed of the tobacco, the cigarette paper or the whole cigarette is controlled at the same time by the aspiration of a water pump and by the cramming into a tube of the sample to be burned and of a piece of glass wool.

While the machine smokes, the tarry substances contained in the tobacco or in the paper adhere to the glass wool, rendering it increasingly compact. At the end of combustion these residual substances are dissolved by the washing of the whole appliance. The solution is then examined by the chemists.

What have the tests carried out at the institute just revealed?

Both in the paper tars and the tars of complete cigarettes, Messrs. Latarjet, Cusin, Hubert-Habart, Muel, and Royer have discovered a substance which they identify as the 3,4-benzpyrene. Now this hydrocarbon is, according to recent works, principally responsible for the cancerization of bronchial and laryngeal cells.

The French team have, on the other hand, definitely established that cigarette paper was far more cancerigenous than tobacco.

The treatment of that paper with ammonium sulfamate by the French scientists prevents to a considerable extent the formation of dangerous substances.

NITROGEN TREATMENT OF TOBACCO

If we take the cigarette alone, the quantity of benzpyrene formed by the combustion of 100 cigarettes is only of the order of 1.2 thousandths of a milligram.

Yet, here also, it is possible to lessen much the formation of tobacco tar. The Latarjet team have even discovered that the addition of certain nitrogenous compounds to the tobacco may lead to a lowering of the benzpyrene proportion, as important as that of the paper treated with ammonium sulfamate.

This is how the French scientific work will result in rendering noncancerigenous the 450 milliards of cigarettes smoked annually in the world.

Dr. LITTLE. That is the 3, 4. I don't know about the quantity of it. It is an interesting suggestion. But I am not enough of a chemist to pass judgment on it.

Mr. PLAPINGER. I want to read a quotation attributed to you. I would like your comment on it. You are quoted as saying that—

Any possible role of smoking in the etiology of lung cancer remains an unresolved question. It cannot be said that smoking has been absolved from suspicion. Neither have the charges been proven.

Does that accurately reflect your position, sir?

Dr. LITTLE. Yes.

Mr. PLAPINGER. I would like also to cite to you a passage from the book, Cancer, a Study for Laymen, in which you are said to have written a chapter which states that—

Although no definite evidence exists concerning the relation between the use of tobacco in the instance of lung cancer, it would seem unwise to fill the lungs repeatedly with the suspension of fine particles of tobacco products of which smoke consists. It is difficult to see how such particles can be prevented from becoming lodged in the walls of the lungs and when so located, how they can avoid producing a certain amount of irritation. One might also question the ultimate results of continued inhalation of the type of atmosphere which characterizes the lower levels of city streets. Experimental work with animals involving these matters is still inconclusive, but it seems probable that the lung as an organ is not immune to the effect of chronic irritation and that it will in this respect resemble the other organs of the body. Such being the case, wisdom in avoiding unnecessary lung irritation seems to be established.

Is that statement made in this 1944 book still applicable?

Dr. LITTLE. To a large degree, yes. If you would be willing to consider it modified to the extent that any prolonged irritation——

Mr. PLAPINGER. Not only of tobacco.

Dr. LITTLE. Not only of tobacco, but of any—that that would be a fairer statement, and that the reaction of different people's lungs to different levels should throw out any sweeping generalization. The trouble with writing anything like that, as I did, or anybody does in a book, you have to put it down to a statement and trust to a chance to qualify it afterward, as I am now doing it with you, talking it over.

Mr. PLAPINGER. Thank you, sir.

Mr. BLATNIK. Doctor, we thank you very much for your patience.

Dr. LITTLE. Thank you very much for your courtesy and your interest.

(Whereupon, at 1:30 p. m. the committee adjourned, to reconvene at 10 a. m., Friday, July 19, 1957.)

FALSE AND MISLEADING ADVERTISING

(Filter-Tip Cigarettes)

FRIDAY, JULY 19, 1957

HOUSE OF REPRESENTATIVES,
LEGAL AND MONETARY AFFAIRS SUBCOMMITTEE
OF THE COMMITTEE ON GOVERNMENT OPERATIONS,
Washington, D. C.

The subcommittee met, pursuant to recess, at 10:05 a. m. in room 100, George Washington Inn, Hon. John A. Blatnik (chairman) presiding.

Present: Representatives Blatnik, Kilgore, Hardy, Meader, and Minshall.

Also present: Jerome S. Plapinger, counsel; Curtis E. Johnson, staff director; and Elizabeth D. Heater, clerk.

Mr. BLATNIK. The Subcommittee on Legal and Monetary Affairs of the Committee on Government Operations will continue in public hearings in further consideration of the question of possible fraudulent and misleading advertising in the matter of several products being advertised, the first of the series being the question of cigarettes—particularly filter cigarettes.

On our second day of hearings we have with us and will hear first this morning Dr. Ernest L. Wynder, of the Sloan-Kettering Institute for Cancer Research.

Dr. Wynder, would you please take the chair.

Doctor, I see you have a lengthy prepared statement. Would you at the outset identify yourself by name, title, and a brief summary of your professional background, particularly how long you were in your current work?

STATEMENT OF DR. ERNEST L. WYNDER, SLOAN-KETTERING INSTITUTE FOR CANCER RESEARCH

Dr. WYNDER. My name is Ernest L. Wynder. I received my medical degree from Washington University School of Medicine in St. Louis in 1950.

In 1947 I began to become interested in the problem of the etiology of lung cancer, together with Dr. Evarts A. Graham, at that time professor of surgery at Washington University.

I have been engaged in clinical and experimental cancer research since that time. I interned at Georgetown University Hospital here in Washington and had 3 years of residency at the Memorial Center for Cancer and Allied Diseases, and have been with Memorial Hospital since 1951.

I am currently heading the section of epidemiology at Memorial Center and in this section we are interested in the cause or causes of all types of cancer and have during the past few years published reports not only on the etiology of lung cancer but also on the etiologic factors of other cancers of the respiratory tract as well as cancer of the cervix and are currently completing studies on cancer of the breast and stomach.

The section also includes a laboratory section where we are investigating the possible carcinogenic activity of tobacco tar and of other agents.

I would like to present to you a summary of evidence which we believe demonstrates a causal relationship between smoking and cancer of the respiratory tract, and make a number of recommendations which we think can successfully overcome this problem, at least in part.

As you know and heard in testimony yesterday, the primary reason why this problem has become so acute is because of the rather high prevalence of lung cancer today. More than 25,000 lives are lost in this country each year due to lung cancer alone.

When we first began studying this problem, we were impressed that this was a real increase for two reasons: One, we felt that improved diagnosis, which so often has been mentioned as accounting for this increase, could not account for the difference in sex ratio because certainly improved diagnosis applies to women as much as it does to men. In fact, it has been our experience in cancer detection work that women are more likely to come to a doctor than men.

Secondly, frequently I hear somebody say "Well, there is more of cancer today because of an aging population." First of all, the studies by the National Cancer Institute and others have shown that you can age-standardize your population group and you still have a marked increase in lung cancer, and secondly, lung cancer occurs in a younger age group than most other major cancers, in whom a similar increase has not been noted.

What is then the evidence that we believe demonstrates conclusively that smoking is a cause of lung cancer? The first of these is what we will refer to as presumptive evidence. It is perhaps like in a trial where you say "Could this particular criminal have been at the scene of the crime?"

In scientific research, or for that matter in any other research problem, an investigator first considers the most likely answer to the given problem. What is the presumptive evidence with respect to lung cancer? It is as follows:

One, the increase in lung cancer has been more marked in men than in women. This is in line with the fact that males currently in the cancer age smoke more heavily than women in the same age group.

I will demonstrate facts on this later. This fact would be difficult to explain on the basis of air pollution since the women in any given city area are as much exposed to polluted air as are men.

Two, in countries in which there has been a sharp increase in the incidence of lung cancer there has also been a sharp increase in the consumption of cigarettes. By itself, of course—and we expressed this repeatedly—this factor means very little, because similarly there undoubtedly has been an increase in the sale of refrigerators and automobiles, and as Dr. Graham often jokingly stated, in nylon stockings. However, in the absence of an increase in the consumption of cigarettes, it would have been difficult to incriminate this smoking product.

Three, in countries such as Norway and particularly Iceland where the consumption of cigarettes is low and yet excellent medical facilities and reporting are available, the incidence of lung cancer is quite low. At the time when in the United States lung cancer accounts for the most common cause of cancer deaths in men, it ranks only ninth in frequency in Iceland.

Four, contrary to most other cancers, the age distribution of lung cancer reaches a peak in the sixth and seventh decades and then declines. This is in line with recent data by the United States Census Bureau that there are more heavy cigarette smokers in the younger age groups than in men now past 65.

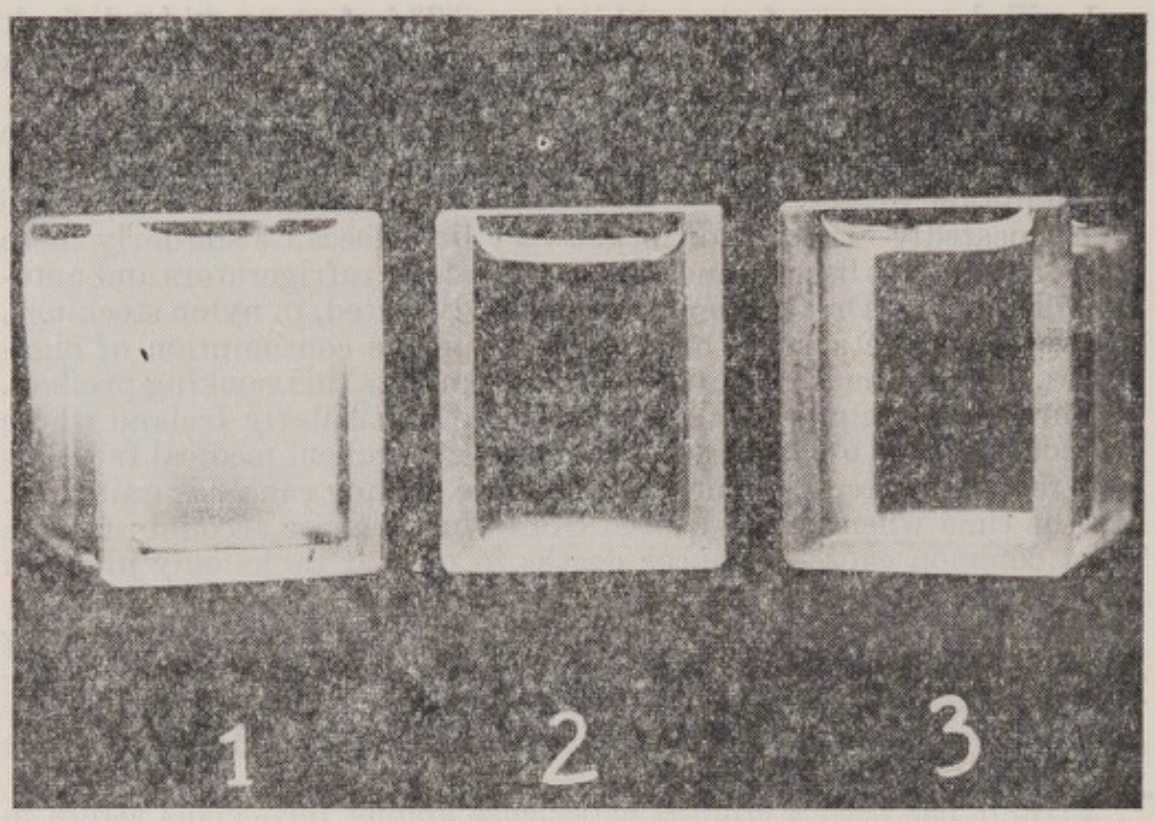
Five, it has been a clinical experience among physicians astute in the taking of medical histories that patients with lung cancer are mostly excessive smokers. It has also been a repeated clinical observation which is known not only to doctors but I am sure also to laymen, that a chronic cough, particularly in the morning, is much more commonly seen among heavy smokers than among nonsmokers. Certainly the existence of a cough indicates an irritation to the bronchial epithelium.

Sixth, one of the established principles in carcinogenesis shows that pyrolysis of nearly all organic matter can create higher aromatic polycyclics which are of known carcinogenic activity. To the cancer worker therefore, it was not a surprise that tobacco, certainly an organic matter, when combusted at such high temperatures that are obtained during smoking, reaching a temperature level of 880° C, can form such higher aromatic polycyclics.

Ernest Kennaway, who is one of the leading cancer researchers in Great Britain, stated he was surprised that in view of the high temperatures reached in the combustion of tobacco, that tobacco wasn't even more carcinogenic than evidence indicates.

These points are our presumptive evidence. They are presented to show that the other evidence which we want to present now is consistent with this presumptive evidence, thus strengthening both.

Now there are two slides I would like to show you which add to this presumptive evidence. The first one is work reported by Dr. Drockrey from Germany.



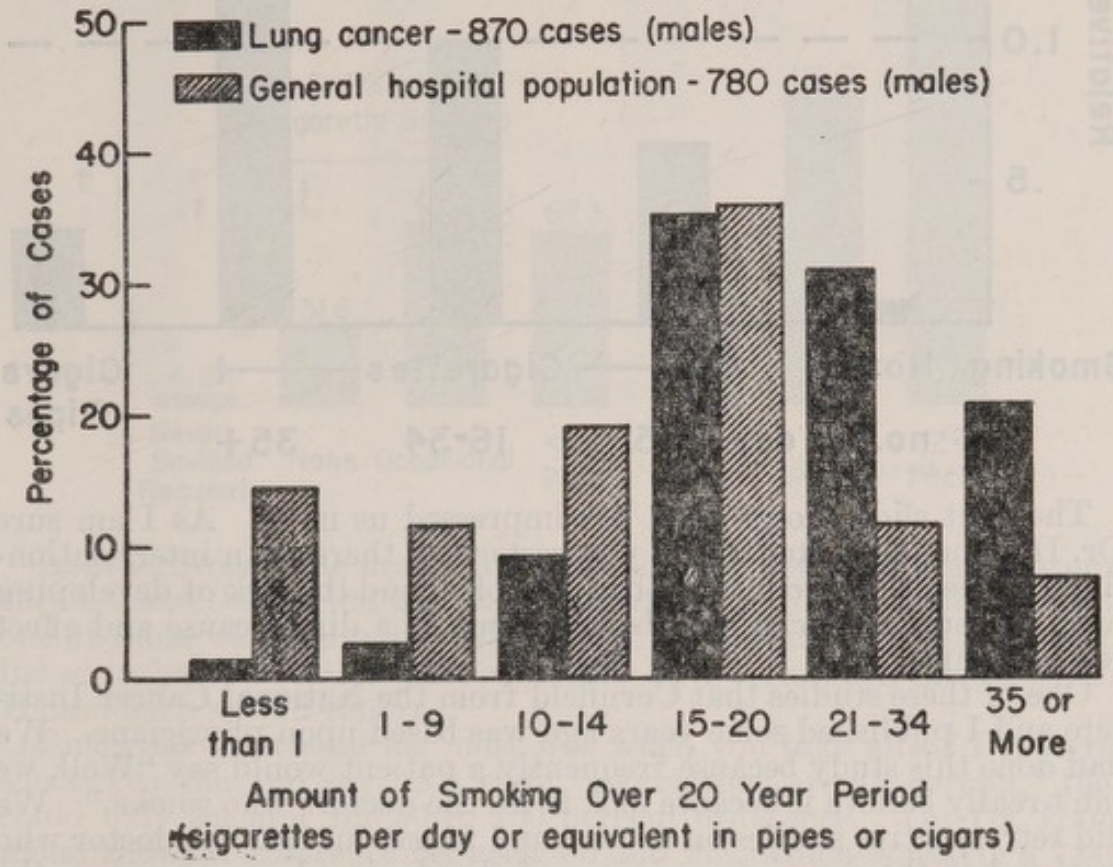
In the section No. 2 you see opaqual free benzol on which you shine ultraviolet light and see practically nothing there. (1) Section No. 1 cigarette smoke blown into optical free benzol without inhalation by the smoker and you will note there is a very marked fluorescence. In No. 3, the smoker inhaled and then exhaled into the optical free benzol and you will see that the fluorescence is much less than in No. 1, which indicates that of the fluorescent particles in the cigarette smoke over 90 percent are retained and therefore absorbed by the lung tissue.



The next slide shows a very interesting study conducted by Dr. Mellors of our Institute with whom we are collaborating. (2) This comes from a patient who has developed leukoplakia which we think is also related frequently to excessive smoking. On the left are cells from his buccal mucosa, which makes up the lining of his mouth, when he had not been smoking. To the right the marked fluorescent cell is a buccal mucosa cell taken a few hours after he had smoked a cigarette. What does it demonstrate? It demonstrates that a

fluorescence component of the smoke of a cigarette can invade the cell. So we do know, we have now ample evidence both from the work of Druckrey and Mezlors that cigarette smoke can enter the human epithelial cell. In other words, we can demonstrate that the suspected agent has been found at the scene of the crime.

So much for what we call presumptive evidence. What about the other evidence? Let's first see epidemiological evidence which in part was summarized for you yesterday by Dr. Hammond. The first slide on this evidence shows work that Dr. Graham and I presented some

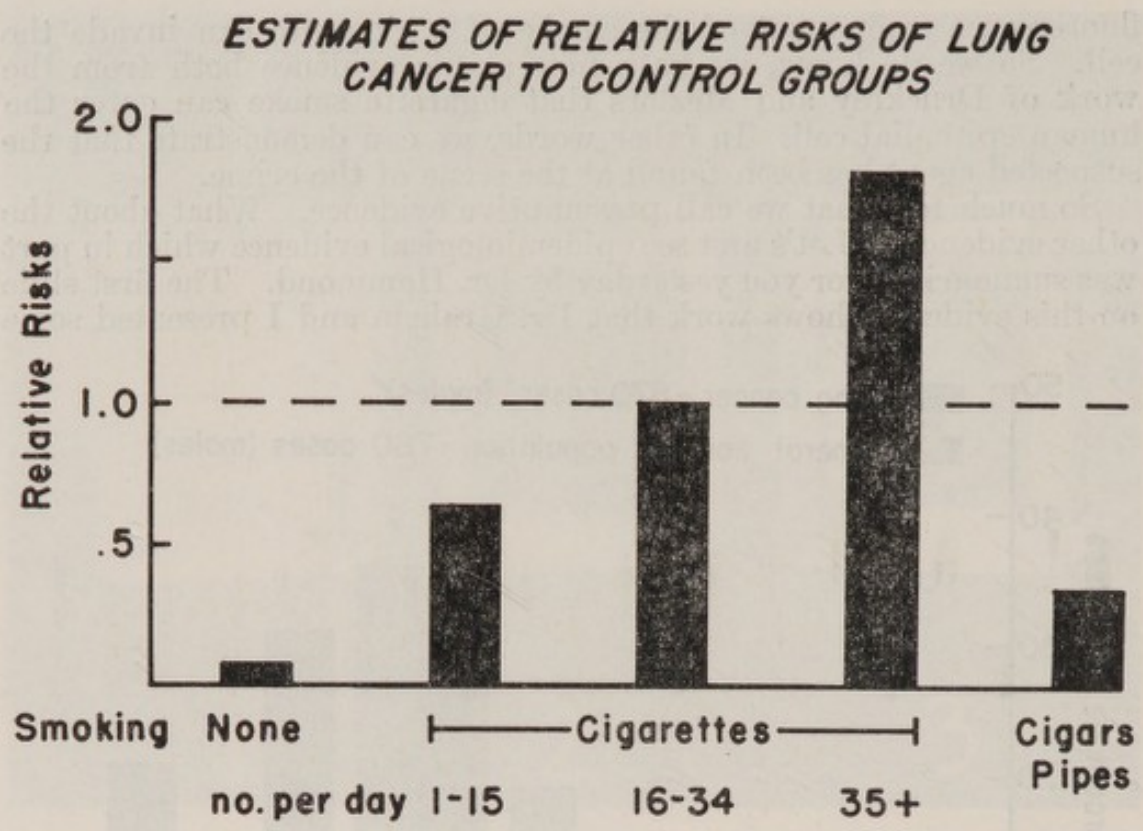


years ago. This is a retrospective study based on 870 lung-cancer patients, all of whom had histologically proved diagnosis. You will see when you compare these to matched controls there are much fewer nonsmokers in the lung-cancer group and many more heavy cigarette smokers in the lung-cancer group.

To me the most impressive thing about this has been the rarety with which lung cancer occurs in nonsmokers. Yesterday you had testimony that lung cancer can occur in nonsmokers and we have never denied it. In fact, we have written a report on cancer of the lung in nonsmokers, summarizing our experience in over a thousand cases in which we could find only 20 who had never smoked.

Similarly, Doll and Hill were so impressed with the rarity of nonsmoking lung-cancer patients that they wrote a special report on this subject. It is that infrequent. In half of these patients we found occupational exposures which could account for their lung cancer.

These studies now have been repeated in 7 different countries, 1 of them, and the most recent one was financed by the French tobacco monopoly. This study by Dendix and Swartz, and our studies have shown in this country a marked relationship between smoking and lung cancer.



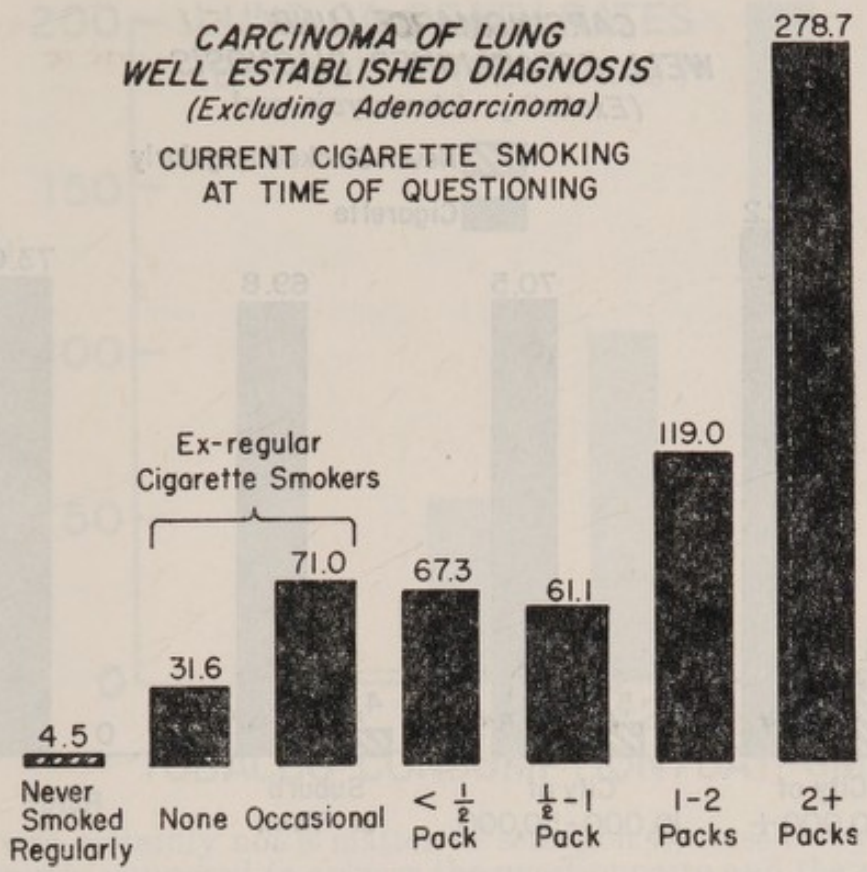
The next slide shows what has impressed us most. As I am sure Dr. Hammond pointed out to you yesterday, there is an interrelationship between the amount of tobacco smoked and the risk of developing lung cancer. This can only be the result of a direct cause and effect relationship.

One of these studies that Cornfield from the National Cancer Institute and I published some years ago was based upon physicians. We had done this study because frequently a patient would say "Well, we can't really believe it because look at all the doctors who smoke." We did retrospective studies on doctors and found out that the doctor who smokes has the very same chance of developing lung cancer as the average layman. To me, all this indicated that on these matters a doctor can be just as stupid as the average laymen.

In other words, the point to be recognized from these retrospective studies is that without exception, in all studies that have had at least 100 lung-cancer cases, the same relationship was seen that you see on this particular graph. In 1950 Dr. Hammond, who you heard yesterday, came to see us in St. Louis, as I am sure he told you. He thought there was nothing to it because he thought that the retrospective studies had certain faults and he therefore started the prospective studies which he reported to you yesterday.

I say it because if there was anybody biased toward the other end, it was Dr. Hammond of the American Cancer Society.

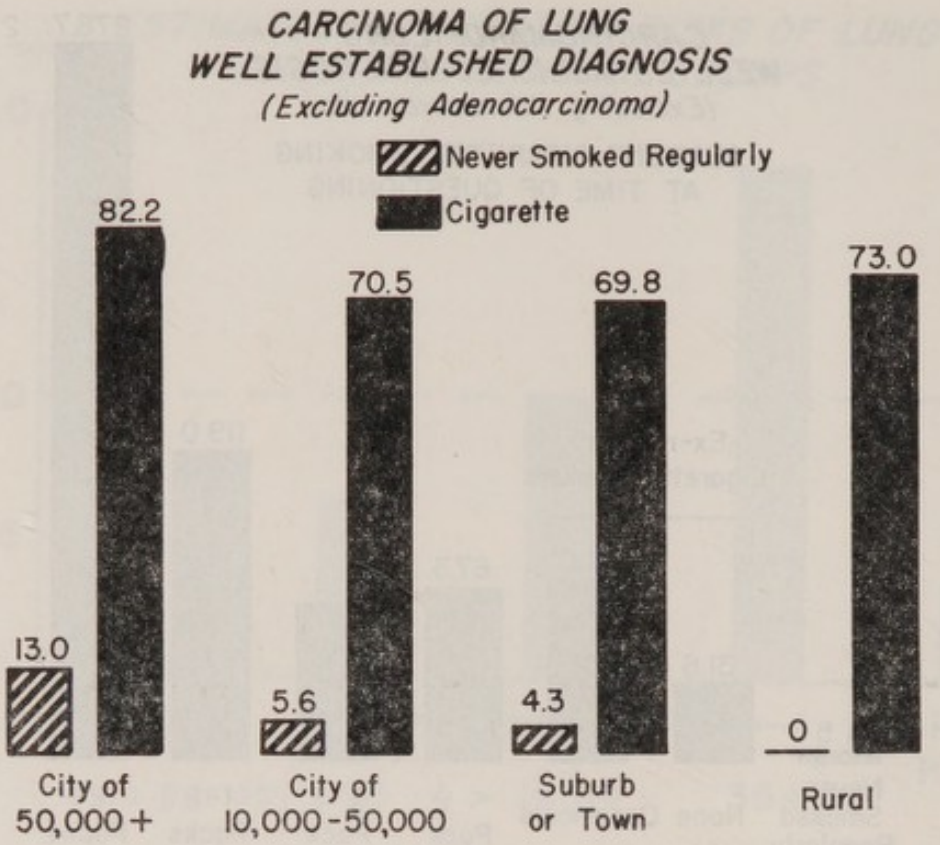
The next slide shows the study which he reported to you in detail yesterday, and the only reason I want to show it again is to demonstrate to you what has impressed me most—that is the enormity of the relationship.



In other words, the death rate of the nonsmoker from lung cancer is only 4.5, and that of the 2-pack-a-day-smoker is 278. Dr. Berkson was one of those who has criticized the Hammond and Horn study as published in 1954. Most of these criticisms have been taken care of by Dr. Hammond's second report.

But what impressed me most was when you take all of Dr. Berkson's criticism and say "All right, maybe they are all correct," they could only account for 50 to 100 percent difference. Yet Dr. Hammond shows a difference between 4.5 and 278, which is nearly a 7,000 percent difference. To declare that this is on a statistical error or bias, you have to demonstrate an error of equal enormity.

The next slide which Dr. Hammond also showed to you is marked "parallelism" for risk of lung cancer between city folk and rural folk if you standardize for the amount of cigarette smoking.

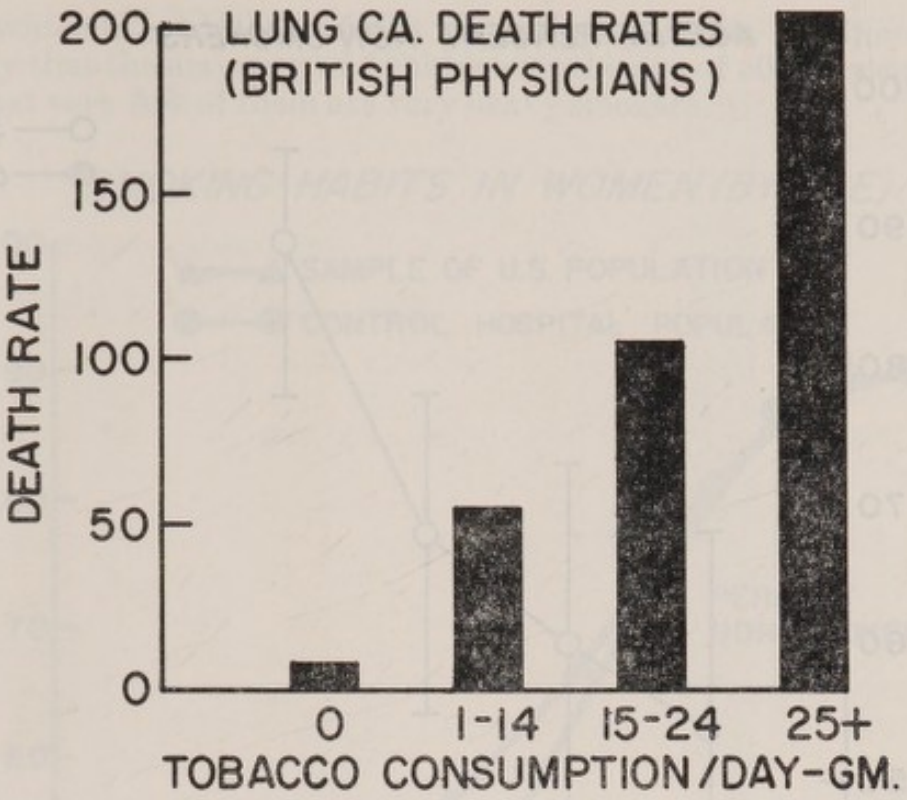


There is a slight increase among nonsmokers who develop lung cancer, in cities. But this is very minimal and it is entirely in line with our belief that there are probably other causes of lung cancer.

Dr. Graham and I have written a special report on occupational influences of lung cancer. We feel that the same preventive measures have been directed against them as against tobacco.

One of the criticisms to Hammond and Horn was that their cases perhaps did not represent the general population, and were artificially selected.

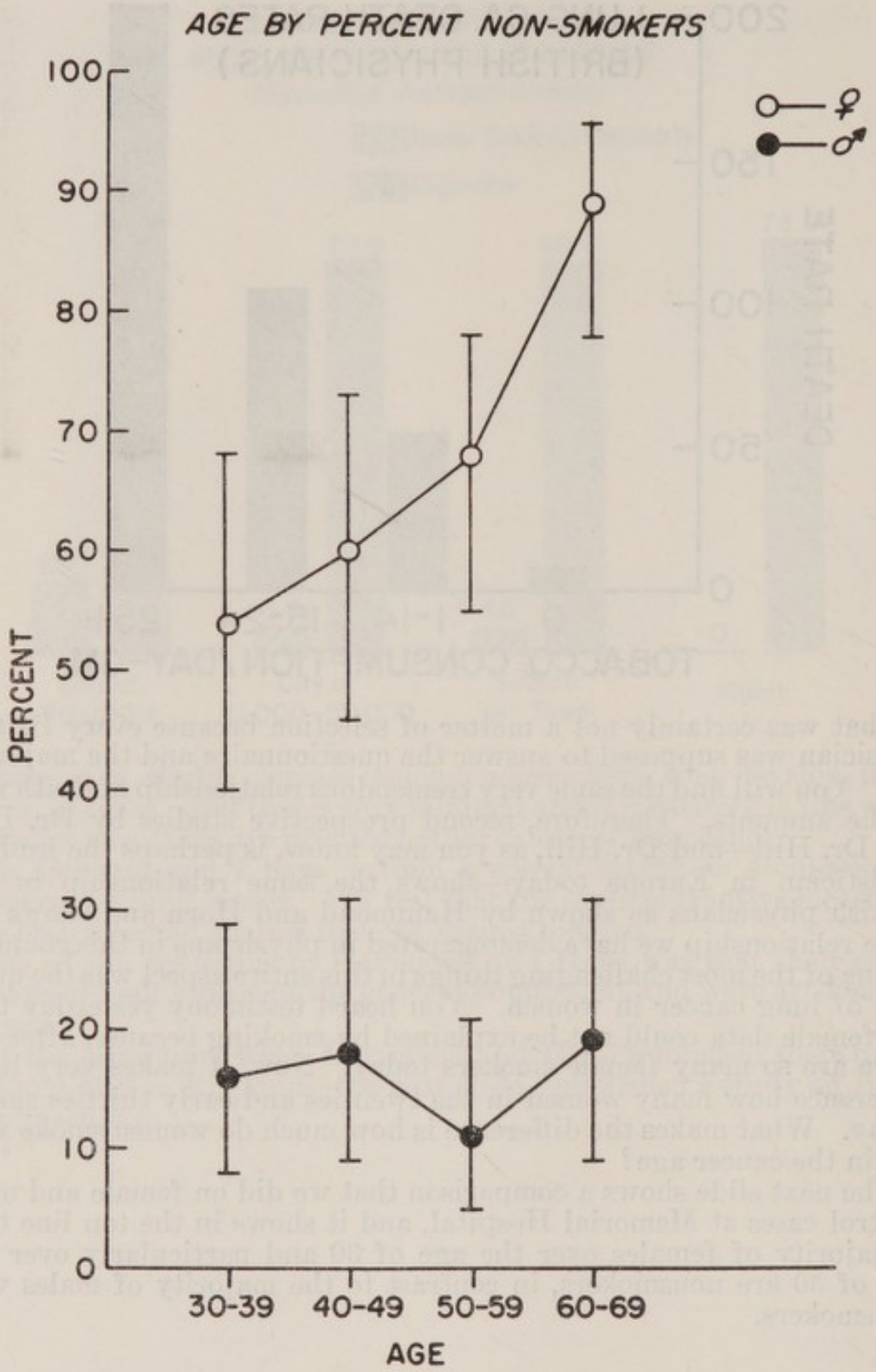
At the same time when Hammond and Horn published their data, Doll and Hill in England—the next slide—published a study on British physicians.



That was certainly not a matter of selection because every British physician was supposed to answer the questionnaire and the majority did. You will find the same very tremendous relationship of death rate to the amounts. Therefore, second prospective studies by Dr. Doll and Dr. Hill—and Dr. Hill, as you may know, is perhaps the leading statistician in Europe today—shows the same relationship in the British physicians as shown by Hammond and Horn and shows the same relationship we have demonstrated in physicians in this country.

One of the most challenging things in this entire aspect was the question of lung cancer in women. You heard testimony yesterday that the female data could not be explained by smoking because, after all, there are so many female smokers today. Now, it makes very little difference how many women in the twenties and early thirties smoke today. What makes the difference is how much do women smoke who are in the cancer age?

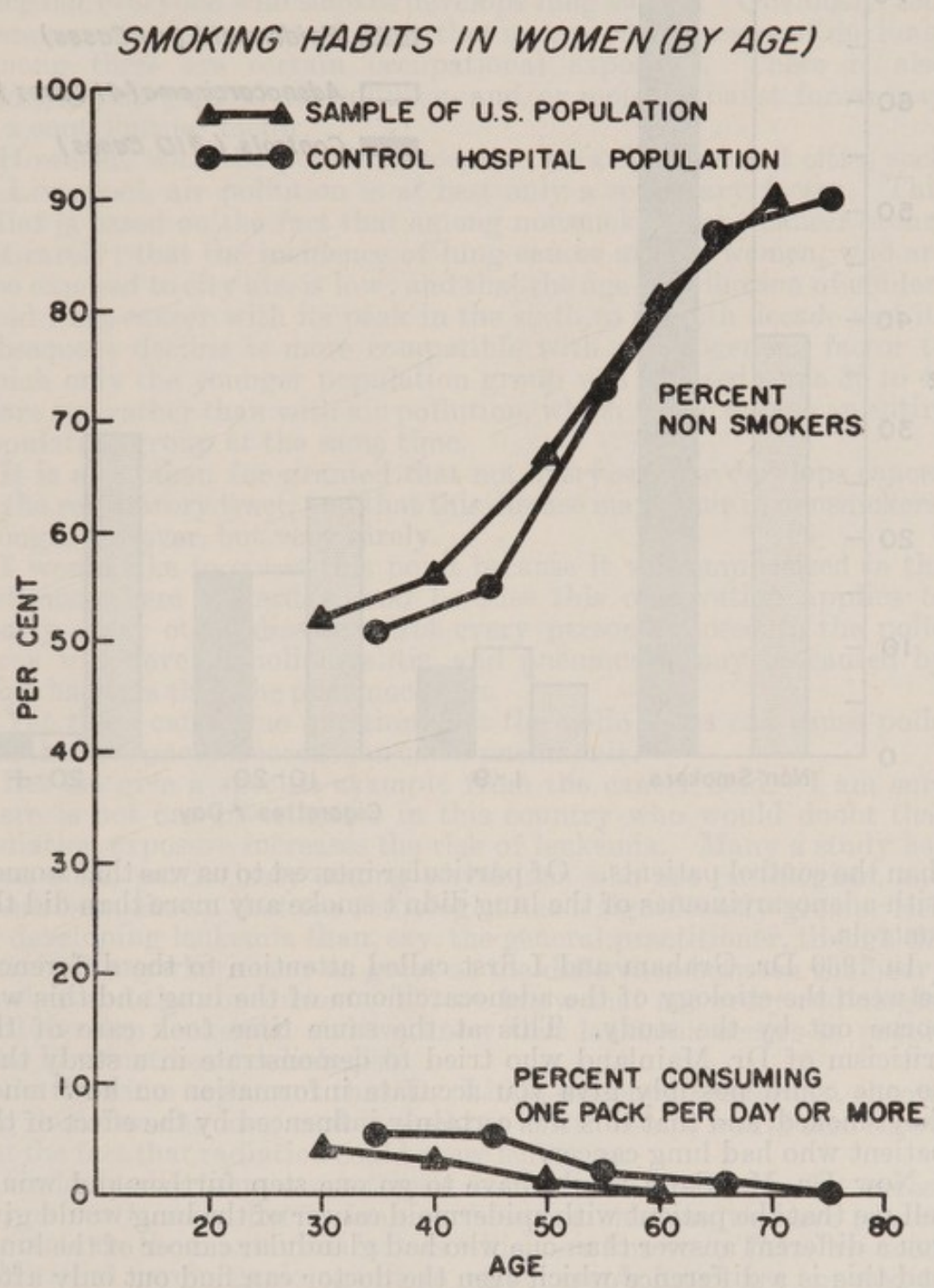
The next slide shows a comparison that we did on female and male control cases at Memorial Hospital, and it shows in the top line that a majority of females over the age of 30 and particularly over the age of 50 are nonsmokers, in contrast to the majority of males who are smokers.



At the same time we found, whereas about 40 percent of the males are rather heavy smokers, only about 2 percent of women in the age group over 45 smoke more than a pack of cigarettes a day.

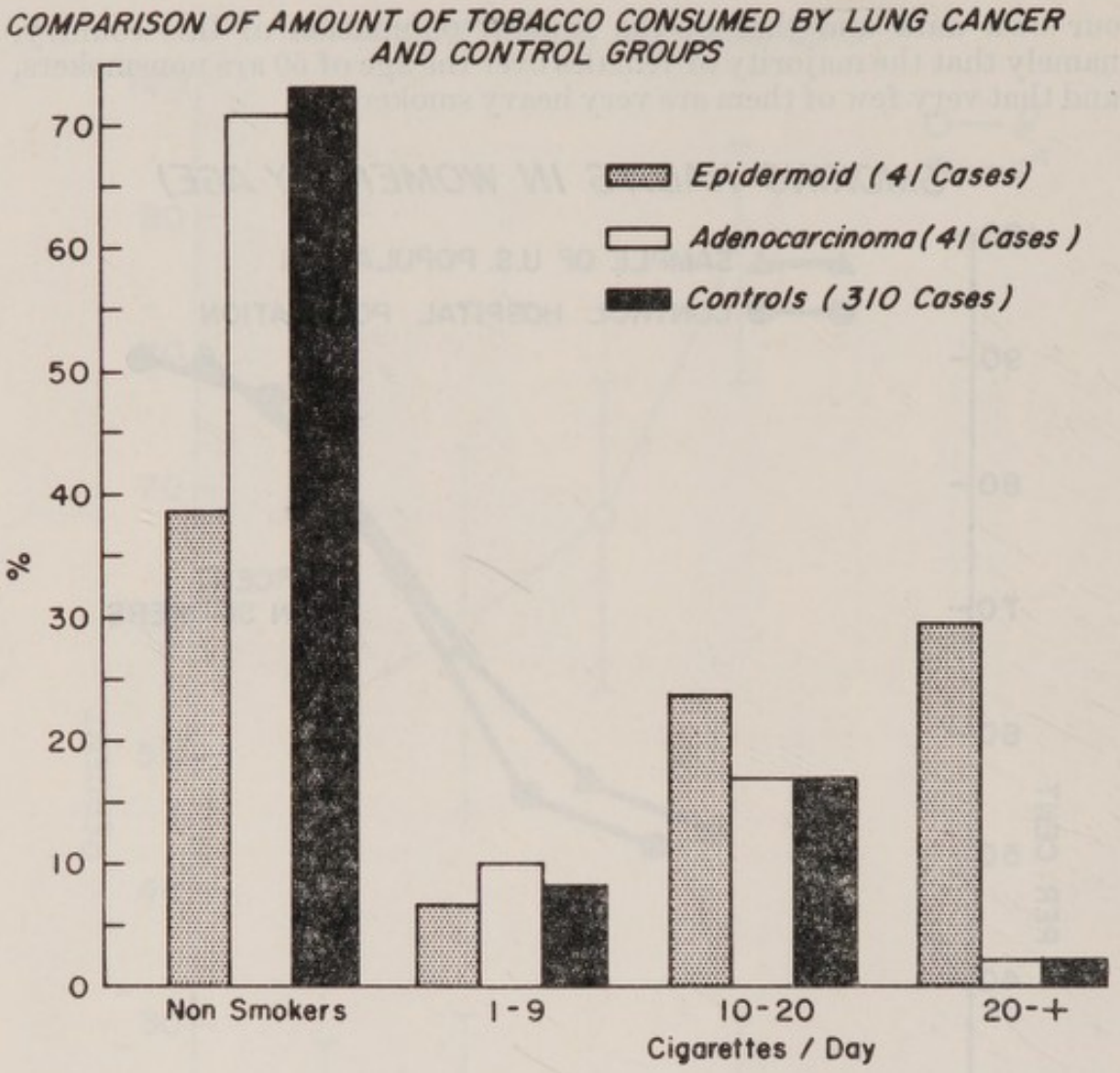
Somebody criticized us and said these are data from hospitals and they do not reflect the general population. We are very fortunate because the United States Census data just came out at the same time—shown in the next slide—and they show a great similarity between

our own data and that of the general population of this country, namely that the majority of females over the age of 50 are nonsmokers, and that very few of them are very heavy smokers.



So we concluded, in line with a detailed study by Shimkin and Haenzel from the National Cancer Institute, that the present sex ratio of lung cancer was entirely comparable with the long-term smoking habits of the two sexes.

What about women with lung cancer. The next slide shows that women with epidermoid cancer of the lung are much heavier smokers



than the control patients. Of particular interest to us was that women with adenocarcinomas of the lung didn't smoke any more than did the controls.

In 1950 Dr. Graham and I first called attention to the differences between the etiology of the adenocarcinoma of the lung and this was borne out by the study. This at the same time took care of the criticism of Dr. Mainland who tried to demonstrate in a study that no one could possibly give you accurate information on how much they smoked, and that this was certainly influenced by the effect of the patient who had lung cancer.

Now Dr. Mainland would have to go one step further and would tell me that the patient with epidermoid cancer of the lung would give you a different answer than one who had glandular cancer of the lung, and this is a difference which even the doctor can find out only after he has studied the lung tissue under the microscope.

So we concluded in a study which I published together with Bross, O'Donnell, and Cornfield that the female heavy smoker reaching the cancer age has chances of developing lung cancer which are roughly the same as that of men. That is the only reason why the sex ratio is as it is today is that the majority of the females in the cancer age are nonsmokers or minimal smokers.

To consider smoking as the major cause of lung cancer does not mean that we have ever said that that is the only cause of this disease. Obviously there is the effect of internal predisposition which must exist since not everyone who smokes develops lung cancer. Obviously, too, there are other exogenous factors that may produce cancer of the lung. Among these are certain occupational exposures. There is also evidence that general air pollution and/or motor exhaust fumes may be a contributing factor.

However, with the possible exception of a few isolated cities such as Liverpool, air pollution is at best only a secondary factor. This belief is based on the fact that among nonsmokers lung cancer occurs but rarely; that the incidence of lung cancer among women, who are also exposed to city air, is low; and that the age distribution of epidermoid lung cancer with its peak in the sixth to seventh decade and its subsequent decline is more compatible with an exogenous factor to which only the younger population group was exposed some 30 to 40 years ago rather than with air pollution, which would expose an entire population group at the same time.

It is also taken for granted that not every smoker develops cancer of the respiratory tract, and that this disease may occur in nonsmokers, though, however, but very rarely.

I would like to stress this point because it was emphasized in the testimony here yesterday, and because this observation applies to nearly every other disease. Not every person exposed to the polio virus will develop poliomyelitis, and pneumonia may be caused by other bacteria than the pneumococcus.

Yet there can be no question that the polio virus can cause polio and that the pneumococcus can cause pneumonia.

Let me give a specific example from the cancer field. I am sure there is not one investigator in this country who would doubt that radiation exposure increases the risk of leukemia. Many a study has been done, particularly among physicians who are radiologists, and it has been shown that the radiologist has a significantly greater risk of developing leukemia than, say, the general practitioner, though the risk isn't anywhere near as great as the difference between the heavy smoker and the nonsmoker. Yet we know that not every radiologist will develop leukemia, and we know that leukemia occurs in people who have not been radiologists.

So the same argument that Dr. Little presented yesterday, throwing doubt on the lung cancer-smoking relationship, would also throw out the fact that radiation could cause leukemia.

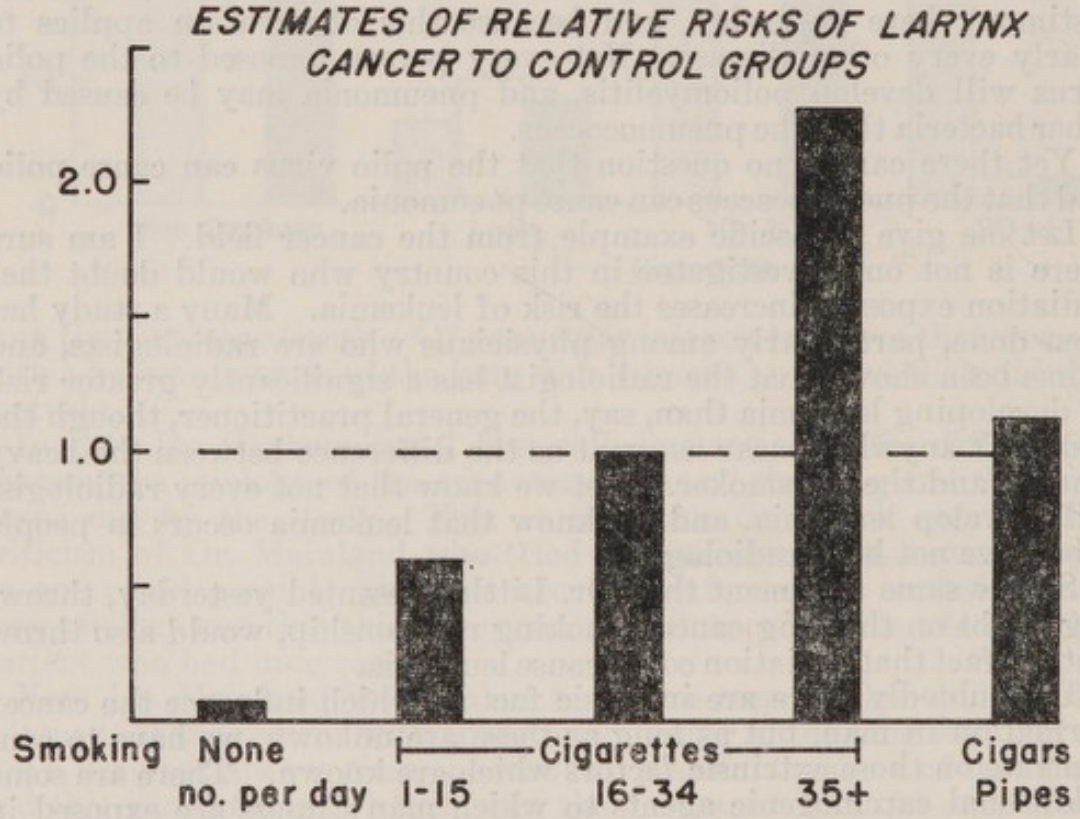
Undoubtedly there are intrinsic factors which influence the cancer formation in man, but as long as these are unkown, we have to concentrate on those extrinsic factors which are known. There are some additional carcinogenic agents to which man's lungs are exposed in regard to which preventive measures should also be developed, but there can be no doubt that smoking represents by far the major extrinsic cause of lung cancer.

While most students of the problem of the etiology of lung cancer admit to an association between smoking and lung cancer, some question whether this association also represents causation. Until proved otherwise, an association at least means a contribution toward, if not a causation of, a disease.

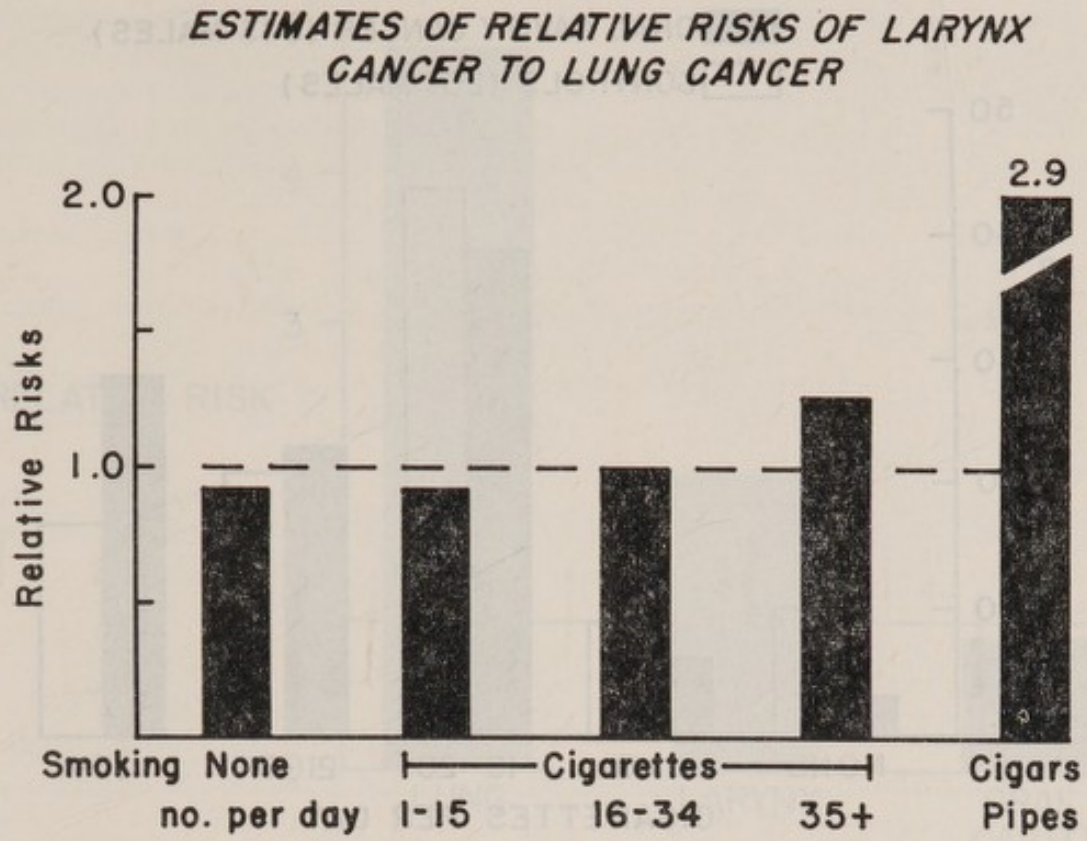
For the patient it matters little whether a factor has contributed to or caused the disease. In either case the elimination of this factor will lead to a reduction of the disease.

In respect to smoking, we have regarded smoking as an important factor in lung cancer and have stated that 80 percent of all lung cancer cases occurring in this country today could be prevented if it were not for smoking. This figure is in complete line with a similar figure presented by Dr. Doll in England, and to those who have stated again in testimony yesterday that lung cancer after all occurs so very rarely in nonsmokers, let me cite you the figures calculated by Cutler and Loveland of the National Cancer Institute that about 5.3 percent of males over the age of 25 who smoke in excess of 20 cigarettes a day will develop lung cancer by the age of 70. And in this line also impressive are the data of Dr. Hammond which show that among the two-pack-a-day smokers, lung cancer was the second most common cause of death.

What about some of the other types of cancer? The next slide shows you the same correlation that we have found with lung cancer to exist with cancer of the larynx, with one major exception. You will notice that the risk becomes now slightly greater for the cigar and for the pipe smoker.

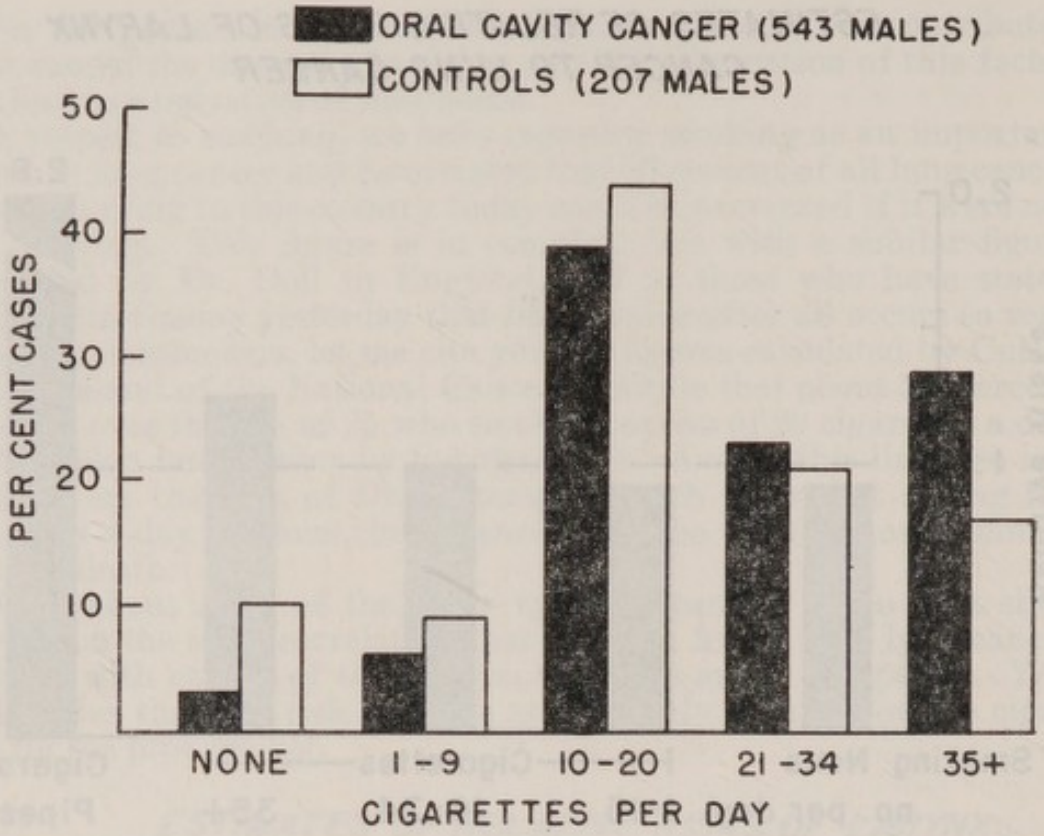


The next slide demonstrates this more clearly. It shows that the risk among cigarette smokers is about the same, but for cigar smokers, the risk of developing cancer of the larynx is greater.

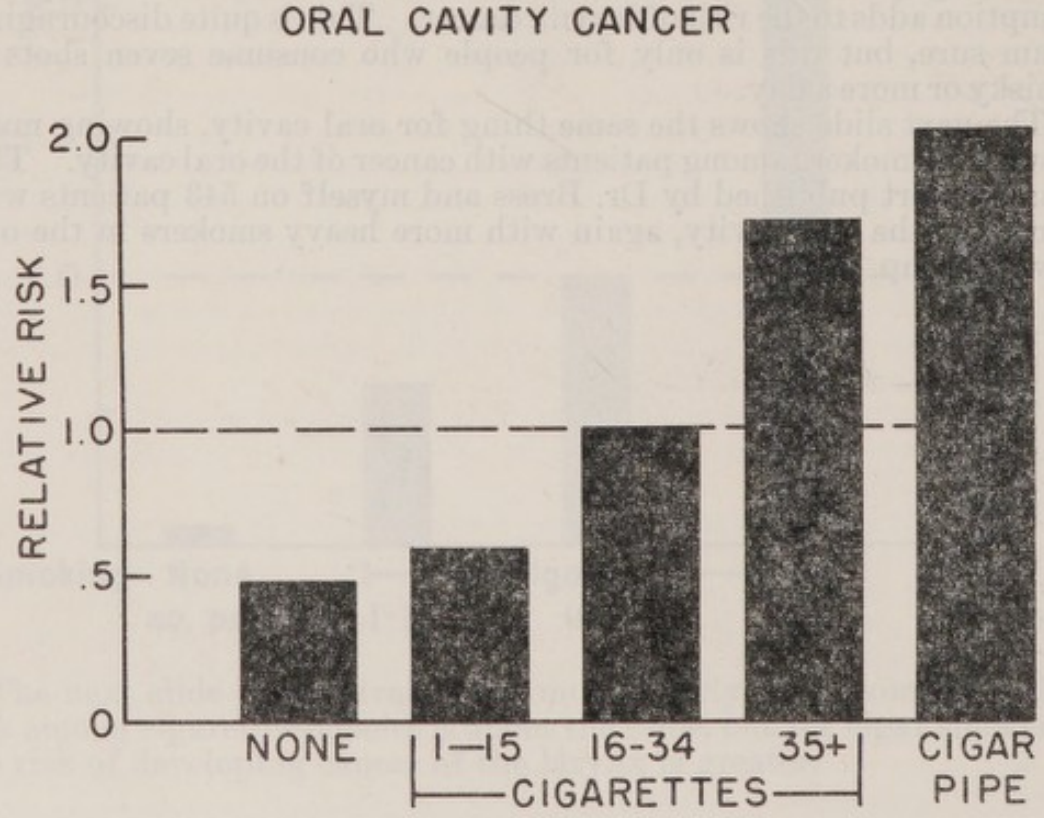


In the larynx cancer group, we found additional etiologic factors about which I don't want to go into detail today because this would discourage you further. We demonstrated that excessive whisky consumption adds to the risk of larynx cancer. This is quite discouraging, I am sure, but this is only for people who consume seven shots of whisky or more a day.

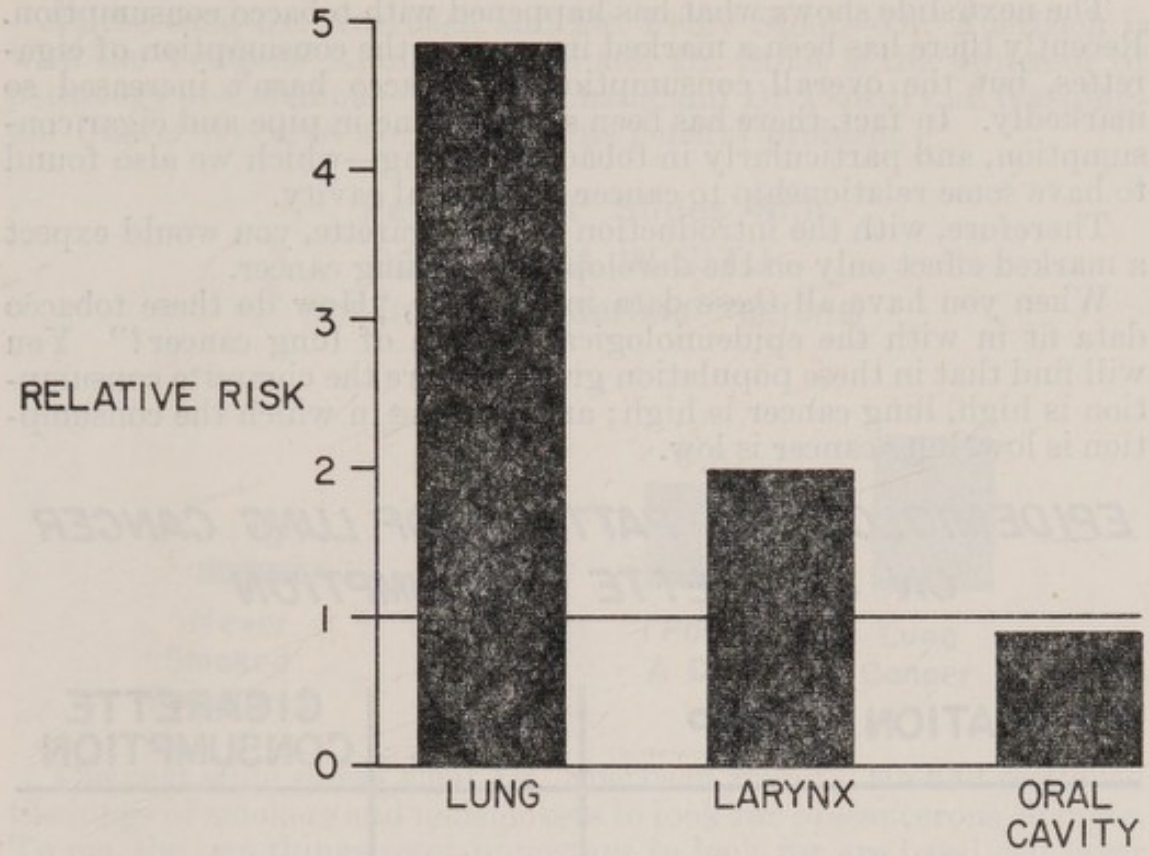
The next slide shows the same thing for oral cavity, showing much fewer nonsmokers among patients with cancer of the oral cavity. This was a report published by Dr. Bross and myself on 543 patients with cancer of the oral cavity, again with more heavy smokers in the oral cavity group.



The next slide shows again the greater risk among cigar and pipe smokers to develop cancer of the oral cavity than among cigarette

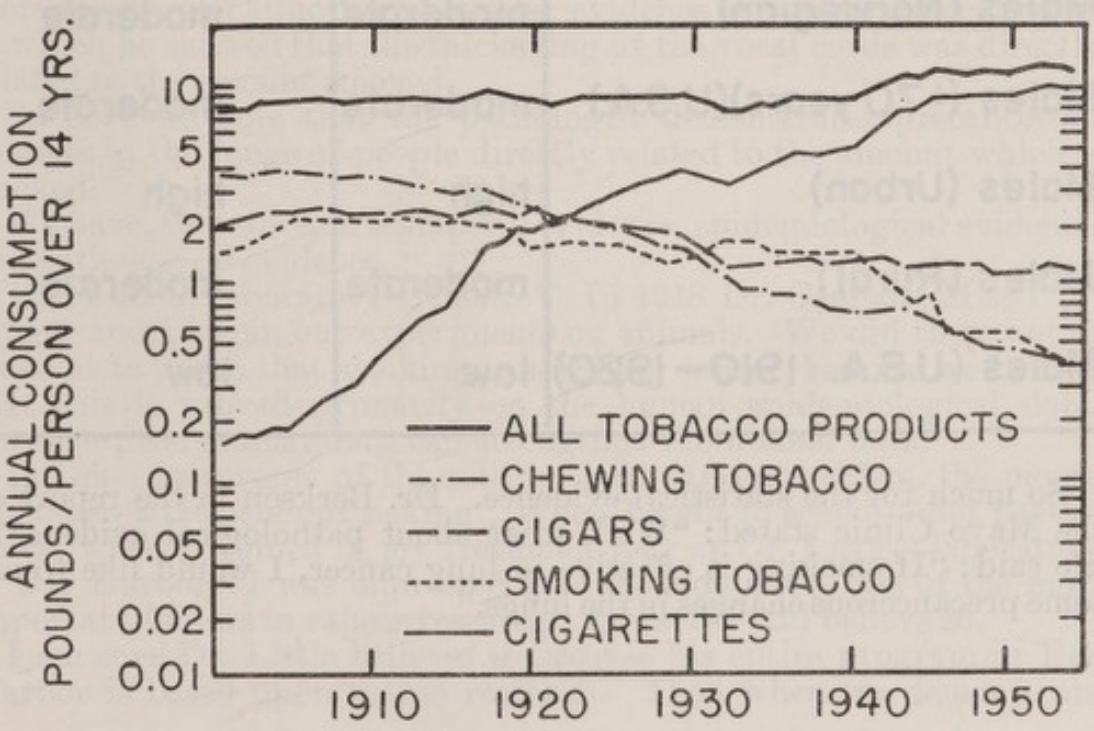


smokers. The next slide summarizes this, showing that the risk for cigarette smokers to develop cancer is greatest for the lung, then the larynx, then oral cavity. With cigar and pipe smoking it would be completely reversed.



This, we believe, is due to the fact—just a physical phenomenon—that the cigarette smoker inhales and gets the stuff in his lungs. Cigar and pipe smokers do not as frequently inhale, and they keep the smoke in their mouths.

I am presenting this, again, because it was pointed out to you yesterday that there was no increase in larynx and oral cavity cancer to compare with that of lung cancer, and it was considered very peculiar if smoking was a factor. But these data do show that the cigar and pipe smoker has a greater risk of developing oral cavity and larynx cancer.



The next slide shows what has happened with tobacco consumption. Recently there has been a marked increase in the consumption of cigarettes, but the overall consumption of tobacco hasn't increased so markedly. In fact, there has been some decline in pipe and cigar consumption, and particularly in tobacco chewing—which we also found to have some relationship to cancer of the oral cavity.

Therefore, with the introduction of the cigarette, you would expect a marked effect only on the development of lung cancer.

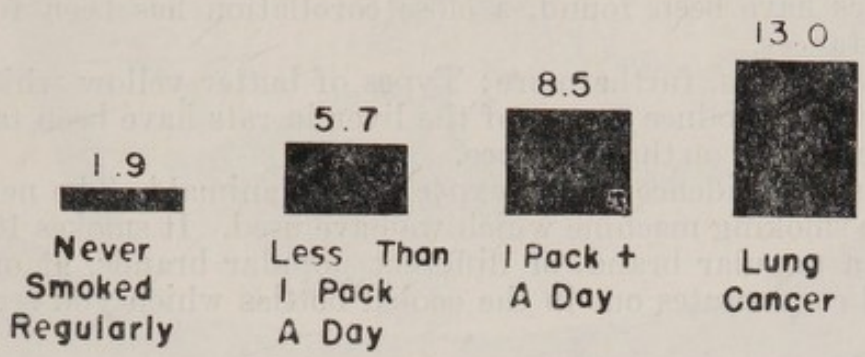
When you have all these data in, you say, "How do these tobacco data fit in with the epidemiological pattern of lung cancer?" You will find that in these population groups where the cigarette consumption is high, lung cancer is high; and in those in which the consumption is low, lung cancer is low.

EPIDEMIOLOGICAL PATTERN OF LUNG CANCER ON CIGARETTE CONSUMPTION

POPULATION GROUP	RATES	CIGARETTE CONSUMPTION
Males (U.S.A.)	high	high
Females (U.S.A.)	low	low
Males (Great Britain)	high	high
Males (Iceland)	low	low
Males (Norwegian)	moderate	moderate
Males (+70 years)(U.S.A.)	moderate	moderate
Males (Urban)	high	high
Males (Rural)	moderate	moderate
Males (U.S.A. 1910–1920)	low	low

So much for the statistical evidence. Dr. Berkson in the report of the Mayo Clinic stated: "Well, what about pathological evidence?" He said: "If smoking is effective in lung cancer, I would like to see some precancerous changes in the lungs."

At the time Dr. Auerbach and Dr. Percy Stout—Dr. Auerbach is with the Veterans' Administration and Dr. Stout is the professor of pathology at Columbia—and Dr. Chang and Dr. Cowdry at Washington University were working on this general problem.



The next slide shows what Dr. Auerbach found. He had sectioned the lungs of smokers and nonsmokers to look for precancerous changes. To me, the two things most important to look for are basal cell layer hyperplasia and carcinoma in situ.

Dr. Auerbach while reading all these thousands of individual slides did not know the smoking history of the patient. In other words, he read them blindly and he found that the precancerous changes were most marked in the heavy smokers and that they were directly related to the amount the patient smoked.

In other words, he—a pathologist—has demonstrated precancerous changes in the lungs of cancer patients which are directly related to the amount they smoked.

Chang and Cowdry found the same thing in St. Louis, and Dr. Ryan from the Mayo Clinic found similar evidence in studying vocal cords in which he showed that the thickening of the vocal cords was directly related to the amount smoked.

So here we note that the pathologist demonstrates precancerous changes in the lungs of people directly related to the amount which is smoked.

We have, therefore, presumptive evidence, epidemiological evidence, and pathological evidence.

What about laboratory evidence? In 1948 Dr. Graham, Miss Croninger and I began our experiments on animals. We did these not so much as to prove that smoking causes lung cancer, because we believe that this is proved primarily on the human epidemiological data. However, the animal data can strengthen the human data.

In this controversy of the tobacco lung-cancer problem, the mouse has taken a tremendous licking. When I went to school—and particularly when I trained in my freshman year at Dr. Little's laboratory in Bar Harbor—I was impressed that the mouse was one of the most important factors in cancer research. In fact, I still believe so.

I am sure Dr. Little believes so because his entire program in Bar Harbor is based upon mouse research. Then when we demonstrate

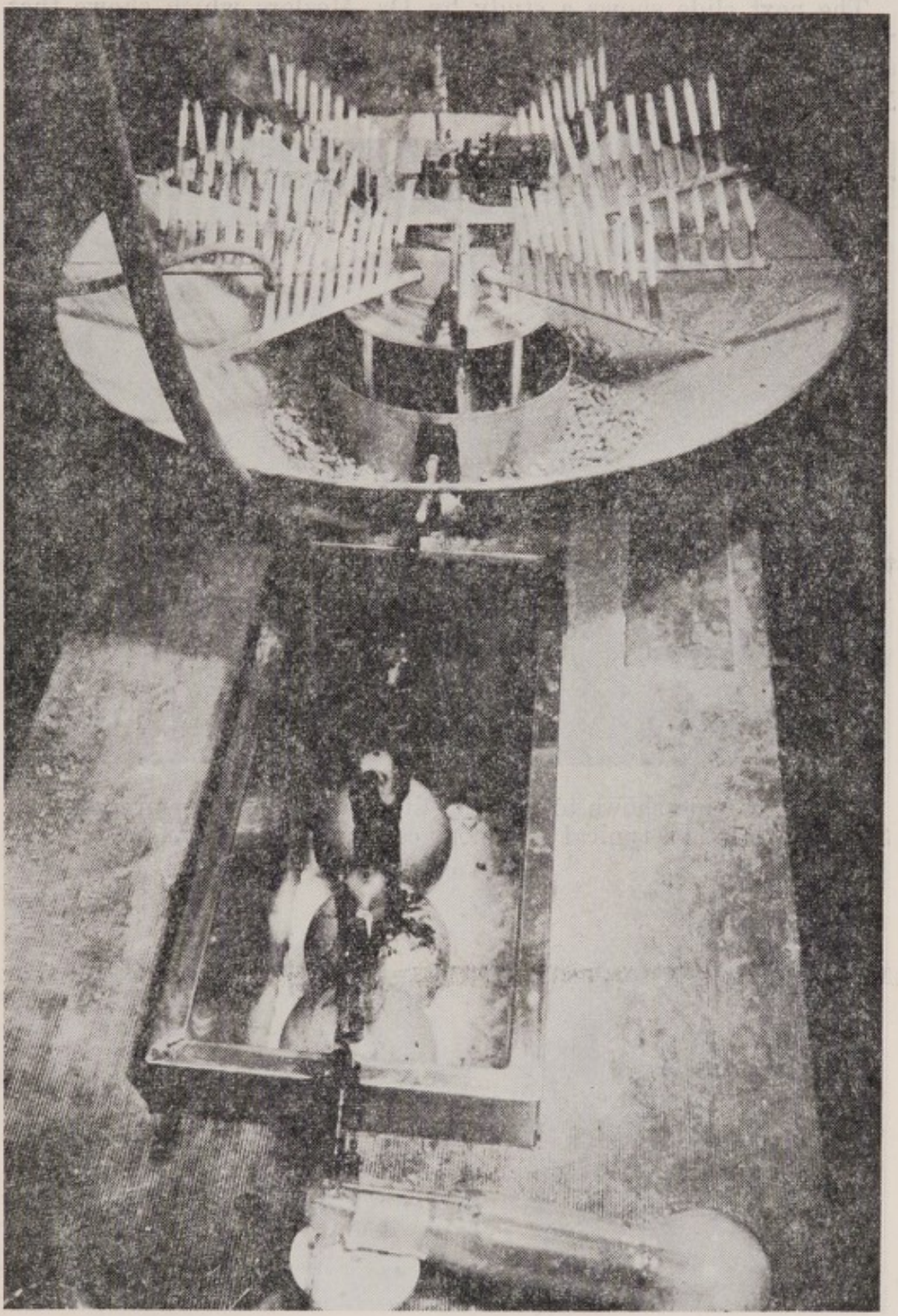
that mice could develop cancer when you applied tobacco tar to them, all of a sudden the mouse lost all the significance that it apparently had ever had.

This is our opinion, as I just stated: The mouse work by itself is not conclusive; but, together with the human data, it is important.

For instance, in the chemotherapy program in the Sloan-Kettering Institute we carry out a major proportion of the screening program on mice. When it works on mice, we try it on men. It doesn't mean necessarily that when you do something on a mouse that it will work on a man, but the chance is much greater; and though some dissimilarities have been found, a close corollation has been found in many instances.

Let me say this, furthermore: Types of butter yellow which have been shown to produce cancer of the liver in rats have been taken off the market purely on that evidence.

What is the evidence on the experimental animal? The next slide shows the smoking machine which we have used. It smokes 100 cigarettes of a popular brand, or different popular brands, at one time and it all condensates out in the cooled bottles which you see at the bottom.



We collect about 50 grams of tobacco per thousand cigarettes. This we dissolve in acetone and apply to the mice.

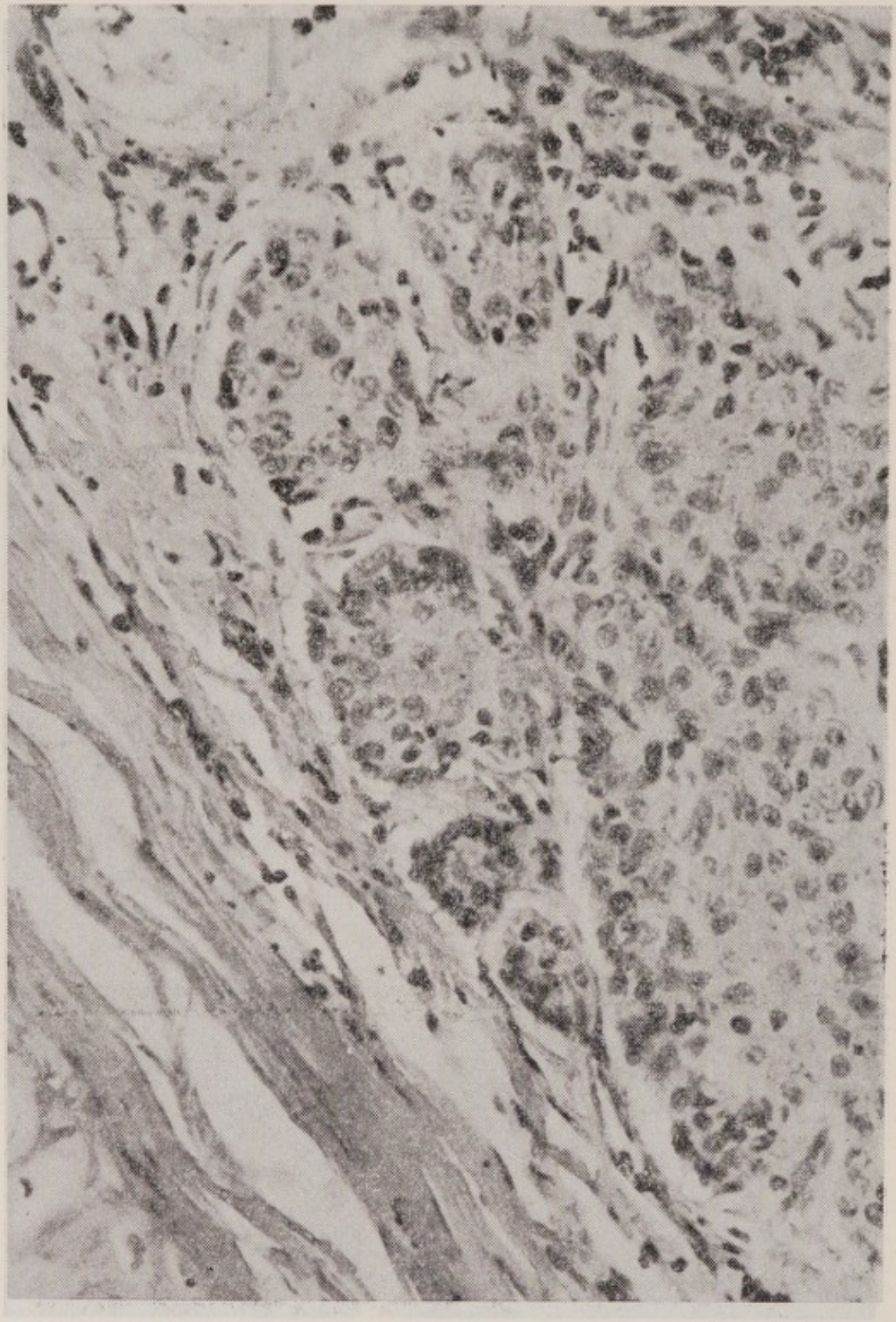
The next slide shows a study by Dr. Mezlors which shows that when you apply this tobacco tar to the mouse, you get very much the same fluorescent absorption that we have previously demonstrated to you in man.



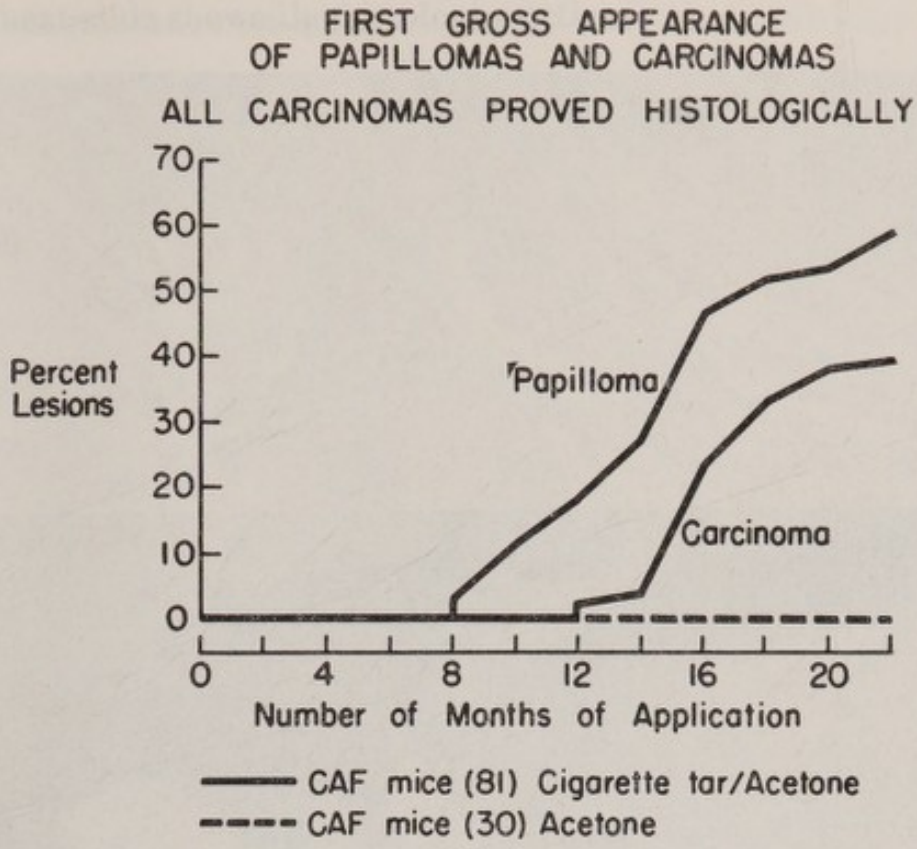
The next slide shows to you the first cancer that we produced in St. Louis. It is a typical squamous cell cancer in a mouse.



The next slide shows you the histologic section of a classical cancer.



The next slide shows to you a summary of these data. They appeared in our first report in 1953, in which 44 percent of the mice developed cancer and 59 percent papillomas.



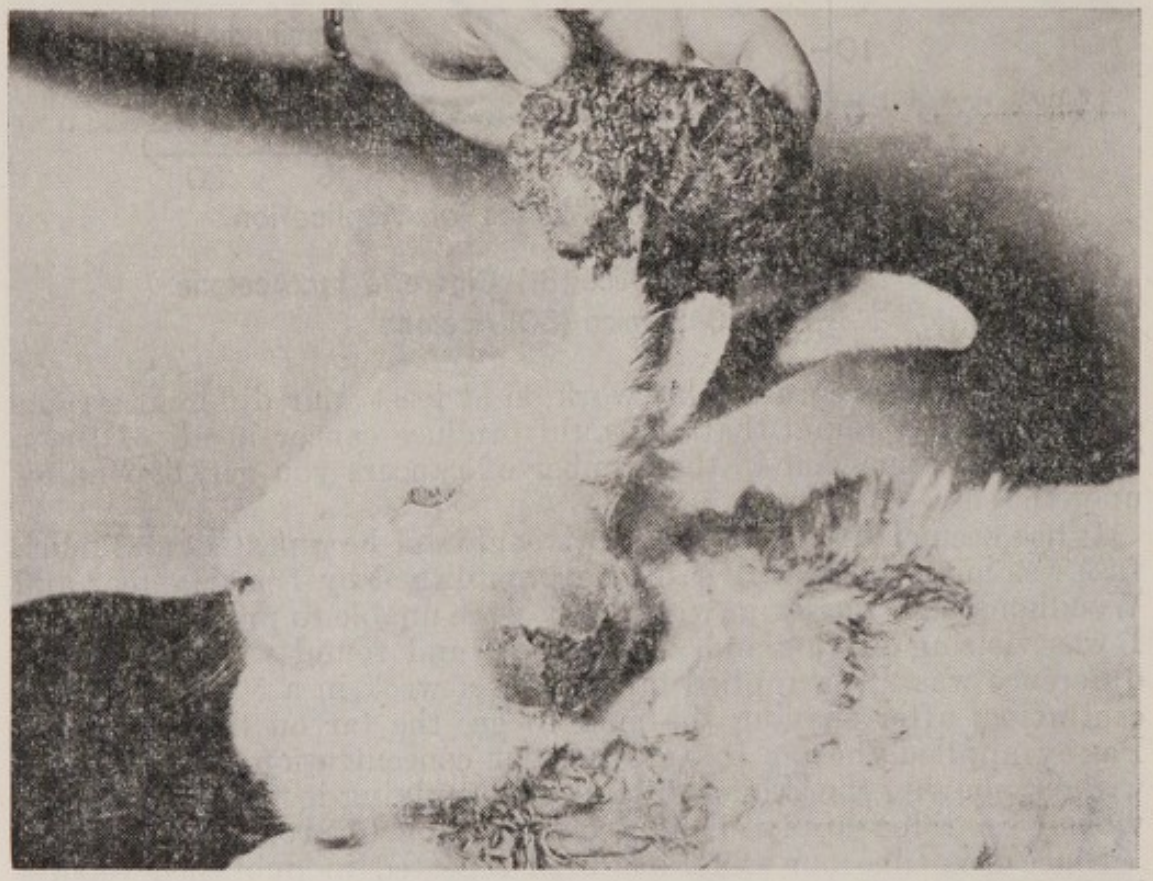
We have since repeated this work on at least four different strains of animals and found that we could produce cancer in all of them. They differ somewhat in the number of cancers you get, depending upon the susceptibility of the strain.

It has been claimed that this work could not be repeated, and much fuss was made about a British study published by Drs. Hamner and Woodhouse and Passey in which they were unable to produce cancers. I was visiting in England at that time and found what the major difference was. We applied tar 3 times a week in a 50 percent concentration, after shaving the mice to get the tar on the skin. Dr. Passey applied the tar in a 20 percent concentration twice a week without shaving the skin at all and just applying it to the hair. Obviously he didn't apply enough concentration to produce tumors.

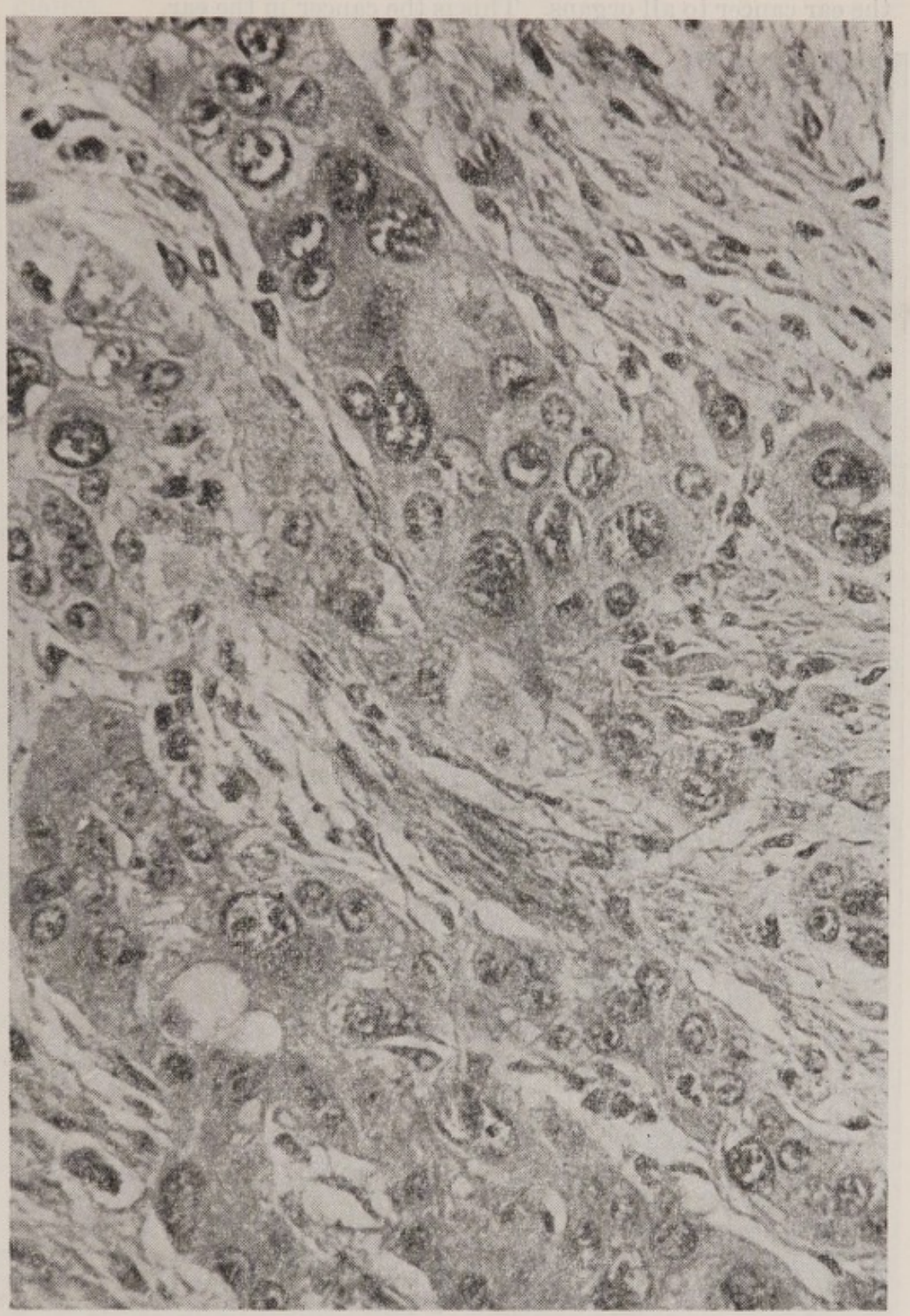
Since this time our work has been repeated in published reports by Dr. Sugiura from the Sloan-Kettering Institute, by Dr. Neukomm in Switzerland, Dr. Blacklock in England has injected tar into the lungs of rats and produced cancer, Dr. Oberling wrote to me a few weeks ago reporting that a study supported by the French tobacco monolopy had been made and he stated he has confirmed our work. These have also been confirmed by studies of Nelson at New York University and Buck and Moore at Roswell memorial.

So there is enough evidence that condensed cigarette tar can produce cancer of the skin in mice.

The next slide shows some new data on a study which we have just completed; a production of carcinoma is rabbits using tobacco tar. This is a rabbit which has been painted for 3 years with condensed cigarette tar.



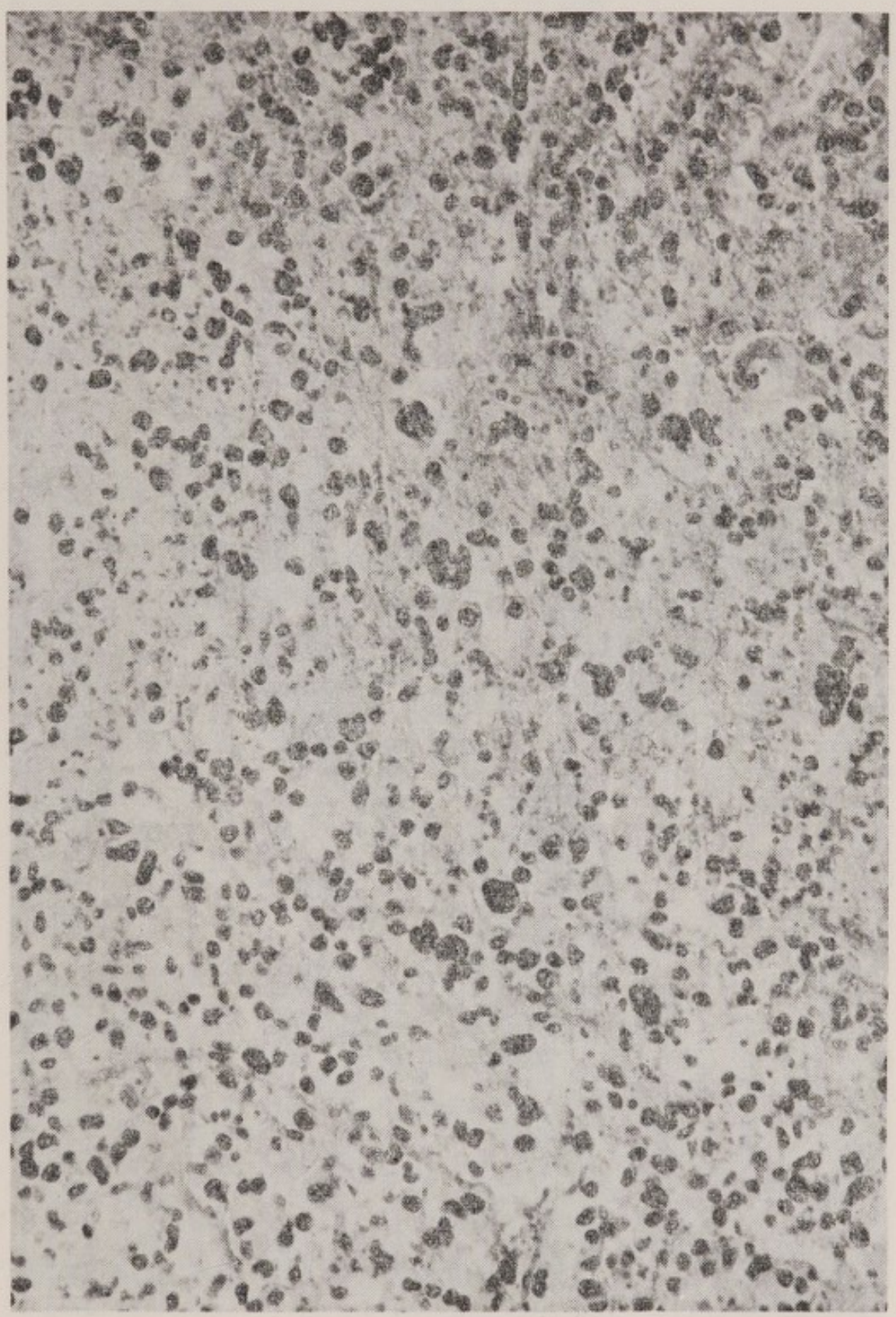
The next slide shows the histologic section.



The next slide is of great interest. It shows wide metastasis from the ear cancer to all organs. This is the cancer in the ear.



The next slide shows the cancer that has metastasized to the cervical glands.

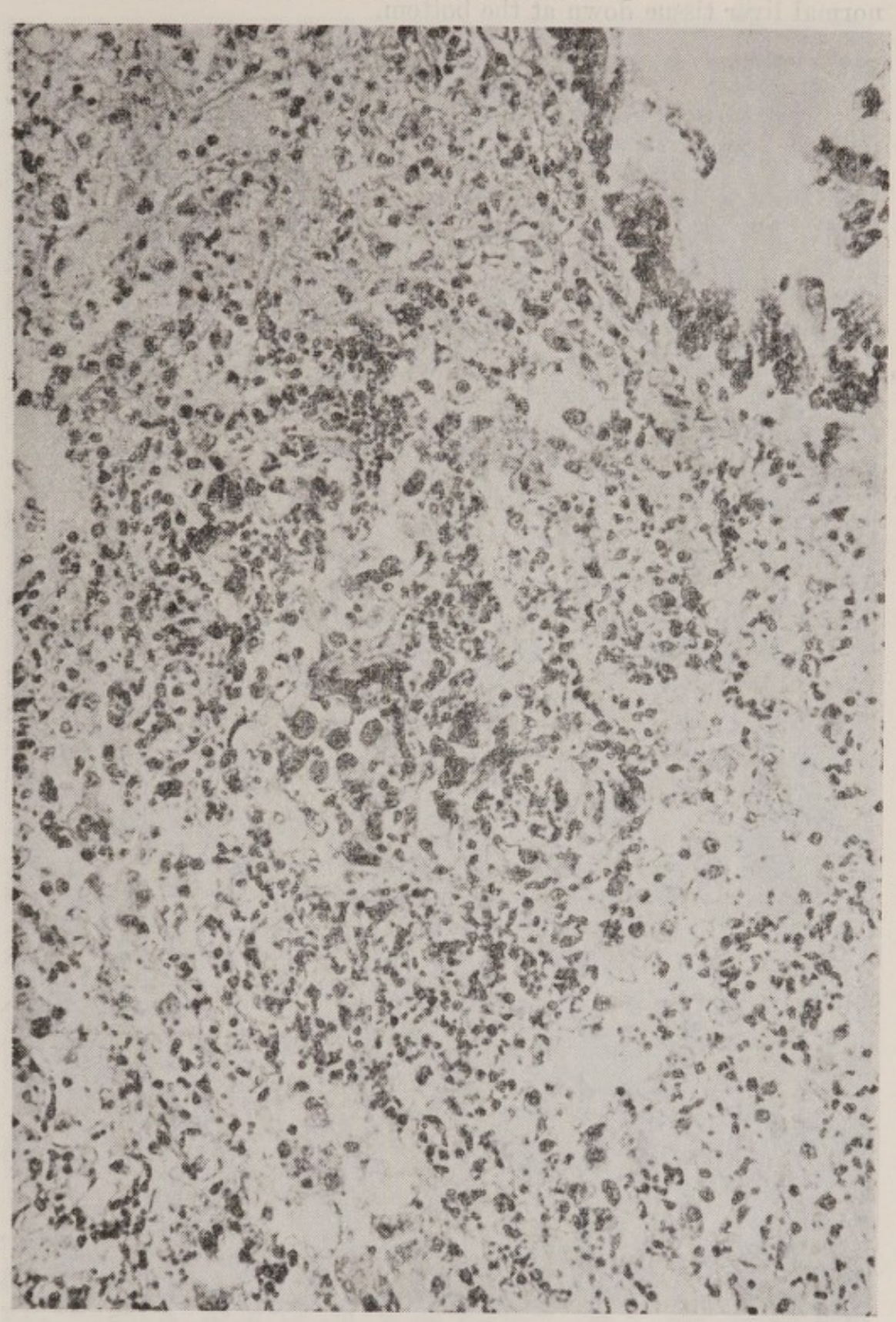


The next slide shows the metastasis of this tobacco-induced cancer from the ear into the thoracic organ.

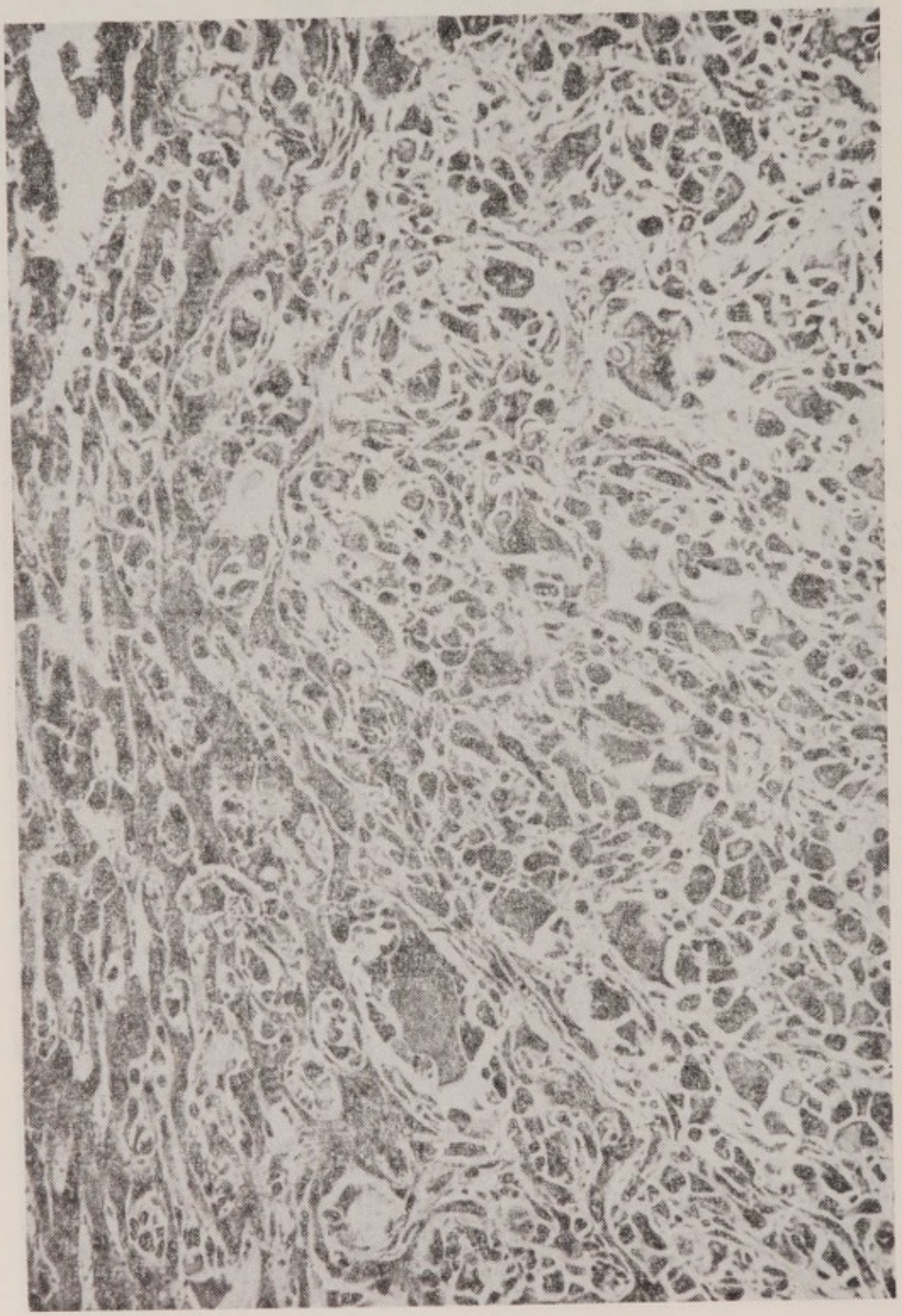


It shows nodules in the lung.

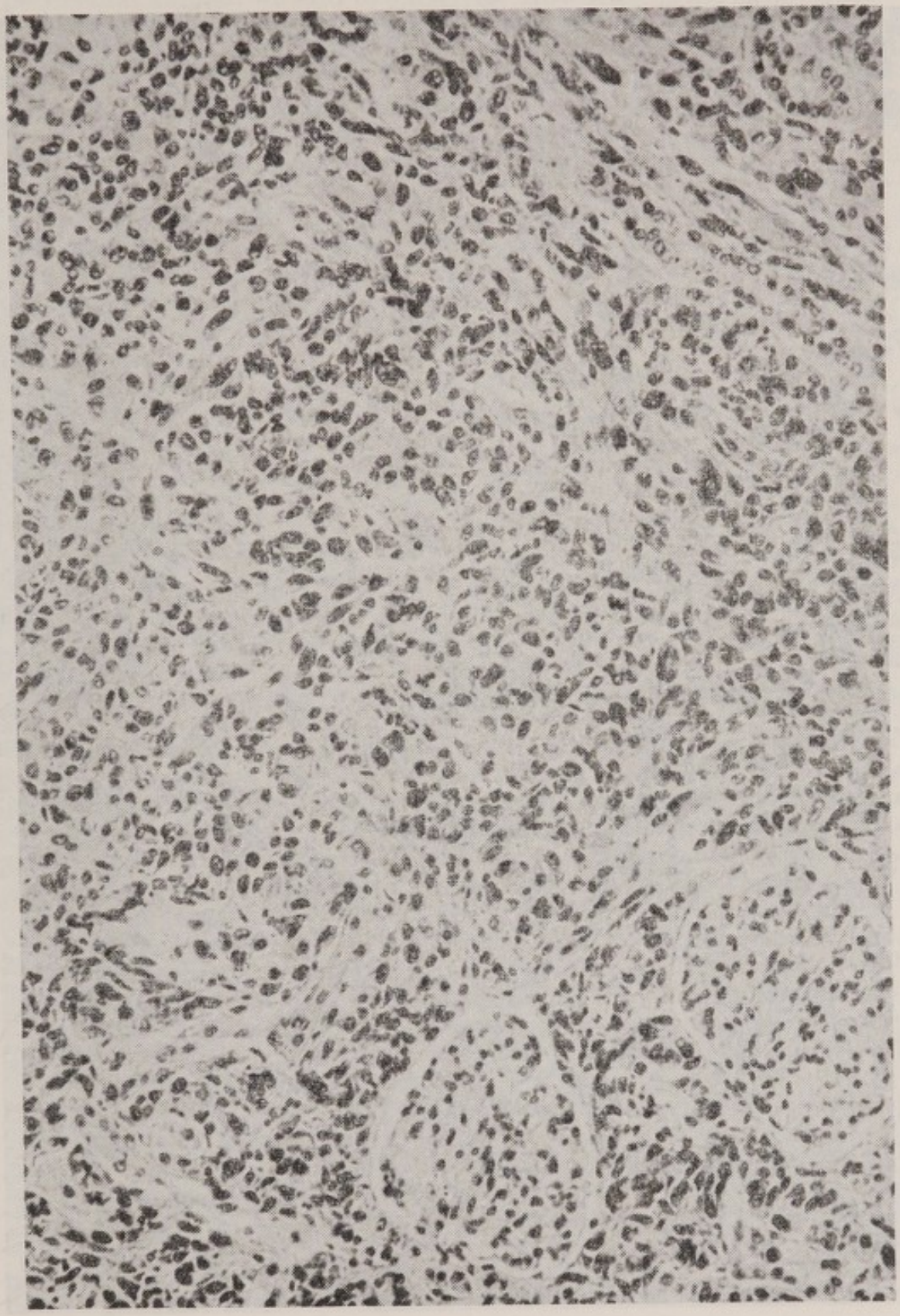
The next slide shows the metastasis into the lung.



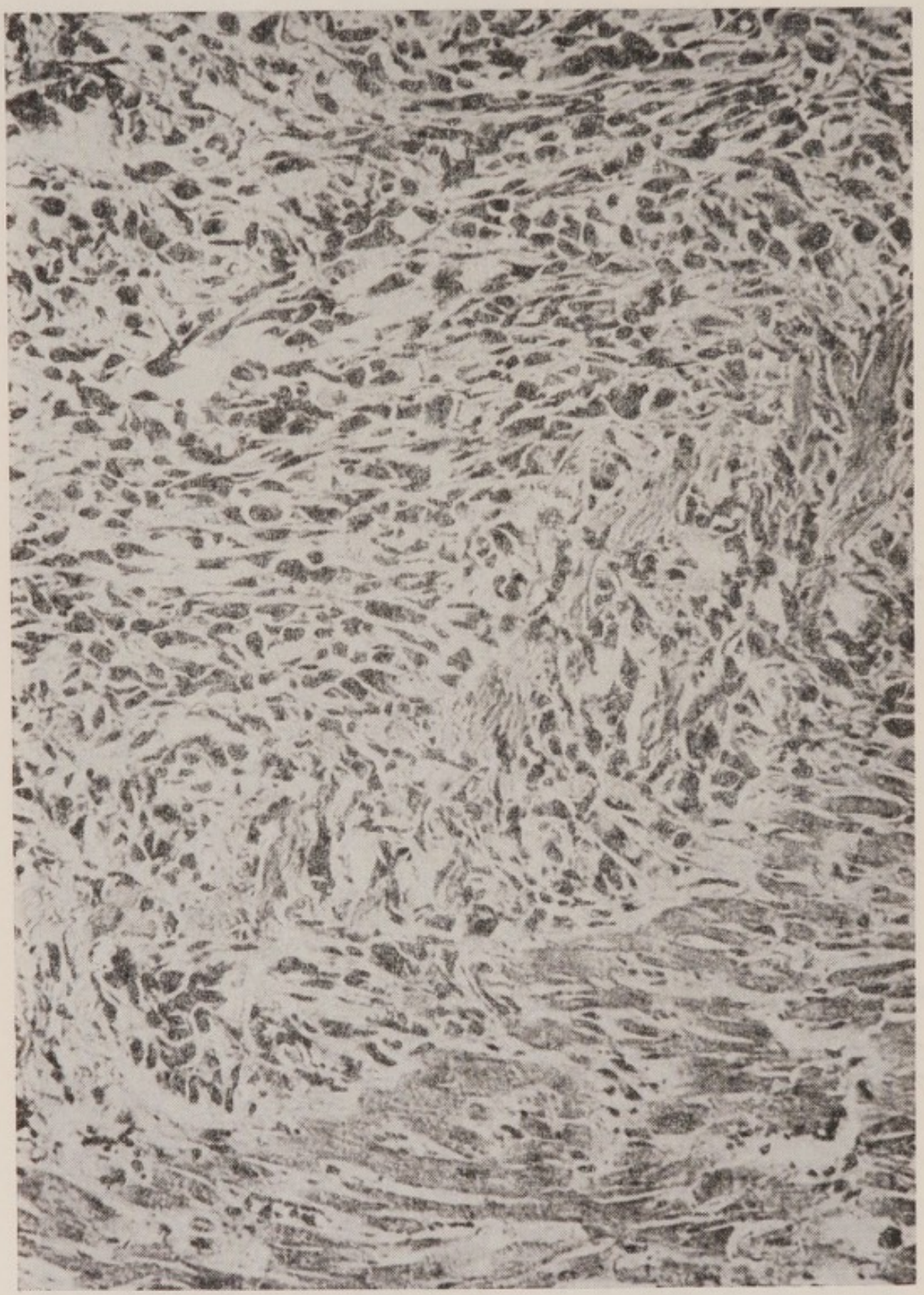
The next slide shows you metastasis into the liver, and you see normal liver tissue down at the bottom.



The next slide shows you metastasis into the kidney.



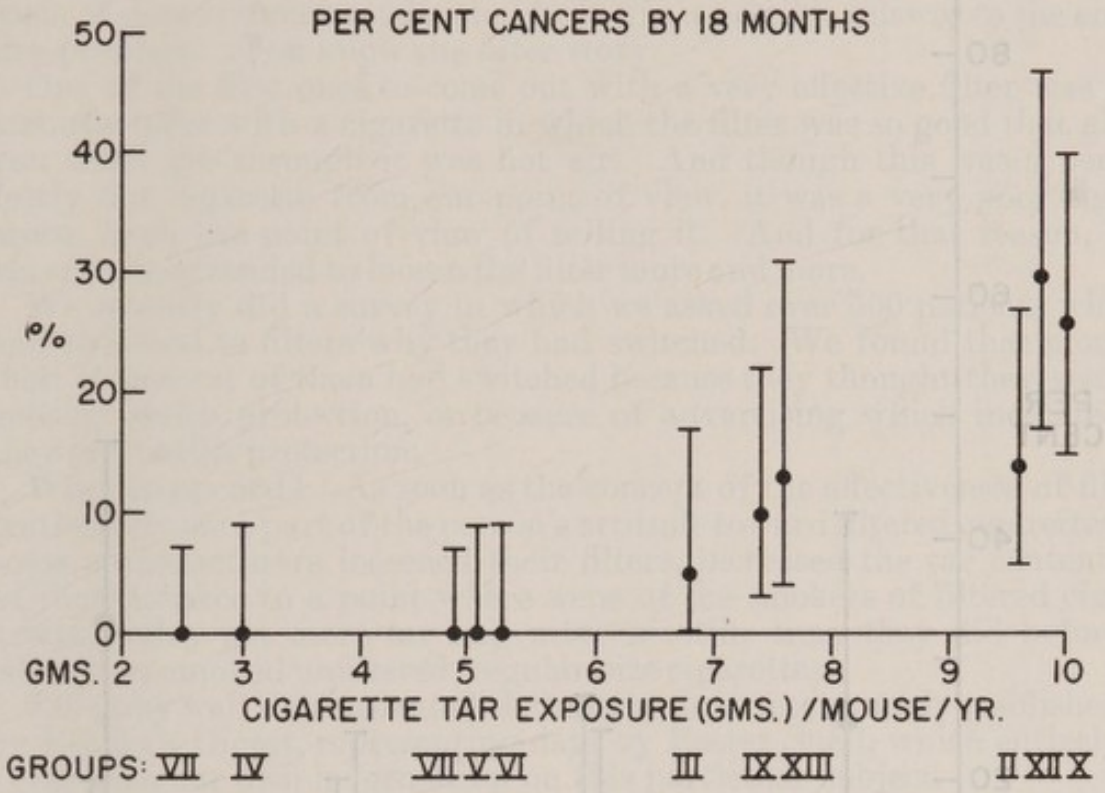
The final slide of this series shows you a metastasis into the heart.



Having demonstrated that tobacco can produce cancer in the experimental animal, we said to ourselves "Now what is the basic significance? What are we going to do with it?"

The first thing we did with this was to try to establish a dose level of the tar.

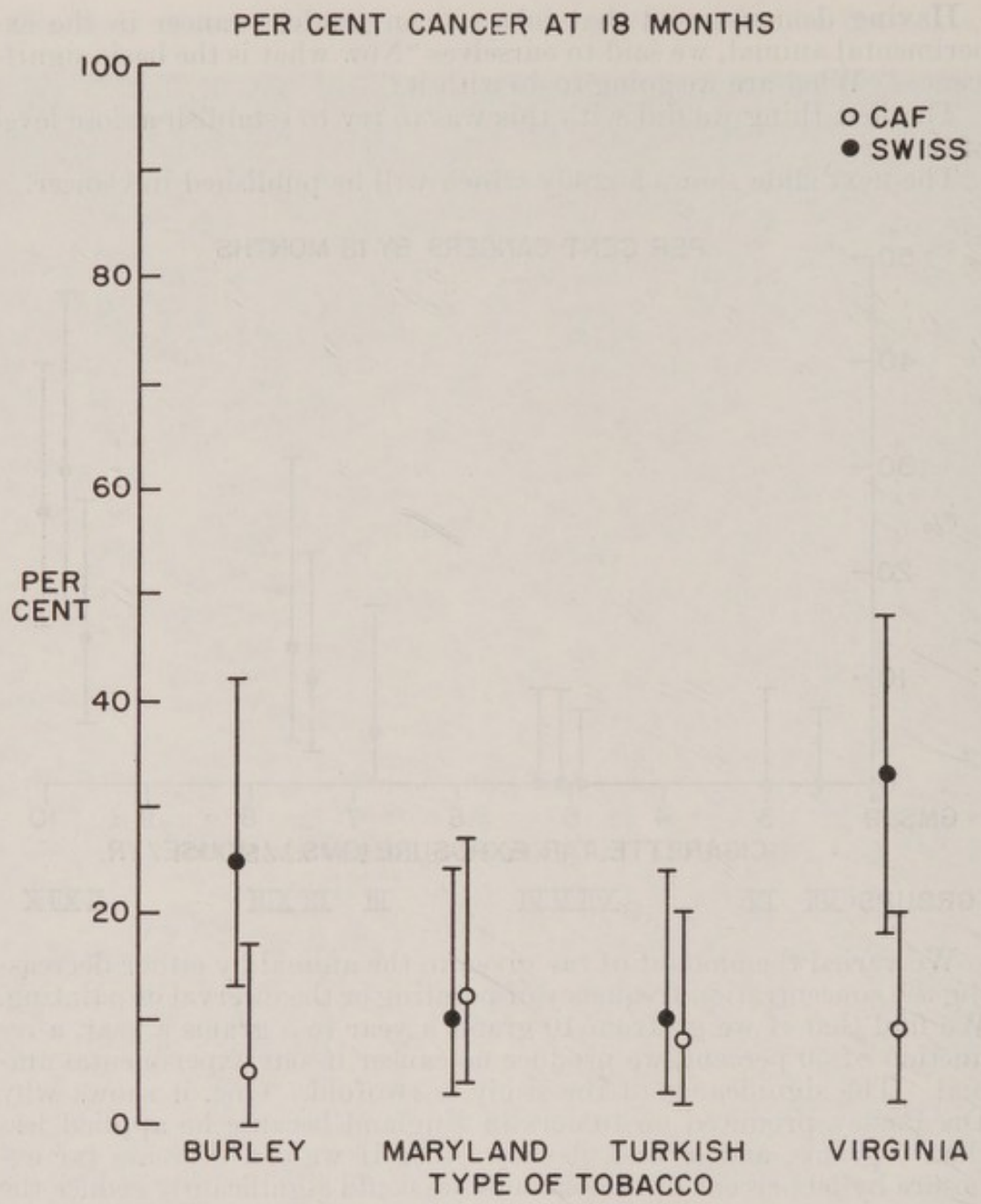
The next slide shows a study which will be published in Cancer.



We varied the amount of tar given to the animal by either decreasing the concentration frequency of painting or the interval of painting. We find that if we go from 10 grams a year to 5 grams a year, a reduction of 50 percent, we produce no cancer in our experimental animal. The significance of the study is twofold. One, it shows why Dr. Passey produced no tumors in England because he applied less than 5 grams, and second, it shows that if we can decrease tar exposure by 50 percent or thereabouts, we could significantly reduce the chances of developing cancer. These dose response studies are quite in line with the human studies which we have previously shown in which we found the same relationship.

The more one smokes, the greater the risk developing cancer.

What is the practical significance of this? It is twofold. One, tobacco types. The next slide shows a study which we published on tobacco types.



We have found that the carcinogenic activity of Burley, Maryland, Turkish and Virginia tobacco on two strains of animals is not significantly different. However, there is a significant difference in the amount of tar that you can get from different tobacco types and from within a given tobacco type. Therefore by blending your tobaccos, and by using different tobacco types, it was clearly shown in studies published by Harlan from the American Tobacco Co., you can change the amount of tar that you get in the smoke.

It seems perfectly feasible to me, and compatible with safe smoking, to choose tobaccos in such a way as to give you a relatively low yield of tar in line with the dose response studies I have just shown.

From that point of view, studies on tobacco types and the tar yield are of great importance. What about filtered cigarettes, which apparently is one of the crucial matters that we want to discuss here? We become very interested in filtered cigarettes because we felt, on the basis of dose response study, that here was a possible answer to the entire problem. You know the filter story.

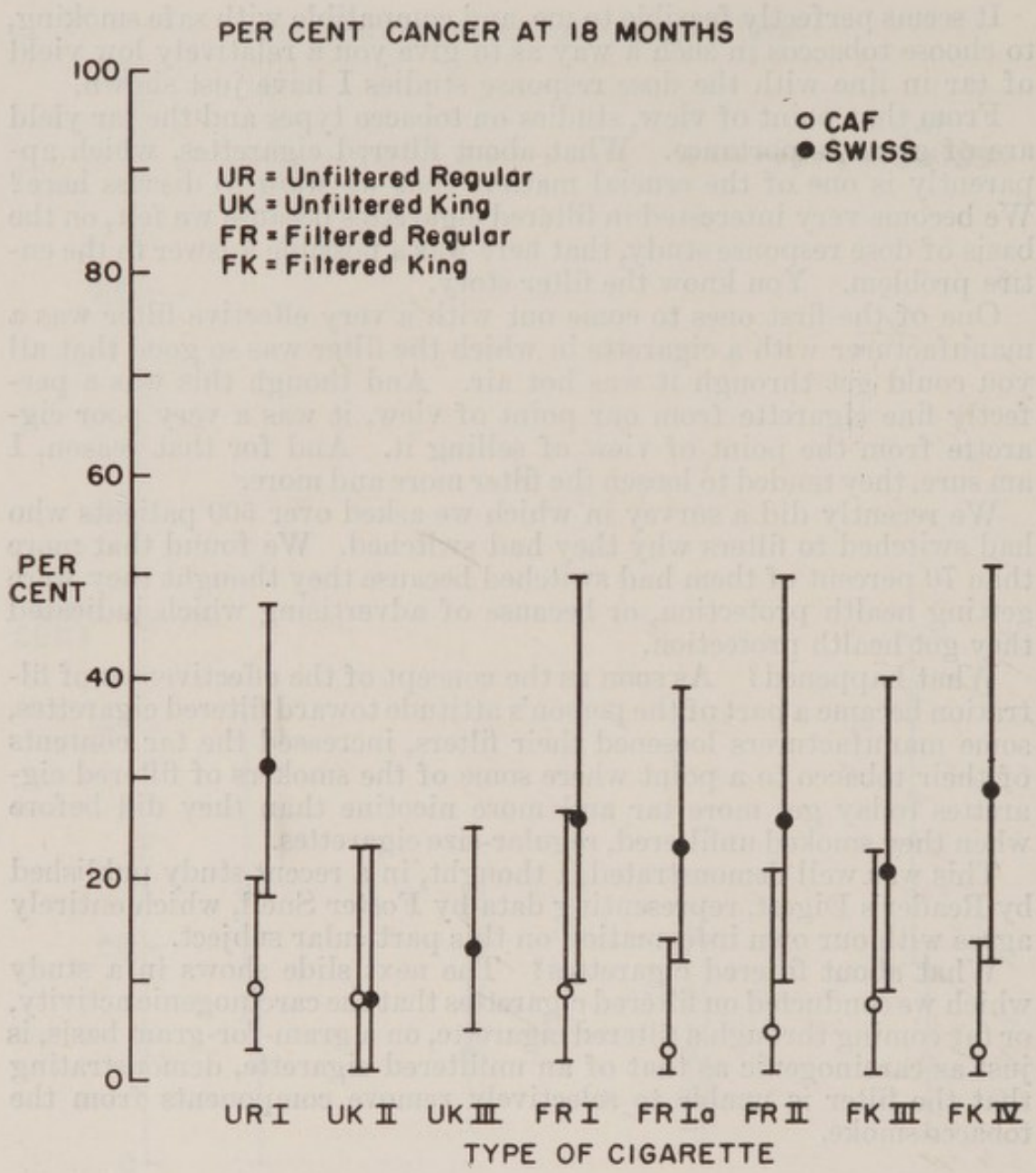
One of the first ones to come out with a very effective filter was a manufacturer with a cigarette in which the filter was so good that all you could get through it was hot air. And though this was a perfectly fine cigarette from our point of view, it was a very poor cigarette from the point of view of selling it. And for that reason, I am sure, they tended to loosen the filter more and more.

We recently did a survey in which we asked over 500 patients who had switched to filters why they had switched. We found that more than 70 percent of them had switched because they thought they were getting health protection, or because of advertising which indicated they got health protection.

What happened? As soon as the concept of the effectivensss of filtration became a part of the person's attitude toward filtered cigarettes, some manufacturers loosened their filters, increased the tar contents of their tobacco to a point where some of the smokers of filtered cigarettes today get more tar and more nicotine than they did before when they smoked unfiltered, regular-size cigarettes.

This was well demonstrated, I thought, in a recent study published by Reader's Digest, representing data by Foster Snell, which entirely agree with our own information on this particular subject.

What about filtered cigarettes? The next slide shows in a study which we conducted on filtered cigarettes that the carcinogenic activity, or tar coming through a filtered cigarette, on a gram-for-gram basis, is just as carcinogenic as that of an unfiltered cigarette, demonstrating that the filter is unable to selectively remove components from the tobacco smoke.

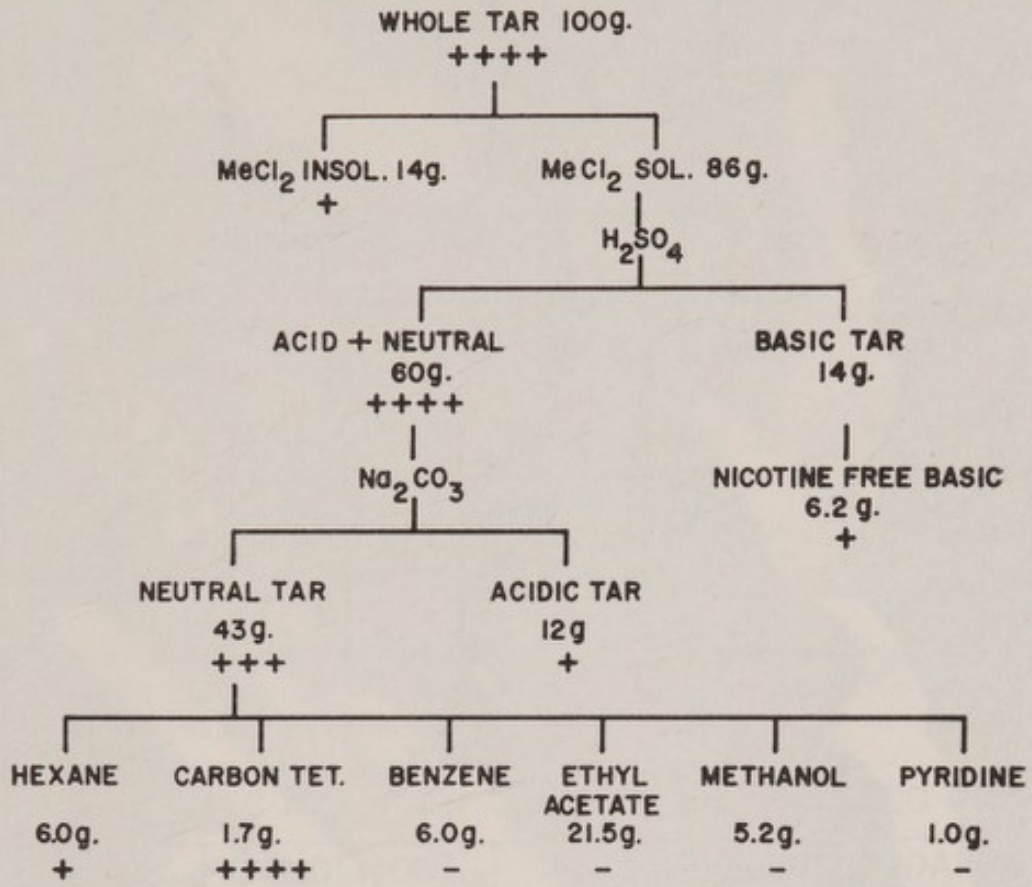


A filter can have a function, however, in line with the dose response studies that we have presented, if it can reduce the tar exposure to the individual. That this can be done seems evident from a second article published in the August issue of the Digest in which they point out that a manufacturer apparently has now succeeded in producing not only a good filter, but by having a certain blend of tobaccos succeeded in exposing the smoker to significantly less tar and nicotine than he received when he was smoking the unfiltered cigarette.

It is a thing which can be done, and I will have specific recommendations on this particular point.

We have discussed animal experiments as they apply to cancer production. What about the chemical experiments that tie in with the animal experiments? The next slide—

CIGARETTE TAR FRACTIONATION - RELATIVE BIOLOGICAL ACTIVITY



And I do not really want to go into detail on this because it is strictly chemical. But in this work which was published by Dr. Wright and myself in the March-April issue of Cancer, we tried to find out which component of tobacco contains the carcinogenic elements.

First of all, it is impressive that the components that are carcinogenic are mostly present in about 1.7 percent of the total tar. In other words, the major proportion of the tobacco tar is not carcinogenic. They are present only in a small group of substances. These we believe—as I will point out to you in a minute—are higher aromatic polycyclics.

We have therefore demonstrated the group of substances in which a major carcinogen in tobacco tar is present.

The next slide shows you the application of this carcinogenic fraction by Mezlors which I previously showed to you and you see again the very marked fluorescence when you apply this to mice.



The next slide shows you cancers, multiple cancers produced with this relatively small fraction of tobacco which proves to be highly carcinogenic to the experimental animal.



How are these carcinogens formed? The next slide shows to you that the formation of carcinogenic tobacco tar is directly related to the temperature at which tobacco burns.

CARCINOGENIC ACTIVITY OF DIFFERENT TOBACCO TARS

TYPE OF TOBACCO	CONDITION	AV. TEMP. OF COMBUSTION ZONE (°C)	STANDARD ACT. RELATIVE TO CIGARETTE TAR
CIGARETTE	STANDARD	884	++++
CIGARETTE	CIGAR LEAF WRAPPED	950	++++
CIGAR	STANDARD	914	++++
CIGARETTE	BURNED IN BRIAR PIPE	905	+++++
CIGARETTE	FILTERED	884	++++
CIGARETTE	HIGH VOLUME PUFF	875	++++
PIPE	STANDARD	767	++
CIGARETTE	HEXANE EXTRACT	—	±

The average cigarette burns at about 880° C. If you raise the temperature higher you produce more carcinogens. If you burn it at pipe tobacco temperature, the standard pipe tobacco burns at 767° C., you get fewer carcinogens. And if you have cigarette extracts which are not burned, you get practically no carcinogens at all.

So the formation of the carcinogens is a direct indication of the temperature condition.

We have since demonstrated that if you reduce the temperature level from 880° to 800°, you get a marked reduction in the formation of the carcinogens, and if you reduce it to 700°, you get very few carcinogens at all. So if we could modify pyrolysis in such a way as to reduce the burning temperature of tobacco, we could reduce the formation of these carcinogens.

The next slide shows a number of organic substances of which we have identified at least traces in tobacco and in tobacco extract which was pyrolyzed.

GROUP B (FRACTION 56-57)

1,2 BENZPYRENE

3,4 BENZPYRENE

1, 12 BENZPYRELENE

1,2,5,6 DIBENZPYRENE

3,4,8,9 DIBENZPYRENE

3,4,9,10 DIBENZPYRENE

1,2,7,8 DIBENZNAPHTHACENE

2,3 NAPHTHO-3,4 PYRENE

CORONENE

1,2,5,6 DIBENZANTHRACENE

UNKNOWN POLYCYCLICS

In other words, as we have reported in Chicago in April, we have demonstrated with Dr. George Wright and Dr. Jergen Lam who worked with us the way in which these tobacco carcinogens are formed. If you take the waxes of tobacco and pyrolyze them at 880° C., which is the temperature at which tobacco burns, you form these substances in great quantity, and, in fact, the pyrolytic material obtained from pyrolyzing tobacco wax at 880° was one of the most carcinogenic materials that I have ever had the chance to test in my laboratory.

So we know the temperature at which they are formed and we know at least one of the precursors that upon combustion leads to these higher aromatic polycyclics.

The last slide shows to you this very highly active pyrolytic material of tobacco wax and you see these animals are just littered with carcinomas.



I have presented to you presumptive evidence, epidemiological evidence, pathologic evidence, animal evidence, and chemical evidence. What about the evaluation of this work?

Rather than citing to you the opinions of individuals, I thought I would just give you the opinions of some organizations and groups that have deliberated on this issue.

As early as 1952 in a meeting at Louvain sponsored by the World Health Organization, they concluded that smoking was a major factor in the development of lung cancer. This has since been confirmed by the Public Health Service in Sweden and the Netherlands, by the American Cancer Society, and by an American study group which concluded that—

the sum total of scientific evidence establishes beyond reasonable doubt that cigarette smoking is a causative factor in the rapidly increasing incidence in the human epidermoid carcinoma of the lung.

A recent statement by Surgeon General Burney goes toward the same point of view. Similar positions have been taken in editorials of such well established medical journals as the New England Journal of Medicine and the Annals of Internal Medicine in the United States, and the British Medical Journal and the Lancet in Great Britain.

Perhaps one of the most thorough statements was published by the Medical Research Council which was incorporated in a report by the British Ministry of Health. They concluded as follows:

1. A very great increase has occurred during the past 25 years in the death rate from lung cancer in Great Britain and in other countries.

2. A relatively small number of the total cases can be attributed to specific industrial hazards.

3. A proportion of gases, the exact extent of which cannot yet be defined, may be due to atmospheric pollution.

4. Evidence from many investigations in different countries indicates that a major part of the increase is associated with tobacco smoking, particularly in the

form of cigarettes. In the opinion of the council, the most reasonable interpretation of this evidence is that the relationship is one of direct cause and effect.

5. The identification of several carcinogenic substances in tobacco smoke provides a rational basis for such a casual relationship.

If I were asked what are the three most important reasons why I think that smoking is a cause of lung cancer, I would say they are, first the enormity of the statistical relationship. As you know, your life insurance data, if you belong to a group which has a 25 percent higher risk than the normal, you pay a higher premium. If it is 100 percent greater than normal, you are going to have a hard time finding someone to insure you.

But here data both from England and the United States have shown an increase in up to 7,000 percent greater.

Second, the relation of the response to the amount smoked in the risk of lung cancer, and third, this is entirely in line with presumptive evidence.

The practical aspects are as follows: (1) Moderation in smoking habits; (2) effective filtration, (3) the removal of precursors which we indicated to you; and (4) modification of pyrolysis.

The specific recommendations we would like to make are as follows: (1) In view of the dose response data established both for men and the laboratory animals, specific tar levels in the smoke of a given cigarette should be prescribed. These tar levels should be at least 40 percent lower than that of the average unfiltered regular-size cigarette. Standard procedure should be prescribed for determining the tar and nicotine values.

(2) The amount of tar yield can be controlled with present knowledge of mechanical filtration and tar yield of different type of tobaccos. The sooner this can be achieved, an achievement which should be entirely compatible with smoking pleasure, the sooner we will lower the health hazard of our smoking population.

(3) Work has to be extended in the field of pyrolysis to determine ways and means of lowering the combustion temperature of smoking products and to determine whether materials can be safely added to tobacco which may interfere with the chemical reaction transforming organic substances into higher aromatic polycyclics. The field of anti-catalysts offers some promise in this direction.

(4) Precursors in raw tobacco which, upon pyrolysis, transform into higher aromatic polycyclics have to be studied further with the hope that by their reduction or removal fewer of the higher aromatic polycyclics will be formed.

(5) In addition to the available dose response studies in respect to cancer reduction, further work has to be done on the tolerance level of nicotine.

(6) The practical aspects outlined above may not individually lead to a tobacco tar that will have less carcinogenic activity, but a combination of these factors will certainly lead to a safer smoking product.

In summary, we believe that because of the presumptive evidence, epidemiological evidence, pathological evidence, and animal evidence and chemical evidence, that smoking represents one of the major causes of lung cancer, and that in its absence, 80 percent of all lung cancer and cancer of the oral cavity occurring in this country could be avoided. This opinion is shared by many responsible agencies in this country and in Europe.

We also believe that there is a practical solution to this problem. This problem, like any other problem, cannot be solved by depreciating its importance or by ignoring it altogether.

It can be solved by facing it squarely. I believe that in line with the recommendations that we have outlined, we can give our public a safer smoking product, and, above all, we can save lives.

It seems to me that the lives of all the people who died of respiratory cancer in recent years and will die in years to come demand that we give this problem our fullest attention.

Mr. BLATNIK. Dr. Wynder, we thank you and thank you most sincerely for the comprehensive statement, certainly a very sobering statement. Do the members of the committee have any questions on my right?

Mr. HARDY. The only question that occurred to me, Mr. Chairman, had to do with the causes which the doctor seemed to ascribe primarily to tars.

Have you determined that the tar content of smoke is substantially the only cause of cancer?

Dr. WYNDER. Mr. Hardy, "tar" refers to the whole brown substance that you condense out.

Mr. HARDY. The total contents?

Dr. WYNDER. That is right. That is made up of nicotine which we have already demonstrated is not part of the carcinogenic problem, and of perhaps thousands of other substances. In one of the slides I demonstrated that we have broken all this tar down to show that the majority of the tar components are not carcinogenic.

Mr. HARDY. I couldn't understand that chart.

Dr. WYNDER. The majority of the components of the tobacco tar are not carcinogenic, and I would estimate that the actual carcinogenic components in tobacco tar are certainly less than 1 percent of the total tar.

Mr. BLATNIK. Have they been identified?

Dr. WYNDER. Let me state this and I am sure this problem will come up repeatedly here. Much attention has been placed on benzpyrene. In effect this has become an issue. It happens to be one of the substances that has been known and everybody tries to blame everything on that one substance. We have stated repeatedly there is not enough benzpyrene in tobacco tar to explain the animal results we have published.

However, we have also stated that there are numerous other substances which are benzpyrene related which are more active than benzpyrene and most likely account for the majority of the activity.

These are, in particular, substituted benzpyrene derivatives. I think it is more or less academic whether it is benzpyrene or dibenzpyrene, or dibenzanthrene, or a substituted benzpyrene derivative because they all form in the same manner.

If we can control pyrolysis and/or the precursors, we can reduce all of these substances at one time.

Mr. HARDY. Of course I am not too concerned with being able to tie a name onto the particular factor. The thing that I am concerned with is whether or not we can get rid of it.

Dr. WYNDER. As I stated, we can get rid of it probably in one or two ways. By modifying the combustion condition of the smoke, and

second, by studying the precursors in tobacco, which we have been studying. We have been extracting tobacco with various solvents. Initial results indicate that by extracting particularly the waxy portion you remove or reduce the higher aromatic polycyclics upon smoking this produces.

Mr. HARDY. To what extent will the filters that are now being used actually remove these carcinogenic tars?

Dr. WYNDER. As I stated and demonstrated in one slide, no filters currently in use—and I think no mechanical filter—can selectively remove any particular component from within the tobacco smoke. These are all very small particles, and they move through the filter very rapidly. It is chemically and physically not possible to pick out certain substances from within the particles.

But the filters do have a use in that they can reduce the overall exposure. If, let us say, a filter removes 40 percent of the total tar, it will also remove 40 percent of the carcinogen.

Mr. HARDY. I am trying to think in terms of what is happening now. If I understand your analysis of it, if we use a proper filter and a proper tobacco mixture, then we could reduce the danger.

Dr. WYNDER. That is absolutely correct. Whereas these studies on precursors and pyrolysis on which we are engaged—and I understand some of the tobacco companies are now engaged—may give a practical answer a year hence, the problem you just stated could be solved today.

Mr. HARDY. Then the question comes down to this, and this is the purpose, as I understand it, of what we are trying to consider here: Whether or not the filters that are currently in use actually do render the particular cigarette, or any of them, free of cancer-causing tars.

Dr. WYNDER. As I outlined to you, the field of filtered cigarettes, as I see it and as brought out in The Reader's Digest story, shows that a majority of the filtered cigarettes give you more tar and more nicotine than you used to get when you smoked a regular sized cigarette.

Mr. HARDY. What you are saying, then, is that the cigarette manufacturers have changed the mixtures to the point that they have more than offset the value of the filter?

Dr. WYNDER. Therefore, in line with the dose-response study that we have demonstrated, any cigarette—filtered or unfiltered—that gives you more tar will increase your risk of developing cancer.

Mr. HARDY. You don't mean to say that the cigarette that is on the market today with a filter on it is more likely to produce cancers than when it was unfiltered a few years ago?

Dr. WYNDER. You shouldn't group all the filtered cigarettes together because some of them are trying to do an honest job. So I don't want to make a statement in general.

Mr. HARDY. I am just trying to understand where we are.

Dr. WYNDER. I do want to say this. In the Digest story they made a certain comparison of changes within a given manufacturer—for instance, manufacturer A who used to produce a regular sized cigarette, A, now produces a filter, filter B.

The Digest story shows that filter B now contains more tar than you used to get when you smoked cigarette A. That, in my opinion, is going in the wrong direction.

On the other hand, there are some other filter makers who have put on a better filter, used a better blend, to give less tars. This is the direction in which I believe the filter industry should go.

I believe that the filter on the cigarette has a potentially great value, provided it is doing the job that it is supposed to do. I believe that a satisfactory smoking product can be produced by a good filter and by a proper blending of tobacco which will lower the tar content.

I would like to see regulations which would encourage or perhaps even require the filter cigarette manufacturers to reduce their tar and nicotine content to certain prescribed levels, or not permit them to use the term "filter."

Mr. PLAPINGER. What is the level you recommend, Doctor?

Dr. WYNDER. The level that we have recommended is a 40-percent reduction.

Mr. PLAPINGER. Forty percent at the top or forty-percent reduction at the bottom?

Dr. WYNDER. Forty-percent reduction over and above the standard regular sized cigarette.

Mr. PLAPINGER. What does that mean, actually, in terms of tar?

Dr. WYNDER. In terms of tars, it depends a little bit on the method that you use for tar determination. But let us say in the studies published by Snell, the tar content of the average regular sized cigarette is 30 milligrams, then it should be reduced to 18.

Mr. PLAPINGER. Suppose it is 40?

Dr. WYNDER. If it is 40, it means that this manufacturer has used specially heavy blends of tobacco, so we must arrive at the average norm that has been kept over the years.

Mr. PLAPINGER. But you would consider 18 the safety threshold. How much give one way or the other? Suppose it is 19?

Dr. WYNDER. It is very difficult to put an exact figure on this. We chose 40 in part because we noted a drop off at about this level in our dose-response studies. This is the line that we like to encourage.

I would never say that this cigarette would be safe, but I would say it will be safer.

Mr. HARDY. Suppose you do reduce it to 18. Then it is up to the individual smoker to regulate his volume of consumption. If he continues at a high rate of smoking, his total intake during a particular day or a particular period of time could exceed what it had been previously.

Dr. WYNDER. You made a very good point there. In the studies that we did on people who smoked filtered cigarettes, we questioned this particular point: Do you now smoke more or less since you shifted to filter cigarettes? There are a certain number of people who smoke more and a certain number of people who smoke less. There was no significant number of people who said they smoked much more.

Mr. HARDY. The encouraging thing in your testimony to me is apparently there is a solution to at least the critical serious effect of smoking.

Dr. WYNDER. That is right.

Mr. HARDY. On the other side of it, you present a rather sad picture to indicate that actually the intake of tars from smoking now is more than it was since before we started using filters.

Dr. WYNDER. This applies to some of the filtered cigarettes. I want to stress the fact that it applies to some of the filtered cigarettes now on the market. This is due to several reasons.

One is that the filter has had to be made more porous. Two, tobacco blends have been used which are heavier in tar and nicotine yield. And three, the cigarettes have been lengthened.

You can demonstrate that the longer the cigarette, the more tar you get as you smoke toward the butt end, because tobacco itself works as the filter. For instance, in the first 10 millimeters of the cigarette, you get only, let's say, 4.5 milligrams of tar in the smoke and in the last 10 millimeters, 11 milligrams.

Mr. HARDY. Then you might recommend that we buy only a king-sized cigarette and don't smoke over half of it. That would increase the sale of cigarettes and reduce the risk of cancer?

Dr. WYNDER. That might make me very popular with the tobacco industry. But I don't want to leave you with the impression that I think filters are bad, because I think they are one of the things that we must encourage. But we must encourage good filtration.

Mr. HARDY. What you are saying is, though, it doesn't make any difference if you use a filter which is good, if you offset its advantage by increasing the tars in your mixtures.

Dr. WYNDER. Correct.

Mr. BLATNIK. Doctor, in all of this discussion of your testimony, you have referred only to the tobacco in a cigarette as the source of tar. Have you considered, or do you have any knowledge of the paper used on cigarettes as a possible source?

Dr. WYNDER. We did one study in which we took cigarettes and wrapped them in a cigar leaf. We thought that that was a good way to find out what the paper does to the total product.

We got just as many cancers with that product as we got with the ordinary cigarette with paper. We therefore believe that the paper contributes very little to the carcinogenic substances in tobacco.

Mr. BLATNIK. After you ran these tests on cigarette tobacco wrapped in a cigar leaf, then did you shift over to use only a cigarette entirely with paper?

Dr. WYNDER. No, we didn't do that because we think it would be very hard to compare this—to take paper pulp, for instance. Dr. Wright published a study in which he compared the benzpyrene content of cigarette paper by burning it in a hollow shell. When he burned it as a pulp, he found some of these higher aromatic polycyclics, but when he burned it as a hollow shell he found nothing.

It seems to me in all of these experiments you want to stick as closely as you can to the way humans consume a product.

Mr. BLATNIK. You mentioned a filter as the possible way to reduce the total volume of intake of smoke, thereby cutting down by the same proportion relatively the intake of your carcinogenic material. Are there any other methods that are being examined or studied?

I am thinking of the chemical which may neutralize or alter or make into a neutral stable compound these carcinogenic compounds.

Dr. WYNDER. This is a very good point. As I outlined, at the Sloan-Kettering Institute we are engaged in research on filtration and dose-response. (See appendix, exhibit 4, p. 370.) We are interested in the pyrolysis of the material and how these compounds are formed. We

are currently working in the field of anticatalysis in which we are adding substances to the tobacco to see whether the formation of these carcinogens can be reduced.

This is very much in line with the statement that you just made. The third one which I outlined—the study of precursors—is to see whether we can remove potential carcinogen substances from the tobacco. Incidentally, in this one study which we did where we extracted tobacco with hot hexane, we did find that you get a marked reduction in the formation of some of these materials, and yet a very satisfactory smoking product comes out of that.

So I think there are practical aspects to the problem short of improved filtration. The only reason I stress the improved filtration is because this is the kind of thing which we can do today.

Mr. BLATNIK. Mr. Meader?

Mr. MEADER. Dr. Wynder, were you present when Dr. Little testified yesterday?

Dr. WYNDER. No, I was not.

Mr. MEADER. You might say that scientists disagree sometimes, and I would like to stimulate that disagreement for just a minute. Dr. Little said in his prepared statement:

> Secondly, after 50 years of research on the origin and nature of cancer, I have the greatest respect for its vigor, versatility, and complexity. I therefore sincerely and deeply deplore premature and oversimplified conclusions and intensive publicity.

Then somewhere else in his statement—I haven't found it yet—he referred to the conclusion of the association of lung cancer cases and the amount of smoking—and deducing from the cause-and-effect relationship. He characterized that as a gymnastic feat of some kind.

I would like to have your comment on Dr. Little's scientific attitude in saying we shouldn't assume that there is a cause-and-effect connection until there is more conclusive scientific evidence.

Dr. WYNDER. On the first statement that this is premature, I don't think that in the field of etiology of cancer there has been ever such widely concerted efforts and research in the field of smoking and lung cancer. Everywhere I have been in Europe and in this country, people are working on it.

As I pointed out to you, at least 16 separate retrospective studies and two prospective studies involving thousands of lung-cancer cases—have all confirmed the same thing. Special studies on women, animal studies, rabbit studies, isolation of known carcinogens in tobacco, plus all this presumptive evidence—this is about as conclusive as you can ever get it to be.

I will entirely admit that there is no scientific subject, and I am sure you in Congress will recognize no political subject—on which you will get complete agreement at any time.

One of my hobbies is history, and one of the things in history of medicine that impressed me most was in the last century when many women, particularly in Europe, died from childbed fever. Dr. Semmelweis, in particular, demonstrated this was due to the fact that doctors didn't wash their hands.

Today we recognize it as an obvious thing. But in those days there were many opinionated people, and he was laughed out of Vienna and died a brokenhearted man in Hungary.

Yet, at that time all he thought was an opinion. It would have been very simple just to wash their hands. Today, unfortunately, we not only fight opinion; we fight a very important industry.

I think Dr. Little, of course, has all the right to present his point of view, though I assume it would be very difficult for anybody associated with the tobacco industry to get up in front of you here today and admit that tobacco is a harmful product.

Dr. Little has often said, "Now, you demonstrate association but not causation." I thought that by showing you all the evidence to find out how it fits in with the presumptive evidence, the causative relationship is the more direct one to assume. But from my point of view it makes very little difference whether we argue about the words "contributing to," "initiating," "causing," "associate," "relating to"—the important factor is that the person who doesn't smoke rarely gets lung cancer. And the more you smoke, the more lung cancer you get. That is the important issue.

I certainly admit there are numerous intrinsic factors of lung cancer about which we know very little. But if we were to apply the same argument of Dr. Little to the point of radiation—and I think it is a good example to use—we can rule out that radiation couldn't possibly cause cancer in men.

Yet I am sure that you will not find a single scientist in this country who will say that radiation cannot cause cancer in men. We have the evidence in men. We have the evidence in the experimental animal. The relationship is identical.

In fact, I am sure that if we were dealing with spinach rather than tobacco, we would not be sitting here debating the point.

The only thing that upsets me are really two points—and I was glad that the Public Health Service recently made a statement in this regard—one: I feel that the public ought to be entitled to know from the Government, from the Public Health Service, what the risks are when they smoke. I deal the same way with my own patients. I don't say to the patient, "Now, if you don't smoke, I will never see you again." I tell them the facts and let them lead their own lives, and I lead mine. That is all I can do as a doctor.

The other point is, that I think there is a solution to this problem. As I pointed out in my last remarks, the solution can only come when we recognize we have a problem here and let's go to it and do something about it.

Mr. MEADER. The only other question I had, Dr. Wynder is this: To what extent are you familiar with the research conducted under Dr. Little at the expense of the tobacco industry?

Dr. WYNDER. I am all in favor of the tobacco industry supporting research in this country. If they spent $2 million to further research in cancer or the tobacco-cancer problem, it is all so much the better.

But I was a little discouraged if after 2½ years Dr. Little publishes his first report, after spending perhaps $2 million, and reported that they found very little.

I am sure that the director of my institute, after I had spent that much money on research and after 2½ years, and I had to report I found practically nothing, would be a little bit unhappy.

The point is that I would like to find out what direction the tobacco industry research committee is taking. They say, "We cannot believe

this animal work." If they don't believe you can demonstrate anything with mice, let's not support any animal work because it doesn't show anything, regardless. Then they think apparently very little of statistics.

If you doubt statistics, why do human work, which is statistics. So actually you have already cut off every possible road to coming to an answer to the problem before you even start it. That is discouraging.

Mr. BLATNIK. Thank you, Doctor. The second bells have rung for a quorum call. If there are further questions of Dr. Wynder, I am sure he may be able to stay over until 2. We thought we would meet at 2 to proceed with Dr. Rigdon here then.

Mr. MINSHALL. Doctor, I have just one question. Do you know of any studies that have indicated that a virus might be a contributing cause of cancer?

Dr. WYNDER. This is a great controversy, as you know. Everyone has his own theory on cancer. I think it is conceivable that a virus could initiate a cancer, very much as a chemical carcinogen could initiate a cancer. I personally believe that the basic cause of cancer is an enzymatic deficiency within the cell itself, and that anything that can induce this deficiency—be it radiation, be it a chemical, be it a physical substance, be it a virus—could initiate cancer.

Mr. MINSHALL. The virus then in your opinion could initiate cancer?

Dr. WYNDER. It could, though I personally do not believe that it does. But certainly there are a number of theories that have been developed along that line; and as long as we do not know what the basic cause of cancer is, this is as likely a theory as anything.

Mr. MINSHALL. Do you know Dr. Little?

Dr. WYNDER. Yes.

Mr. MINSHALL. Have you ever discussed with him the merits or demerits of filters?

Dr. WYNDER. No, I have not. Let me give you one final example—and sometimes examples are very good. There was a cholera epidemic in London—and apparently a lot of these good examples come from England.

At that time Dr. Snow—who did the first major study on epidemiology—got the idea this might have to do with a Broad Street pump where much water was consumed. He did a study and found out that the people who drank water from that pump got more cholera than consumers of other water.

At that time they didn't have the vaguest idea what the basic cause of cholera was. Yet he went to the board of governors one day and said, "Let's try to remove the handle of that pump just on the basis of epidemiological evidence." They removed the handle and the cholera epidemic ceased.

At that time the basic cause of cholera was not known. The same thing applies to the cancer problem. We do not have to find out or know the basic cause of cancer in order to prevent many of the cancers that occur in men today.

Mr. BLATNIK. Thank you very much.

The committee will continue at 2 o'clock this afternoon with Dr. Rigdon.

I have received several articles on the relationship of cigarette smoking and lung cancer written by Dr. Alton Ochsner, president of the Alton Ochsner Medical Foundation in New Orleans, and, if there is no objection, I shall have them inserted in the record. (See appendix, exhibit 5, p. 391.) Dr. Ochsner, one of our most eminent surgeons, is chairman of the department of surgery, school of medicine, Tulane University, and a former president of the American Cancer Society, American College of Surgeons, and the American Association for Thoracic Surgery.

(Thereupon, at 11:25 a. m., the committee recessed, to resume at 2 p. m. of the same day.)

AFTERNOON SESSION

Mr. BLATNIK. The Subcommittee on Legal and Monetary Affairs of the House Committee on Government Operations will resume public hearings on the role of Federal agencies in the field of fraudulent and misleading advertising in the current series.

The first is concerned with cigarettes, particularly with the filter cigarettes.

We have this afternoon Dr. R. H. Rigdon, professor of pathology, School of Medicine, University of Texas.

Doctor, we appreciate your bearing with us. You were scheduled to be on in the latter half of the morning session, which was interrupted by the House convening at 11 o'clock this morning instead of the usual 12 o'clock.

The mutual foreign aid program is in debate. It is a matter of considerable congressional interest and activity and we regret we have to run the hearings into the afternoon. Many of the members are tied down in debate on the floor who would like to have been here, and of course they asked me to express their regrets because of this unavoidable conflict. It is literally impossible for them to be here.

Dr. Rigdon, I notice you have a prepared, written summary statement of about three pages. Would you proceed with either reading your prepared statement or we will insert it in the record and you can give us an oral presentation.

STATEMENT OF DR. R. H. RIGDON, PROFESSOR OF PATHOLOGY, SCHOOL OF MEDICINE, UNIVERSITY OF TEXAS

Dr. RIGDON. I would like to say that I am a pathologist, and a pathologist is one who is interested in the changes that occur in the body in disease. Cancer is one disease process. I teach pathology to medical students, and I am interested in research.

I have been working on both, so my interest is in human diseases as well as in the experimental study of disease.

I have prepared a summary of the problem as I see it, and I also have a copy of a recent publication which sums up my ideas on lung cancer and smoking.

This problem of cancer of the lung is not a new problem. The first case of cancer of the lung was reported in 1810. Since this time there has been a progressive increase in the number of cases of lung cancer.

In fact, you can find a very good correlation between the number of increases relative to the introduction of new techniques for diagnosis.

The X-ray was invented in 1895 and that was a very good technique to pick up more cases of cancer in the lung. And then the bronchoscope, where you go down and get out biopsies of the lung—has been a very important factor in this problem of an increase in the number of cases of cancer of the lung.

As I see it, cancer of the lung is not something to itself. It is a part of the picture of cancer. So when we are talking about cancer of the lung, we must think about cancer as a disease process wherever we find it.

I have been interested in this problem of cancer of the lung. The first publication was in December 1950, we started in this problem before this controversy arose. So it is of interest to note that in 1933, only 24 years ago, we had the first successful operation for cancer of the lung. This immediately brought into focus attention on this problem.

It is of interest to note from a statistical standpoint some of the problems referable to cancer. One of the first things that I did in this study was to check on the number of lung cancers that had been reported in the different States. When I speak of statistics, I refer to vital statistics and not to those that I got myself. I refer to vital statistics of the United States Public Health Service.

We took the number of cases of cancer of the lung that had been reported, and the number of doctors practicing medicine in the different States, and the number of hospital beds. With these we were able to find out a correlation between the number of doctors, the number of hospital beds, and the number of cases of lung cancer.

We had to conclude from this study that if you didn't want to die from cancer of the lung from a statistical standpoint, you would have to go to a State with few doctors and few hospital beds, because they had fewer cases of lung cancer than other States.

We realized at that time that there are some problems in such a statistical study. We then started to work on this problem referable to cancer of the lung and death certificates because we felt that the death certificate, which is a basis for vital statistics, is not an accurate record of whether you have lung cancer or not.

We know that during the past 100 years there has been an improvement in diagnosis, and with this improvement, our vital statistics are better. All of those factors enter into this question of vital statistics and death certificates. We know that many men practicing medicine don't have facilities to X-ray a patient, take a biopsy of the lung and do all of the examinations that are necessary. They do the best they can under the conditions in which they are working.

If they are practicing medicine, in the back side of some county, they don't have all of these facilities. They have to do the best they can and their diagnosis will not be as good as one who is in a teaching institution.

We therefore began to question whether these vital statistics were an accurate source for determining whether or not cancer of the lung is as frequent as it is reported to be.

In this problem of vital statistics there has been a continuous change in the coding of cancer of the lung. I think there have been 4 or 5 changes in the classification. At one time they just lumped cancer of

the lung with all other cancers that occurred in the chest. Because of this you don't have an accurate baseline. With the last revision which came in 1939, it is a more specific classification.

Since 1939 we have had a tremendous increase in cancer of the lung. But I don't think that you can actually determine how many cases of cancer of the lung that occurred preceding that time. As I see it, we really haven't a base line to talk about.

If we change coding every few years, we don't have anything really to base our opinion on. Those are some of the things that I am interested in in this problem of the frequency of lung cancer.

There are many people who have gone into this problem from a statistical standpoint, all these opinions are not my own about the value of statistics and the way they are changed. I might cite for you a few.

In 1931 it was pointed out by Downes that—

> The general limitations of official mortality statistics as scientific data are already so well known that none save the tyro in statistics will fail to go behind the published figures in order to take into account their grosser faults. The full extent of their limitations will not be appreciated adequately by anyone, however, until he makes the attempt to ascertain what the death rate actually is from a given disease in a given area.

The vital statistics office of the United Nations in 1955 said:

> All medically certified cancers are not equal in quality. The knowledge and skill of the physician in making the diagnosis, his willingness to report completely, and the existence of facts for reporting certain diseases all affect the ultimate results.

Mr. PLAPINGER. Where are you reading from, Doctor?

Dr. RIGDON. I am reading from the second page in the manuscript that I showed you—Review of Literature, the last paragraph.

Mr. PLAPINGER. Thank you.

Dr. RIGDON. We question the statistics as being a satisfactory source to draw fine conclusions from. I personally have to be cautious in drawing conclusions from these data.

We can find here—quoting from Gilliam, which is the last part of the paragraph on the first column on the following page, reported in 1955:

> I have previously stated that the rate of increase in recorded mortality was greatest in this country between 1914 and 1930 and it has been declining since.

There are many references in the literature which emphasize this problem of statistics and the significance that one can attach to them.

The second thing that we have been interested in is a survey with regard to the smoking habits of individuals. It was published in the Journal of the National Cancer Institute in 1955. I mention this only to emphasize the fact that we have been personally interested in gathering statistics on this problem of smoking and lung cancer.

In this study we obtained the smoking habits of about 12,000 people. In this we began to learn something about the practical side of getting such data. We find that it is very difficult to establish a fact by asking a man how much he smoked 20 years ago. They would usually come up with a statement, but it was always a question in my mind as to whether I could accept it as being correct. I don't smoke; I have never smoked. So I find that it is difficult to establish exactly what a man did 20 years ago with regard to smoking.

The next thing is, how much do they smoke? Because one man says he smoked 3 packages of cigarettes in a day, and another man says that

he smokes 1 package. Statistically you would put them in two different groups. But really and truly, this man that smokes three packs may discard every cigarette when it is only a third smoked, while another will smoke it down very short—to a butt.

We therefore have to be cautious about such data. It is not a matter of just collecting these data. They have to be critically analyzed. I don't know how you are going to avoid a certain number of errors.

So the question of smoking—how much you smoke—is hard to establish from a statistical standpoint. Then I find that there is a lot of difference in the division—whether you are a heavy smoker, if you smoke one pack; whether you are a heavy smoker if you smoke a pack and a half. So that is another difficult problem.

Personally I have been very much disappointed in some of these surveys in which they have converted a cigar smoker into a cigarette smoker as far as the statistics were concerned.

Mr. PLAPINGER. Has the Cancer Society done this?

Dr. RIGDON. I don't think the Cancer Society has, but the United States Public Health Service, and Doll and Hill in England did in their studies.

In other words, they say that 1 cigar is equal to 10 cigarettes. You can see what it would mean. If a man smokes three cigars, then he is really a heavy cigarette smoker as far as statistics are concerned. Then if you smoke a pipe, it is 2½ cigarettes.

Personally I don't follow that line of reasoning, shifting these cigar and pipe smokers over into cigarette smokers. Because of these conversions it becomes a difficult problem to arrive at data referable to this point.

It has been of interest to me to note some of the data that have been given referable to the smoking habits and how much they smoked. I have one point that I would like to call your attention to, to emphasize this point.

In a study by Doll and Hill—these are English investigators, and they reported this in 1950—of 649 males with cancer of the lung, there was 647 smokers—a percentage of 99.7. In the control group of 649 patients with diseases other than cancer of the lung, there were 622 smokers, a percentage of 95.8.

My comment is, I can't see much difference between 99.7 and 95.8 in these statistics, especially when you have as many defects in getting these as I have mentioned. We have no argument at all with statisticians. They can take figures and do a wonderful job with them. But as far as I am concerned, their significance lies in the figures that they used.

So that is why I fuss and argue with them. It is not from what they have done, because they do a swell job. But these are the things that bother me, and therefore I have to consider them in interpreting the data.

There have been many problems to arise with this smoking business. One of them, the argument is as to whether or not the inhalation is bad. Referable to this, it is of interest to observe—also in the statistical studies made by Wynder and Graham in 1950—that:

> The greater practice of inhalation among cigarette smokers is believed to be a factor in the increased incidence of the disease.

Doll and Hill, English investigators, in 1950 in a similar statistical study state:

> It would appear that lung carcinoma patients inhaled slightly less often than other patients.

There are two statistical studies. One says it influences causes of the lung, and the other one says it doesn't. So I have to consider that.

There have been several statistical studies made on this problem. I think 14 are frequently cited. But one of the interesting things in those data is the fact that one is by Schrek. In 1940 he says:

> This relatively low percentage of deaths by cancer of the respiratory tract as compared to the higher percentage of smokers indicates that smoking is at most only a weak carcinogenic agent.

Now, that is included in these 14 studies, but they don't emphasize that point, don't you see? So to me, in trying to evaluate this problem and form an opinion, I have to look at such points. That is the basis that I have to form an opinion.

In these studies, then, there are certain things that come out, I think, as far as my personal opinion is concerned, that make me wonder about some of these things.

Now I would like to go into another phase of this problem, and that is the experimental study of cancer. I have been studying cancer since about 1937. We did some work with viruses, and I might mention that there are many other agents that are said to cause cancer.

If you want to really hear the argument in favor of virus, you should get that group of people together. They almost convince you that all cancer is due to virus. Then there are others who say it is due to chemical agents. Then there are others who say it is due to hormones. And then there are other problems referable to heredity and mutation in the etiology of cancer.

So there are many theories about cancer. Experimentally is one of the ways to tackle this problem. I have been interested in the study of the experimental production of cancer. In this study I have used one of the most potent cancer-producing agents that is known, which is methylchlolenthrane. It is recognized as being an experimental agent only for the production of cancer.

I have used a white Peking duck in this study. We have found several things that I think would be of interest to you in the overall problem.

We have put this agent on the skin beneath the wing of these birds and we get a variety of tumors. Then we put it on the web of the foot and we don't get very many tumors. If you put the same agent into the trachea and down the lungs, we still don't get the type of tumor that we get on the skin.

Therefore, in one host, we get a variation depending upon where this same chemical agent is going.

We have been interested in putting this cancer-producing agent on the skin, and then we plucked the feathers. That is the technique that we were using to produce trauma. We found that after having put this cancer-producing agent on the skin and the feathers were plucked, we got more tumors to develop in the skin than we did in those birds where we didn't pluck the feathers.

This observation suggested to us that this cancer-producing agent, plus the trauma, made this point more significant. This work has been published in the Archives of Pathology.

That brings into the problem of cancer cocarcinogens—that is, certain things acting together that may produce cancer. We have here a very important factor. That is, if you traumatize the skin and then put on a cancer-producing agent, that will be entirely different than just putting on the skin only the cancer-producing agent.

That brings us to a point that I think is very important in experimental cancer and that is, if you put an agent on the skin over a period of a year and traumatize that skin frequently during that time, you have a combination of things to worry about that produce the tumor.

We are seeing this thing in Texas, in ranchers. They are exposed to sun. We are seeing these people develop skin cancer—we know that they develop skin cancer very easily because of the sunshine; the ultraviolet rays produce some changes in the skin cells and trauma comes along and may be the immediate cause of producing it. Sometimes you get skin cancer without any trauma.

There are many things that go into this problem of cancer rather than just one particular process.

I believe in trying to evaluate this thing of experimental cancer, it would be wise to take several experimental hosts to work on. Since I was using the duck, I took the chicken and I put the same cancer-producing agent on the skin of the chicken and I got a different type of tumor.

For a third host I took a turkey, I put this agent on the skin of turkeys and I just don't get anything to compare with the tumors I get in the duck. So in the skin of the chicken, the duck, and the turkey we have an entirely different situation referable to the development of tumors. I have to be cautious in concluding that what is going to happen in the skin of a mouse is what is occurring in the lungs of man.

I can't accept this theory. The reason I can't accept it is because of my experience in the experimental study of cancer.

I know that you can develop strains of mice that will develop cancer of the breast very, very quickly, or cancer of the lung very quickly, while another strain won't. So if you select certain strains of animals, you can bring out many points, while you couldn't if you used other hosts.

Although the mouse has been used for a long time, I don't accept observation as being conclusive evidence as to what is going to occur in man. So I have to be cautious about such conclusion.

Mr. MINSHALL. Mr. Chairman, I wonder if I could interrupt at that point, please. Have you heard of any investigators who have attempted to grow a cancer on human skin?

Dr. RIGDON. No; I have not. I don't know. I developed one (squamous cell) on the ear, my ear, and somebody said it was due to the fact that I had been using methylcholanthrane. We got it out 4 years ago and it hasn't recurred.

But I don't know any human investigation on human with this carcinogenic agent that would throw light on the problem. I am not familiar with it. It may be.

Mr. MINSHALL. That answers my question. You just don't know.

Dr. RIGDON. I just don't know.

Mr. PLAPINGER. Doctor, have you experimented with tar after the fashion of Dr. Wynder on your turkey and your Peking duck?

Dr. RIGDON. We are working on that now. The experiment has been in progress now for a year. After all, it has taken me 3 years to get a technique, and it takes me a year after I set up the experiment. This is nothing that you can do quickly.

Mr. PLAPINGER. Please don't misunderstand me. I am not criticizing. I am just asking.

Dr. RIGDON. No; I just mentioned the time factor, and these experiments are not completed. But you asked me, and I will say that I am using tobacco tar. I do not make the tar. I obtain it from a commercial plant.

We are putting it in the trachea of ducks, and we are comparing it with methylcholanthrane.

Mr. PLAPINGER. Is there methylcholanthrane in tobacco?

Dr. RIGDON. Not as far as I know, no. That is not in tobacco. It is an entirely different agent.

So we may say we have been disappointed, if you want to look at it that way, that we haven't found any tumors in the lungs. We mix this tobacco tar with mineral oil. That is not like you get it when you are smoking, but we are trying to work out a technique that may be of help to us later on.

We have not been able to obtain any tumors so far with this tobacco tar. We find that it goes down into the lungs. It comes in contact with all the cells there. This brings up a very interesting problem because we can see that it gets all down in the lungs and the tumors alleged to be due to tobacco occurs at one site in the larger bronchi. That is hard for me to put together, but those are the facts the way we have them.

We are studying lung cancer. We have found that tobacco tar is widely distributed through the lungs, and that brings up the question that, after all, as these agents go down into the lungs they go by the larynx, the voice box. There has been a lot of discussion about cancers that occur here.

The British have made a nice study on the frequency of cancer of the larynx over a period of the last 25 years, and they haven't found a significant increase in cancer of the larnyx—nothing to compare with cancer of the lung.

Now it is the same epithelium; it is the same individual. It is interesting, this variation, because the concentration goes right by the vocal cords and down in the lungs. That is another thing that is of considerable interest.

Mr. MINSHALL. Doctor, one other question—a hypothetical one in a way. Assuming you took some of this cancer-producing agent that you have been talking about that is used on the Peking ducks, chickens, and turkeys, and you put some of that on the arm of a human being or some other parts of his skin and it produced tumors or cancerous tissue. Would you think that was significant?

Dr. RIGDON. I would only say that it did produce it in man, but I would not conclude that everything that will produce it in a chicken you can transfer to man.

Mr. MINSHALL. Wouldn't you think it was significant if they produced cancer on human skin in numerous instances with this same

agent about which you speak? Wouldn't you think that would have some bearing on it?

Dr. RIGDON. It would be an interesting observation, but I wouldn't be able to conclude—just because they parallel each other—that this was "cause and effect." In the skin of the duck we get one thing and in the trachea, we get another.

Mr. MINSHALL. Even if it did 100 percent of the time in human beings?

Dr. RIGDON. Yes. I would consider that as being a significant factor. But I don't think it would prove that that is the thing, because we don't see too much difference between the skin of the duck and the skin of the chicken and the skin of the turkey and we find a different reaction, don't you see, so that is why I am careful in drawing my conclusions.

Mr. MINSHALL. I am talking about a human being now.

Dr. RIGDON. O. K., if you got it in a human being, I would say that this human being was acting like this particular animal. But I wouldn't be able to say that it would act like all the other animals——

Mr. MINSHALL. We are not worrying about what it does to chickens. We are worrying about what it does to human beings. If it does this to a human being, wouldn't you say that that is a cancer-producing agent on human skin especially if it produces cancer 100 percent of the time, don't you think that is significant?

Dr. RIGDON. I certainly would.

Mr. MINSHALL. That is the end of my question.

Dr. RIGDON. Sure, that would be the most important thing. If we could get some human beings to do this, it would be a wonderful experiment.

Mr. MINSHALL. I think some have. If you don't think it would do it to human skin, why don't you try it on yourself?

Dr. RIGDON. That is a very good point—for the simple reason that I feel like, as a scientist, I could contribute more by trying to study and work out these things than to do something to myself and probably cause death in the next 5 years.

So I don't think it is a wise thing.

Mr. MINSHALL. You have never tried to get any volunteers?

Dr. RIGDON. No; I never have. But I think it would be a swell thing to do. I don't object.

Mr. MINSHALL. I am not volunteering.

Mr. KILGORE. I would just like to inject here, as a fellow Texan, if the doctor seeks any volunteers, I would rather he would go to Ohio.

Dr. RIGDON. I have mentioned about the experimental procedures and some of the things you would have to watch out for. Then I would like to comment on the problem of the production of cancer in man and other pathological processes that might occur in man as a result of a carcinogenic agent—and to be specific we will talk about tobacco smoke.

It is alleged that it is the cause of cancer of the lung. It is also alleged that it produces heart disease.

Since I am a pathologist and since I do autopsies and I look at people, examine tissues, I am a little critical of what we mean when we say heart disease. I know you have several things that produce heart disease. One of them is syphilis, another is a bacterial process, one

is rheumatic heart disease, one is congenital heart disease, another is atherosclerotic heart disease, one is a heart disease you get with high blood pressure.

When people say, "Well, it will produce heart disease," I have to know what kind of heart disease they are talking about. So I think that it becomes a very important thing in evaluating this problem as to the specific disease processes that they relate to.

It is very interesting to me to note that we have an agent that is alleged to produce cancer and any type of heart disease that you want. Cancer, as I know it—in man or the duck—is entirely different from heart disease. Here we have an agent that is producing both. We not only have that, but we have got it producing prostatic cancer.

I just can't see how you are going to connect this agent producing cancer in the lung, let us say, with a prostate. That is just too far-fetched for me, and that is why my conclusion has been that I don't agree with what has been done. I can't see that. I can't see it by any stretch of my imagination.

We know that hormones play a part in cancer. We know the age in which men get these cancers, and we know that women get cancers of the breast, and we know the effect of hormones on these things.

So I can't see how we can get all of this in tobacco to account for everything that we get, don't you see. So that is the reason why I have difficulty in accepting these data.

So, Mr. Chairman, I will close. I could go ahead and discuss this thing further, but I think this will give you the basis for my opinions.

I understand that is what you would like to have.

Mr. BLATNIK. Would you like to have this report you have, Doctor, inserted in the record?

Dr. RIGDON. It has been published in the Southern Medical Journal. (See appendix, exhibit 12, p. 583.)

Mr. BLATNIK. This is largely a résumé of other literature, is it not?

Dr. RIGDON. We have a review of that. We have an additional article which hasn't been published yet, Cancer of the Lung From 1900 to 1930, in which we review much more of the literature than what is in that.

So that will give a basic idea of what we have, commenting on the observations of others.

Mr. BLATNIK. Any questions? Mr. Kilgore?

Mr. KILGORE. Doctor, I haven't had an opportunity to go through this publication. It may answer the question. The study that you are doing, particularly with reference to what you described in the use of some of these agents on animals: Is that study carried on at the University of Texas branch at Galveston?

Dr. RIGDON. It is, sir.

Mr. KILGORE. It is carried on under your direction, is it?

Dr. RIGDON. Yes, sir.

Mr. KILGORE. How many people have you associated with you in connection with this particular study?

Dr. RIGDON. I have in the laboratory of experimental pathology seven people.

Mr. KILGORE. In addition to the studies that you have made and are making in connection with the animal studies you describe, have you also made any evaluations on the incidence of lung cancer in pathological reports that have come to you in the hospital?

Dr. RIGDON. Yes. In one of our earlier reports, we were studying the frequency of cancer in the white and colored. So from our study we found that cancer of the lung was less frequent in the colored than it was in the white.

So we have made such studies.

May I develop that point a little?

Mr. KILGORE. Certainly.

Dr. RIGDON. A few years ago the surgeons in the hospital got very excited about the number of cases of cancer they were seeing. So I said, "Well, now I am going to see what is happening here."

I began to check to see where these patients were coming from. I found out that—as you know—at the University of Texas we have a State hospital that brings in patients from all over the State. We were seeing that people were coming from a large area of the State into this particular clinic, don't you see.

It occurs in other clinics in the State. It occurs throughout the country in this way. We find that these doctors are the ones who write the papers for medical journals, don't you see. They are getting lots of patients concentrated there. This we figured results from a better cooperation between the University and the doctor out in the rural areas. It is better hospital facilities. It is better roads getting those patients into these clinics.

That is one reason why we are concentrating these patients, we are seeing lots of them, in these centers. We have made a study that emphasizes this problem.

Mr. KILGORE. I am certainly familiar with the fact that they funnel in—I think it might be well to describe it—the hopeless cases from many areas.

Dr. RIGDON. Yes, diagnostic problems that the men don't have facilities to study. They send them in. We see them there, and it is an impressive thing. There is no two ways about it.

Mr. KILGORE. In addition to the matter of affording them medical treatment, it affords clinical information to the medical students.

Dr. RIGDON. Sure.

Mr. KILGORE. What is the source of financing for your studies?

Dr. RIGDON. The sources of financing are this. In 1952 I believe it was the American Tobacco Co. gave me money for 3 years. Since that time the United States Public Health Service and the Tobacco Industry Research Committee have given me money.

Now, let me say a word about how that is handled. That money comes to the University of Texas. I never see it. I draw on it, and when it is gone, it's gone, don't you see. I have to be responsible to the University of Texas for what is done. They send in a report on that, and I never do see the money. They fuss about the 15 percent overhead, you know, and they get that out of the grants. But I don't ever see it. So that's a very good point. It is a point that has caused considerable discussion because some people think it is a personal thing, that I get that money. I don't get the money. It is just exactly like any other pharmaceutical house gives me money. I put it in the business office and I draw on it.

That is the way the thing is handled.

Mr. MINSHALL. Will the gentleman yield? How much money is that? How much is involved?

Dr. RIGDON. It is just so little that I am ashamed to say. I got $7,500 from the American Tobacco Co. My grant for 3 years from the Tobacco Industry Research Committee is between $5,000 and $5,400, maybe. It is just in that area.

Mr. MINSHALL. What is the total amount that you received from them?

Dr. RIGDON. I have received 3 grants for 3 years from the tobacco industry research committee. I believe it is either 2 or 3. I have got a new one that starts the 1st of July, and I no longer get the money from the American Tobacco Co. I have been getting money from the United States Public Health Service for several years.

Mr. MINSHALL. I am just concerned with how much money you have gotten from the tobacco interests in total. What is your best estimate as to how much you have received?

Dr. RIGDON. I can tell you exactly; $7,500 for 3 years, plus $5,000 for 3 years.

Mr. MINSHALL. That is outright grants. Do they also give any fellowships out there?

Dr. RIGDON. No; I don't have any of that.

Mr. MINSHALL. That represents all of the money you received?

Dr. RIGDON. That is all the money I have received.

Mr. PLAPINGER. Is the Federal Government any more generous with you?

Dr. RIGDON. Well, the Federal Government gives me a grant. It is about $5,000 or $6,000. I hope we get one of about $10,000 this September to carry on some of my work, don't you see. So that is my budget, plus the money and the facilities that I have from the University of Texas, which is added to this.

Mr. KILGORE. Doctor, on a little bit of a different subject, earlier when you were relating the increased incidence, or we will say the incidence of lung cancer, to the number of hospital beds and the number of doctors in the various communities, it occurs to me that that might also be related in an almost direct line to population, with minor variations, from one area of the country to the other.

There would be, wouldn't there, a pretty strong correlation between the number of hospital beds and the number of doctors and the population?

Dr. RIGDON. That is not a very good correlation, don't you see, and that is why we are able to pick up this difference. In other words, in some of the Eastern States the ratio of hospital beds to population is different to what we have in Texas. So it is because of that that I can pick up that variation. Of course, you are aware of the fact that there have been many correlations made that go along with smoking. In 1925, it was automobiles. You correlated the cancer of the lung with automobiles. Then they said it is due to the tarring of the roads, and you can get a curve there. So it just depends upon what period of time you are in as to the correlation you use.

Mr. KILGORE. I think you mentioned that since 1939 the information contained on death certificates or the vital statistics with respect to the reports of deaths has contained a sufficiently detailed breakdown on the types of cancer that you can get a pretty fair picture on the incidence of lung cancer. Assuming that situation, has there been any marked increase in the incidence of lung cancer since 1939?

Dr. RIGDON. We are seeing a tremendous number of cases of cancer. But now the question is: Is it an actual increase or is it an apparent increase? That is the thing that bothers me.

Not having a baseline, don't you see, I have difficulty in answering that problem.

Mr. KILGORE. It may be that there is an increase, and it may not be.

Dr. RIGDON. It may be, and it may not be. I don't know any way to determine that. We just have to do the best that we can with what we have. But it does present that problem.

Mr. KILGORE. One other question. You were discussing the producing of skin cancer on these test animals after the trauma resulting from the plucking of the feathers; is it possible that the situation with respect to the relationship of smoking and lung cancer could be—I say is this possible, not is it probable—but is it possible that the existence at repeated intervals or at repeated times of the cancer-producing agent on the lung tissues might combine with some sort of an independent traumatic condition in the lung very similar to the situation on the skin to explain the correlation between smoking and lung cancer, making it a contributory rather than a causative relationship? Is that a possibility?

Dr. RIDGON. I think that that is a very definite possibility that should be explored. You are aware—or you may not be, not being familiar with this cancer literature—we have talked about influenza in the past. In 1919 they said, "We have got the answer to cancer of the lung, because it follows influenza," and that was the idea at that time. Preceding that, along about 1890, they said, "It is due to tuberculosis." You see, we were having lots of tuberculosis at that time. And you associate cancer of the lung with these things.

That is an illustration of a change in the tissue, because you do get changes in the epithelium of what we might call metaplasia as a result of infectious processes like influenza, and they felt that was the answer to the problem of lung cancer.

Silica was thought to be the cause of cancer of the lung; tuberculosis and influenza, aspirating the tar from the roads, and all those things have been one of the factors suggested.

So, some people have felt very strongly that it was the chronic inflammation associated with tuberculosis that was the basis for it. That would fit in with your idea, as I see it.

Mr. KILGORE. So, in general summary, your studies and your experiments have caused you to conclude that smoking may be a cause of lung cancer or it may not be?

Dr. RIGDON. That is right. We haven't got the data to warrant the conclusion. That is the way I see it.

Mr. KILGORE Thank you very much.

Mr. MINSHALL. In other words, you don't believe in statistics, but still you don't want to become one yourself, is that it, by refusing to test this out on your own skin?

Dr. RIGDON. That is a very interesting approach to it. I think, if that could be arranged through legislation, that we could use these people in mental institutions for such as this; they absolutely are not going to be able to return to society——

Mr. MINSHALL. I don't think you need any legislation. We out in Ohio have had volunteers in the Ohio Penitentiary who submitted

themselves to medical science. I think in this case it would be a very worthy sacrifice to make.

Dr. RIGDON. I agree with you 100 percent. If that could be encouraged, I think it would help us solve some of these problems, and we would know what is occurring in man and not mouse; don't you see?

Mr. MINSHAL. I think we are going to have some testimony later on, Doctor, before this committee that will show that these cancer-producing agents will produce, with regularity, cancer on the human skin. You said that in itself would be significant.

Dr. RIGDON. It certainly would. But now you want to be sure to keep straight the different carcinogenic agents that you are talking about. I was talking about methylcholanthrane. You may be talking about some other chemical.

Mr. MINSHALL. I am talking about the chemicals that are derived from cigarettes.

Dr. RIGDON. I have no information on that.

Mr. BLATNIK. Doctor, did you use any carcinogenic substances from tobacco tars in your tests?

Dr. RIGDON. Yes; I used the entire tar. I didn't try to break it down, because I am not in a position to break them down and get these different elements. I just used the whole tar.

Mr. BLATNIK. What were the results when you used the whole tar?

Dr. RIGDON. We haven't got any tumors. We haven't found any irritation in the trachea of the ducks any different to those that used only the vehicle, which was mineral oil.

Mr. BLATNIK. Did you try it on any other animals?

Dr. RIGDON. No. The duck is the only one. I have put it on the skin of chickens, and I don't get anything there. I discontinued that experiment. I haven't tried it on any other animals.

Mr. BLATNIK. Did you write any report on that?

Dr. RIGDON. No.

Mr. BLATNIK. What is your conclusion, on the basis of your observations?

Dr. RIGDON. That experiment is still in progress. I want to repeat it. It will be, I think, 6 months or a year before that piece of work would be completed, so I feel that I can publish it.

Mr. BLATNIK. What I am trying to find out is, is there anything in cigarettes that could be considered as a source of cancer or inducing cancer? Is there anything at all that is deleterious or harmful in cigarettes, is what I am driving at?

Dr. RIGDON. I am no chemist and I can't answer that. I can only say, upon what I have used in experiments that I have done, I haven't been able to get anything out. But from a chemical standpoint, I am not in a position to answer that. I just don't know. That is the reason.

Mr. BLATNIK. The reason we are interested is because there is a lot of money in the American economy going first to produce and to promote the filters, and secondly, to purchase them. A lot of money is involved in that alone. A lot of money is spent in advertising in implying that it does serve some function—perhaps for protecting, that it does something. I am trying to find out why the filters.

We have had two top witnesses—one for the tobacco industry, Dr. Little, who made a very fine presentation. But when it came to the subject of filters, he said, I forgot the exact language, but he indi-

cated he was not at all concerned about them either positively or negatively.

A lot of money is going then for the manufacture and the sale and the promotion and the consumption of something that a scientist of over 50 years' experience, says, according to his words, is of no consequence, either positively or negatively.

Today we had another scientist who feels that filters could be of consequence if made efficient enough that they would reduce the volume of smoke, and therefore the tars and nicotine; and that their statistics show—I am referring to Dr. Wynder's testimony of this morning—that those who smoke less are least inclined to get lung cancer. If they smoke more, they are more inclined to get lung cancer.

So you see, we are trying to find out some basis for it. It may be perfectly valid. We would like to find some basis for the claims that are being made for filter tips.

Dr. RIGDON. As I see that problem, if we filter out, let us say, some tobacco tar, some is going on through. We don't have any idea as to how much is necessary to produce cancer, even if it produces it, don't you see.

So suppose you filter out 50 percent. What are you doing? You don't know the answer.

Mr. BLATNIK. You say you have not been able to induce any cancer with the smoke containing the tars, without filters?

Dr. RIGDON. That's right.

Mr. BLATNIK. Without any filters?

Dr. RIGDON. That's right. This is just the tobacco tar.

Mr. BLATNIK. You gave it full, without any reduction in volume, getting the full dose of the tars and all the condensed substances out of cigarettes?

Dr. RIGDON. Yes. So I think we should establish whether we have got anything there before we get so involved in this thing. I don't know how we can get a quick answer to it. But that is a basic problem right there.

Then we should know how much is going to be necessary to filter out. What would we accomplish if we filtered out 50 percent and a person smoked 2 cigarettes instead of 1? He would get the same amount.

So we have got to know some of these basic things before we can get the answer over here, it looks to me, as I see it.

Mr. BLATNIK. This is more of a side issue, but I am curious. It is true, if I have some understanding of what you are saying on the reliability of these statistics, that a man smoking three packs a day may smoke a third of the cigarette. But don't you also think in terms of probabilities?

It is also likely that the man smoking one pack a day habitually smokes only a third of the cigarette. There is certainly probability—not specifically, you can't say that— that every man will do it.

Dr. RIGDON. That is right.

Mr. BLATNIK. But in general if a man who smokes 1 pack versus a man with 3 packs, there is some sort of a measure—not absolutely precise, but not too rough—that the man who smokes 1 pack will be smoking only one-third the cigarette like the man who smokes 3 packs, if you have a large group, or if your sampling is typical enough.

So to protect you, we don't know in either case, you see.

Dr. RIGDON. But what about the large number that smoke and don't get it, don't you see. How are you going to figure those in? That is the thing that bothers me. There are a tremendous number of people who smoke three packs and they don't develop cancer of the lung.

So that must be evaluated into this overall problem as well as those that smoke three packs and get it.

Mr. BLATNIK. Not to get into an argument, which I don't want, but as a discussion to help clarify my thinking: If I recollect correctly, Dr. Wynder was not saying that all who smoke heavily—there will be many who smoke heavily who will never get cancer. There are many things they don't know about it.

But he did say emphatically there will be a larger number of those getting cancer who smoke the heaviest—a direct correlation. The less you smoke, the smaller the number that will get it.

That was true of every group. It is true in the cities, it was true in the rural areas. It is true of every group if you tried a large enough sample.

Dr. RIGDON. I find this very interesting, on the next to the last page of the article that you have, to see many different diseases in which smoking is occurring. For instance, lots of people who have tuberculosis smoked, trauma, a high percentage of those smoke, and benign tumors of the skin, lots of these smoke.

I don't know how to interpret that.

Mr. PLAPINGER. How long did this study last, Doctor?

Dr. RIGDON. I started in in 1952, and it went to 1954, accumulating these data, compiled them.

Mr. PLAPINGER. You mentioned on the previous page a clinical diagnosis on 12,000 hospital and clinical patients has been correlated with their smoking history and that "This will be reported more fully in a future publication." Has that been done?

Dr. RIGDON. It hasn't been done. There is a manuscript right there.

Mr. PLAPINGER. Can you tell us something about your results?

Dr. RIGDON. We are finding that a higher percentage of the people smoke regardless of what they have. We found a higher percentage of those with cancer of the lung smoking. Our results were just exactly what the others had. But we also found such a large number that smoke and didn't have it until I have difficulty in evaluating that point, don't you see.

I don't know what it means. The only thing that I can see is that there are some other factors that are involved in those cases that we don't know. I think it would be important in evaluating the thing to learn something about that. That is the best I can do.

Mr. PLAPINGER. I think almost all of the witnesses who have indicated that tobacco is a factor, have conceded that there are other factors.

Dr. RIGDON. O. K. But the point is that it was alleged that 80 percent of the apparent increase in cancer of the lung was due to smoking. That was Doll's statement, and he got the ball rolling, don't you see. And that is the thing that has occurred there. It is the tremendous number of cancers of the lung which are alleged to be due to tobacco with which I can't go along on the basis of my knowledge of cancer and my knowledge of the statistical study.

Mr. PLAPINGER. Incidentally, you say that the frequency of cancer of the lung is based upon records obtained from death certificates.

Dr. Hammond yesterday citing his statistics, said that 79 percent were microscopically proven. He was addressing himself to this particular point.

Dr. RIGDON. That is a very good way. That is the only way we can get good data. It is a well-known thing that cancer of the lung is hard to diagnose. Dr. Weller, from the University of Michigan, has done an excellent piece of work. He died last year. He worked on this problem of cancer of the lung along in 1927 and 1928. So he was interested in it and had done an excellent piece of work and commented on the necessity of doing an autopsy, and a good autopsy, in order to establish the diagnosis.

He wouldn't even accept any of these diagnoses of lung cancer in 1927 if an autopsy had not been performed.

Gentlemen, at the time of death I have trouble making some of these diagnoses, to determine whether it is a primary tumor in the lung, or whether it has spread from some other point up to the lung.

Mr. PLAPINGER. I think Dr. Hammond conceded that point also, yesterday.

Are you familiar with the joint Report of the Study Group on Smoking and Health, Doctor? (See appendix, exhibit 6, p. 421.)

Dr. RIGDON. The only thing I know about it is what I have read in the paper.

Mr. PLAPINGER. Let me just cite the basis for their conclusions. They cite:

At least 16 independent studies carried on in 5 countries during the past 18 years have shown that there is a statistical association between smoking and occurrence of lung cancer.

They say also that—

epidemiological studies cannot always indicate that cigarette smoking cannot account for all cases of epidermoid cases of cancer of the lung.

There are other causes, the most important of which are probably various atmospheric pollutions; as in other diseases, various others such as sex, nutrition, and heredity may modify its occurrence.

Two prospective studies further suggest that cessation of smoking by chronic smokers decreases the probability that such individuals will develop lung cancer.

They say that the—

epidemiologic evidence is supported by laboratory studies on animals. At least five individual investigators have produced malignant neoplasms by tobacco-smoke condensates, and they say that studies on pathogenesis of human-lung cancer also are compatible with the causal relationship, and fluorescent substances present in cigarette smoke have been shown to enter the cells of the buccal mucosa.

And they finally conclude that—

the sum total of scientific evidence establishes beyond reasonable doubt that cigarette smoking is the causative factor in the rapidly increasing incidence of human epidermoid carcinoma of the lung.

This study was sponsored by the American Cancer Society, the American Heart Association, the National Cancer Institute, and the National Heart Institute and was the basis for the Surgeon General's conclusion.

To the untutored layman, that seems to be a pretty formidable array of evidence, and certainly is impressive. I just wondered whether you would care to comment on the basis for these conclusions by this joint study?

Dr. RIGDON. Yes; I would.

In the first place, in the United States Public Health Service there is a wide variation with regard to the role of cigarettes and cancer. You have certain ones that say it just can't occur, and others who say that it might be, but it hasn't been proven. So you have got a split in there from the people that I know in that organization.

So you can't disregard one group and say "Well the others are right, and we will base our opinion on that." I personally have tried to take all this work into consideration in arriving at my conclusion.

Secondly, there may be 5 studies, but I think it would be of value to check into it and see if this work on the mouse was confirmed and whether they got 50 or 75 percent cancers after treating the skin, or whether they got 1 mouse out of a group of 100 to produce a cancer.

I think that factor needs to be evaluated—just how much—and then I think they should evaluate how many people repeated this experiment on mice and didn't get the same results.

They took the positive approach to it, and everything is positive there and no comment on the negative side.

As I see it, they didn't even mention about what Schrek said. They put him in the group, and he is the man who is really going along with us on it. But they didn't quote him as saying if it is anything, it is certainly a poor agent.

Mr. PLAPINGER. Schrek is cited as a reference in the article. You are not suggesting that all of the evidence wasn't considered in arriving at this conclusion, are you?

Dr. RIGDON. I don't know what evidence they had. I am commenting on the fact that in that report they didn't bring out this other side of the situation. They just took the positive side and didn't discuss the negative side of the thing.

Had they come out with an equal study of the people who didn't agree with that, to me it would have been a much better report.

Second, I would like to comment on another point—

Mr. PLAPINGER. I understood you hadn't read this, Doctor.

Dr. RIGDON. But you read it to me.

Mr. PLAPINGER. But you didn't read the rest of the report. There is an appendix here which is some 3 or 4 pages long. They discuss a number of other studies.

Dr. RIGDON. That was published in Science, wasn't it?

Mr. PLAPINGER. That's right.

Dr. RIGDON. I saw it in Science. Your comment on this regression is a very interesting thing. That brings up a basic problem in the consideration of neoplasms, and that is this, that in experimental work—and this was done by Andivant here at the National Cancer Institute, he put a pellet of methylcholenthrane into the subcutaneous tissues of the mouse and let it stay in, some animals for a week and took it out, and in others let it stay for 2 weeks and took it out.

Then after a month, they took it out at other mice. What he found was that after it stays in there for a period of time, although you remove it, the animal goes ahead and produces a cancer.

So it is an interesting point to me that if the mechanism of cancer due to carcinogen as alleged in tobacco acts differently because some say that if they cut down on the number, or if you stop smoking, the number of cancers will likewise decrease. This is different to the work on other cancer-producing agents, methylcholenthrane specifically. This is a very interesting point, I think.

I don't know how that fits into this report.

Mr. PLAPINGER. In citing the various factors here leading up to their conclusion of the evidence that they have accumulated, they go on to say, as you may recall, there are a number of areas for a number of questions that are unresolved on which research is continuing.

Dr. RIGDON. Yes.

Mr. BLATNIK. Thank you very much, Dr. Rigdon.

The hearings will be continued on Tuesday morning at 10 o'clock in the same room, so for today the committee is adjourned.

(Whereupon, at 3:25 p. m., the committee adjourned, to reconvene at 10 a. m. Tuesday, July 23, 1957.)

FALSE AND MISLEADING ADVERTISING

(Filter-Tip Cigarettes)

TUESDAY, JULY 23, 1957

HOUSE OF REPRESENTATIVES,
LEGAL AND MONETARY AFFAIRS SUBCOMMITTEE
OF THE COMMITTEE ON GOVERNMENT OPERATIONS,
Washington, D. C.

The subcommittee met, pursuant to recess, at 10 a. m., in room 100, George Washington Inn, Hon. John A. Blatnik (chairman) presiding.

Present: Representatives Blatnik, Mrs. Griffiths, Meader, and Minshall.

Also present: Jerome S. Plapinger, subcommittee counsel; Curtis E. Johnson, staff director; and Elizabeth D. Heater, clerk.

Mr. BLATNIK. The subcommittee on Legal and Monetary Affairs of the House Committee on Government Operations will continue with public hearings on the role of Federal Government agencies in the effective administration and enforcement of law and regulations pertaining to false and misleading advertising.

This morning we have for the first time the Government witnesses, we have our two top medical authorities.

We welcome you, Dr. Burney and Dr. Heller.

We have Dr. LeRoy Burney, the Surgeon General of the Public Health Service, Department of Health, Education, and Welfare, and Dr. John R. Heller, Director of the National Cancer Institute.

The first two hearings held on Thursday and Friday of last week, Doctor, we heard witnesses, pro and con, from the professional world, the object being not to go into the medical problem with a view of trying to make a medical determination. That is neither within the competence of the committee nor within its legal jurisdiction.

However, we do feel it is essential and vital that we have, although in somewhat summary and general terms, a complete statement on the pros and cons of the problem of the effect on the health of the public.

To do that more correctly, and in better perspective, we felt we should have a background of the latest available medical opinion on the subject, and against that we can get the true relative proportions of the nature of the problem and whether or not there is a Federal responsibility for you men in the executive offices, and for us in Congress in the legislative field, to do something about it.

We will first hear Surgeon General LeRoy E. Burney.

Dr. Burney, we welcome you this morning. Will you please give us a brief introduction, which I am sure is well known to most of us, but for the record a brief summary statement of your background and your record of experience, and then proceed with your statement.

STATEMENT OF SURG. GEN. LeROY E. BURNEY, PUBLIC HEALTH SERVICE, DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Dr. BURNEY. Thank you, Mr. Chairman, and members of the subcommittee.

I was appointed Surgeon General about 1 year ago. My previous experience has been almost entirely in the United States Public Health Service. I have been a career officer in the Commissioner Corps for 26 years, coming into the Public Health Service in 1932, although I interned in the service in 1930.

Following that I had a year at Johns Hopkins University in the School of Hygiene and Public Health, and then came into the regular corps of the Service.

I served in various assignments since then in the Public Health Service, and was detailed to Indiana as a State health commissioner of that State for 9 years. I came back to Washington in 1954, as an assistant surgeon general, deputy chief of the Bureau of State Services, and, as I say, last August was appointed by President Eisenhower as the Surgeon General.

With your permission, Mr. Chairman, I would like to read this very brief statement, and will certainly be very pleased, together with Dr. Heller, who is much more of an expert in this area than I am, to answer any questions which members of the committee might have.

The Public Health Service, Department of Health, Education, and Welfare, has participated actively in research on lung cancer since the establishment of the National Cancer Institute in 1937. This participation has included research at the Institutes, grant-supported research, in institutions outside the Federal Government, and close administrative and informational liaison with organizations and with investigators concerned with the problem.

Up to 1952, emphasis was placed on laboratory investigations on the genetic and other factors in lung tumors of mice and other animals, the histogenesis of lung tumors, and environmental carcinogenesis including the search for carcinogens in atmospheric dusts and in tobacco. A recent selected bibliography of contributions by the staff lists over 75 titles.

Since 1952, the emphasis has turned toward epidemiological investigations. One such retrospective study by the staff on smoking and cancer was published in 1953. Another one, supported in part by a grant, was published in 1954. A national survey of tobacco-smoking habits in the United States was conducted with the Bureau of the Census and published in 1956.

Mr. PLAPINGER. May we have a copy of that for the record, please?

Dr. BURNEY. I will be happy to supply you with a copy.

(See appendix, exhibit 7, p. 431.)

Dr. BURNEY. Several statistical analyses of available data on the subject were also prepared.

In light of these and other studies the Public Health Service stated on July 12 (see appendix, exhibit 8, p. 554) that it is clear there is an increasing and consistent body of evidence that excessive cigarette smoking is one of the causative factors in lung cancer. At the same

time, we expressed our belief that heavy and prolonged cigarette smoking is not the only cause of lung cancer.

Three major investigations on the relation between smoking and lung cancer are now in progress by the Public Health Service staff. The first is a large study in collaboration with the Veterans' Administration of the smoking habits of 220,000 veterans of World War I. The second is a national retrospective analysis of lung cancer in women, and its relation to smoking. The third is a national survey on the relation of occupation and lifetime residence, as well as smoking, to lung cancer. The method for this study has been tested in Pennsylvania and found to be feasible.

Since 1952, the Public Health Service has not undertaken in its own facilities direct laboratory studies on tobacco. Close liaison with the American Cancer Society and many other individual investigators has led to the judgment that such studies are being pursued with sufficient resources, and that additional expansion is not required.

Research work on tobacco products and on filters at present is being supported by a number of industrial concerns. A number of requests for grants also have been made to the American Cancer Society.

Insofar as cigarette filters are concerned, research data available to the Public Health Service are insufficient to warrant a conclusion at this time.

Modification of cigarettes so that their harmful effects would be reduced is of obvious importance to public health. It has been proposed, theoretically, that such modifications could be achieved in three ways: (1) by changing the tobacco leaf and other initial constituents of the cigarette, (2) by reducing the temperature of the burning cigarette, and (3) by the removal of certain constituents from the smoke. The last method, of filtration, preferably should remove harmful materials from the smoke and retain those materials that provide smoking pleasure.

It is apparent that further research, both on the general question of tobacco and its relationship to lung cancer, and on the question of filtration or other means of modifying cigarettes, is needed. The Public Health Service is prepared, of course, to play its part in this research, of importance to industry, to our economy, and to the health needs of the people of the Nation.

Thank you, Mr. Chairman.

Mr. BLATNIK. Thank you, Dr. Burney.

Dr. Burney, Dr. Heller's testimony will tie in and give the scientific details to support the general statement you have made?

Dr. BURNEY. Yes, sir.

Mr. BLATNIK. I suggest you proceed right on with the statement and then we can come back with our questions.

Dr. BURNEY. That will be fine, Mr. Chairman.

Mr. BLATNIK. Dr. Heller, we welcome you to the committee this morning. Would you give us for the record a short biographical background of your professional background experience and then proceed?

Do you have a written statement, Doctor?

STATEMENT OF DR. JOHN R. HELLER, DIRECTOR, NATIONAL CANCER INSTITUTE

Dr. HELLER. I do not, Mr. Chairman. I am prepared to answer any particular questions you or the members of the committee may have and provide any additional information you or others may wish.

Mr. Chairman, I am, like Dr. Burney, a career medical officer in the Commissioned Corps of the Public Health Service. I have been in the Public Health Service approximately 26 years. During that time my activities have been primarily in the field of preventive medicine and public health. During the war I was Chief of the Venereal Diseases Division, which played a very active role in a very active program.

In 1948 I was appointed Director of the National Cancer Institute and have served in that capacity since, up to the present.

I am also an Assistant Surgeon General in the Public Health Service and have been primarily interested in medical research administration.

Mr. Chairman, I might amplify, for the information of the committee, one point which Dr. Burney mentioned; namely, the prospective study among veterans of World War I, upon which no report has been made. This study, in my judgment, is a very important one and one which should elucidate many angles of this problem generally.

Mr. PLAPINGER. This is still a statistical study?

Dr. HELLER. It is a statistical study; that is correct. It is prospective in the sense that we take a group of people and follow them to find what happens to them from the standpoint of health.

There were approximately 290,000 men of World War I who had national service life insurance. Questionnaires were furnished them through the cooperation of the Veterans' Administration. Part of the questionnaire incorporated smoking habits of this group.

The questionnaire had other features, such as occupation and other elements which probably should be of usefulness and value in consideration of their health history.

The reason that recipients of national service life insurance were included is that when they die, someone claims the insurance. We have knowledge of the death fairly promptly. Frequently they die in veterans' facilities and frequently post mortem examinations are performed. We, therefore, have a fairly accurate medical background upon which to append an analysis of these cases.

These individuals, of course, at this age group are dying at an almost predictable rate, something on the order of perhaps 5,000 per year, with an increasing increment each year.

This study has been progressing now about 3 years, or 3-plus years. We have made some preliminary checks and the data which are derived from this study would seem, in general, to parallel other prospective studies for men of this age group.

The importance of this study is that these individuals are spread over the United States geographically and represent a variety of occupations. Of the 290,000, some 260,000 answered the questionnaires and we have now about 220,000 whom we are studying because their questionnaires are complete and otherwise statistically significant.

We hope to make a preliminary report on these data, these studies, probably next spring. This should give us considerable insight into

the problems generally of smoking and health, among other things. It may give us some additional insight as to the types of individuals who die from whatever diseases, as correlated with their smoking habits.

This study, gentlemen, is another, but probably the best prospective study, and one upon which we are depending to give us additional information to enable us to understand better and more intelligently the entire problem so that additional programs might be implemented, if necessary.

Dr. Burney has indicated the statement he presented on July 12, which reflects the best judgment that we of the National Cancer Institute and others of the Public Health Service are capable of rendering.

We believe that this problem of smoking, particularly excessive cigarette smoking, is one which needs to be brought to the attention of the American public as being a health hazard, the magnitude of which we are not completely prepared to state at this time.

Previous testimony before this group would indicate that it is enough, certainly, to direct our attention to the problem, and for us as health people to undertake every action that we know to take in order to find out more about the problem leading toward possible remedial action.

Mr. Chairman, I believe that is about all I have to comment upon at this particular time, since my role was that of being in a position to assist in answering questions, or present any additional information which you or your committee members may wish to have.

Mr. BLATNIK. Thank you, Dr. Heller.

Doctor, did you give this statistical study which you have undertaken with the veterans of the First World War—there is a larger group than Dr. Hammond has in his statistical groupings. His was about 186,000 and yours is about 260,000.

Dr. HELLER. Mr. Chairman, that is correct. Dr. Hammond said it was something like 186,000 or 188,000. Our studies from a statistical viewpoint will consist of better than 220,000, although we have received replies from 260,000. However, we had to eliminate some as being not desirable to include in the study because of insufficient information or data which otherwise would render them statistically invalid, so that our study will represent 220,000, but is pretty well distributed over the United States.

The Cancer Society prospective study is an excellent study, and reflects a statistical sample of 9 States involving 394 counties. I wouldn't say this isn't statistically valid. I believe that it is. Our study in which we are very much interested, and which we believe will be significant, is a larger national study and comprises the entire country, and we believe that its real significance will be the additional number of post mortem examinations we will have which will completely define the diagnosis which is made. However, the Cancer Society study did have a very good sample of post mortem examinations to indicate that correct diagnoses were being made and, therefore, could be established as being statistically valid.

Mr. BLATNIK. You feel that these statistical studies do have merit, and are positive in their findings? The reason I ask that question is that it was indicated by some of the medical witnesses last week that Dr. Hammond's study was merely a statistical study, and gave no

proof of smoking being a direct causative factor in the inducement of lung cancer.

Dr. HELLER. Mr. Chairman, I believe that statistical studies are worth while in the hands of good statisticians, in the hands of competent people, who interpret the studies. They are tools in the hands of the public health administrator and epidemiologists, and have proved exceedingly valuable in the assessment and management and control of other diseases, such as polio, syphilis, tuberculosis, and other diseases which for the most part are fairly well under control.

We therefore believe that such statistical studies are extremely valuable and extremely significant, and it is our belief that on the basis of the studies which have been presented, and of which you have information from testimony of others, these studies reflect a situation of which we must take cognizance, and which considered in connection with laboratory studies, brought about the statement the Surgeon General made on July 12, 1957.

Mr. BLATNIK. Doctor, as head of a very important Government health agency dealing directly and almost exclusively with cancer, its origin, nature, and possible cure, what would be your official recommendation, your official thinking on tobacco as a source of cancer-inducing substances?

Dr. HELLER. Mr. Chairman, it is our belief, on the basis of the information which is available to us—and we have indicated a mounting evidence that when cigarette tobacco is burned (pyrolysis is the technical name) at about 800° Centigrade, we believe there is a chemical change in certain hydrocarbons in tobacco which produces certain cancer-causing compounds. We do not know which of these compound is the culprit. We do not know whether it is a combination of these compounds, but we believe from the best knowledge that we have at this time that the cancer-causing properties or agents are in the tar fraction in smoke condensates. There is reason to believe that these are some of the higher polyclic, many ringed, hydrocarbons, which are very complicated hydrocarbons, and about which we need to know more.

At present much research is going on in several parts of the world, and in our own country, on these very important and very complicated chemicals, and we hope that they can be chemically characterized, they can be identified more precisely, and their role or possible role in the production of factors in the causation of lung-cancer can be more precisely defined.

It may be that there are combinations of such compounds, it may be that some promoting factor which interacts with another substance or compound is responsible.

These are some of the things, Mr. Chairman, that we believe must be established before we can precisely put our finger upon all of the compounds.

We do believe, however, that at this juncture enough is known and enough has been demonstrated from laboratory and from epidemiologic investigations that we, as Federal health officials, should direct to the public information concerning these compounds and rely upon the laboratory, rely upon other investigators throughout the country, and, in fact, throughout the world, for additional information in order that we may be better prepared to know what are the next steps that might be taken.

Mr. BLATNIK. How would you reach the public to disseminate information, especially when you have the competition of this terrific advertising encouraging all people to smoke? If you are fat, smoke and you will reduce. If you are weak and lethargic, smoke and it will pep you up and you can keep up with the athletes.

Dr. HELLER. With your permission, Dr. Burney can speak on that.

Dr. BURNEY. Thank you, Dr. Heller.

Mr. BLATNIK. Dr. Burney, please.

Dr. BURNEY. I don't know, Mr. Chairman, that we are competent to answer that particular question. We are primarily a Federal health agency; naturally, we have a responsibility in health education, in informing the public as to health facts which influence their own individual health needs. That is the reason we felt a responsibility to put this statement out at this time.

As Dr. Heller said, we felt that, as a result of the research we had done, and that others had done, at this time there was increasing and consistent evidence that this excessive and prolonged cigarette smoking was one of the causative factors.

Now, we, as you know, have sent out this statement together with the backup information to the State and Territorial health officers, and to the American Medical Association, suggesting they pass that information on to their State medical societies and local medical societies and local health departments.

We recognize that this may not be sufficient. On the other hand, the facts have been furnished to the public through the press, as a result of this statement, together with the facts brought out in your hearings, and the State and Territorial health officers have definite responsibilities relative to public information and health education.

I would agree with Dr. Heller that our position at this time, I think, relative to this whole matter, is that there is a lot that is unknown in this area. It is a controversial area in which some well-known scientists have opinions which differ from some of the other people whom you have heard in these hearings.

Our position is that we have informed the public through the excellent coverage of the press, radio, and TV. We have informed the official health agencies in the States who are responsible for this area, and we have informed the American Medical Association, recognizing that many people will go to their own physicians for advice.

We believe that is as far as we should go at this time until and when we have additional information. If, for example, next spring we have some definite findings coming from this survey, then I think we have an obligation and responsibility to make those facts known, and any additional information that may come out.

Mr. BLATNIK. Doctor, the British, we understand, are circulating posters through local health authorities, samples of which are included in the July 26 issue of U. S. News & World Report, accompanying your interview, Dr. Heller, on the truth about smoking and cancer. (See appendix, exhibit 9, p. 558.) Would that be a proper function of the Government, in view of the concern expressed here as to the seriousness of smoking?

Dr. BURNEY. I don't believe so, Mr. Chairman. In the first place, the British Ministry of Health is a centralized organization. In other words, the district health departments are really branches of the Minis-

try of Health, whereas in this country the States are sovereign in matters of health, and we have certain interstate responsibilities, but are not the directors or administrators of State and local health departments; so, there is that difference.

In other words, when the British Ministry of Health prepares posters and sends them out to their district health officers, they are sending those out to their own employees over whom they have administration. We have never in any of our public health programs, whether it is the Salk program or the fluoridation program, prepared that type of poster, and so forth, to send to the States for distribution. We have sometimes helped them in the preparation of material, but they, in turn, do their printing and put their own byline on it and that sort of thing.

Mr. BLATNIK. Would, perhaps, labeling on the package containing the articles itself help? For example, I notice rather innocuous substances, such as "low salt content," in articles purchased for saltless diet, and I believe the bottle or the product is required to be labeled with a complete description saying there is a salt content of a certain figure. With regard to beer, they indicate 3.2 percent alcohol.

Now, why are they compelled to label the contents of small amounts of things that are not really harmful? Do you think there is any validity to the recommendation that, perhaps, you ought to have a label on a cigarette package saying the total content does not exceed 17 milligrams of tar, or so much nicotine? Would that help in passing on information to the people? If it is a filter-tip cigarette, should it contain a label saying, "This filter removes 10 percent of the tars, or removes 20 or 30 percent," so the people have some idea of how much protection or how much filtration they are getting from the filtered cigarette? Would something like that, do you think, be a sound suggestion?

Dr. BURNEY. We, as you know, do not have responsibility for labeling, and that sort of thing, but if the Federal Trade Commission—in answer to your question, my thought would be, and Dr. Heller is able to add or subtract to this—we do not have sufficient evidence at this time in the identification of the agent, and the relationship of that agent to, perhaps, cofactors, whether it is air pollution or what else it might be, as the causative factor in this. In other words, even though I am firmly convinced that we were right in making this statement at this time, and in letting the public know that, in our opinion, we believe there is a relationship between excessive and prolonged cigarette smoking, I do not believe that we would be on firm ground in recommending such a warning sign at this time until we have much more specific information.

Mr. PLAPINGER. This wouldn't be a warning, Doctor; this would merely be a statement that "this cigarette contains blank milligrams of tar and blank milligrams of nicotine." What would be your objection there?

Dr. BURNEY. I, personally, wouldn't see any objection to that.

Mr. BLATNIK. I notice along the same line, Doctor, many medicines for the common cold; many of them will have a label on them saying, "You take 1 tablet every 4 hours, not to exceed 6 in any 36-hour period." That is just an example. "One every 4 hours, not to exceed 6 in every 36 hours." Suppose you had a pack which said, "Not to exceed 18 or 15 in any 24-hour period," as a threshold? How will the people get these facts unless you have them printed on the package?

Dr. BURNEY. I am not sure too many of us read too much of the fine print on anything when we buy it, as far as that is concerned. Also, I think there is a little difference between taking a medicine for a periodic ailment as contrasted to the matter of smoking, which most of us enjoy and which we do routinely. I enjoy smoking myself, although I smoke a pipe and have for about 15 years. I think there is a little bit of difference, Mr. Chairman, between labeling pills or other medicines, which we take occasionally for something, as contrasted to this. I would question whether too many people would read the fine print.

Mr. BLATNIK. Doctor, getting back to the main thing that concerns me, Dr. Little, speaking in opposition, more or less, on behalf of the tobacco industry—and we will have to get his exact quote, but, in essence, he said there was not only no evidence proving there was any substance in the cigarette, or in the smoke of a cigarette, that would induce or tend to induce cancer, but neither was there anything in the smoke of the cigarette which might be considered harmful to the body. That is his general position.

Then he followed up in answer to my question, "Then why the filters?" He said he doesn't know; he is not interested in filters, and regards them as a thing so inconsequential that they are of no concern at all. That is his position. He is a scientist, a medical man, an adviser to the tobacco industry.

What we are trying to find out, and we hope to do it before the week is over, is: Why the filters? If we accept your statement, that there is a rather substantial significance to the statistical relationship between heavy smoking and prolonged smoking, and the high incidence of deaths in lung cancer, if that is true, then how effective are the filters? How effective are the filters? What do they filter? What do they remove, and how much do they remove?

What I am trying to get to, Doctor, What is the responsibility that falls upon those of us now representing the Government—you in the executive agencies, we in the legislative branch, in protecting the health of the people and discharging our responsibilities in protecting the health of the people, in this case from false or misleading advertising, that encourages them to smoke filter-tip cigarettes, that gives them some notion that they are being in some way protected or that something is being filtered. What is our responsibility, or is there any Federal responsibility there?

Mr. BURNEY. May I refer that to Dr. Heller, Mr. Chairman?

Mr. BLATNIK. Dr. Heller.

Dr. HELLER. Mr. Chairman, as indicated in Dr. Burney's opening statement, we have not ourselves undertaken any definitive research in filters. It has been our viewpoint that there is a desirability in elucidating the fundamental problems in this question of excessive smoking and lung cancer, which by no means depreciates the possible importance of filters.

We do not know what in the tars cause, or represent one of the causes, in the producing of lung cancer. Filters, for the most part, are made up of cellulose acetate, and, as I am informed the tightness of the filter is the "gimmick" involved in filtering out the smoke. Obviously, since the tars, or the condensates from the smoke contain the material believed to be carcinogenic, anything that filters or strains

these tars would prevent this material from getting down into the bronchi, or the tubes of the lungs.

Now, the original filters, I believe, were found to filter out somewhere in the area of 40 to 50 percent of the smoke. I am informed that the company that originated this filter found that the cigarette was not as acceptable to individuals as they would hope because something was wrong with the taste. They missed something. Therefore, as I understand it, there was a loosening up of the filters; the manufacture was so adapted that smoke could get through more freely, and tars and smoke condensates would go through.

I am informed at present that filters will eliminate something on the order of 15 to 20 percent of the smoke and, therefore, a proportionate amount of tar.

It has also been stated, as you have heard from previous witnesses, that in order to compensate for the possible loss of taste and the loss of effectiveness of cigarettes, that some different types of tobacco were introduced, and in some instances, that result was an increase in amounts of tar as compared with that originally obtained in nonfilter cigarettes.

I do not know whether or not this is true. We have not done this work in our own laboratories, but I believe the work has been done by responsible individuals under circumstances which would lead one to believe the work is good.

We do not know, Mr. Chairman, that any filter can selectively sort out that component or components in the tars which are responsible.

As I related earlier, there may be a promoting factor involved which along with the tars is responsible, or partially responsible, for lung cancer in certain individuals. Therefore, as far as my knowledge goes, I would say that the important thing is to find out what is in the tobacco to chemically characterize those constituents which we believe to be incriminated. If it is possible to filter them out, I think it is perfectly acceptable and proper. I do not know what is involved from the advertising viewpoint, where filters are concerned. I do not know the claims of companies for the virtues of filters, whether it provides a cooler smoke and that sort of thing.

But I do know that we are intent, as scientists, upon obtaining the fundamental basic information concerning those things which cause or tend to cause lung cancers. Then the proper preventive steps can be taken, and we hope soon. We have no desire whatever to join the antitobacco league. As far as we are concerned, if people enjoy smoking, and can do it with little or no hazard, that is perfectly fine. We do feel, however, that as long as there is an agent which is possibly causing lung cancer, or is one of the causative factors, we should undertake studies and to assist in supporting studies which will be directed toward finding out these compounds, or the compound involved in this process.

Mr. Chairman, I am not sure your question can be completely, directly, and thoroughly answered, but that represents a reasonable attempt on our part.

Mr. BLATNIK. You would, then, recommend further research on the part of the Government to find out more about what substances in the cigarette cause cancer, and perhaps how that substance may be removed or reduced to protect the people?

Dr. HELLER. By Government, Mr. Chairman, by others who are interested, and by Government support of research, which as you know is one of our traditional roles.

Mr. BLATNIK. How much is the Federal Government spending, itself, either directly or through grants to other independent research groups, or universities in the form of grants for this cancer research?

Dr. HELLER. Mr. Chairman, I am unable to give you that figure. If you will allow me to, I will insert it in the record.

Mr. PLAPINGER. May we have that by year, Dr. Heller?

Dr. HELLER. Yes.

Mr. PLAPINGER. May we have the names of the grantees?

Dr. HELLER. Yes. Would you like it for the last 5-year period?

Mr. PLAPINGER. That would be fine if it is not too much trouble.

(See appendix, exhibit 10, p. 572.)

Dr. BURNEY. These would include other causative agents that might produce cancer or lung cancer.

Mr. BLATNIK. Are other governments concerned about excessive smoking as a possible cause or source of cancer? Could you name the governments, give us a brief idea of what they are doing?

Dr. HELLER. Yes, Mr. Chairman. We have knowledge of studies on the part of individuals in nine different countries, such countries as England, Finland, Sweden, the Netherlands, France, and Italy. Four governments now have put out a national statement concerning their alarm, or I should say presenting information to the public concerning this question, and their alarm over the causative factors involved in excessive smoking. These four countries are Great Britain, Sweden, the Netherlands, and our own.

Great Britain, I believe, was the first to come out in about 1954 with a statement. Only recently the Medical Research Council of Britain, a very distinguished and authoritative body, has come out with a more definitive attitude toward this entire question, and this has resulted in the action on the part of the Ministry of Health to which you referred earlier.

The Netherlands has put out an official statement saying they believe that excessive cigarette smoking should be brought to the attention of the public and individuals should be guided accordingly.

Sweden has put ont a similar declaration. To the best of my knowledge, no other countries have come out with an official document, or a statement which reflects official attitude.

The work that is being done, Mr. Chairman, in other parts of the world to some extent parallels that going on in this country. They have approached the problem of the constituents of tobacco tars in a slightly different manner. In France, for example, Dr. Latarjet and his associates in Paris have been primarily concerned with how much benzpyrene may be formed from the burning of cigarette paper.

Now, Mr. Rand, and his associates in Cleveland, in this country, have been similarly preoccupied.

The group in England had been concerned with air pollution, with the correlation between excessive smoking and individuals subjected to heavy pollutants in the atmosphere. They also have been working on 3,4-benzpyrene in tobacco tars. Their results, in general, confirm that of Dr. Wynder and his associates in this country, and also those of Arthur D. Little in Boston.

There are 4 or 5 individuals in this country who have been working in this area, and we can expect in the future that additional good work will originate from these areas.

I would say, Mr. Chairman, and members of the committee, that one of the very hopeful things about this problem is the willingness of the investigators throughout the world to share quickly their information with us, and we in turn share with them. They recognize that this is a problem which is international in scope. As a matter of fact, the incidence of lung cancer in England is greater than in this country, and it is similarly true of certain other countries in Europe.

Norway, for example, I am informed, has much less lung cancer, as has Sweden and Iceland practically no lung cancer at all. Attempts are being made to correlate the smoking habits of the population of these countries with the occurrence of lung cancer, to determine if there are factors there of which we have no knowledge in this country.

We believe that the pooling of information which we get from investigators throughout the world who are proceeding along lines which are believed by our scientists generally to be proper and productive, and we believe the immediate availability of these data will enable us to move much faster in meeting this problem from a preventive medicine standpoint.

Mr. BLATNIK. I have this last question: Doctor, just what is being done in America, then, to protect the people or inform them on the possible dangers from excessive smoking or just smoking?

Dr. HELLER. There are several things being done, Mr. Chairman. One, as Dr. Burney pointed out, officially, there have been furnished to State health authorities for transmission down to local health departments in some three thousand-odd counties, information indicating our official concern over this problem.

Similarly, the American Medical Association, the spokesman for organized medicine, will pass this information down to local individuals.

Newspapers, radio, TV, and other media have done an excellent job covering this problem, and a very objective job. This is an exceedingly valuable way of informing the public. There has been an interpretation of the scientific literature by us, insofar as possible, by other interested agencies and groups, and there have been many discussions and additional discussions of planning in the future, of ways which are dignified and proper, that would not be productive of a scare to the public, and yet be consistent with the facts that we have.

We wish to proceed in an orderly, dignified, and proper manner in order to present the facts as they become available to us, to the public.

Mr. BLATNIK. Have all those things been done to help inform or help protect the public from the possible dangers of smoking? Again I come back to what puzzles me most and that is that no one seems to know anything about the filters. The scientists representing industry, their own people—don't know. We are spending, we will find out, literally millions of dollars to promote that one filter, and I will venture to say, sight unseen, and I am open to correction—this is just a statement and I shall seek that information, I venture to say that in our country as a whole, more is being spent just to promote the smoking of filter-tipped cigarettes than is being done to find either the cause of cancer, or how much that filtration protects. I can't understand why such a large expenditure is being carried out with no purpose.

Here we have great difficulty in a congressional committee to find out why there are filters. If we don't know, how many people don't know?

Dr. Little told us right here last Thursday, on the matter of filters, he said it was a matter of no interest to him. It might be of no interest to him, and he might have good reasons for saying that, but we would like to know where we can find this out.

Could you advise us, Doctor? I know it is a little out of the field of your medical research.

Dr. HELLER. Mr. Chairman, this, of course, comes completely into the field of developmental, industrial research. We know that physically the filter simply removes certain constituents of the smoke. Now, whether or not it can do a complete job, we hope it can, if our plans continue to hold up as they have. Whether or not there are better filters that can be made, whether or not industry proposes to undertake additional work on filters, we do not know. I do know that individuals such as Dr. Wynder and others, some of whom are not known to me, are working on this problem. They are fitted by training, by interest, to undertake this sort of research and we are keenly interested in it.

I, personally, Mr. Chairman, propose to go into this much more exhaustively and completely than we have in the past.

May I repeat again it has been our belief that it is eminently desirable for us to elucidate the basic mechanism involved here and not necessarily introduce another variable. If we knew all of the constituents, if we could identify the particular tar or tars, then we could turn to such factors as filters, or washing of tobacco to remove wax before the cigarette is made, or whatever the remedial measures that should be instituted might be. But, since we do not know those things, we believe that it is desirable to find out all we possibly can about these other factors and then see where filters fit.

After all, filters are merely mechanical strainers and, as far as my knowledge goes, there is no chemical action involved. Therefore, it is developmental research.

Mr. BLATNIK. Doctor, does the Federal Trade Commission seek medical information from either you or, perhaps, from the Surgeon General in their analyses and evaluation of the propriety and correctness of cigarette smoking? Could you give us some idea on that?

Dr. HELLER. Yes, Mr. Chairman.

The Chief of the Division of Consultation of the Federal Trade Commission has been in constant touch with me concerning this item. He is very anxious to know what the Surgeon General's attitude is, what the Surgeon General's pronouncements are. I have been in touch with him. We have kept him informed. He has been, indeed, most cooperative and helpful and since this problem came to his and our attention, has indicated a desire to work closely with us, and to be guided by the medical facts which are developed and the medical opinions which may be derived from the scientific endeavors generally. I can only indicate our extreme pleasure at cooperation with this agency of the Federal Government.

Mr. BLATNIK. Thank you very much.

Mr. Meader?

Mr. MEADER. As the chairman mentioned, we had Dr. Little before the committee last week. I don't know whether you are familiar with his testimony originally given before this committee.

Dr. HELLER. Yes, I am.

Mr. MEADER. I would like to refer to a few passages from his testimony.

On page 2 of his prepared statement he said:

After 50 years of research on the origin and nature of cancer, I have the greatest respect for its vigor, versatility, and complexity. I therefore sincerely and deeply deplore premature and oversimplified conclusions and intensive publicity.

Then on page 6 of his statement he refers to the study of Dr. Hammond and says:

A positive association is claimed by the American Cancer Society through the Hammond-Horn Study of 1957 between excessive smoking and death from cancer of the lung.

And then he lists a whole lot of other organs of the body and diseases that are associated with excessive smoking.

On page 7 he makes this comment:

It is a difficult if not impossible scientific gymnatic feat to imagine a cause-and-effect relationship in this overall mixture of human ailments, especially with a strikingly increased life span of our population, and increased national tobacco consumption staring us in the face.

Then on page 64 of the committee's transcript, Dr. Little testified:

To establish a cause-and-effect relationship on statistical association without the experimental evidence is not safe. It cannot be done. You may get an indication of something to look for, but to say that the case is finished, the evidence is all in, and that you can satisfy experimental scientists, all of them, that is not possible because too many of us have seen too many statistical relationships which have not helped the cause-and-effect relationship.

In the statement presented to the committee this morning, I quote from page 1—I guess the statement is in the name of Dr. Burney, the last paragraph:

There is an increasing and consistent body of evidence that excessive cigarette smoking is one of the causative factors in lung cancer.

Now it strikes me the comments I have quoted from Dr. Little and the comment I have just now quoted from your prepared statement seem to be in conflict. You say there is a causative relationship that is established by the evidence and Dr. Little says it is dangerous to take the statistical associations and from them deduce a cause-and-effect relationship.

I would like your comment on what appears to be a conflict.

Dr. HELLER. Certainly, Mr. Meader.

Dr. Little is a distinguished and beloved scientist of the Nation. I think, perhaps, you may be aware of his accomplishments in the field of genetics. Dr. Little is indeed a very fine gentleman, both as a man and as a scientist. But there are differences in interpretation among scientists as perhaps you are well aware.

Dr. Little is sincerely of the opinion that these data do not warrant the inferences which we have indicated.

We do believe, however, Mr. Meader, that while Dr. Little is entitled to his interpretation and we respect him, still we feel on the basis of our background, our knowledge, our experience, and our information, that our stand is a proper one for us to take. We feel

impelled to, after much study, and we believe that we respectfully disagree with Dr. Little in this matter.

Mr. MEADER. I believe you do say, after saying that excessive cigarette smoking is one of the causative factors in lung cancer—your next sentence says:

> At the same time we expressed our belief that heavy and prolonged cigarette smoking is not the only cause of lung cancer.

Let me see if this isn't a reconciliation of what apparently is a conflict between the position you take, and Dr. Wynder, and Dr. Hammond, and so on, with that of Dr. Little.

He asserts that there has been no logical demonstration of a causative agent in the tobacco or in cigarette smoking which produces cancer, and I believe that is conceded by all of the rest of the scientists who testified. He takes the position that the statistical association, while it might show the direction in which to conduct research, might be misleading and, as he said, cause one to chase one fox instead of looking for some other foxes, and might retard the discovery of the logical relationship between cause and effect in smoking and cancer, because it might discourage exploration of other avenues, if people are satisfied that they have the culprit already identified. So it seems to me that there isn't really any too great disagreement on basic scientific attitude between Dr. Little and the other scientists who have testified—simply, perhaps, a greater question of causation on his part than on the part of the other scientists.

Would you agree with that?

Dr. HELLER. Partially, Mr. Meader. I would say fundamentally it is a difference in interpretation.

May I say, Mr. Meader, and members of the committee, that generally in cancer causation there is a belief that there are certain products within the body—endogenous products—which may be responsible for cancer. It may be hormones, it may be an anatomical anomaly, such as polyps, which we know in certain cases usually proceed on to cancer, or there may be exogenous factors, or things brought from the outside in contact with the body, or inhaled or ingested.

What we say here is that we believe there is an exogenous product which is brought into the body, or in contact with susceptible parts of the body, which may interact with an endogenous factor or other exogenous factors. Dr. Little does not quite interpret these products in the tars as being exogenous factors. We are led to the inescapable conclusion that we believe there is a causative factor, but we are unable to identify that particular factor. We hope that it can be identified and then we can proceed much more effectively.

Therefore, truly as scientists, we are all seeking the facts, and there is always a difference in interpretation. I certainly give Dr. Little every respect and admiration for differences which he may have. But we believe that this subject is so important—and as protectors of the public health in general, or at least in our particular roles as protectors—that we feel compelled, nevertheless, to indicate our interpretation. This represents a consensus after long and careful study of our medical staff people who are working with it for the most part, and with others with whom we have talked. The advice comes from the outside and represents a body of opinion, and not just simply in-

dividual personal opinions which may be correct but, therefore, might be considered only personal opinions. This represents a body of opinion.

Dr. BURNEY. Mr. Meader, may I supplement this just briefly.

We would all like to have the ideal situation in any public-health problem, to know the causative factor before we proceed to invoke preventive measures, but it has been our experience throughout the 150 years of the existence of the Public Health Service that many serious public-health problems have been solved very largely before the final causative agent or its method of spread has been determined.

In pellagra, for example, we didn't know what the final dietary part was that prevented pellagra before certain remedial measures were carried on. We didn't know the same about malaria, about yellow fever, and a number of things. We did have, however, statistical and epidemiological information which pointed up certain factors, and upon which we could proceed to eliminate malaria, yellow fever, and pellagra before we could identify the specific cause.

We would like to wait until we have the specific agent, and be able to identify it, and isolate it, before we pick any approach. On the other hand, it has not been our experience in public-health work that that has either been possible or has been a responsible course to follow.

Mr. MEADER. Thank you.

Dr. Heller, Dr. Little also told us about the $2.2 million that the tobacco industry has supplied for research in this field. Are you familiar with the grants made under Dr. Little's supervision, and what progress has been made as a result of expenditures of the tobacco industry in research in this field?

Dr. HELLER. I have seen a list of the grantees and the titles of the grants. In general, those grants are basic, fundamental studies in what we call carcinogenesis; that is, those things which cause cancer.

To the best of my recollection and knowledge, there are no studies in progress which are related directly to such things as filters or other applied-research components. I do know that the recipients of these grants are reputable scientists who are also recipients of grants from the Public Health Service, and from other grant-giving bodies. The work they do we can expect to be good work, and certainly, the research which is being conducted under the auspices of the tobacco industry research committee can be classed as good research.

I do not know of any outstanding results which have come from these studies. Perhaps it is a little early, inasmuch as they have only been in operation, perhaps, a couple of years. So that, while my information is not detailed, in general, I think that represents the situation.

Mr. MEADER. Dr. Wynder commented that if he had spent $2 million on his cancer research, and had so little to show for it, he would receive some very severe criticism from his superiors.

Mr. BLATNIK. His superiors wouldn't be very happy about it.

Dr. HELLER. I don't quite agree with that, Mr. Meader. I have been following cancer research, and have been responsible, for cancer research, generally, for a long time. It is a long, slow, laborious, heartbreaking, frustrating experience. Research, generally, in whatever field, medical research, industrial research, or what have you, is expensive and time consuming. It is frequent that much money goes

into something, and no results are obtained. If $2 million had been spent for cancer research, certainly, one would expect some results to come out of that. Probably some results are coming out of these studies. I just don't happen to know of them.

I would not be too critical of the results of research of $2.2 million worth of cancer research, on the basis of our experience in the National Cancer Institute over a period of years, in which much more than $2.8 million has been spent. Truly, I would certainly accept the proposition that any grant-giving group has a right to expect reports of progress and information as to the direction of the research, the accomplishments of research, attitudes of the investigators, as to whether they feel they are on the right track, and that sort of thing. I expect that is what Dr. Wynder is referring to, as well.

Mr. MEADER. Now, Dr. Heller, there was a comment on page 2 of Dr. Burney's statement, the last paragraph:

> Modification of cigarettes so that their harmful effects would be reduced is of obvious importance to public health—

and I think a similar attitude toward this question is expressed by Dr. Wynder and, perhaps, by other witnesses; that there isn't any desire or any hope that tobacco smoking is going to be wiped out, but that the aim should be to identify the harmful agents in tobacco and see if they cannot be eliminated, and, thus, the bad effects of smoking could be reduced.

That leads me to this question, which I haven't heard explained in our record so far: Just what are the effects of cigarette smoking or the elements in cigarette smoking on the human body, the stimulating effect, how does it operate, and is it possible that whatever pleasurable effects derived from smoking can be separated from the harmful effects? In other words, that gets back to this question of the filters that filtered out so much of the smoke that nobody wanted to smoke filter cigarettes any more, and now the manufacturers apparently have put in cheaper tobacco with more tars and some of these elements that are suspected of being harmful. As a result, people get a kick out of the filter cigarettes, but they are getting as much of the harmful elements as they were before.

Now can you, as a result of your research explain the operation of the elements in cigarette smoke upon body processes, and what element stimulates, and through what means there is a stimulation?

Dr. HELLER. Yes, Mr. Meader. My understanding is this: There seem to be three constituents, or major constituents, in cigarette smoking from which the individual derives the so-called pleasure of smoking. One is the effect of the nicotine on the individual. Now, nicotine is a very complex drug which has a complicated action on the human body from the standpoint of physiology. It brings about increased pulse rate, stimulation of what is called the parasympathetic nervous system, and involuntary nervous system, a stimulation of saliva, and a very complex train of physiological action which varies somewhat from person to person, but, in general, can be predicted. The second is in the tars, themselves. This is a mass of many hydrocarbons which haven't been completely identified chemically, although 5 or 6 of them have been very definitely established as being present in tobacco tars. We happen to believe that the carcinogenic, or the cancer-producing

elements, is in these tars, as we have indicated previously. The tars apparently give the taste the individual gets from smoking a cigarette.

Mr. MEADER. Rather than in the nicotine?

Dr. HELLER. That is right. So far as we know, nicotine itself is not involved, even remotely, as one of the causative factors in lung cancer, as far as we know.

Mr. MEADER. The nicotine is tastless?

Dr. HELLER. It is tasteless, and produces a physiological action, both of which are associated with the taste, and they are indistinguishable, as far as the individual is concerned. If he doesn't get the slight stimulation from nicotine, then the cigarette doesn't quite satisfy him. But the same token, as I understand it, when the tars are removed he doesn't quite get the same taste, and it is, therefore, not what he is accustomed to.

The third factor involved, as I understand it, is the psychological factor, which is doing something with the hands, or seeing the smoke curl up, or otherwise the satisfaction that one gets in release of his nervous tension, by merely holding a cigarette.

Considerable work has been done by psychologists as well as physiologists as to what really constitutes pleasure in smoking, and I believe that represents a consensus. Much more work needs to be done on the effect of nicotine, as far as the possible deleterious effect on the human body is concerned. We have reason to believe, for example—and I, certainly, am not expert in this field—that the action of nicotine does not have a beneficial effect upon certain cardiovascular diseases such as a disease called Buerger's disease, or endarteritis obliterans.

It is the technical name, in which the terminal arteries are constricted by the disease, and there seems to be a further constriction by the action of nicotine and, therefore, worsens the disease.

Now, this has not been elucidated, and I do not claim that this is a particular factor in cigarette smoking, but it needs to be elucidated, and much more work needs to be done. But physicians generally will advise individuals with coronary heart disease not to smoke, or to moderate their smoking, and this is true in other similar cardiovascular diseases. Nicotine probably has a much more direct action in cardiovascular diseases.

Again, I repeat as far as my knowledge goes, nicotine is not involved in lung cancer except as it promotes the pleasure in smoking that the individual might have and, therefore, he is less likely to smoke moderately if he is accustomed to the effects of nicotine.

Mr. MEADER. In other words, I take it that your research activities are reflected primarily toward the identification of elements in the tars, and their effect upon possible cancerous conditions?

Dr. HELLER. I would say generally, Mr. Meader, that is correct. We by no means ignore the possibility of such things as arsenic on the tobacco when it is growing, the constituents of the paper in which cigarettes are wrapped, the waxy coating of the leaves which Dr. Wynder undoubtedly mentioned in his testimony, might be removed by hot hexane before the tobacco is processed for cigarette making.

We believe as a very practical measure that the most direct approach is toward these tars and particularly the neutral fraction, which comprises only about 2 percent of the total tars; nevertheless, we are not eliminating or ignoring any of the other possible factors involved. In fact, we pride ourselves that we keep an open mind, and

that we are alert to every possibility in research which may be quite important in this entire question.

Mr. MEADER. I believe you agreed to give the chairman a list of your cancer grants over a 5-year period. Could you give us an approximation of the specific research on this problem of the relationship between smoking and cancer, in proportion to the total cancer research program? How much of it is devoted to this particular problem?

Dr. HELLER. From our work, or that of the Nation generally?

Mr. MEADER. I refer to your grants.

Dr. HELLER. It would be completely a guess.

We do not support an awful lot of work, Mr. Meader, and frequently work may be going on in this direction in connection with certain other grants which we are supporting, of which we may not be aware until they report on it. I would say that several hundred thousand dollars are now utilized in support of this type of research, as of the moment.

Mr. MEADER. Probably not as much as the $2.2 million that the tobacco industry is spending?

Dr. HELLER. I would say not, Mr. Meader. We may be supporting as much as a half million dollars, depending upon the definition of what constitutes research in this area.

Mr. MEADER. Could you describe so that a layman could understand it, some of the avenues that you are exploring in this particular field?

Dr. HELLER. Yes.

In addition to the epidemiological investigations with which you are, I think, reasonably familiar—the veterans' study and the study the cancer society is carrying on—we have engaged in studies to determine the smoking habits of a cross section of the population.

Mr. MEADER. I was thinking more in terms of the purely scientific rather than the statistical research.

Dr. HELLER. All right, sir.

In the purely scientific approach which the laboratory scientist can work at, we have been quite interested in finding out what are the earliest changes which occur in the epithelium or the lining of the bronchioles of the lungs.

Two or three studies with which you may be familiar, as referred to by Drs. Wynder and Hammond, have indicated that since cancer of the lung occurs at a certain predictable rate, there would be reason to believe that early changes which precede the actual cancer lesion might be observed at autopsy. These studies have indicated that there are very early changes, indicated by a diminution in the activity of the little cilia which propel waste material outward in the bronchial passages, in the nose and other parts of the bronchial system. There are varying degrees of change of this tissue, proceeding to the actual changes in some of the basic cells, and to what is called carcinoma-in-situ, or the very earliest stage of cancer that is clinically demonstrable before it becomes clinically recognizable. That is a very important approach, from our viewpoint, inasmuch as it enables us to know, or at least partially to know, how long it takes from the time that exogenous factors are applied to a sensitive epithelial or mucous membrane, until a fullfledged carcinoma of the epidermoid type is produced.

Mr. MEADER. Are you financing any chemical research into the composition of the smoke in tobacco?

Dr. HELLER. Yes, Mr. Meader. We have financed Dr. Wynder and Dr. Graham, who died last March, from lung cancer, incidentally. Their initial work was financed by us and partially so continuing.

We have financed work of the French investigators in 3,4-benzpyrene. Some work has gone on in our laboratory in this regard but we don't have individuals particularly interested in this field. We have financed some of the work at Sloan-Kettering. Of course Mr. Rand and his associates in Cleveland are independently financed, as is Arthur D. Little, of Boston, independently financed, and a group in Boston, I believe.

There are only 4 or 5 workers immediately in this field, as far as my knowledge goes. There may be others of whom I do not have knowledge.

We believe excellent work is going on and probably enough work is going on to give definitive results without blanketing the country with this sort of research. These investigators have to go through a very careful and quite complicated approach which, of course, requires considerable study and considerable knowledge.

Mr. MEADER. Mr. Chairman, I didn't mean to monopolize so much of the time, but perhaps, since I made a personal reference to Dr. Little being president of the University of Michigan when I graduated in 1927, I might also mention that my brother, Ralph, has been associated with Dr. Heller in the Cancer Institute.

Mr. BLATNIK. On the question of expenditures, Doctor. I recall quite well there was a special interest in medical research when appropriations were made by this Congress.

The President asked the Congress for $46,902,000 for the National Cancer Institute for this coming fiscal year of 1958. Congress upped that by almost $10 million. The Congress voted $56,402,000 for this period.

Now for this first quarter of fiscal year 1958, could you tell us at this time, or supply for the record upon checking up, whether the National Cancer Institute is spending one-fourth of the funds which the President asked for, which is $46 million-plus, or are you spending one-fourth of the funds which Congress appropriated which is over $56 million?

In other words, are you spending a larger figure or a smaller figure?

Dr. HELLER. Mr. Chairman, as of yesterday, to the best of my knowledge, the apportionment had not come down from the Public Health Service and the Bureau of the Budget, so I cannot answer that accurately at this time. I will be pleased to supply that for the record as of today or as of whatever day the apportionments are made.

(Dr. Heller advised the subcommittee that the first quarter apportionment totalled $33,258,000.)

Perhaps Dr. Burney might have some additional information.

Dr. BURNEY. I assume we will do the same as we did last year, Mr. Chairman. In other words, we received quite a sizable increase last year and there was no ceiling put on the amount of funds, and the Institute, through their advisory counsels and study secretaries were told that "We have this amount of funds and if we have the projects to go ahead and spend the money. We hope that the same quality of review and appraisal of project will continue, even though you have

more money to spend," and I would expect that the same policy would be followed this year.

Mrs. GRIFFITHS. May I ask when did you first make a determination that there was a relationship between lung cancer and excessive smoking?

Dr. HELLER. It was brought to our attention in 1950 by the work primarily of Dr. Evart Graham and Dr. Wynder. However, part of that time in our own laboratories as early as 1939, some investigations had gone forward which were inconclusive, in attempting to establish a possible relationship between smoking and the production of lung cancer in animals. Not until 1950 did it come to our attention that there was a real problem involved.

We recognized at that time that there was probably a statistical correlation.

Subsequent studies were made in this country and abroad and in 1954, an official viewpoint was expressed by the Surgeon General of the Public Health Service—I don't think there was an official document put forth as has been, here, but simply a statement to the press and others that we believed there was a statistical correlation between excessive smoking and the occurrence of lung cancer, but we did not believe that there was a cause-and-effect relationship, as expressed at this time.

Subsequent studies, and particularly laboratory studies, have brought us additional information, and the increasing weight of evidence caused us to recommend to the Surgeon General that we believed there was a desirability of calling to the attention of the public the information which had been derived from work, not only at the National Cancer Institute but in the world in general.

Mrs. GRIFFITHS. When did you suggest that?

Dr. HELLER. Just recently.

Mrs. GRIFFITHS. Within the last 60 days?

Dr. HELLER. Within the last 6 weeks, I would say.

Mrs. GRIFFITHS. Is this the first written statement that has ever been issued by the Public Health Service, or have there been others?

Dr. HELLER. In this regard the Surgeon General issued a statement on July 12, concerning the attitude of the Public Health Service, which I believe is the first written one that has been so formalized by the Surgeon General.

Mrs. GRIFFITHS. May I ask you, had other countries made these determinations ahead of the United States?

Dr. HELLER. Yes, Britain in about 1954 came forth with an official statement by the Ministry of Health that there was at that time a statistical correlation, and they believed it to be a cause-and-effect relationship.

Mrs. GRIFFITHS. Have you already sent this information to the health departments in various States?

Dr. BURNEY. Yes, ma'am. This statement was given Friday, July 12 and the material went out, I believe, the following Monday.

Mrs. GRIFFITHS. Have you notified the Federal Trade Commission?

Dr. HELLER. Yes.

Mrs. GRIFFITHS. And have you notified the Department of Justice?

Dr. HELLER. I have notified officially the Federal Trade Commission. I have not officially notified the Department of Justice.

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Mrs. GRIFFITHS. Did you consider doing that? Would that be part of your job?

Dr. HELLER. Ordinarily, it would not be a part, Mrs. Griffiths. However, the Federal Trade Commission is directly concerned, and have been in touch with us quite closely as I have indicated to the chairman.

Mrs. GRIFFITHS. Have they asked you what you have found? Do you know of any official inquiry that the Federal Trade Commission ever made of you concerning lung cancer and smoking?

Dr. HELLER. Oh, yes.

Mrs. GRIFFITHS. How long ago?

Dr. HELLER. Mr. Grandey, the Chief of the Division of Consultation, I believe it is called, of the Federal Trade Commission, and several of his associates, visited me several months ago, 3 or 4 months ago, and we discussed at length this growing problem. I indicated we were studying this problem through a smoking and study group—which gave a report last June—and that probably we would recommend some action to the Surgeon General, but we were not completely sure of what it would consist. We have been in telephone conversation with them since then.

Mrs. GRIFFITHS. They have never asked you to check filters or asked your advice on filters?

Dr. HELLER. No, only in a very general fashion.

Mrs. GRIFFITHS. Do you know if they are checking filters?

Dr. HELLER. I do not know.

Mrs. GRIFFITHS. Would you consider it a wise expenditure of public funds to put this information that you have given us in every school in the country?

Dr. HELLER. As a part of a general health-information program, if the State health authorities approve of it, yes. Not as an official action on our part; as Dr. Burney indicated, the health authorities are sovereign.

Mrs. GRIFFITHS. Why don't you think it is a wise official action?

Dr. HELLER. Because, generally, we as a Federal agency do not indicate the way that health departments should approach their particular problems.

Mrs. GRIFFITHS. But it has nothing to do with your conclusions?

Dr. HELLER. Oh, no.

Mrs. GRIFFITHS. So if the Congress suggested this, actually you would feel it would be wise to warn children, would you?

Dr. HELLER. I would say if the State health authorities, in view of their knowledge of their problems, and the situations with which they are confronted, believe that this information warrants a conclusion in such a program as they wish to make, then I think that is their determination.

Mrs. GRIFFITHS. Have you or any official of the Public Health Service, ever appeared on a television program and announced your findings?

Dr. HELLER. In this particular regard?

Mrs. GRIFFITHS. Yes.

Dr. HELLER. I have not.

Mrs. GRIFFITHS. On any others? Do you sometimes warn the public of various dangers? Is that one of the means that the Public Health Service has traditionally used since television has come into use?

Dr. BURNEY. I don't believe we have ever asked, Mrs. Griffiths, to appear on any particular program, whether it is on polio vaccine, or anything else, but occasionally we are asked by a certain moderator of a program, radio or TV, to appear and discuss it.

Mrs. GRIFFITHS. If you consider this a peril, and the Federal Government controls the airways, would you consider it a legitimate use of those airways for the Public Health Service on a public-service program to announce their findings?

Dr. BURNEY. I would be very pleased to present my statement on either TV or a radio activity.

Mrs. GRIFFITHS. Then I am going to ask for you to have some time.

Have any States requested information on this problem?

Dr. BURNEY. I cannot answer that specifically, I am sorry. I can find out for you.

It just happens that the executive committee on the State and Territorial health authorities was meeting with us the day before the statement was coming out—it had nothing to do with the statement; we did inform them that day of our statement coming out the following day, and Dr. Heller gave them the background which he has given you all here this morning, and told them we were sending this material to them.

But, as far as I know, we have not received any specific requests.

Dr. HELLER. I don't believe we have.

Mrs. GRIFFITHS. When you were considering the relationship between lung cancer and smoking, what contrary evidence did you consider?

Dr. HELLER. The fact that there was no proved connection between the chemicals involved, and their ability to produce cancer in man; the fact that prospective studies were preliminary, and as yet not defined as precisely as Dr. Hammond has defined it. Lack of knowledge of the processes of the pathology involved in lung tissue, which only in the last year or so has come to the public attention; I would say in general these represent the basis for our hesitancy before coming out—the fact that these particular factors were not elucidated properly to our satisfaction. Subsequently we believe that happened.

Mrs. GRIFFITHS. Thank you.

Mr. BLATNIK. Mr. Minshall.

Mr. MINSHALL. Mr. Chairman, at the outset I should like to ask unanimous consent of the committee to make part of the record a very excellent and concise article that appeared in the New York Times, Sunday, July 21, 1957, by William M. Blair, entitled "Huge Tobacco Industry Again on Defensive." I think it has some background information that would be of very much assistance to this committee.

Mr. BLATNIK. Without objection, it is so ordered.

(See appendix, exhibit 11, p. 579.)

Mr. MINSHALL. Dr. Heller, you said you were familiar with Dr. Little's testimony that he gave before this committee.

Dr. HELLER. In general, sir.

Mr. MINSHALL. Would you give us an opinion of his testimony—not as to whether or not you disagree with it, but of his findings?

Dr. HELLER. Well, Mr. Congressman, I would say that Dr. Little's testimony reflects the attitude of a scientist who has been preoccupied with——

Mr. MINSHALL. Dr. Little is not a doctor of medicine?

Dr. HELLER. That is right, he is a doctor of philosophy and particularly noted for his work in genetics and as an educator.

I would say that Dr. Little's viewpoint is one of a very well-informed scientist who is interested in the basic components of research. He, by his own admission, was not interested in filters or the developmental research that is inherent in a discussion of this problem.

Dr. Little's testimony in general—at least the impression I received from it, was that he simply did not believe that the epidemiologic and statistical evidence submitted was sufficient to allow an interpretation of a causative factor being involved in tobacco smoke.

I think we agree certainly with Dr. Little that more research needs to be done. We disagree fundamentally in the interpretative aspects.

I will not take exception to Dr. Little's basic attitude, I think he is entitled to that. I differ with respect to his basic interpretation.

Mr. MINSHALL. The question was also asked you, Dr. Heller, as to how soon you thought some definite findings might be made known to the public—whether it was just a manner of speaking but you said something about next spring.

Dr. HELLER. I was referring primarily to the report which we hope to make of this very large prospective study among veterans. Now, as to what pronouncements might be made in the future would be dependent upon what findings emanate from the laboratories, or from the work of other investigators, and I do not know how to predict just when that might be.

I can predict, because we believe that the initial reports will come out next spring on this prospective study which is a very important one, in our opinion.

Mr. MINSHALL. Your studies thus far are the results of analyzing the statistics of the World War I veterans. Am I correct that you said those studies thus far parallel the reports and statistics as given before this committee by Dr. Wynder?

Dr. HELLER. Primarily by Dr. Hammond.

Mr. MINSHALL. Dr. Hammond, yes. Pardon me.

Dr. HELLER. The fragmentary reports dipped into the stream of information.

Mr. MINSHALL. What do you mean by fragmentary?

Dr. HELLER. Well, in the sense that they take out a little piece of data and look at it, and see which way it seems to be going. This dipping into portions which they have examined seems to be consistent with results reported previously.

The idea was, as we processed these data, to ascertain that they were consistent with existing ideas or findings which previously had been reported.

Now, obviously, one is unwise to make a dogmatic statement that this is going to be the same as any other statement. We are waiting until all the statements are analyzed, statistically interpreted, and studied by people who make it their business to interpret properly and put into a practical usage the results of such studies.

Mr. MINSHALL. Have you at any time conferred with the chemists or the doctors, other than Dr. Little, that represent the tobacco industry?

Dr. HELLER. I know most of the members of the tobacco industry research committee. I have attended a social gathering or so of this

group. I know most of them personally, and we have discussed it in very general terms. I called Dr. Little, for example, before Dr. Burney's statement came out, to keep our liaison. We are good friends and we have always enjoyed pleasant relationships which I hope will continue.

I do not know of the work of the investigators who are supported by the tobacco industry research grants.

Mr. MINSHALL. You, of course, have discussed, the general effects of smoking and cancer with these people representing the tobacco industry; have you not?

Dr. HELLER. Yes, sir.

Mr. MINSHALL. Have you ever discussed the relative merits or demerits of various kinds of filters that they have used?

Dr. HELLER. No; I have not. The times at which I have discussed the problem with them, filters have not been introduced into the conversation, and, as a matter of fact, there seems to be a lack of knowledge, generally, of the results.

Mr. MINSHALL. Well, the cigarette companies are making fantastic claims and have over the past several years as to what these filters do. Are you familiar with their laboratory tests?

Dr. HELLER. No. sir.

Mr. MINSHALL Do you know that they do have laboratory tests?

Dr. HELLER. We understand that tobacco companies have their own research laboratories. We do not not know the results of any work which they have done.

Mr. MINSHALL. Have you ever requested that information from these research laboratories of the various companies?

Dr. HELLER. No, sir; we have not requested it.

Mr. MINSHALL. Don't you thing that would be a good idea?

Dr. HELLER. I think it probably would.

From past experience with industrial laboratories, they traditionally or conventionally do not give out the results.

Mr. MINSHALL. This is a different matter. This is in the interests of medical science. Like a gasoline company constantly sample tests the quality of its gasoline it puts in its service stations, and I am sure the tobacco people do the same thing with their tobacco as well as the filters. They test their filters periodically, do they not?

Dr. HELLER. Yes; I presume they do.

Mr. MINSHALL. Have you ever discussed that matter with the tobacco people at all?

Dr. HELLER. No; I have not, sir.

Mr. MINSHALL. Where did you get the information that the filters were 40 to 50 percent effective when they first came out?

Dr. HELLER. Primarily from the work of Dr. Wynder and his group, and also from the work—reported in the Reader's Digest, of which I had some knowledge before it came out—done by Snell & Associates, which is believed to be good work.

Mr. MINSHALL. From your knowledge of reading these various publications, and talking with Dr. Wynder, are these filters as effective today as they were when they came out?

Dr. HELLER. The information I have from these discussions is that these filters are not as effective today as they were when they first came out.

Mr. MINSHALL. Did you happen to see the New York Times article that appeared in last Sunday's paper, where they stated that they are using all kinds of stems and roots in the tobacco that they put in the cigarettes now?

Dr. HELLER. I did not happen to see that one, sir. I have heard that statement made by others, but I did not see this particular report.

Mr. MINSHALL. Doctor, you also mentioned some statements put out by the health departments of foreign countries—Mr. Chairman, I think it would be also advisable if we had those statements put into the record.

Dr. HELLER. I will supply them, sir.

Mr. BLATNIK. Without objection, it is so ordered.

(See appendix, exhibit 13, p. 593.)

Mr. MINSHALL. I have no further questions, Mr. Chairman.

Mr. BLATNIK. After a 3-day hearing we have come to this point:

First, we have from the layman's point of view a rather comprehensive picture of the extent, statistically, of lung cancer, and its high statistical relationship with heavy, prolonged smoking.

The medical witnesses representing more or less the point of view of the tobacco industry, say there is no direct evidence to show any causative factor in smoking that would cause cancer. Nor, as Dr. Little said, is there anything that he knew of in the smoke or in the cigarette that was harmful to the body.

We have other testimony, and these two witnesses this morning state that there is something which justifies deep concern on the part of both official and private agencies and bodies, protecting the people from possible medical harm from excessive smoking.

Last year, in 1956, a study group on smoking and health was organized including four organizations: The American Cancer Society, the American Heart Association, and the National Cancer Institute, and the National Heart Institute.

Dr. Heller, were you involved in the forming of this joint Study Group on Smoking and Health?

Dr. HELLER. I was, sir.

Mr. BLATNIK. In their report the statement is made—

the sum total of scientific evidence establishes beyond reasonable doubt that cigarette smoking is a causative factor in the rapidly increasing incidence of human epidermoid carcinoma of the lung.

Does that statement hold as of this moment, Doctor?

Dr. HELLER. Yes.

Mr. BLATNIK. Is it correct, Dr. Burney, to say that is the basis, the combined collective judgment of these professional people who work in the field of cancer and heart disease, studying the smoking and health, that that would be the basis for this statement, the policy made by yourself, on behalf of your department?

Dr. BURNEY. That was the major factor, Mr. Chairman. In addition, there was the report of Dr. Hammond and Dr. Horn; also the additional laboratory biological data that Dr. Heller discussed, the hyperplasia, the changes that occur in the cilia, and the bronchioles, the study group appraisal of existing evidence, and their comments—these constituted the major evidence upon which we based our conclusions.

Mr. BLATNIK. In the conclusions of this joint Study Group on Smoking and Health it says:

The study group concludes that the smoking of tobacco, particularly in the form of cigarettes, is an important health hazard. The implications of this statement are clear in terms of the need for thorough consideration of appropriate control measures on the part of official and voluntary agencies that are concerned with the health of the people.

Would you say that statement sets forth the latest thing and the latest area of agreement on this important subject?

Dr. BURNEY. We believe, Mr. Chairman, that in that report we had this responsibility to express an opinion and make these facts known to the public, and our interpretation of the facts.

I would like to say again, however, that we do not believe the final answers have been secured and that there is a limit to what a responsible, official Federal agency can or should do before they have all available information. That is why I think we have stopped at a certain point, using our particular judgment, and that until such time as we have much more definitive information, we should not go all out on a campaign and put stickers on cigarettes and certain other things. Maybe our judgment is wrong in that, but I think we have gone as far as we should go at the present time. As new information comes out, as a result of what Dr. Heller is doing, or other things, we will make those facts known to the public and to our counterparts in the States.

Mr. PLAPINGER. But you said in your published statement "it is confirmed beyond a reasonable doubt." It seems to me you are now saying, "on the one hand," but, "on the other hand." But you have said in the first instance that "it is confirmed beyond a reasonable doubt that there is a high degree of statistical association between lung cancer and heavy and prolonged cigarette smoking." That is an unequivocal statement, Dr. Burney.

Dr. BURNEY. That is right. We believe that, and Dr. Heller's group agrees with that.

Mr. PLAPINGER. You are not prepared at this time to do more than make this information available to State agencies concerned with health. Having made this pronouncement, apparently that ends the public information-education phase, or is that a premature conclusion?

Dr. BURNEY. I would suspect this subject will be discussed this fall when the Surgeon General has his annual conference with the State and Territorial health officers. I would also suspect at the same time that we will discuss accident prevention in children, which is one of the very serious causes of death.

This is not the only health hazard, and we certainly have some very definite information on those indications, and so there are other health problems that State health officers must consider. But, at the present time, yes, sir; I would say this is as far as I would intend to go, and which my scientific group have recommended.

Mr. PLAPINGER. How about information in your own installations, such as the public-health hospitals, and among Federal Government employees?

Dr. BURNEY. This information has been pretty widely dispensed, as Dr. Heller mentioned. The press has given, I would say, very complete and very factual reporting on this.

I would also say that we are getting into an area here where individual likes many times overcome one's fears and prejudices. I have smoked a pipe for 15 years. If pipes were more seriously implicated

in this, I think now that I might not stop smoking, but might not smoke so much. I wouldn't swear to that, because I get a lot of pleasure out of smoking and, even though the threat is there, I might still continue.

Mr. PLAPINGER. We have some evidence along that line from Dr. Hammond that I might turn over to you. Dr. Rigdon, the head of the department of pathology at the University of Texas, mentioned in his testimony that in the United States Public Health Service—

there is a wide variation with regard to the role of cigarettes and cancers. You have certain ones who say it just can't occur, and others say it might be but it hasn't been proven, so you have a split in there from people who are now in that organization.

Now, I would assume that on any issue there is divergence of opinion—and I have been professionally trained to believe that there is such a thing as a 5-to-4 decision in the Supreme Court—but how wide is the variation of opinion within the Public Health Service or within the National Cancer Institute?

Dr. BURNEY. I believe Dr. Heller should answer that, but I should say it was my understanding when we prepared this position that the consensus of the scientists at the National Cancer Institute were firmly behind this particular position.

Dr. HELLER. That is true.

Mr. Chairman, there are many scientists in the Cancer Institute, and many differences of opinion, scientists being scientists. However, I would disagree with Dr. Rigdon that there is a wide variation in attitude. Even a particular scientist who believes that air polution is much more of a factor, for example, than smoking, says, however, that there is no doubt that smoking is incriminated in this process and it is simply a matter of degree.

There are others who are of the opinion that there might be such lung-cancer increases as has been stated, but that, through better diagnosis, it is coming to the attention of the medical profession and the people. We have better doctors, we are dagnosing more quickly, and so forth. However, I would say that the consensus in the Cancer Institute—I can't speak for the entire Public Health Service, but certainly in the Cancer Institute and in the National Institutes of Health—the consensus is reflected in the statement which the Surgeon General has promulgated.

Mr. PLAPINGER. Does that mean 51 percent, or is it overwhelming?

Dr. HELLER. An overwhelming majority. I would say with the exception of only 1 or 2 who do not agree completely with this viewpoint, but the overwhelming majority of the scientists in the National Institute of Health agree.

Mr. PLAPINGER. With regard to the 1 or 2, what happens to their research that may have been going off in one direction or another as a result of this pronouncement of Government policy?

Dr. HELLER. They go right along, sir. It is not stopped.

Mr. MINSHALL. You have mentioned what they do with it in the National Cancer Institute. Have you had the opportunity to discuss this with, for example, the Mayo Clinic, or any other private institutions?

Dr. HELLER. With individuals of the Ochsner Clinic, and the Mayo group, and so forth. I have discussed it with individuals.

Mr. MINSHALL. And what was their feeling?

Dr. HELLER. Taking the country as a whole, sir, and speaking of individuals whom I respect and who are responsible individuals, I would say the majority of them concur in this viewpoint. There are certain individuals, like Dr. Berkson at the Mayo Clinic, and others around the country, who do not agree. They agree, perhaps, with Dr. Little's viewpoint. This is characteristic of science in general, where there is a difference of opinion on many subjects. However, when one analyzes it to the utmost, there is not as much difference as one might think on the surface.

Mr. MINSHALL. Can you give us a ratio among the private surgeons and cancer experts as to what the ratio would be?

Dr. HELLER. It would be purely a guess on my part.

Mr. MINSHALL. What is your best guess?

Dr. HELLER. My best guess is that 75 percent of the physicians or scientists who have knowledge and some competence in this area would concur with this formula.

Mr. PLAPINGER. Dr. Heller, in an article that appeared in the July 19 issue of the New York Times, there is a reference to the fact that a Dr. Smith stated that the National Cancer Institute, after protests from chemical companies, had abandoned a study aimed at determining the extent to which chemicals might cause cancer. Would you care to comment on that, please?

Dr. HELLER. Yes, sir. I am aware of that testimony. That is not a correct statement of action by the National Cancer Institute. The Public Health Service has not stopped any field or laboratory investigation into cancer at the request of any individuals on the outside. Such projects as have been terminated have been terminated because they logically had completed their course, or because we found it was fruitless, or for other reasons. I would say this is either a misunderstanding on the part of the individual or individuals who made the statement, and not founded upon fact. And we, in fact, have increased our studies in environmental cancer over a period of years.

Mr. BLATNIK. In conclusion, we are certainly pleased and relieved to hear that our top medical agencies in the Government are working so closely and intimately on this important problem. Without trying to self-evaluate, would it be proper or correct to say that our Government officials, those of you in the top, executive positions, and those of us in the legislative, are doing about what is being done in other countries in Europe concerned with the problem of health and smoking, Doctor?

Dr. BURNEY. I believe we are Mr. Chairman.

Mr. BLATNIK. In terms of research and in terms of keeping the people informed, and in terms of keeping the Government agencies informed?

Dr. BURNEY. I think we are. We are doing more than many countries are doing, as Dr. Heller mentioned, and I would also point out that in addition to specific research on this particular area we have to recognize that some of the research in cancer, which is not directly aimed at this field, may have a bearing in giving some answer to this problem.

Mr. BLATNIK. Well, thank you very much. We appreciate your excellent cooperation, and your splendid assistance, and commend you for your straight-forward and forthright statements.

The Chair wishes to announce that the hearings will continue tomorrow when we hope we will finally get some information on filters. I have never seen anything that has been advertised so extensively and persistently, and at great expense, about which I finally discover I know so little about.

The witnesses tomorrow will be Mr. Irving Michelson of the special projects division, the head of the Consumers Union.

Dr. Walter Wolman, director of the chemical laboratory, American Medical Association, and Dr. C. S. Kimball, executive vice president of the Foster D. Snell, Inc., the research agency in New York which made the laboratory tests and the report for The Reader's Digest, which is reported in their articles of this July and the coming issue of August.

So until 10 o'clock tomorrow morning, the hearings are adjourned.

(Whereupon, at 12:15 p. m., the subcommittee adjourned to reconvene at 10 a. m., Wednesday, July 24, 1957.)

FALSE AND MISLEADING ADVERTISING

(Filter-Tip Cigarettes)

WEDNESDAY, JULY 24, 1957

HOUSE OF REPRESENTATIVES,
LEGAL AND MONETARY AFFAIRS SUBCOMMITTEE
OF THE COMMITTEE ON GOVERNMENT OPERATIONS,
Washington, D. C.

The subcommittee met, pursuant to notice, at 10 a. m., in room 100, George Washington Inn, Hon. John A. Blatnik (chairman) presiding.

Present: Representatives Blatnik and Meader.

Also present: Jerome S. Plapinger, subcommittee counsel; Curtis E. Johnson, staff director; and Elizabeth D. Heater, clerk.

Mr. BLATNIK. The Subcommittee on Legal and Monetary Affairs of the House Government Operations Committee will come to order.

We are proceeding with further public hearings on the effectiveness of Federal agencies in regulating advertising, restricting false and misleading advertising and in this case we are continuing with the hearings on cigarettes, primarily the filter-tip cigarettes.

In announcing these hearings about a week ago, on July 15, I made a note that the cigarette sales declined for the first time in 21 years of uninterrupted growth following reports that cigarette smoking is a causative factor in lung cancer. Sales fell again in 1954, but the cigarette industry rescued itself through filter cigarettes promoted with a tremendous advertising campaign. Filter cigarette sales boomed and in spite of additional medical reports that cigarette smoking is a hazard to health, sales are greater today than at any time in history. Some reports we have received indicate that many filter cigarettes afford little, if any, protection to the public, although cigarette advertisers have emphasized the superiority and effectiveness of their particular filter.

The testimony which follows will give us reports on the quantities of nicotine and tar in the smoke from various brands of cigarettes. Tobacco tar has been identified by medical testimony as the suspected cancer-causing agent. Nicotine has been linked with cardiovascular diseases.

The protection afforded to the public by the filters on cigarettes can be measured roughly by the degree that the nicotine and tar are reduced.

Our first witness today is Irving Michelson, head of the special projects division of Consumers Union. Mr. Michelson will be followed by Dr. Walter Wolman, director of the chemical laboratory of the American Medical Association. After Dr. Wolman, we will hear from Dr. C. S. Kimball, executive vice president of Foster Snell, Inc., the

laboratory that conducted tests contained in the July and August issues of Reader's Digest. (See appendix, exhibit 14, p. 604.)

Consumers Union, in its publication Consumer Reports, published tests on cigarettes in 1953, 1955, and 1957. (See appendix, exhibit 15, p. 622.) These tests afford us an opportunity to observe the evolution of filter cigarettes—their effectiveness or lack of it and also a comparison of filters with regular cigarettes.

I would like to note that the test methods used by Consumers Union and the American Medical Association, whose report will follow later this morning (see appendix, exhibit 16, p. 668), are the same and are comparable.

Foster Snell, Inc., which conducted the Reader's Digest tests, used a different laboratory technique in extracting tar which gives higher quantities and thus are not comparable to those obtained by Consumers Union or the American Medical Association.

We will now proceed with our first witness, Mr. Irving Michelson of Consumers Union. Mr. Michelson, we welcome you to the committee and appreciate your willingness to assist in gathering information for the committee on the effectiveness of filter cigarettes.

Mr. Michelson, I notice you have a prepared statement with the printed reports of your three studies of 1953, 1955, and 1957.

Mr. MICHELSON. Yes, Mr. Chairman.

Mr. BLATNIK. Would you please give us a brief background of your technical experience and association with these Consumers Reports and then proceed with your statement?

STATEMENT OF IRVING MICHELSON, SPECIAL PROJECTS DIVISION, CONSUMERS UNION

Mr. MICHELSON. My statement covers my background and association with Consumer Reports and I would like to proceed to read the statement with the chairman's permission.

Mr. BLATNIK. Please proceed.

Mr. MICHELSON. My name is Irving Michelson. I am head of the special projects division of Consumers Union, whose laboratories and offices are located at Mount Vernon, N. Y. I have been a division head at Consumers Union for the past 10 years. Prior to that I was an analytical chemist in the laboratories of the United States Bureau of Customs at New York and Boston.

To this introduction of myself, let me add a few words by way of explaining the work of the organization I represent. Consumers Union is a nonprofit organization, chartered under the membership corporations law of the State of New York. For more than 20 years it has been providing consumers with information and counsel on consumer goods and services. A major part of its work over these years has been in the field of comparative product testing—that is, it buys on the open market various brands of widely sold products, submits them to appropriate tests, and reports its findings in its monthly magazine, Consumer Reports.

Consumers Union has no connection of any kind with any commercial interest, accepts neither advertising nor samples of products from manufacturers, and permits no promotional use of its test results. It derives its income from subscription fees and, to a lesser extent, from

newsstand sales. At present the circulation of Consumer Reports is approximately 900,000—a figure which makes it, I believe, by far the largest publication of its kind in the world.

Along with its product testing and publishing activities, Consumers Union has initiated and participated in a variety of other educational efforts related to the welfare of the public as consumers. Next month, for example, it is sponsoring jointly with Rutgers University, a conference on quality control and the consumer. In the past it has taken an active part in similar conferences held at other educational centers; has cooperated with other consumer organizations, with the American Standards Association, with teachers, trade unions, and professional groups; and has worked with technical socities toward the end of improving test methods for consumer goods. Consumers Union has also cooperated with agencies set up by Congress in fields affecting the consumer interest—including the Federal Trade Commission, the Food and Drug Administration, and the United States Department of Agriculture.

In short, my organization has been doing what it has been able to do, and what has seemed proper and useful to do, to contribute toward the development of that "intelligent and responsible citizenry" of which Dr. Clarence Cook Little spoke in his testimony here the other day.

With particular reference to the cigarette-cancer controversy, and other health aspects of smoking, Consumers Union over the past 4 years has made 3 sets of tests covering all leading cigarette brands along with a number of brands less widely sold. Our procedure has been the same for each test project. Consumers Union shoppers in various parts of the country have bought packages of various brands at retail; carefully controlled laboratory tests for nicotine and tar content have been run on the smoke of the cigarette thus purchased; and the findings concerning each brand have been published in the pages of Consumer Reports.

In the February 1953 issue of the Reports our findings covered 27 brands; in the February 1955 issue, 37 brands; in the March 1957 issue, 33 brands. (See appendix, exhibit 15, p. 622.) Our test results over the past years have convinced us that cigarettes vary considerably from brand to brand in the nicotine and tar contents of their smoke, and that an individual brand may vary considerably from time to time.

It is therefore necessary to test many brands, and to retest the brands now and then, to obtain useful information about the general situation, about individual brands, and about trends. In this connection, at the request of this subcommittee we have just completed another set of tests, this time on one brand—the Kent cigarette, which has recently announced a new filter tip described in its advertisements as revolutionary. Results of these newest tests will be published in the September issue of Consumer Reports, but are summarized for the subcommittee as an attachment to this statement. (See appendix, exhibit 17, p. 677.)

In our opinion, the Consumers Union test reports taken together constitute the most complete set of objective data available on the nicotine and tar content found in the smoke of cigarettes, and on the effectiveness of various filter tips to date in reducing such nicotine and tar content. Copies of the issues of Consumers Reports contain-

ing these test results are also attached to this statement. (See appendix, exhibit 15, p. 622.)

On the average, we have found that filter-tip cigarettes give the smoker about the same amount of nicotine and somewhat less tar (about 15 percent) than cigarettes of the same size without filter tips. It is interesting to note also that the average tar content of the smoke of the filter-tip cigarettes reported on in March 1957 was no lower than the average found in cigarettes reported on in 1953.

Mr. PLAPINGER. What kind of cigarettes are you referring to there in the last part, are they filter cigarettes, or nonfilter?

Mr. MICHELSON. We are referring specifically there to the regular size without any filter and also to the king size with filter that existed in 1953. In both those cases there is no advantage in the present filter tips over the previous ones.

Certainly, no filter tip of the many subjected to tests by Consumers Union can be considered in any sense to have eliminated whatever health hazard is presented by smoking.

Without affecting the truth of this generalization, two brands in particular are worthy of special comment. The King Sano filter-tip cigarettes, covered in our 1957 reports, was found to give the smoker about 40 percent less tar than the average king-size filter cigarette, and about 50 percent less than unfiltered king-size cigarettes.

Our recent tests on the new Kent cigarettes show that these also give the smoker about the same reduced quantity of tars as the King Sano cigarettes.

For the information of the subcommittee, I submit the following notes on the methods used in the Consumers Union tests for tar and nicotine contents of the smoke of cigarettes.

Mr. Chairman, would you want me to read these or should we just put them into the record?

Mr. BLATNIK. The three pages you have here?

Mr. MICHELSON. On smoking, nicotine determination, and tar determination. Would the committee be interested?

Mr. BLATNIK. Will you read them, Mr. Michelson?

Mr. MICHELSON. Smoking: The apparatus and technique used were those described in the July 1936 issue of Industrial and Engineering Chemistry, in an article entitled "Technic of Experimental Smoking," by Bradford, Harlan, and Hanmer.

(See appendix, exhibit 18, p. 677.)

Mr. BLATNIK. Just to identify these people, the article was in July 1936—I see—Bradford, Harlan, and Hammer were not men in your laboratories, they were men who developed this technique?

Mr. MICHELSON. Yes, sir; the name of the laboratory in which they did their work I can provide.

Mr. BLATNIK. We can get that later. I am sorry to have interrupted you.

Mr. MICHELSON. The same apparatus and technique are widely used by laboratories in the United States working on this problem. In our tests, all cigarettes regardless of size were smoked to the same butt length (23 millimeters). The smoke was collected in an alcohol-water mixture containing a small quantity of acid.

Mr. MEADER. Is this the same test made by the tobacco companies?

Mr. MICHELSON. I am sorry I did not hear the question.

Mr. MEADER. Is the technique you use in making these determinations of nicotine and tar content, the same technique that is used by the tobacco companies themselves in any experimentation or test that they make?

Mr. MICHELSON. I believe so. As a matter of fact, this test was a result of work by the American Tobacco Co., and these three gentlemen who wrote the article were working for the American Tobacco Co. at the time and so far as I know, all the laboratories in this country are using this technique of testing modified only to the extent of making it more or less mechanical, or automatic, but in all cases the techniques are substantially the same in that they apply the same burning conditions of a cigarette, the same puff rates and the same technique for collecting the smoke.

Mr. MEADER. Thank you.

Mr. MICHELSON. Nicotine determination: The nicotine was separated from most of the other smoke components by a method described in the Official Methods of Analysis of the Association of Official Agricultural Chemists, section 5.106. The quantity of nicotine present was then determined by an ultraviolet spectrophotometer, according to a method described in the March 1950 issue of Analytical Chemistry, in an article entitled "Spectrophotometric Determination of Nicotine" by Willits, Swain, Connelly, and Brice, of the United States Department of Agriculture.

Tar determination: That portion of the collected smoke which was soluble in chloroform was separated from the water-soluble material by extraction of the smoke solution with chloroform; the chloroform was then evaporated and the residue weighed.

The material obtained in this way is what we have been designating as "tar." Because "tar" is a nonexact term, other definitions are possible. There is no generally accepted definition for "tar," so each laboratory is free to use any reasonable method it may choose. The chloroform extraction method was also used by the chemical laboratory of the American Medical Association in preparing their reports (Journal of the American Medical Association, July 4 and 11, 1953, and April 9, 1955, see exhibit 16, pp. 668, 671), and by other laboratories. If a different definition of "tars" is used, a different test method consistent with the definition is necessary. Direct comparisons of results can be made only when the same definition, and, hence, the same test method, has been used.

Another method used by some laboratories is to define tar as being the residue from the evaporation of all the trapped smoke—this may be designated as the "total smoke" method. The smoke is collected in the same manner in an alcohol-water mixture, and the solution is evaporated to dryness directly. This was the method used by the Foster D. Snell Laboratories in preparing their report for Reader's Digest, and appears to be the method used in preparing the figures in the current Kent ads. Our tests on the current Kents were done both ways, to get comparisons with our previous work and with the Kent figures advertised. Both sets of figures are given in the attachment.

Consumers Unions' tests were performed by the Fitelson Laboratories of New York, acting as consultants to and working under the direction of the Consumers Union technical staff. Dr. Jacob Fitelson,

an expert on analytical chemistry, was formerly a chief chemist of the New York district of the Food and Drug Administration.

Attachment A gives the results of Consumer Union tests on Kent cigarettes over a 4-year period. A table is given which I believe would be difficult to visualize if read but, since everybody here has a copy, I guess I can leave that, Mr. Chairman. (See appendix, exhibit 17, p. 677.)

Kent cigarettes are now available in 3 sizes, although in previous years they have been available in only 1 size, sometimes 2, and only in the last month or so they have become available in 3, so that, in order to keep the record complete, we are giving the figures on all of these different sizes and, where there are blanks, it is to be understood there either were no such sizes available at the time or we, for one reason or another, did not test them at the time.

Mr. MEADER. May I ask, Mr. Michelson, on this chart, are the milligram figures on just one cigarette?

Mr. MICHELSON. In that sense, yes. The actual test was made on 16 cigarettes in each case, but the result is in terms of milligrams per cigarette.

In 1953, the Kent regular size filter cigarettes had 8.5 milligrams of tar per cigarette, and 1 milligram of nicotine per cigarette.

For 1955, the regular size had 12 milligrams of tar and 2 milligrams of nicotine. The king size had 15 milligrams of tar and 2.8 milligrams of nicotine.

The Kent cigarettes reported upon in our March 1957 issue: The regular size had 16 milligrams of tar per cigarette and 2.7 milligrams of nicotine. However, the current Kents now on the market have, according to our tests last week, 11 milligrams of nicotine per cigarette in the regular size, 12 in the king, and 10 in the long, and these same cigarettes have nicotine to the extent of 1.3, 1.7, and 1.6 milligrams, respectively. When we also tested by the total smoke method to compare their advertising with what we would find in the cigarettes themselves, we found 17 milligrams of tar in the regular and 22 milligrams in the king size. They did not give any figures for their long cigarettes in their ads. They compare quite well with the current Kent ad figures which are listed in the last column, 17 and 21 milligrams of tar, respectively. Similar agreement can be noticed——

Mr. MEADER. These are all filter cigarettes?

Mr. MICHELSON. Yes.

Mr. MEADER. And the tests were made, drawing through the filter?

Mr. MICHELSON. Yes.

Mr. PLAPINGER. Is it reasonable to conclude that the current Kent offers less protection than the 1953 Kent?

Mr. MICHELSON. That is a valid conclusion.

Mr. BLATNIK. Are you suggesting—the Kent and Sano are considered the more effective filter cigarettes; is that correct?

Mr. MICHELSON. At the present time?

Mr. BLATNIK. Yes.

Mr. MICHELSON. Yes.

Mr. BLATNIK. On page 4 of your testimony you gave 2 illustrations of the high percentage of total stream reduction; 40 percent of the tars were removed by King Sano, and the same for Kent; is that correct?

Mr. MICHELSON. It may very well be that what I have said can be interpreted in that way, and I think it would be best if I were to clarify an important point here. In all of these tests that we have made—and we have made many on many kinds of filter-tip cigarettes—we have never tested the effectiveness of the filter, per se. We have always been testing only the amount of tar and nicotine which has come through the filter from the tobacco which was being smoked. We, therefore, have been careful to phrase our report in terms of, if I may be allowed to reread that paragraph:

> Without affecting the truth of this generalization, two brands in particular are worthy of special comment. The King Sano filter-tip cigarette, covered in our 1957 reports, was found to give the smoker about 40 percent less tar than the average king-size filter cigarette, and about 50 percent less than unfiltered king-size cigarettes.

You will notice this statement does not say whether the filter is effective, because we do not know how much would have come through had there been no filter. We feel that the King Sano and the Kent cigarettes, as tested at the time we tested them, gave the smoker less tar and nicotine—less tar, particularly—than the average of other cigarettes, filtered and unfiltered.

Mr. BLATNIK. I see. You didn't test the cigarette without the filter?

Mr. MICHELSON. Our comparison is made between other cigarettes—the tars delivered to the smoker by the other cigarettes and the tars delivered by these particular cigarettes.

Mr. MEADER. You say you never have made a test of the effectiveness of the filter, per se?

Mr. MICHELSON. That is correct.

Mr. MEADER. In other words, you wouldn't be able to tell from your experimentation whether or not a better filter was used but cheaper tobacco with harsher elements in it and more tar in the tobacco. All you get is a net result. You never made any examination of the components and the effectiveness of the filter?

Mr. MICHELSON. That is correct. We felt that the sum total of these two factors, the kind of tobacco used and the filter, reacts on the smoker as a combination. To the smoker it does not matter whether he has a very effective filter and a terribly strong tobacco, or a very mild tobacco and a very ineffective filter. If he gets the same amount of tar from either combination, they are equivalent, so far as he is concerned. And so we kept our tests on that basis.

Mr. MEADER. I would like to ask about tar being a loose term and not identifiable. Have you, in your research and experimentation, sought to identify the various materials that are lumped together under the term of "tar"?

Mr. MICHELSON. No, we have not done that. There are many people in the tobacco industry and in other laboratories who have been working on that. There is very extensive literature on the subject. Dr. Wynder, I believe, testified to some of his efforts in separating various components and mentioned the fact that these components will change depending upon the burning temperature of the cigarette.

If the committee would desire it, I could furnish a list of references to articles bearing on the subject, none of which is definitive because nobody knows everything yet that is in cigarette smoke.

Mr. MEADER. Mr. Chairman, I believe it would be useful to have that bibliography on the components loosely lumped together under the term of "tars."

Mr. MICHELSON. I can mail that to the committee.

Mr. BLATNIK. You will supply that for the record? We will appreciate it. (See appendix, exhibit 19, p. 678.)

Mr. BLATNIK. Getting back to this chart on your Kent cigarettes, let's start off with your regular. The tar content of your regular cigarette, in 1953 there is 8.5 milligrams; is that correct?

Mr. MICHELSON. Yes, sir; corrected to the same basis as the later test.

Mr. BLATNIK. 1955 is 12?

Mr MICHELSON. That is correct.

Mr. BLATNIK. 1957, it jumped way up to 16 and only very recently within a few weeks in July, they put on a new filter—these are not filters, these are just regulars?

Mr. MICHELSON. No.

Mr. BLATNIK. What does "regular" refer to?

Mr MICHELSON. The Kents are all filter cigarettes and the only reason I have not so specified in the left-hand margin is that these data are only on Kent cigarettes and Kent cigarettes do not come in any other form than with filter tips.

Mr. BLATNIK. We are getting them in 1957 with the total-smoke method and you have about twice the tar content out of the same type of cigarette that you had 4 years ago in 1953; is that correct?

Mr. MICHELSON. Mr. Chairman, I believe that this comparison is the kind of comparison that I stated would not be valid. You cannot compare tar contents determined by two different methods. The comparison between the 1953 results has to be made by basically the same method where figures are given as 11 for the most recent ones.

Mr. BLATNIK. At any rate you have more tar and nicotine in the same cigarette with the filter than you had 4 years ago?

Mr. MICHELSON. That is correct.

Mr. BLATNIK. The reason I am trying to get this clear in my mind is, if I recall the advertising—and we will go into all of them we possibly can, not just this one—but in 1953 this was called a micronite filter but now it is called "a new exclusive micronite filter"

If I get this "new exclusive micronite filter" it reduces less tar and less nicotine than the original micronite filter did 4 years ago; is that correct?

Mr. MICHELSON. Are you suggesting that the current advertisement would lead a reader to believe that this new current filter would be more effective than previous Kent filters?

Mr. BLATNIK. I couldn't say what they are trying to do but it implies that it is new and exclusive. I can only speak for myself. I don't know what the tobacco companies are trying to do, but it is a new and exclusive filter. I would expect when they say more efficient, it would be a newer model or up-to-date version but what you prove is that there is more tar and more nicotine getting in the 1957 Kent cigarette than we got in 1953?

Mr MICHELSON. That is absolutely correct. That conclusion is clearly shown by these data and it also appears to me to be clear that the current Kent ads can be interpreted to imply that they are actually giving the smoker less tar and nicotine, even though they do not say so

explicitly in comparison with the 1953 Kent cigarettes. Their advertisements do not compare their cigarettes with the 1953 Kents.

Mr. BLATNIK. May I ask you why you just select Kent here? What is the pattern for other filter cigarettes?

Mr. MICHELSON. The pattern in other cigarettes in general is somewhat similar, that they go up and they go down without any apparent explanation.

If the committee is interested I have a chart which traces the history of many cigarettes, brand by brand, in the same way as the Kent cigarettes have been studied in this chart. I have it available.

Mr. BLATNIK. May we see it?

Summary

Brand	Tars			Nicotine		
	1953 [1]	1955	1957	1953	1955	1957
Philip Morris:						
Regular	17	21	18	1.9	2.7	2.8
King size	------	26	------	------	3.4	------
Long	------	------	21	------	------	3.1
Parliament:						
Filter tip	17	14	------	2.8	2.4	------
Long, filter	------	------	20	------	------	3.4
King size, filter	------	15	------	------	2.6	------
Camels, regular	18	20	17	1.9	3.3	3.2
Pall Mall, king size	18	26	24	2.6	3.5	3.0
Kool:						
Regular	19	20	20	2.2	3.4	2.9
King size	------	23	------	------	3.7	------
King size, filter	------	------	16	------	------	3.1
Lucky Strike, regular	19	21	19	2.1	2.7	2.6
Murad, regular	20	21	17	1.1	1.8	1.8
Cavalier, king size	23	26	26	2.8	3.9	3.8
L & M:						
Filter	------	11	15	------	1.5	2.6
King size, filter	------	18	15	------	2.5	2.6
Tareyton:						
King size	------	22	22	------	3.1	2.9
King size, filter	------	19	19	------	2.5	2.3
Regent, king size, filter	------	19	21	------	2.4	3.1
Winston, king size, filter	------	20	22	------	3.1	3.8
Sano:						
Regular	12	15	------	1.2	1.2	------
King size, filter	------	12	11	------	1.0	1.0
Yorkshire:						
Regular	13	15	15	1.2	1.7	1.8
King size	------	18	------	------	2.0	------
Chesterfield:						
Regular	13	17	17	2.0	2.3	2.1
King size	16	22	22	2.5	2.7	3.0
Wings, king size	13	20	------	2.3	2.9	------
John Alden, regular	14	16	21	.4	.4	.7
Old Gold:						
Regular	15	18	18	2.0	2.9	2.7
King size	------	25	19	------	4.0	3.2
King size, filter	------	14	19	------	2.9	3.1
Raleigh:						
Regular	16	------	------	2.2	------	------
King size	------	25	23	------	3.7	3.7
Viceroy:						
Filter	16	------	------	2.4	------	------
King size, filter	------	18	18	------	3.0	2.8
Fatima, king size	16	24	24	2.4	2.9	3.1

[1] Different methods were used for tar extraction in the 1955 and 1957 tests. The 1953 figures have been increased by 20 percent in an attempt to make them directly comparable.

Mr. MICHELSON. This listing, Mr. Chairman, that I have just given you, contains only those cigarettes which we tested more than 1 year. There are many other cigarettes which we tested one year and which either failed to remain on the market, or were changed to a different

kind of cigarette the next year, or were so low in distribution that we did not feel it was warranted that they be tested by us again.

So that these figures actually constitute a part of the whole picture but it gives you a complete picture so far as we have it, of every cigarette which we have tested on more than on one occasion.

For example, the first cigarette listed, the Philip Morris, under "tars" is found to vary, in 1955, it was higher than in 1953. In 1957 it was back down somewhere in the 1953 level.

Mr. MEADER. Mr. Michelson, might I ask, do you have any figures prior to 1953?

Mr. MICHELSON. We have no figures that would be comparable to these, Mr. Meader.

Mr. MEADER. What I am wondering is whether this reflects a reduction in tar content in cigarettes as a result of some publicity about possible deleterious effects of the tar in cigarette smoke.

Mr. MICHELSON. I am unable to answer that question because I don't the information that would be necessary to——

Mr. MEADER. My thought was that in 1952 or 1953, the first adverse publicity of any importance on this subject came out?

Mr. MICHELSON. I believe that is so, yes.

Mr. MEADER. I wonder if the low figures in 1953 throughout the entire listed cigarettes could be connected with that adverse publicity and that perhaps prior to the adverse publicity the tar content had been comparable to the 1955 figure?

Mr. MICHELSON. Mr. Meader, I would be willing to make a guess that the cigarette companies had not been paying much attention to controlling, back then, the tar content of the cigarettes. It is my impression they were more interested in controlling the flavor which would appeal to smokers, but this is entirely a personal opinion of mine. I have seen no references in the technical literature to any attempts made prior to Dr. Wynder's work on the waxes of tobaccos, of efforts to reduce tars by treating the cigarettes so I know of no attempt on the part of the cigarette manufacturers to control the tar levels.

Mr. MEADER. So far as you know, no similar figures exist with reference to periods prior to 1953, on tar and nicotine content of cigarettes?

Mr. MICHELSON. They may exist in the files of the tobacco companies or other laboratories who have not published them. So far as being in the public prints are concerned, there are none that I know of.

Mr. PLAPINGER. Mr. Michelson, in a study prepared by the staff, by company, not unlike your listing of tar and nicotine in the 3 years, the following was found. With respect to the American Tobacco Co., it was noted that in 1953, Lucky Strikes had 2.1 nicotine content and 16 milligram tar content.

This is lower than all of the cigarettes produced by the American Tobacco Co., both with respect to tar and nicotine in 1957. That includes Lucky Strikes, Pall Mall, king size Tareyton, filter king Tareyton and filter king Hit Parade.

The same is true with respect to Liggett & Myers' products, regular size Chesterfield in 1953, it had a nicotine content of 2 milligrams, a tar content of 11 milligrams. This is lower than the nicotine and tar

content for 1957 of the regular sized Chesterfield, the regular filter, the L & M, the king size Chesterfield, the king size Fatima and the filter king L & M.

With respect to the Reynolds Tobacco Co., regular size Camel in 1953 had 1.9 milligrams of nicotine and 15 of tar. This was lower than the Camel, king size Cavalier, filter king Winston and the mentholated filter king Salem in 1957.

With respect to Philip Morris in 1953 the regular size Philip Morris had 1.9 milligram nicotine content and 14 milligrams of tar. Lower than regular sized Philip Morris, king size Philip Morris and the filter king Marlboro in 1957. This is also true with respect to the regular sized Old Gold in 1953 with the 2 milligram content of nicotine, and 12 of tar, as against the Old Gold regular size—not the Murad but the regular filter Kent prior to this new advertisement and the king size Old Gold and the filter king Old Gold currently.

The filter king of Viceroy was 2.4 milligrams of nicotine in 1953 and 16 of tar, as against 2.8, in 1957. The king size Raleigh was 2.1 of nicotine, and 16 of tar in 1953 as against 3.7 nicotine and 23 in tar in 1957.

Tests of cigarettes for nicotine and tar—Consumers Union reports

[Numbers are quantities in milligrams]

Firms and brands	1957		1955		1953	
	Nicotine	Tar	Nicotine	Tar	Nicotine	Tar
AMERICAN						
Lucky Strikes (regular)	2.6	19	2.7	21	2.1	16
Pall Mall (king)	3.0	24	3.5	26	2.6	15
Tareyton (king)	2.9	22	3.1	22	.....	.....
Tareyton (filter king)	2.3	19	2.5	19	.....	.....
Hit Parade (filter king)	3.0	20	.....	.....	.....	.....
LIGGETT & MYERS						
Chesterfield (regular)	2.1	17	2.3	17	2.0	13
L & M (regular filter)	2.6	15	1.5	11	.....	.....
Chesterfield (king)	3.0	22	2.7	22	2.5	16
Fatima (king)	3.1	24	2.9	24	2.4	16
L & M (filter king)	2.6	15	2.5	18	.....	.....
REYNOLDS						
Camel (regular)	3.2	17	3.3	20	1.9	18
Cavalier (king)	3.8	26	3.8	26	2.8	19
Winston (filter king)	3.8	22	3.1	20	.....	.....
Salem (menthol filter king)	3.7	19	.....	.....	.....	.....
PHILIP MORRIS						
Philip Morris (regular)	2.9	18	2.7	21	1.9	17
Philip Morris (king)	3.1	21	3.4	26	.....	.....
Marlboro (filter king)	2.9	18	.....	.....	2.2	16
Parliament (filter king)	.....	.....	2.4	14	2.7	17
LORILLARD						
Old Gold (regular)	2.7	18	2.9	18	2.0	15
Murad (regular)	1.8	17	1.8	21	1.1	17
Kent (regular filter)	2.7	16	2.0	12	1.0	9
Old Gold (king)	3.2	19	4.0	25	.....	.....
Old Gold (filter king)	3.1	19	2.9	14	.....	.....
BROWN & WILLIAMSON						
Viceroy (filter king)	2.8	18	3.0	18	2.4	16
Raleigh	3.7	23	3.7	25	2.2	16

Do those figures generally gibe with yours, or have I been reading too fast for you? These are figures we correlated from the Consumers Union test by company.

Mr. MICHELSON. Can this be off the record?

Mr. BLATNIK. Off the record.

(Discussion off the record.)

Mr. PLAPINGER. I am citing the increases in nicotine and tar content between 1953 and 1957. It is interesting to note in every case in comparing the regular size cigarette produced by a given company in 1953, that the nicotine and tar content is lower than all of the cigarettes produced by that company, regardless of whether filter, filter king, or regular, today.

Mr. MICHELSON. I believe that your conclusion is well founded. The figures you have used to make that point, do make that point; but I believe that a further hypothesis is possible along the lines suggested by Mr. Meader, and that is, had we had more information going back before 1953 and had we had information, let us say, in between the ones we have in 1954 and 1956 for example, we might have found that there is generally a great fluctuation.

The fact that when we tested our samples and reported on them in 1953, we found fairly low—compared to 1955 and 1957—nicotine and tar, that this was just one point on a curve which we happened to hit in a low part of its swing.

You will notice many of the figures in 1955 are somewhat higher than the 1957, as though the figures are beginning to go down again in 1957. This indicated to me there probably is continual variation, from time to time, and that any figures used apply only to the cigarettes at the time the tests were made.

The generalizations about what happened in intervening times are only guesses.

Mr. PLAPINGER. How marked is that variation, generally? Would it be as marked as a nicotine content of 1.9 to 3.2, for instance, over a period of 4 years?

Mr. MICHELSON. I believe that is easily to be predicted from variations in nicotine of the same type of tobacco grown in the same field in a different year. The tobacco literature that I have perused has figures which indicate that a given tobacco type grown on any particular field may vary by as much as 100 percent from year to year and in one particular case a thousand percent over previous occasions.

So that it is my belief that these figures would show variations from year to year in the nicotine content of cigarettes and may be based on the fact that any given kind of tobacco will vary in nicotine content from time to time and that the cigarette companies may make no effort to control the actual amount of nicotine in the cigarette.

Mr. MEADER. Now do you know anything about that, Mr. Michelson, whether cigarette companies do regularly in the production of cigarettes, make periodical tests on each batch of cigarettes and each batch of tobaccos used in the cigarettes to determine the yield of tar and nicotine?

Mr. MICHELSON. I do not know that this is generally true except that a few companies are doing it on a regular basis. The Sano ads indicate they are doing that on a regular basis and the Kent cigarette ads indicate they are going it at present but to what extent other companies are doing it, I have no knowledge, at all.

Mr. MEADER. Have you ever sought information of this character from the tobacco companies, themselves?

Mr. MICHELSON. No, I have not, sir.

Mr. BLATNIK. To sort of summarize what you are stating here, would you look at your March 1957 Consumers Report, which is the last of your three reports, on page 102. Perhaps this will help consolidate this general summary picture.

There you have tests for the average type of filter, regular size, no filter; king size, no filter, king size with filter. For the years 1953, 1955, and 1957. Would you explain that chart?

Mr. MICHELSON. In 1953, regular size, no filter cigarettes, which at that time was the common type of cigarette, had on the average, 2 milligrams of nicotine per cigarette. In 1955, the average of the same type of cigarette had increased to 2.6 milligrams and in 1957, the average was 2.5 which is not significantly different from the 2.6.

During the same period, the regular size with filters of which there were few, had 2.1 milligrams in 1953; in 1955 there was a distinct drop to 1.9 milligrams—this is on the average and does not apply necessarily to any particular cigarette—and in 1957 the average had gone up again—this time to 2.7.

This figure, incidentally, included the Kent cigarette which was tested for this report but which has now been displaced by the new Kent, and so this figure, while true in March 1957, may no longer be true.

Mr. BLATNIK. Let's stop right there for a minute. You have only regular size, no filter; with filter. In both cases we have more nicotine today then we had in 1953, regardless of whether it is a no filter regular or a filter, is that correct?

Mr. MICHELSON. That is correct.

Mr. BLATNIK. In the filter, regular size, you have a little more tar than you have in the nonfilter. Is that right? I mean nicotine. You had 2.7 nicotine in your regular size with filter, and 2.5 today, without the filter.

Mr. MICHELSON. Yes, sir, that is correct.

Mr. BLATNIK. Am I correct in assuming as far as the nicotine content goes in a regular filter, in fact I am better off if I buy a regular cigarette with no filter, is that correct?

Mr. MICHELSON. In regard to nicotine.

Mr. BLATNIK. Now, with regard to tars—let's stick to the filter cigarette. I have never seen anything advertised and publicized as much and that we know as little about as these filter cigarettes. It is somewhat exasperating. It is not directed at you because you are trying to help us. But look at the tars. On a regular cigarette—they have all this fancy business, king size, small, and we don't know what they are—but the regular cigarette, in 1953, the tar content without filter is 17 milligrams, 1955, 18; and 1957, 18. So the regular has a little more tar than before.

Mr. MICHELSON. That is correct.

Mr. BLATNIK. The filter then does reduce the tars—14 in 1953, it dropped down to 12 and went right back up to 16. So on the tars, what is your conclusion on the regular cigarette? You have some protection, you have 2 milligrams less in the filter regular cigarette than in the nonfilter, is that correct?

Mr. MICHELSON. That is correct, sir.

Mr. BLATNIK. How about the king-sized cigarettes?

Mr. MICHELSON. The king-size filter cigarettes' advantage over the regular-size no-filter cigarette is absolutely none, by our figures, on the average. However, they offer an advantage over the king-size no-filter cigarettes to some extent. Whether this extent is significant, I would rather leave to the medical authorities in the field, but at best, according to our tests, the king-size filters at present give no less tar than a regular-size cigarette without a filter, either for 1957 or 1953. According to this latter comparison, they offer no significant advantage for the smoker.

Mr. BLATNIK. Repeat that again now. Do I understand that the king size with filter, which yields in 1957 18 milligrams of tar, gives me just as much tar, no more, no less, as a regular-size no-filter 1957 cigarette; is that correct?

Mr. MICHELSON. That is absolutely correct, sir.

Mr. BLATNIK. So I get nothing here by way of reducing tar level in buying the filter on a king size—do I get any more tobacco in a king-size filter tip?

Mr. MICHELSON. According to the Department of Agriculture, there is no more tobacco in a king-size, filter-tip cigarette than in a regular. In fact there may be even a little less. But the smoker smokes a good percentage more when he smokes a king-size cigarette, regardless of whether there is a filter present or not.

Mr. BLATNIK. Will you repeat that? By smoking more, you mean with a filter tip he smokes most, if not the entire part, of the cigarette; is that the idea? And without the filter tip he leaves a little butt; is that it?

Mr. MICHELSON. Perhaps I would do best to give you some figures: If a regular-size cigarette is 70 millimeters long and a king-size cigarette is 85 millimeters, and a filter is 15 millimeters, the entire difference in length between a regular cigarette and a king-size cigarette may be taken up by the filter. But when a smoker smokes down to about a 1-inch butt on a regular-size cigarette, he has only smoked 47 millimeters of that cigarette; but on a king-size cigarette, by the time he has reached that 1-inch butt, he has smoked 62 millimeters, I believe it is, an increase of 15 millimeters more of tobacco that he has burned.

It may very well be that he is getting more for his money in that sense, more smoking time; but if we compare the total effect on a per-pack basis, then he is getting just as much tar out of a king-size filter cigarette as he is getting out of a regular-size nonfilter cigarette, regardless of the difference in length between the cigarettes. He is actually smoking more tobacco and getting the same amount of tar.

There was a time when some cigarette companies made a virtue of the fact that their cigarettes were longer, they were king size and, therefore, there was more filtering by the tobacco itself. This we firmly believe, on the basis of test results, is fallacious because there is actually more tobacco burned in the longer cigarettes when smoked to the same size butt and that accounts for the differences in nicotine and tar found between regular-size cigarettes and king size. What the filter on the king size seems to have accomplished—and I say "seems to" because I don't know whether it is a change in tobacco or the filter—it seems to have reduced the tar level of a king-sized cigarette to that of a regular size without a filter. Have I made myself clear?

Mr. BLATNIK. Yes; you have. The amount of tobacco is still the same in the king-size filter as it is in a regular but you smoke actually more tobacco in a king-size filter.

Mr. MICHELSON. In any king-size cigarette.

Mr. BLATNIK. On page 100 of that same report you state in the opening column there in the second paragraph, the left-hand side of the page:

A number of interesting facts stand out from a study of the data obtained in this and previous tests.

Would you read those or summarize those? Are those your conclusions?

Mr. MICHELSON. Those are conclusions.

Mr. BLATNIK. Of yourself and your organization after these current 1957 tests?

Mr. MICHELSON. Yes; these were our conclusions at the end of the 1957 tests.

Mr. BLATNIK. Would you read those?

Mr. MICHELSON. I would like to point out before I begin reading these that some of the conclusions may relate to particular brands and as I said before, there are constant changes in the particular brands from time to time so that some of them may no longer be true at present.

Mr. BLATNIK. We are not interested in individual brands at this time but those five general summary statements or conclusions. Would you read those?

Mr. MICHELSON (reading):

King-size cigarettes produce more nicotine and tar in the smoke than the shorter regular-sized cigarettes, if both are smoked to the same butt length. As between filters and no filters there is very little to choose so far as nicotine content of the smoke goes. While the nicotine content is about the same, the average filtered cigarette smoke contains somewhat less tar than unfiltered smoke. So-called low-nicotine cigarettes do show low-nicotine content as compared with others, about a third as much on the average as the ordinary brands.

Unfiltered cigarettes, both regular and king size, are remarkably similar in nicotine and tar levels to their levels of 2 years ago. In contrast, the average nicotine and tar levels of filter-tipped cigarettes have risen.

Mr. BLATNIK. Are there any further questions?

Mr. MICHELSON. Mr. Chairman, I would like to make a few general remarks.

Mr. BLATNIK. Please do.

Mr. MICHELSON. The statement which I have presented on behalf of Consumers Union Laboratory has been confined to technical material. We did not want to editorialize in this factual presentation and we would like the committee to know that we have an interest in the general situation as well as the technical data.

It is our feeling that the Federal Trade Commission can play an important role in protecting the American public in this situation. We feel that they may have been less than the most effective organization in the world in this sphere, partly because of the procedural difficulties they may be operating under, partly because of budget difficulties, but whatever the difficulties that have beset them we feel that Congress would do well to try to find legislative remedies to give them every possible help in this situation.

The fact that the general cancer-cigarette problem has not been completely resolved to everybody's satisfaction (as to whether ciga-

rettes do or do not give people cancer of the lungs) does not warrant ignoring the situation. If there are misleading advertisements in the field of filter-tip cigarettes, we feel that action should be taken by the FTC.

Our organization probably will have an editorial on the subject in one of our coming issues.

I wonder whether it is your intention in this hearing to ask the opinion of Consumers Union any more extensively on the subject of the FTC?

Mr. MEADER. Mr. Chairman, in the light of this voluntary statement made by Mr. Michelson, I would like to ask him whether his organization has taken any position on the question of whether the advertisements of the cigarette industry have been misleading to the general public.

Mr. MICHELSON. The last time we took a position—in print, that is—was some time I believe around 1940 when there was a report by Reader's Digest on cigarettes and I believe it was Old Gold came out lowest at that particular time. Reader's Digest made certain limiting statements about this finding, they felt it didn't have much significance because it was not much lower. Old Golds launched an advertising campaign on the basis of the fact that they were lowest in that particular test and we called the attention of the FTC to this, I believe, and the FTC tried to take action but were unsuccessful because of the time involved. By the time they got to a cease-and-desist order, Old Gold had already switched to another advertising slogan and there was no effect in such a cease-and-desist order. The company wasn't interested in using that advertising any more.

Since then we have felt that the FTC needs prodding but under its procedures and other difficulties, there isn't much possibility of effective action.

Mr. MEADER. Now we are getting off on the second question as to whether FTC has or has not discharged its responsibilities in protecting the public under its law. What I want to get at first was whether your organization had made any study of the advertisements of the cigarette companies and made a determination or at least formed an opinion as to whether or not those advertisements were misleading and misrepresented to the general public—the facts in the situation.

Mr. MICHELSON. We have studied the advertisements in our offices and laboratories. We have come to conclusions but we have not published our conclusions in this field so far as I recall.

If the committee would be interested in our feelings about these advertisements, I would be glad to give you some examples or cite you some examples.

Mr. MEADER. I think that is the subject matter of our hearing.

Mr. BLATNIK. That is exactly what we want.

Mr. Michelson, while I am not quite satisfied for the record—I don't mean I am not satisfied with your testimony—because the three articles I have read, and I have read them carefully and one I have read before, your March 1957 article, is about as good a report on the operation and functioning and the purpose of the filter that I have come across anywhere. I think you have done as careful a job as was possible and I believe you are fair, you are fair in your conclusions, you still leave it open, you suggest there is probable need for further testing on it.

We are having great difficulty in trying to find out just what the filter does, so then we can proceed to see if it does what they claim for it and if you have any further material in your organization or any analysis you have made of claims—I believe you do study the claims made for a product and you run a test on a product, don't you, to see if the test substantiates the claim, and Mr. Michelson, if you or your organization has any information on analyses of the claims made in advertising and see how it stacks up with what you find in a laboratory, we would certainly appreciate it and would like very much to have it.

Mr. MICHELSON. You already have in attachment A, an analysis of the calims of the Kent. The current Kent ads claim certain quantities of tar and nicotine for their regular- and king-size cigarettes and we have found that they are substantially correct. This, however, is a type of case which I would like to come back to, if I may.

There are, in our opinion, two kinds of false advertising, at least. With one the correct facts are given, the material presented to the public is absolutely correct, but through some manipulation of possibly a copywriter—and a clever one, I might add—a very misleading impression can be given to readers. I can cite you one example which was startling to me when I first ran across it a few years ago. Parliament cigarettes were advertising that they had no more than one-quarter of 1 percent of nicotine in their smoke. We finished our tests and found that Parliament had about as high a nicotine content as any of the other cigarettes—higher than most of the others.

That quarter percent was correct, but there was no comparison figure given to show whether others were higher or lower. They made no claim as to being higher or lower. Any implication that it was lower than other cigarettes was purely in the reader's mind. And, by George, it was there in the minds of most people who read it, I believe.

On the other hand, there are ads like Kents, where comparisons are given, and the figures are correct. There the danger lies in the implication that this gives complete protection. I believe Dr. Wynder had something to say about the quantitative aspects of reduction of probability of getting lung cancer depending on the quantity of tars. These figures in the ads are not given in the context of Dr. Wynder's remarks and the reader is left to assume for himself any conclusion, to reach a conclusion on a basis of very limited knowledge but with great desire to continue smoking if at all possible and he may very easily delude himself into feeling that this gives him all the protection that he needs.

So again, there are data, there is some comparison, but again the implication in the reader's mind may be misleading. I believe that even when a company does not make any other claim than that it has a filter tip on the cigarette, it can be a misleading advertisement.

Mr. BLATNIK. What does the word "filter" mean, Mr. Michelson?

Mr. MICHELSON. A filter is a functional word in the sense that it does not define the material that is doing the filtering but it defines the function of filtering. A piece of felt can be a filter. A piece of paper can be a filter. A piece of steel with holes in it, a piece of sintered glass—all of these things can be called filters because they can remove from the materials passing through them undesirable materials, or materials desired to be separated for one reason or another.

Because of this, almost anybody who is told that a cigarette has a filter tip—just the very fact that it has a filter tip—is led to believe that things are being taken out of the smoke. At this particular time when the cancer scare has reached such proportions that officials of various organizations are being quite positive that there is a correlation, when the public mind is very much aware of the danger of cancer, when every time they read an ad of a cigarette, they have this in mind, the presence of the words "filter tip" even without any other claim will give the readers the idea that they are going to get protection from this horrible danger.

Mr. BLATNIK. Mr. Michelson, will you furnish us further material for examination by the staff—our time is running out and we have two more witnesses.

Mr. MICHELSON. I will be glad to.

Mr. BLATNIK. At this point, if there is no objection, I would just like to include in the record articles on cigarettes in your Consumer Reports of February 1953 and February 1955 and March 1957. (See appendix, exhibit 15, p. 622.)

Our next witness is Dr. Walter Wolman, director of the chemical laboratory of the American Medical Association.

STATEMENT OF WALTER WOLMAN, DIRECTOR OF THE CHEMICAL LABORATORY, AMERICAN MEDICAL ASSOCIATION

Mr. BLATNIK. Doctor, we welcome you to the committee. I am sorry we have run a little longer than we anticipated. You have a prepared statement with you, Dr. Wolman?

Mr. WOLMAN. Yes, sir, I have.

Mr. BLATNIK. Will you give us a short background of your professional experience and proceed with your statement.

Mr. WOLMAN. I obtained my bachelor's degree in chemistry from the University of Wisconsin in 1938. I obtained a master's degree from Michigan State College in 1940, and a doctor's degree from the University of Minnesota in 1943. The latter was in chemistry.

Mr. Chairman and members of the committee, I am Dr. Walter Wolman, director of the chemical laboratory of the American Medical Association, in Chicago, Ill. I am a doctor of philosophy in chemistry, not a doctor of medicine. I am here today in response to the request of this committee to discuss a series of studies made by our chemical laboratory on the nicotine and tars in the smoke of several types of cigarettes and the effectiveness of various filter devices in removing nicotine and tars from cigarette smoke. These studies were made during 1952, 1953, and 1954. (See appendix, exhibit 16, p. 668.)

The procedures and methods used were those generally recognized in the scientific literature as acceptable and accurate. The results of the studies were presented in four papers published in the Journal of the American Medical Association. Attached to my statement are copies of these articles with the published graphs summarizing the nicotine and tars contents in the mainstream smoke of the cigarettes and holders tested.

Mr. WOLMAN. My statement shall be a brief summary of these reports. No further work of this type has been done at our laboratory, and, as a consequence, we have no data on cigarettes and filters marketed since 1954.

As you will note, the various cigarettes tested were not identified as to brand names in the articles reporting the results of these studies. Therefore, I have made available to the committee several copies of the key identifying the brand names of these cigarettes.

Mr. BLATNIK. In showing your percentage of reduction, you don't have a third column which would give the level of tar which passes through? That would be the important thing, wouldn't it?

Mr. WOLMAN. That is right.

Mr. BLATNIK. If you have a weak tobacco with low tar content and a very weak and ineffective filter that reduces 10 percent of this, the end product may be a low tar level?

Mr. WOLMAN. That is right.

Mr. BLATNIK. If you have a high tar content cigarette and a more efficient filter—let's say it reduces 40 percent of the tars—you may still end up with a higher tar level.

Mr. WOLMAN. That is true, depending upon the original amount of tar-forming substances present in the tobacco.

Mr. BLATNIK. Do you further clarify that?

Mr. WOLMAN. I believe so. The statement explains that.

Acceptance of laboratory smoking data demands that the procedure used must simulate human smoking and must be reproducible. These conditions were met to a considerable degree by the use of an automatic smoking machine. There are a large number of variables in human smoking, including the rapidity, time, volume and number of puffs, the duration of smoking, and the length of the butt. With an automatic smoking apparatus, it is necessary to assign, arbitrarily, certain values of these factors—values which are reasonably close to the conditions of human smoking. The results and data obtained will depend on these assigned values, but, as long as the conditions are constant, certain comparisons between cigarettes and filters can be made.

The results of such laboratory experiments will depend also to a certain degree upon variables inherent in the cigarettes themselves. Variations are due to type, weight, cut, and tightness of packing. Some of these variations were minimized by using cigarettes that varied no more than 20 milligrams from the average weight of a lot. The weight of nicotine found in cigarettes and tars in their smoke can vary in different lots of the same type of tobacco. The nicotine content of tobacco is subject to a variation depending on growing conditions and cultural practices.

Five cigarettes were smoked for each single determination and the smoking was done in a room kept at constant temperature and constant humidity. For the determination of nicotine, the smoke was collected in a flask containing a given amount of alcohol and acid. After the smoke deposited in the solution, it was made alkaline and then the nicotine steam-distilled into a dilute acid. The nicotine was then precipitated as the silicotungstate, ignited, and weighed. The amount of nicotine in the tobacco was also determined by the methods of the Association of Official Agricultural Chemists.

Tars were determined by collecting the smoke over a given amount of sulfuric acid in a flask. The smoke was allowed to settle for 20 minutes and then the contents of the glass smoke tubes and supplementary tubes were washed into the flask for chloroform. The material in the acid solution of the flask was extracted with chloroform.

The chloroform was evaporated and the residual tars dried for 3 hours at 100° C. and weighed.

Attached to my statement are two pages explaining in detail the methods used in analyzing the nicotine and tars in the cigarette smoke. (See appendix, exhibit 18, p. 677.)

Our first report, published in July of 1953, dealt with the effect of the filters of the three largest selling filter-tip cigarettes on the market at that time. They also represented the three types of filter tips available then, namley, paper, asbestos, and cotton.

The efficiency of the filters in the removal of nicotine and tars was determined by smoking cigarettes with the filter mouthpiece and with the filter mouthpiece removed. The percentage reduction effected by the filter mouthpiece is based on the difference between the two values obtained. The values we reported actually favored the filter, because if the filter, after removal, had been replaced by an equal length of tobacco, the additional tobacco would itself act as a filter.

During the course of the study, changes were made in the filters of each of the three brands and in order to avoid publication of data on obsolete filters, the work was repeated on the new filters. The results of the tests were as follows:

	Percent reduction by filter in the mainstream smoke of—	
	Nicotine	Tars
Brand A1	9	5
Brand A2	14	17
Brand B1	60	55
Brand B2	41	44
Brand C1	14	16
Brand C2	27	23

NOTE.—Fig. 1 in 1st column refers to the original set of filters and fig. 2 to the set of cigarettes with the modified filters.

Actually the filter of brand C2 trapped more nicotine than that of brand B2, but the percentage efficiency was smaller. This is a result of the fact that brand C2 has a larger nicotine content in the tobacco. The overall effectiveness of filters A and C was not large. The brand B filter was much more effective, although B2 was considerably less effective than B1 because of changes made in the original filter.

Our second report, also published in July of 1953, was concerned with special low-nicotine cigarettes. Two brands, E and F, were so-called denicotinized cigarettes, and the third, brand G, contained a tobacco bred for low-nicotine content. The nicotine content for all 3 tobaccos was less than 1 percent in contrast to the 1.8 or more percent found in the tobaccos of other types of cigarettes. Brand G had only about 0.3 percent of nicotine present, but it also contained 0.45 percent of another alkaloid similar to nicotine, called nornicotine. It also contained a significantly greater amount of tars. The tar content of brands E and F were about the same as for other tobaccos.

The third report, published in February of 1954, covered our study on cigarette holders that contained filters. The efficiency of these were found by determining the amount of nicotine and tars remaining

in the holders in addition to that appearing in the mainstream smoke (that is, the smoke which reaches the smoker's mouth in contrast to sidestream smoke.) Holders 1 and 2 used a metal trap such as those found in many pipes. The reduction of nicotine was 5 and 4 percent; and in tars, 8 and 11 percent, respectively, for these two holders. Holder 3 used cylindrical paper filter containing a number of small paper baffles. It reduced the nicotine content of the mainstream smoke by 7 percent and the tars by 9 percent. Holder 4 used a plastic cylinder filled with granules of silica as a filter. It reduced the nicotine by 14 percent and the tars by 21 percent. The most effective holder, No. 5, was one which used an inserted cigarette as a filter. It reduced the nicotine and tar content by about 41 percent. The efficiency of the holders dropped as additional cigarettes were smoked, although holder No. 5 maintained its efficiency even after the 20th cigarette was smoked.

Mr. BLATNIK. At that point, Doctor, could you give us, just on the holders—now—would I be correct in assuming from the testimony you have here, that the best filter is a cigarette, itself?

Mr. WOLMAN. In these particular tests, this showed up best.

Mr. MEADER. Is that because of the material in the cigarette or because it was a longer filter?

Mr. WOLMAN. I am of the opinion that it is largely a matter of the length of the filter.

Mr. PLAPINGER. You say the nicotine and tar was reduced 41 percent in No. 5. Would this depend upon what cigarette was used as a filter?

Mr. WOLMAN. We didn't make tests to that effect, but I would imagine that it would be fairly constant, depending on the tightness of the packing of the cigarette that is used as a filter and the length of the cigarette.

The final report, published in April of 1955, covered regular cigarettes, king-size cigarettes, and additional filter-tip cigarettes which had appeared on the market since the work was done on the first report. In these instances, the same length was smoked in both the king-size and the regular-size cigarettes, but in a second test, king-size and regular cigarettes were smoked down to the same size butts.

The attached reproduction of this fourth article summarizes the results of these tests in table 2. Columns 4 and 9 of this table record the weight of nicotine and tars found in the mainstream smoke. Columns 5 and 10 show the percent reduction of nicotine and tars by the filter cigarettes. If a king-size cigarette is smoked, only the same amount as a regular cigarette, the longer butt will serve to filter out nicotine and tars. However, if the king-size cigarette is smoked to the same size butt as a regular cigarette, more nicotine and tars are in the mainstream smoke.

That concludes my formal statement, Mr. Chairman. I will be happy to attempt to answer any questions that the members of the committee may have.

Mr. BLATNIK. Without objection, we have included with your statement, Doctor, the four reports you have submitted with the detailed information and tables.

May I ask you if the method or the technique used for determining the percentage or the actual amount of tars and nicotine was similar to those used by Consumers people.

Mr. WOLMAN. Yes, sir. There were some minor modifications but it is essentially the same.

Mr. BLATNIK. And the results are the same?

Mr. WOLMAN. They compare very closely, yes.

Mr. BLATNIK. Could you give us a summary statement in general terms of the advantage of using a filter?

Mr. WOLMAN. A filter will reduce the amount of certain constituents of the smoke, or of smoke particles that pass through the filter.

Mr. PLAPINGER. What is the effect of the filter on the nicotine and tar content of the cigarette if the nicotine and tar content of the cigarette is stepped up?

Mr. WOLMAN. We didn't make tests along that line, but I would imagine the efficiency would be pretty much the same. It might drop a little bit because of the volume that goes through but it would be pretty much the same.

Mr. BLATNIK. Doctor, you have brand A1, A2, B1, B2, C1, and C2, and so forth. Not for publication but for study of the staff to check with other tables we have which identify the brands, do you have a key we could have?

Mr. WOLMAN. I thought that had been presented to you.

Mr. BLATNIK. I see we have the key here.

I have no other questions, Doctor. Thank you very much for your cooperation and for your assistance.

The next witness is Dr. C. S. Kimball, executive vice president of Foster D. Snell, Inc., Laboratories. We thank you, Doctor, for making a special effort to come down and give us information which I am sure will be of assistance to the committee.

STATEMENT OF C. S. KIMBALL, EXECUTIVE VICE PRESIDENT, FOSTER D. SNELL, INC.

Mr. KIMBALL. Mr. Chairman and members of the subcommittee, may I state I am Mr. Kimball and I am not entitled to the doctor's degree.

My name is Cyril S Kimball. I am executive vice president of Foster D. Snell, Inc., New York, N. Y., consulting chemists-engineers. I reside at 80 Chittenden Avenue, Tuckahoe 7, N. Y.

I am a chemist and I have been employed by Foster D. Snell, Inc., since the business was incorporated in 1931. We engage in a fairly broad spectrum of activities, largely centering on applied chemical research and development work for industry and the Government. It will be apparent to the committee that neither myself nor my firm are qualified to draw conclusions or express opinions on the physiological effects of smoking cigarettes.

My first experience in carrying our work on tobacco smoke dates back to 1936. At that time we did some work in our laboratories which showed that an equal length of tobacco was a more efficient filter for tars and nicotine than the then current cellulose or paper filters. The following year three investigators at the Aluminum Company of America Research Laboratories confirmed our findings in an article published in the July 1937 issue of Industrial and Engineering Chemistry, a publication to do work on tobacco smoke from time to time up to the present.

We recently carried out tests for Reader's Digest and the results were published in the July issue of that magazine. (See appendix, exhibit 14, p. 604.) It is believed that you will be interested in knowing the technical details of the methods of testing we employed and, therefore, I have turned over to counsel of the committee a report giving these details and literature references. (See appendix, exhibit 14, p. 619.) Copies of this same report have been made available to anyone requesting such information from our client, the Reader's Digest.

It is presumed that you are concerned with the reasons why the data published in Reader's Digest is not in complete agreement with data obtained and published by others. We believe that the discrepancy is due to differences in methodology and in the case of data published several years ago there would be differences in the tobacco used.

As regards the tar content of cigarette smoke, we think it is in order to define what is meant by tar content. We have never seen an official definition for the word "tar" as applied to tobacco smoke. Therefore, it seems logical to accept what I will call the classical definition for the word. We think it means a dark brown or black condensate obtained by the pyrolysis or destructive distillation of organic materials such as wood, coal, shale, and petroleum.

It has a variable composition depending upon the original material, the temperature and other conditions employed in producing it. Tar is a generic word describing a complex mixture of substances of uncertain composition. It is often possible to separate from tars, by extraction or distillation, pure substances of known chemical composition, as for example phenol and naphthalene are derived from coal tar. Tar may also contain inorganic matter, that is to say mineral substances as well as organic matter or carbon compounds.

According to the article published in Consumer Reports for March 1957, showing the tar content of cigarette smoke, the figures are based upon a selective extraction with chloroform of the so-called smoke solution. This smoke solution was obtained by passing the smoke through acidified alcohol. We have determined that upon extraction of the acidified alcohol-water solution containing the tar, with chloroform, we obtain a fractionation with approximately 69 percent being soluble in the chloroform and leaving a residue of 31 percent remaining in the acidified alcohol-water solution. On evaporation of this residual alcohol-water solution we obtain a dark brown tarry residue. I have with me some beakers containing residues which I would like to show you.

No. 1 contains the tar from cigarette smoke as we determine it—for purposes of clarity I will call it total crude tar.

No. 2 contains the chloroform extractable tar from an equivalent amount of cigarette smoke as in beaker No. 1.

No. 3 contains the residue remaining in the acidified alcohol-water solution after extracting out the chloroform soluble tar.

We cannot distinguish the residue in beaker No. 2 from that in beaker No. 3. We hold that our method of analysis measure the total tar in cigarette smoke whereas a chloroform extraction technique measures some unidentified portion of the total tar. It seems reasonable to us that if we are concerned with tars which may be condensed or absorbed by a filter in a cigarette then the total tar content should be measured.

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The purpose for which an analysis is made is important in selecting a method of analysis. In this case, we are trying to find out what the average smoker gets in the mainstream smoke of his cigarette, whether it is a filter-tip or a plain-tip brand, and irrespective of any physiological effects.

The two important components we are familiar with are tar and nicotine. To have significance for human smoking, we think it is necessary to recover and weigh all—or as much as possible—of the tarry substances in the mainstream smoke.

Until some recognized authority tells us which portion or fraction of this tarry substance has some particular significance, we cannot justify extracting selectively only some specific part of the total tar. We should extract and weigh all of the substance that the smoker would take in.

In determining nicotine content of the smoke, we used the standard method of the Association of Official Agricultural Chemists, an organization whose membership comprises workers in State and Federal Government agencies. We obtained good reproducibility of results. We have not had experience with ultraviolet spectrophotometric method of analysis for nicotine content, the method employed in the Consumer Reports data. We believe that the data we obtained for nicotine content is comparable with other data which was obtained by the same method of analysis.

There is one additional point which may be of interest to the committee. We smoked all cigarettes to the same butt length, namely 23 millimeters or just under 1 inch. This has been the practice of most investigators in the United States and it dates back before the advent of king-size cigarettes.

The 23 millimeter butt length has been applied in most published studies that since have included king-size cigarettes.

However, there is the question of what is the average butt length to which the average smoker smokes a cigarette, and does this average vary with regular- and king-size cigarettes.

Frankly, we have made no statistical survey to try to establish these facts. As an individual I find that when I smoke a filter-tip king-size cigarette I smoke it to approximately the same butt length as a regular cigarette and my direct observation of other smokers indicates that this may be the general practice.

That concludes my formal statement.

Mr. BLATNIK. Without objection, we will insert in the record a 2-page methodology employed in the interpretation of tar and nicotine content of cigarette smoke for Reader's Digest, signed by Mr. Kimball, who testified here this morning, and to include the two Reader's Digest articles for July and August of 1957, in the July issue, The Facts Behind Filter Cigarettes; and in the August issue, Wanted: Available Filter Tips That Really Filter. (See appendix, exhibit 14, pp. 604, 612.)

Mr. BLATNIK. Doctor, some of us have gone over the two articles on filter cigarettes. Were those articles written under your direction or supervision or did the authors merely have access to the laboratory findings which you have made available to The Reader's Digest?

Mr. KIMBALL. We made the data available to The Reader's Digest and had no connection with the article.

Mr. BLATNIK. Did you review the article? Were the conclusions justified?

Mr. KIMBALL. I read the article prior to publication but only for the purpose of making certain that there was no reference to our name in connection with same.

Mr. BLATNIK. The interpretation or evaluation of your laboratory findings is entirely the responsibility of the authors and The Reader's Digest publishers?

Mr. KIMBALL That is correct.

Mr. BLATNIK. Would you have any comments to make from your experience and your laboratory work on the effectiveness of filters? In general what is their efficiency?

Mr. KIMBALL. Filters do remove tar and nicotine taken from the smoke. Now, I think we may be confused when we resolve this down to a per cigarette basis. Inasmuch as previous witnesses have pointed out that when you smoke an 85 millimeter cigarette, king size, and relate that to a regular cigarette of 70 millimeter size, you are smoking more tobacco in the case of the king size cigarette. You expect, therefore, that you will find more tars and more nicotine when you smoke more tobacco.

Mr. MEADER. Mr. Kimball, with respect to these beakers with the tarry content in the bottom, can you tell us how much cigarette smoke was used to produce the amount of tar you have in beaker No. 1?

Mr. KIMBALL. I am not exactly certain but I believe that was from five cigarettes. This was made for demonstration purposes and it may have been more.

Mr. MEADER. Do I understand that beaker No. 2 contains that portion of the tar which can be dissolved in chloroform?

Mr. KIMBALL. Yes, sir.

Mr. MEADER. And the content of beaker No. 3 is the remainder that was in the acidified alcohol?

Mr. KIMBALL. That is correct.

Mr. MEADER. You say you have no way of distinguishing as far as chemical composition is concerned from the chloroform soluble tars and the alcohol soluble tars?

Mr. KIMBALL. That is correct.

Mr. MEADER. You did not attempt to break down the chemical components of this tar?

Mr. KIMBALL. We made no attempt, sir. In the first place, the tars have not been completely identified. They are of unknown composition. Therefore, one would hardly analyze to determine whether or not you get all of the tars when you make an extraction with chloroform. How to distinguish between the two when you are starting with something of unknown composition is impossible.

Mr. MEADER. It would be logical to assume that the tar contained in beaker No. 2, soluble in chloroform, would be of different chemical composition than the other which was not and remained in the acidified alcohol solution?

Mr. KIMBALL. It would be logical to think there would be some differences in composition. We have made a selective extraction by extracting the chloroform but we have not tried to identify exactly what the composition is that was soluble in the chloroform. From appearances, the matter soluble in chloroform and the matter which remained in the acidified alcohol water solution appear to be the same.

Mr. MEADER. That is simply from examining it with the naked eye and not testing of any kind with scientific instruments?

Mr. KIMBALL. Yes, sir.

Mr. PLAPINGER. Apparently your findings confirm that of the Chesterfield, Lucky Strike, Camel, and Old Gold, each of those cigarettes have less tar content than the king sized filter tips made by the same company. I am not sure I am correct there.

The Chesterfield regular has 32.7 milligrams of tar. L & M king filter has 38.5.

Lucky Strike regular has 31.5 milligrams of tar. Hit Parade has 36.3.

Camel regular has 31 milligrams of tar and the Winston king filter has 32.6 milligrams.

Old Gold regular has 30.9. Old Gold filter-tip king has 39.

Mr. KIMBALL. That is correct.

Mr. BLATNIK. Would that indicate that in each case the regular non-filter cigarette has less tar in the main-stream smoke than the king size with king-size filter?

Mr. KIMBALL. Yes, when both are smoked to the same butt length.

Br. BLATNIK. If there are no further questions, we thank you very much Mr. Kimball. We appreciate your assistance.

Mr. Stephen E. Wrather, Director, Tobacco Division, Agricultural Marketing Service, Department of Agriculture.

STATEMENT OF STEPHEN E. WRATHER, DIRECTOR, TOBACCO DIVISION, AGRICULTURAL MARKETING SERVICE, DEPARTMENT OF AGRICULTURE

Mr. BLATNIK. Will you please proceed with a brief biographical summary background? Do you have a prepared statement with you?

Mr. WRATHER. I do not. Mostly statistical tables showing the price of tobacco.

I was born in Kentucky. I grew up on a tobacco farm and attended the University of Kentucky. I was on the staff of the Kentucky Experiment Station for about 6 years. I moved to Washington in 1940 and have been here since then. I have been with the Department of Agriculture most of this time. I did serve a sentence of about 2 years in the OPA during the war. Currently I am Director of the Tobacco Division of the Agricultural Marketing Service.

Basically as I understand the interest of your committee, we establish and promulgate the grades for tobacco. After setting up the standards or the grade for tobacco, we proceed to inspect the tobacco, on the auction markets before it is sold. That is a matter over which the growers have jurisdiction in that before we move in and inspect the tobacco on an auction market a referendum would be held and if two-thirds of the growers voting approve the inspection, and along with the inspection goes the Market News Service, the quoting of the prices—then we would take it on as a regular operation.

All of our auction markets are inspected. That includes our southern types of tobacco. All of our flue-cured, all of our burley, all of our southern Maryland, all the dark types of tobacco. This program does not prevail in the cigar-leaf producing districts largely because of the types of markets they have. They have no centralized market where tobacco is assembled for purchase, and so forth. In the cigar-leaf areas

tobacco is bought by private treaty between the purchaser and the grower at the producer's farm.

As I indicated, I do not have a prepared statement. I would appreciate some indication as to what I could contribute to the Committee.

I would say to begin with that the inspection and grading of tobacco is an extremely complicated operation. We have in flue-cured and burley tobaccos, 120 grades. Actually we have more than that. We have some off-factored grades. We would make in flue-cured and burley tobacco perhaps 140 or 150 grades. So you can see when you arrange tobaccos into grades and have that many grades, it gets to be a rather involved grading system. I don't know how far we can get into the grading system. I do have some pamphlets here that I thought if you were interested I might distribute and point out a few pertinent factors in connection with our grading system for tobacco. I thought it might help in the interpretation of the grade prices which I think you are interested in.

Mr. PLAPINGER. Mr. Wrather, I think some of the committee's principal interest might be summarized in this statement from Consumers Reports of March 1957, and we would like to hear your comments on it. The last paragraph in the first column, page 104. It starts:

> What kinds of tobaccos are going into filter cigarettes these days seems to be largely each company's secret. Manufacturers are reported to be switching from light-colored, light-bodied, flue-cured tobacco, to darker, leafier bodied leaf in an effort to compensate for loss of flavor that occurs as the smoke paces through the filter. In making this switch they are turning the tobacco market upside down. The price of low-grade darker leaves which used to be much lower than that of flue-cured leaf is climbing rapidly while high-grade flue-cured tobacco is bringing bids below Government support prices and going begging at that. At the end of last year, the supply of flue-cured leaf on hand had mounted to a record three and a half billion pounds, enough to last almost 3 years. Even at the old rate of consumption. As a result, the Government, which in connection with the price-support program exercises strict control of the tobacco acreage, has ordered a 20 percent cut in flue-cured growing for 1957.

Mr. WRATHER. Basically I will have to talk to you in terms of prices in the market place. I would like to point out in general the situation that we seem to be facing. I think it is true that for the filtered cigarettes, manufacturers are using a different blend from what they used in what we have called standard cigarettes. It also follows that as we approach the introduction of the filtered cigarettes, they had rather large inventories of leaf tobacco bought to put in standard cigarettes.

Now, with their volume of standard cigarettes going down each day, their inventory of those tobaccos for those cigarettes durationwise becomes longer each day without acquiring any additional tobacco.

It also follows that they were short inventorywise on these tobaccos they are using as a new blend for their filter-tipped cigarettes.

We have had rather intense pressure in the market for this new blend of tobacco.

Mr. BLATNIK. What is that blend? The tobacco that was used earlier was not used in the old quantities? Could you identify it more specifically than call it a new blend?

Mr. WRATHER. Largely they have moved up the stalk, I would say. As you begin with the lower part of the plant you have certain grades of tobacco which we classify as flyings, or lugs. As we go up the plant we get into cutters and then we wet into leaf tobacco.

Now, the leaves on the lower part of the stalk are extremely mild. They show ground injury and during the era when we were talking about a lot about a mild cigarette, those tobaccos were in tremendous demand because we were making milder cigarettes and then we made them milder and milder.

In the meantime, plant breeders and agricultural experiment stations bred tobaccos in that direction and they too got milder and milder tobacco until I wouldn't be surprised, as far as the consumer is concerned, but what we somewhat overshot the mark. I think our standard cigarette boys were in trouble with a mild cigarette—I am talking now about consumer acceptance. I am not speaking from a health point of view.

So when we began to introduce a cigarette which had a little different blend of a little heavier-bodied tobacco——

Mr. BLATNIK. Was that a less expensive tobacco?

Mr. WRATHER. To begin with, Mr. Chairman, it was because there was a substantial supply of these tobaccos, including loan inventories, and they were immediately acquired.

Mr. MEADER. In loan inventory, do you mean in the Government warehouses?

Mr. WRATHER. The tobacco was collateral for loan under the Government price-support programs. These tobaccos regularly are stored in commercial warehouses.

Currently this price spread does not prevail. You take the burley market last year, there was very little difference pricewise in these tobaccos. Regardless of what quality you are thinking about. The flue-cured markets are in progress now and I understand that in Georgia and Florida, these tobaccos are being bid up in the price ranges of the thinner tobaccos.

Mr. BLATNIK. Would you repeat that last part? Your flue-cured tobacco is your lighter, finer, and is less in tar and nicotine content and in short, it is your more expensive and better tobacco?

Mr. WRATHER. Historically those tobaccos brought the higher prices because our domestic manufacturers year after year were going back for their requirements for a mild popular standard cigarette and those tobaccos were bid up.

I indicated earlier that manufacturers have large holdings of these tobaccos and I rather expect their requirements for these tobaccos are declining along with the sale of standard cigarettes. As a result of this inventory position, manufacturers are concentrating their purchases on the heavier bodied tobaccos. We must remember that manufacturers carry or attempt to carry about 2½ years' supply of tobacco. Accordingly, if he has an annual manufacturing requirement of 10 million pounds, the manufacturer would be in the market trying to buy 25 million pounds of tobacco.

So with all manufacturers having success with filtered cigarettes, and in trying to accumulate an inventory of tobacco for these blends, tremendous price pressures have been exerted on those grades of tobacco. Particularly in relation to the tobaccos which they are long on, inventorywise, due to falling off in consumption of standard cigarettes.

I have some tables here showing the breakdown of tobacco in grade groupings and showing how the prices of these different kinds of tobacco have behaved, beginning in 1952 and continuing through 1956.

Mr. BLATNIK. Without objection, they will be placed in the record at this point.

(The documents above referred to are as follows:)

Flue-cured prices, loan receipts, and holdings by grade groupings, 1952–56 crops

[Cents per pound]

Grade groupings	Market price					Loan rates				
	1952	1953	1954	1955	1956	1952	1953	1954	1955	1956
Thin and fairly thin bodied	66	67	66	65	64	64	63	63	63	62
Medium bodied	63	64	62	61	62	57	56	56	57	58
Fairly heavy bodied	50	48	51	54	53	42	40	40	40	44
Dark and green	34	32	32	40	40	29	27	26	27	31

Grade groupings	Loan receipts (percent of total)					Current loan inventory (percent of total)
	1952	1953	1954	1955	1956	
Thin and fairly thin bodied	35.3	15.9	35.4	50.2	43.7	49.9
Medium bodied	38.9	26.2	32.7	46.8	51.5	46.7
Fairly heavy bodied	7.4	19.6	8.0	.9	1.8	1.1
Dark and green	18.4	38.3	23.9	2.1	3.0	2.3

	1952	1953	1954	1955	1956
Market price (cents per pound)	50.3	52.8	52.7	52.7	51.5
Overall loan level (cents per pound)	50.6	47.9	47.9	48.3	48.9
Average value loan receipts (cents per pound)	37.6	31.7	41.6	55.1	49.4
Loan receipts (million pounds)	165.0	151.4	130.3	298.9	319.9
Loan receipts (percent of crop)	12.2	11.9	9.9	20.2	22.5

Source: Tobacco Division, Agricultural Marketing Service, July 1957.

Burley prices, loan reecipts and holdings by grade groupings, 1952–56 crops

[Cents per pound]

Grade groupings	Market price					Loan rates				
	1952	1953	1954	1955	1956	1952	1953	1954	1955	1956
Thin to medium bodied	68	66	65	64	66	65	64	64	63	63
Medium bodied	56	54	51	58	65	50	48	47	47	49
Medium heavy bodied	48	47	43	55	65	41	38	38	38	41
Red, dark, green	30	29	28	46	61	27	23	22	22	26

Grade groupings	Loan receipts (percent of total)					Current loan inventory (percent of total)
	1952	1953	1954	1955	1956	
Thin to medium bodied	14.8	49.6	54.4	97.9	99.2	84.0
Medium bodied	13.5	15.2	31.5	2.0	.8	15.9
Medium heavy bodied	14.5	4.5	8.5	.1	----	.1
Red, dark, green	57.2	30.7	5.6	----	----	----

	1952	1953	1954	1955	1956
Market price (cents per pound)	50.3	52.5	49.8	58.6	63.5
Overall loan level (cents per pound)	49.5	46.6	46.4	46.2	48.1
Average value loan receipts (cents per pound)	34.9	46.0	53.7	62.7	60.0
Loan receipts (million pounds)	104.0	102.0	221.0	73.0	6.0
Loan receipts (percent of crop)	16.0	18.2	33.4	15.3	1.2

Source: Tobacco Division, Agriculture Marketing Service, July 1957.

Mr. MEADER. May I ask for a description of the criteria through which they grade these tobaccos. I presume the tables are geared to those grades.

Mr. WRATHER. That would be true.

Mr. MEADER. We should have something in the record that describes the grading.

Mr. BLATNIK. Give us a greater description of the procedure and the method of grading. Do I understand that the United States Department of Agriculture makes the final determination in the grade?

Mr. WRATHER. That is true.

Mr. BLATNIK. And the same procedure is used in all tobaccos in determining the grade and thereby, their possible price on the open market.

Mr. WRATHER. If you people will open that pamphlet on pages 6 and 7—I wondered if we could discuss this pamphlet for a couple of minutes here without trying to put it in the record because I am afraid it won't make very good reading in the record. It depends on what your interests are in connection with it.

Mr. BLATNIK. Off the record.

(Discussion off the record.)

Mr. BLATNIK. Without objection, the statement, "Tobacco Inspection, Market News, and Demonstration Services (Type 31–Burley)" will be included in the record.

(See appendix, exhibit 20, p. 684.)

Mr. WRATHER. We would have the same thing for the flue-cured and the other types but this is the pattern.

Looking at pages 6 and 7—particularly 6—a grade of tobacco really has three symbols. The first one would indicate the group. We have on the tobacco plant, three groups of grades. The flyings, that would be X as far as our grade symbol is concerned.

If we had a flying of third quality in lemon color, the grade symbol would be X3L. That means it is in the lug group, third quality and lemon in color.

That would be true on up the stalk or in all groups. We get a large number of grades when you consider all groups, qualities, and colors. On page 10, the first column to the left, the long column, are the groups referred to. At the top of the column we have the B groups. That is leaf tobacco and reading across the page you will note you could have leaf tobacco in any of the five different qualities and in any of the different colors. So you get a tremendous number of combinations of grade.

Now historically——

Mr. MEADER. Mr. Wrather, how do you determine this "choice," "fine," "good," "fair," and "low"?

Mr. WRATHER. We have spelled out grade specifications for each grade of tobacco. Maturity would be a factor, freedom from injury would be a factor, porosity would be a factor and for each of these 120 grades, we have a description of the physical factors making up the grade. All of our grade work in tobacco is predicated on physical factors and characteristics which we can distinguish by an examination of the tobacco.

Mr. MEADER. Now would they have any relationship to the chemical composition or amount of tar or nicotine that would be produced by that leaf?

Mr. WRATHER. I am afraid you are getting me in the wrong laboratory as far as chemistry is concerned.

I would say this and I am not prepared to discuss it because I am not a chemist, some years ago we initiated a project trying to relate our grades which were established on a physical basis, to chemical constituents in an effort to determine what relationships we might get.

But we did that for a purpose I think quite different from your interest. If there is some better way of setting up tobacco grades, we would like to be aware of it.

As I say, these physical standards sometimes seem rather crude. If you know tobacco, you know it is a pretty difficult commodity to judge and there is always a lot of—oh, some disagreement about the grading of tobacco. So in an effort to determine if there was some better or, more concrete basis for establishment of grades, we did explore the chemical constituents of tobacco, on a Federal-grade basis.

Relating chemical analysis to Federal grades is, I think, quite different from the chemical analysis that is done generally on tobacco.

Mr. PLAPINGER. What does heavier-bodied tobacco mean, Mr. Wrather? You referred to that before.

Mr. WRATHER. Just what the term indicates. That the leaf is a little thicker——

Mr. PLAPINGER. The physical characteristics?

Mr. WRATHER. That is right. If you look at this tobacco plant here, beginning at the ground and working up, you would generally have your thinnest tobacco near the ground and as you move up through your flyings, through your cutters, into your leaf tobacco, you get into a little heavier-bodied tobacco.

Now historically, the stronger demand was in these flyings and cutter grades, these milder, thinner tobaccos.

Mr. PLAPINGER. Milder isn't a visual, physical characteristic. Does "heavier bodied" mean stronger tobacco?

Mr. WRATHER. I think generally that would be true. I think these extremely thin, tissuey tobaccos are mild and as you get into a bodied leaf of tobacco you would have what we think of as stronger tobacco.

Mr. MEADER. That would mean that it most likely would have greater tar content and greater nicotine content, the higher up the plant you go?

Mr. WRATHER. I am really not prepared to say chemically what the relationships would be.

Mr. MEADER. I don't know whether you completed this study that you made to see whether or not there was any chemical composition of the various classes that you have here of the tobacco plant, as related to your grading.

Mr. WRATHER. This would be a different category altogether from what you are talking about. For example, I don't think we made any analysis on tar. I just heard a witness say he didn't know what it was chemically.

We are interested in nitrogen content, nicotine content, ash content, and a few substances like that, rather than tar and some of the things I have heard discussed in this hearing.

Mr. PLAPINGER. Mr. Wrather, haven't there been chemical analyses made by the Department of Agriculture, on the various types of tobacco?

Mr. WRATHER. Well, I know only of the work that we are doing and we have made rather extensive analysis on a grade basis of these burley tobaccos and of the flue-cured tobaccos.

Mr. PLAPINGER. Chemical analyses?

Mr. WRATHER. Yes.

Mr. PLAPINGER. Would you tell us something about that, please?

Mr. WRATHER. I could submit for the record the tables and the analytical work that was done. As far as attempting to discuss the chemical properties of tobacco to you——

Mr. PLAPINGER. What are these chemical analyses directed toward?

Mr. WRATHER. In our work we are responsible for the development——

Mr. PLAPINGER. Incidentally, I don't mean to foreclose your submitting anything for the record.

Mr. WRATHER. I understand that.

We are responsible for developing tobacco grades. Any work we do we have to relate it to the development of tobacco grades. Now when it gets into the field of chemistry of tobacco, per se, or classical chemistry, we don't have that kind of money, so to speak. We are restricted.

Now the work we did as I indicated a while ago, was exploratory, trying to determine whether or not we had a rather logical basis for establishing grades, in these physical standards which we have established, and whether or not we could find some other criteria that might help us in establishing grades of tobacco.

Quite frankly, I don't know how familiar you people are with the tobacco market.

Mr. PLAPINGER. I think we would like to be familiar. That is one of the things we would like to hear from you.

Mr. WRATHER. It is like a house afire. You are likely to get run over mighty quick at a tobacco sale.

It moves on pretty rapidly, I would say. These tobaccos are placed on huge warehouse floors in baskets about 3½ or 4 feet square and the tobacco is placed on the basket and arrayed in rows across the warehouse. We go in front of the sale and grade the tobacco. The sale moves very rapidly. Say 400 baskets an hour.

We have got to have some system of grading tobacco where we can move on and get out of the way of the sale, or else the method of sale would have to be modified. As far as placing a chemical analysis on each basket of tobacco, you are talking about something we aren't prepared to do. We haven't explored it with that in mind.

Mr. BLATNIK. Not to interrupt but let's get back to some things you might be able to help us on. Give us a better picture on what has happened in the tobacco market, let's say since 1952. Give me a picture in terms of the shift in the type of tobacco which is selling faster and on which the price has gone up, the shift in another volume of tobacco and so forth.

In short, what has been the change in the price and the volume?

Mr. WRATHER. That is the information I have presented for the record.

I show that rather completely here for the two big cigarette tobaccos, and that is burley and flue-cured.

Mr. PLAPINGER. Are manufacturers switching from light colored, flue-cured tobacco to darker, heavy-bodied leaf?

Mr. WRATHER. In flue-cured we have had a development that perhaps I should explain. As we were looking more and more for thin, mild tobaccos, the plant breeders kept breeding in that direction. Some 3 years ago they introduced some varieties that were extremely light in color. They were mild to the extent that they have been referred to as neutral tobaccos.

The domestic buyers did not want this tobacco and bought very little of it. The importing countries said that they could not use this tobacco, since it was lacking in flavor or aroma and if they had to buy a neutral tobacco they might as well conserve their dollars and buy Rhodesian tobacco or tobacco from other countries.

As a result of that, the Commodity Credit Corporation for this immediate crop reduced its price-support level on those tobaccos by 50 percent of the support on comparable grades of the varieties. So they are trying to discourage the production of these extremely light-colored tobaccos as far as flue-cured is concerned.

Generally speaking as I have said, there has been more pressure on these medium, heavy-bodied grades in the past 3 or 4 years.

Mr. PLAPINGER. What do you mean by pressure, have there been more sales?

Mr. WRATHER. There is more demand for them and there is more interest in acquiring tobacco of that type by our domestic people than we had experienced prior to some 3 or 4 years ago.

Mr. PLAPINGER. Is flue-cured tobacco bringing bids below government-support prices?

Mr. WRATHER. Currently?

Mr. PLAPINGER. Yes.

Mr. WRATHER. A very small percent. The market just opened last Thursday and less than 2 percent is moving under loan, which is a very low percentage but they have a short crop.

As it is referred to in this statement here, the acreages were reduced 20 percent. It is a short crop, the production is definitely below annual requirements so we don't anticipate much tobacco moving under loan in flue-cured.

Mr. PLAPINGER. Incidentally, is this statement accurate? Are there any inaccuracies in the statement?

Mr. WRATHER. I would tell the story differently from what he has told it. I think if he wants to give it that sort of a slant, he would have some basis for telling it that way. The thing he leaves out as far as I am concerned is the fact that you are really buying the market today, for two kinds of cigarettes. On the one hand you have excessive inventories. The manufacturers have excessive inventories of certain kinds of tobacco.

Mr. PLAPINGER. The mild tobacco?

Mr. WRATHER. For their standard cigarettes. So they are not buying much of those tobaccos and they are rather choosy.

On the other hand they are short of bodied or upstalk tobacco and their requirements for these tobaccos, because of sales of filter cigarettes is going up daily. So if you are going to build up an inventory of a 2½ year's supply and your competitor is trying to do the same thing, you have tremendous pressure on the kind of tobaccos you are talking about.

Mr. PLAPINGER. But this has been truer since 1953 than it was before?
Mr. WRATHER. That is true.
Mr. PLAPINGER. It wasn't true before?
Mr. WRATHER. That is right.
Mr. PLAPINGER. The demand in the period prior to 1952 and 1953 was for the mild tobacco?
Mr. WRATHER. That is right.
Mr. PLAPINGER. Now what was determined to be an inferior grade is now in demand and in short supply?
Mr. WRATHER. I wouldn't agree with the inferior grade.
Mr. BLATNIK. What word would you use, is it a lower grade?
Mr. WRATHER. It is a heavier bodied tobacco.
Mr. BLATNIK. Perhaps it was cheaper in price before?
Mr. WRATHER. It was because it moved under loans and the demands were for the other kinds of tobaccos.
Mr. BLATNIK. Give us an example of what was the price in 1952 of this heavier grade?
Mr. WRATHER. In flue cured in the table I have presented here, "Market prices in 1952," are you following that? (See p. 191, supra.)
Mr. BLATNIK. Yes, I have it.
Mr. WRATHER. Let's move down to the table under 1952. Thin and fairly thin bodied, that was 66 cents a pound. Medium bodied, 63; fairly heavy bodied, 50; dark and green, 34.
Mr. BLATNIK. That is your flue cured?
Mr. WRATHER. That is right.
Now move across the table to 1956 and come down in the same sequence.
Thin and fairly thin bodied, 64; medium bodied, 62; fairly heavy bodied, 53; dark green, 40 cents. It moved up.
Mr. BLATNIK. In fact the lowest part in 1952 is your dark and green at 34 cents a pound and now that is up to 40 cents a pound?
Mr. WRATHER. That is true.
Mr. MEADER. It has gone up 6 cents while your thin and fairly thin bodied has gone down 2 cents a pound.
Mr. WRATHER. That is true. That is true.
Mr. JOHNSON. Mr. Wrather, wouldn't it be correct to say that the cigarette manufacturing industry since the introduction of the filter cigarette has sought a tobacco for the filter cigarette which would give to the smoker essentially the same flavor, the same strength of smoke that he would get with a regular cigarette?
Mr. WRATHER. I think that statement is true. I am not at all sure, Mr. Johnson, but what he has wanted to go a little further than that. I think we were in difficulty with our standard cigarettes in 1952. I think we had gotten them so darned mild they were having trouble peddling them.
Mr. JOHNSON. With regard to filter tips as opposed to other types of cigarettes, in effect what the tobacco industry has done, while it puts a filter on the end of the cigarette because the public wants it, they have introduced a modified blend of tobacco so that essentially the consumer will get the same smoke whether he smokes regular or whether he smokes filters? Isn't that right?
Mr. WRATHER. I think that is true.
Mr. BLATNIK. Mr. Wrather, let's go to your burley prices.

Mr. WRATHER. You get more contrast in burley.

Mr. BLATNIK. Much more contrast. We start off with what you call your prime part of the tobacco leaf, your thin to medium. In 1952 it was 68 cents a pound and now it drops slightly to 66. It dropped by 2 cents.

In medium bodied in 1952 it was 56 cents a pound. And in 1956, 65 cents a pound. Going down to your medium leafy bodied at 56 cents a pound in 1952, today it is up to 65 cents, just a cent below your premium quality thin to medium bodied. (See p. 191, supra.)

Mr. WRATHER. That is right.

Mr. BLATNIK. Going down to your lowest grade, your dark green burley tobacco, in 1952 it was 30 cents a pound and now is 61 cents a pound, which is more than twice.

Mr. WRATHER. That is right. We have this difference between the burley and flue cured. For the past two seasons and for the crop coming up, burley production has been running below disappearance. Where in flue cured it has been going the other way, very substantially.

Mr. BLATNIK. How much tobacco of all types does the Government have in the warehouses stored as surpluses now? Do you have these figures?

Mr. WRATHER. 1,040 million pounds held as loan collateral.

Mr. BLATNIK. We will correct that but it is approximately 1 billion pounds. Can you divide that between your burley and your flue cured?

Mr. WRATHER. I can give that for the record.

Mr. BLATNIK. And compare it with 1952 to see if there is any shift.

(The information is as follows:)

Loan stocks, July 1, 1957

[Million pounds, farm sales weight]

Flue cured	643
Burley	281
All other	116
Total	1,040

Mr. WRATHER. I show that, in a way, in the tables I have presented here. Look at the bottom part of the table there.

Mr. BLATNIK. On which sheet?

Mr. WRATHER. Either one of them. It is in the same place, whichever one you are looking at.

Mr. BLATNIK. I have the burley sheet now.

Mr. WRATHER. It shows down here on the bottom portion of the table, the loan receipts in millions, the quantity they took out of each of those crops, 1952 through 1956.

Mr. MEADER. That is not broken down with respect to these grades, is it, Mr. Wrather?

Mr. WRATHER. In the middle table if you will notice, the right-hand column of the table in the center, "Current loan inventory, percent of total," that will show you what their inventory holdings, what percent are in these different grade groupings which we are talking about.

Do you see that, on burley? Eighty-four percent is in the thin to medium bodied, 15.9 in the medium?

Mr. PLAPINGER. In the middle, the red, dark and green, would be the heavier.

Mr. WRATHER. That is right.

Mr. PLAPINGER. And there you have no inventory, is that right, no inventory. While in the mild you have 84 percent.

Mr. WRATHER. Eighty-four percent of their holdings in the burley would be in those grades.

Mr. BLATNIK. Mr. Wrather, years ago did the Department of Agriculture participate in developing and encouraging the development and growth of these mild tobaccos?

Mr. WRATHER. Yes.

Mr. BLATNIK. What is the purpose in encouraging that type of tobacco?

Mr. WRATHER. Well, I think it is rather obvious that we went through a decade, or perhaps more than a decade, of a rather standard product, a very mild, acceptable cigarette which seemed to get milder—the consumer wanted it milder year by year, and we had rather standard requirements as far as leaf was concerned.

Naturally, the plant breeders tried to affect the crop composition as much as they could in that direction. So, all of our breeding and all of our educational work, not only with the Department, but through the experiment stations and the extension forces and everywhere, we thought we knew what we wanted, and we were encouraging people to move in that direction.

It was a natural sort of a thing to do. I would like to add that there is a lot of confusion, I think, about quality of tobacco. For example, the tobacco you use in smoking or pipe tobacco, back over the years we would not have used that in cigarettes at all because it was a heavy-bodied tobacco. What constitutes quality in some measure, particularly from a manufacturer's point of view, depends on what he intends to use it for.

Quality tobacco for a pipe is not quality tobacco for cigarettes. I am talking about historically, what we grew up on. If I may go back over a few years ago when we were just getting underway in the cigarette industry, these lower leaves here, which I have referred to as flyings, were literally thrown away. They weren't even marketed. They wouldn't bring enough to justify the stripping and placing them on the market.

Mr. BLATNIK. How far back was that?

Mr. WRATHER. I would say in the early twenties, and along in there. But then, as the cigarette industry developed and as we turned more and more in the direction of the mild cigarette, these darned trashes that we used to throw away turned out to be, in your terminology, choice, fine, first-quality tobacco, because they brought the high dollar.

Now it seems that we are turning in the other direction, where the manufacturers are making a little different product, and what he thinks will do the most for that product is not the tobacco that he has been using in his other product during this stable decade of mild cigarette production and consumption which we have experienced.

Mr. MEADER. On this table of burley prices, in the center and to the left I notice that in 1952 the red, dark, and green, which in the terminology we have used is the lower grade tobacco, represented 57.2

percent of all of the burley tobacco that went into Government warehouses.

Mr. WRATHER. That is true.

Mr. MEADER. Whereas in 1955 none of it went in, and in 1956 none of it went in there.

Mr. WRATHER. That is exactly what I wanted to show.

Mr. MEADER. And the high-grade tobacco, on the other hand, in 1952 represented only 14.8 percent of the burley tobacco moving into Government warehouses but, in 1956, 99.2 percent of it.

Mr. WRATHER. That is another way of saying exactly what we have been saying. I wanted to go back far enough with my figures to latch onto the patterns which prevailed before the filter cigarette gained the momentum it now has. I thought 1952 would be far enough. So, in 1952, if you please, you can see from these tables where the demands were, what tobaccos were being bought, what tobaccos were moving under loan, and you can see currently, also.

The figure referred to by Congressman Meader is a good figure but it is like a lot of percentages; it is a wee bit tricky—99.2 percent. However, in 1956, only 6 million pounds moved under loan, if you will notice in the bottom table, this burley table that you are talking about; 99.2 percent were in those thin- to medium-bodied grades, but the takings under loan in 1956 in burley were almost nil. Burley production in 1956 was 506 million pounds, so 6 million pounds which moved under loan was negligible.

Mr. MEADER. That compared with 104 million in 1952.

Mr. WRATHER. That is true.

Mr. BLATNIK. Mr. Wrather, we wanted to have the staff check these figures to get the complete story. We thank you very much for your assistance.

Mr. WRATHER. If I may add, these are rather comprehensive tables. I think it will point out pretty clearly the direction in which we are going, and I know, from the questions you are raising, there are quite a few things reflected in the tables that you aren't aware of since you have not had an opportunity to study the different parts of the different tables.

Mr. BLATNIK. Do you have any other comments or statements?

Mr. WRATHER. I could present for the record the production of cigarettes going back to 1951 and how that is broken down between the different kinds of cigarettes, regular, king-size, and filter cigaettes.

Mr. BLATNIK. I am sorry. Would you please repeat that?

Mr. WRATHER. I have a table here showing the production of cigaettes annually from 1951 through 1956, broken down by regular, king-size, and filter cigarettes, if you want it for your record.

Mr. BLATNIK. We would like that very much. Without objection, it will be included.

Cigarettes

Calendar year	Total production	Domestic consumption	Type of cigarette (domestic) [1]		
			Regular	King size	Filters
	Billions	*Billions*	*Percent*	*Percent*	*Percent*
1951	418.8	379.7	86.6	12.7	0.7
1952	435.5	394.1	80.3	18.3	1.4
1953	423.1	386.8	69.8	26.9	3.3
1954	401.8	368.7	61.0	28.9	10.1
1955	412.3	382.1	53.2	27.1	19.7
1956	424.2	391.2	45.8	24.3	29.9

[1] Estimates of Harry M. Wootten, Printers' Ink.

Source: Tobacco Division, Agricultural Marketing Service, July 1957.

Mr. BLATNIK. Do you have any other tables?

Mr. WRATHER. I may have some additional copies of those I have presented.

Mr. BLATNIK. Mr. Wrather, we thank you very much. We will recheck these tables and information we have here and get a more comprehensive, chronological and more orderly picture of the problem with which you are thoroughly familiar and have the staff consult further with you. We would appreciate your further assistance on it.

Mr. WRATHER. In terms of prices, I think we can give you any information you want.

Mr. BLATNIK. Thank you very much.

We have no further witnesses this morning. I would merely like to announce that thus far we have had people testifying on the filters and the type of tobacco in cigarettes that are not directly connected with the industry.

We have hoped to hear from representatives of industry, in order to get a more complete and accurate picture to help us come to the point of just what is the function of the filter cigarette. To date we have had only one direct, formal accounting or reply. We had Mr. Robert DuPuis, vice president of research of Philip Morris who was tentatively scheduled to be one of the witnesses tomorrow speaking for the tobacco industry or for the cigarette manufacturing industry.

I have a letter here addressed to Mr. Curtis E. Johnson, the staff director of the subcommittee, a letter dated July 18, and I would like to insert this in the record and briefly state that Mr. DuPuis will be unable to appear before the committee. I quote from the letter:

> I understand that no other tobacco industry technical man is scheduled to appear. I do not feel that I alone could represent the entire industry. I hope you will understand my feeling in this.
>
> In view of the above, I feel that I must decline your kind invitation to appear.

He had an appointment at some laboratory and there was a conflict in date.

(The letter referred to is as follows:)

PHILIP MORRIS, INC.,
Richmond 15, Va. July 18, 1957.

Mr. CURTIS E. JOHNSON,
Staff Director, Congress of the United States,
House of Representatives, Legal and Monetary Affairs Subcommittee,
Washington, D. C.

DEAR MR. JOHNSON: Thank you for your invitation of July 15 to appear before your subcommittee on Thursday, July 25.

I understand that no other tobacco industry technical man is scheduled to appear. I do not feel that I alone could represent the entire industry. I hope you will understand my feeling in this.

When I discussed my possible appearance with you on the phone Monday, I realized that I would have to cancel a week-long trip to the laboratory of one of our subsidiaries. This meeting had been planned for over a month.

In view of the above, I feel that I must decline your kind invitation to appear and reactivate my original plans.

I am sorry that I shall not be able to meet you next week in Washington.

Sincerely,

ROBERT N. DUPUIS,
Vice President, Research.

Mr. BLATNIK. Our job is to get spokesmen from the industry in the interests of trying to get a complete and fair record of all points of view. We do hope we will be able to succeed in getting testimony from the people who know most about this whole problem of the filter-tip cigarette and who are doing the most to promote its use.

If there are no further comments, we will adjourn the meeting until 10 o'clock tomorrow morning.

(Whereupon, at 12:45 p. m., the hearing adjourned to reconvene at 10 a. m. the following day, Thursday, July 25, 1957.)

96946—57——14

FALSE AND MISLEADING ADVERTISING
(Filter-Tip Cigarettes)

THURSDAY, JULY 25, 1957

HOUSE OF REPRESENTATIVES,
LEGAL AND MONETARY AFFAIRS SUBCOMMITTEE
OF THE COMMITTEE ON GOVERNMENT OPERATIONS,
Washington, D. C.

The subcommittee met, pursuant to recess, at 10 a. m., in room 100, George Washington Inn, Hon. John A. Blatnik (chairman) presiding.

Present: Representatives Blatnik, Mrs. Griffiths, Meader, and Minshall.

Also present: Jerome S. Plapinger, subcommittee counsel; Curtis E. Johnson, staff director; and Elizabeth D. Heater, clerk.

Mr. BLATNIK. The Subcommittee on Legal and Monetary Affairs of the House Government Operations Committee will please come to order.

We are in further continuation of public hearings on false and misleading advertising within the jurisdiction of the Federal Trade Commission and with special reference to filter cigarettes.

Today the subcommittee begins its fifth day of hearings on cigarettes; their effect on health, the promotion of filter cigarettes, and the effectiveness of these filters.

We expect to conclude these hearings tomorrow with an examination of some of the filter-cigarette advertising and with testimony from Mr. Robert Secrest, Acting Chairman of the Federal Trade Commission. We have attempted to secure the most authoritative and complete testimony possible on all phases of this problem.

Today we had hoped to hear the cigarette-industry people state their point of view and their thinking on the use of filters and on the heavy promotion of filter cigarettes. The cigarette industry, though vitally involved and affected by our inquiry, has to date been conspicuously absent. Invitations have been extended to the industry. A representative of one company accepted but later withdrew.

Testimony before this subcommittee from some of the most competent medical authorities in the country, including the Surgeon General of the United States, charges that excessive cigarette smoking is a contributor to the incidence of lung cancer. We have been told that this is the conclusion of an overwhelming majority of the medical and scientific personnel who have conducted work in this field. They have identified tobacco tar as the suspected caustive agent. Nicotine has been named as a possible contributor to heart disease.

We have received testimony that the cigarette industry has placed relatively ineffective filters on their cigarettes and in addition, have used stronger tobaccos to nullify the effect of these filters.

We have been informed that in spite of the mounting medical evidence that tobacco tars and nicotine are deleterious to public health, the cigarette industry is marketing a product with as much or more nicotine and tar than ever. To date, the cigarette industry's response to all of these charges has been, in effect, "no comment."

Without passing judgment on the charges that cigarette smoking is a causative factor in lung cancer—and may I note that the evidence to date is impressive—I find it difficult to fathom the behavior of the cigarette industry in deliberately flying into the face of this evidence.

We have had testimony that the case against cigarettes has not been proved. However, even Dr. Clarence Cook Little, the scientific adviser to the Tobacco Industry Research Council, has stated here that "neither have cigarettes been absolved."

Dr. Ernest Wynder, of the Sloan-Kettering Foundation for Cancer Research, has stated his belief that a "safe" cigarette can be produced.

We would like to know if the cigarette industry is conducting any research toward producing such a cigarette or whether they are just sitting out this controversy in the hope that it will blow over.

In the hearings tomorrow, the subcommittee will give attention to specific examples of cigarette advertising which claim everything from "priceless health protection" to a "smoother, milder smoke."

I find it difficult to understand why this industry, which spends millions of dollars to advertise its products, should remain utterly silent in these proceedings in which it is the central figure.

In the light of these observations, I again extend an invitation to leaders of the cigarette industry to appear before us in order that we may have a record which reflects all facets of fact and opinion on the cigarette question. Today we will hear from two very eminent medical experts.

We have first Dr. Harry S. N. Greene, chairman of the department of pathology of Yale University. Dr. Greene, will you please take the chair?

STATEMENT OF HARRY S. N. GREENE, CHAIRMAN, DEPARTMENT OF PATHOLOGY, YALE UNIVERSITY

Mr. BLATNIK. Dr. Greene, welcome to the committee. We are certainly honored by your presence here and we appreciate your cooperation in giving us the broader point of view from men of great professional standing and experience in the medical profession on this very serious problem which is of very deep concern to literally millions of people in America.

Doctor, do you have a prepared statement?

Dr. GREENE. I do, Mr. Chairman.

Mr. BLATNIK. Will you give us a brief background for the record?

Dr. GREENE. Mr. Chairman, members of the committee, and ladies and gentlemen, my name is Harry S. N. Greene. I was graduated from McGill University School of Medicine in 1930 with a degree of M. C., C. M. At the present time, I am professor of pathology and chairman of the department of pathology at Yale University School of Medicine.

I have been intimately concerned with cancer research throughout the past 25 years and throughout this time have had a considerable interest in cancer of the lung. I thought today I would try to give you a brief presentation of my understanding of the lung cancer problem and also some evaluation of present-day research.

Now, there are several distinctive anatomical features of lung cancer that are quite suggestive from the point of view of ideology. The function of the lung as a respiratory organ requires continued contact between its internal surfaces and inspired air. Thus, the lung, more than any other internal organ of the body, is brought into immediate association with the external environment. Generally, organs and tissues subjected to prolonged environmental contact are provided with a covering made up of cells of a special type, known as epidermoid cells. Such cells cover the skin, the tongue, the lining of the mouth and nose, etc., and being of special structure and present in a number of layers, supply a degree of insulation or protection.

In contrast, the cells lining the tubules of the lung are of a different type, being columnar or glandular in structure and not stratified in multiple layers. It is from these cells that cancer of the lung arises, and their exposed surface as well as the type of cancer evolved are pertinent points in a discussion of causative factors.

Now, this supposition is enhanced by another anatomical point and that is that cancer of the lung is an epidermoid carcinoma, similar in all respects to the carcinomas derived from the skin or the mouth or lips.

Now, an epidermoid rather than an arsenal carcinoma is one that might be expected from columnar cells. Elsewhere they produce an abscess.

Now, before a glandular cell, a columnar cell can produce an epidermoid carcinoma, it has to undergo a change in cells. This change from a columnar to epidermoid type cell is known as metaplasia. Now, metaplasia is not an uncommon finding in the body and all evidence that we have suggests that it is due to chronic irritation. Therefore, the suggestion is that preceding the development of cancer of the lung of an epidermoid type there must be a chronic irritation of the columnar cells to convert them into epidermoid cells from which the cancer then arises.

Then we have two points. First, the intimate relationship between cells of the lung and the inspired air places them in immediate contact with all sorts of environmental agents that may be present in the atmosphere. And second, the vast majority of the lung cancers are epidermoid in type similar to those derived from the skin rather than glandular would be.

Now, cancer-inducing agents, metaplasia-inducing agents, as judged from research are an extremely common contaminant of the everyday atmosphere in which we live.

This has been very well expressed by Dr. William Boyd who used to be professor of pathology at Toronto. He said, "The trouble is we probably swim in a sea of carcinogens (cancer-inducing substances)." He goes on to say that the task of tracking down any particular one is difficult.

Now I would like to say a word about the term "metaplasia" which appears to be so intimately concerned in lung cancer. Now, it is supposedly due to the action of chronic irritants, but not all chronic irri-

tants would produce metaplasia. And surely not all metaplasia lesions are precancerous. Let me give an example. Some years ago people used to continuously wear stiff, starched collars which were a continual irritant to the skin of the neck. Yet no cancers arose in the skin of the neck in that region. In contrast, cancers of the mouth is not associated with ill-fitting dentures. So that the nature of the chronic irritant is of extreme importance in the production both of metaplasia and precancerous relation.

There is also considerable evidence that the constitutional makeup of the individual is very much concerned in the production of cancers in areas of irritation.

Let me emphasize the fact that not all areas of metaplasia resulting from chronic irritation are precancerous. Almost any individual in this room if subjected to a thorough post mortem examination would show areas of metaplasia, or areas that could be interpreted as precancerous. It is obvious, however, that not all of us will die of cancer. It is clear, therefore, that not all of these areas so designated are truly precancerous.

How is the determination of a precancerous lesion made? First they are made from organs removed from human patients and sent to the surgical pathologist. He looks at them after the tissue has been killed and stained under microscopic slides. If there are certain structural changes or changes in the relationship between cells, he says the lesion is metaplastic, shows cellular atypism, basal-cell hyperplasia, or many such terms as he considered to be precancerous.

Now, how does he know it is precancerous? The lesion is not left in the individual to go ahead and develop. It goes in a can of formula and killed, where further development does not occur.

In other words, the assumption that a lesion—the diagnosis of a lesion being precancerous is based purely on an assumption and not observation. One has, therefore, to be careful in interpreting these lesions.

There have been many instances of that sort of thing at the present time and it applies particularly to cancer of the cirvex. There a lesion has been noted with some frequency recently and called a carcinoma in situ or a noninvasive carcinoma.

Now that word, of course, is a complete absurdity. Carcinoma by definition is an invasive lesion. It is not possible, therefore, to use the term "noninvasive carcinoma." In fact, all it means is the relationship of the cells in a thin layer has been disturbed and its significance is not known. By many people, it is true, it is thought to be precancerous. There is some evidence, however, coming now that that is not true. A number of women who were subjected to cervical biopsies—some 5 to 6 years ago—refused treatment and the cervicis were not removed. Those women have recently been examined to see the outcome of this so-called precancerous lesion.

Now, in some instances there has been a cancer present. These have been very few, however. In the majority of cases the same precancerous lesion, so-called, is still present, or has completely disappeared. So before we have some more, better observational data, one must take this business of precancerous relations with many grains of salt.

Another point in relation to metaplasia—I mention this specifically because there have been a number of papers recently, or one paper in

particular, which has purported to show that smokers—cigarette smokers in particular—have many areas of metaplasia, and so on, in the lung.

I would like to point out one point in relation to that, and that is that way back, following the great influenza epidemic of 1917, Dr. M. C. Winternitz, who was my predecessor at Yale, observed in the lungs of people who died with this disease an extreme degree of metaplasia, and at that time said that, if this was a precancerous lesion, then all individuals who recovered from influenza would subsequently die of cancer. Now, that hasn't occurred.

I would like to also emphasize the fact that viruses are of great importance in the production of metaplasia. I have seen mouse lungs from mice which have been infected with influenza virus that, under the microscope, one could only with great difficulty distinguish from cancer because of this metaplastic lesion. If, however, these mice are allowed to live, or if a fragment of that lung is transplanted to another mouse, it never eventuates this cancer.

Well, this has all been said simply to bring some doubt into your minds as to the significance of the term "precancer." It is not of very great significance.

Now, the frequency of lung cancer is thought by many to have increased during the past two decades and, on that basis, a search has been made for some environmental factor showing a parallel increase. It should be emphasized, however, that there is some question as to whether the purported increase is real or only apparent—that is, a question arises: Has the increase of lung cancer actually increased, or are we only now becoming aware of the true frequency of the disease?

This is an extremely difficult question, and let me emphasize that there is a great deal of doubt in critical minds as to whether or not a true answer is possible.

The statistics that have been used in the recent studies, when based upon death certificates, are practically valueless. In the great majority of cases, the cause of death is recorded in a death certificate and is a clinical impression and is as limited in value for scientific purposes as is the clinical diagnosis of a living patient.

This applies particularly when the assigned cause of death is a popular disease, such as cancer, to which the attention of the attending physician has been directed by frequent reference.

Acute gastritis was formerly a common, inclusive term employed when the actual cause of death was not obvious. But, as a result of the present widespread educational propaganda, the diagnosis of cancer has been substituted. There is usually no greater basis in fact.

The point to be emphasized is that, in the absence of an autopsy, the cause of death is always in doubt, and death-rate statistics based on clinical evidence rather than on post mortem findings are entirely too unreliable for use in scientific investigation.

At the present time, the diagnosis of cancer rests on microscopic examination and there is no acceptable substitute. Moreover, microscopic examinations must be performed on adequate speciments of tissue, and the desquamated cells found in a smear of sputum are not sufficient. Further, it must be shown that the tumor found represents a primary growth and not a metastasis.

Now, this latter point applies particularly to lung cancer, for the lung is an extremely common site for the location of secondary

growths arising as metastasis from primary tumors elsewhere in the body.

The common lung cancer, as I said, is an epidermoid carcinoma, and does not differ in appearance microscopically from epidermoid carcinomas originating in other bodily regions.

Thus, microscopic confirmation of a lung cancer means confirmation not only of gross findings, but also an examination not only of the tumor, itself, but of the individual as a whole. Therefore, the loose term—the loose use of the term "microscopic confirmation" in the hands of statisticians is valueless without these qualifications.

Thus, the only material available for analysis of the incidence of lung cancer are biopsy or autopsy reports. Figures so obtained—that is, from biopsies or autopsies—are necessarily based on a hospital rather than a general population and are, accordingly, biased, as they represent the incidence among people sick enough to be brought to the hospital.

Now, the latter figure varies with many extraneous, unrelated factors, including hospital facilities, which have been greatly augmented during the past 40 years. Nevertheless, the figures derived from such a population—that is, a hospital population—do indicate an increased incidence of lung cancer, but, again, one doesn't know whether or not the increase is due to the coincident improvement in diagnositic facilities. These have advanced in a remarkable manner.

Almost all hospitals now have one or more chest services, and these are manned by trained individuals who take X-ray pictures of one's lungs, examine the cells in the sputum, look at the interior of the lung through illuminated tubes, snip off suspicious areas for pathological study, or even open up the chest wall for firsthand observations.

Now, if such a screw failed to find an increase in the incidence of something or other, their diligence would be in doubt and their function in hazard.

Autopsy records would appear to be a better source of material for investigation, inasmuch as pathologists have been performing post mortem examinations according to the same technique and with little increase in facilities or imagination for well over 100 years. But, again, experience breeds suspicion. One sees what one knows, and this is as true at the autopsy table as it is elsewhere.

The great pathologists, Virchow and Rokitawsky, fail to recognize the chronic areas in the heart characteristic of coronary occlusion, yet present-day, first-year interns, as a result of the increased emphasis on heart disease, spot the lesions with facility.

Further, and more to the point, the eminent pathologist, Sir John Bland-Sutton, who was the dean of cancer pathology in his time, wrote a textbook on tumors and, in the 1922 edition, pictured the typical lung cancer over a caption bearing a completely wrong diagnosis of "mediastinal lymphosarcoma." Let me emphasize the fact that this man was one of the best histologists of his day.

If one goes through pathological museums at the present time and picks out old specimens labeled "Hodgkin's disease" or "lymphosarcoma" and gets sections of these tissues, one finds in many cases they are not Hodgkin's disease or lymphosarcoma, but epidermoid carcinoma of the lung. It was not recognized at the time.

And without accurate knowledge of the past incidence of lung cancer, which is of course impossible, it is obviously unreasonable to state that the present incidence represents an increase.

Despite the evidence of definite proof of an increase, the assumption has generally been made that the incidence has increased, and this has been considered as a clue in the search for related environmental agents.

Many factors in man's environment have changed in the past 50 years, and several of these pertain directly to the lung. The atmosphere has become polluted with the exhaust gases of industry and the automobile; tobacco smoking has become a universal habit.

X-ray examination for early detection of tuberculosis has become a standard procedure to which many individuals are subjected at frequent intervals. There are many others, any one of which shows an increase in prevalence comparable to that thought to obtain in lung cancer.

But for reasons which appear to me to be more subjective than objective, major attention has been directed toward smoking.

The statistical studies purporting to establish a causal relationship between smoking and lung cancer have been the subject of a barrage of propaganda both in the press and over the radio, and as the controversy has provided a great deal of free entertainment for the populace.

The arguments, of course, go on endlessly for, like the question of how many angels can sit comfortably on the head of a pin, there are no pertinent data on which to base a definitive answer.

Several comments are required with respect to the published statistics. Two points have been brought out in these studies that strongly suggest a conclusion contrary to that drawn by the investigators.

First, the incidence of lung cancer has been found to be greater in cities than in the country and, second, the incidence is much greater in males than in females.

The discrepancy here relates to the fact that country people smoke as much as do city people. I was brought up in the country. And the consumption of cigarettes by many females equals or surpasses that by many males. This latter discrepancy is further enhanced by the fact that there are more and more smoking females who reach the lung cancer age.

The difference in cancer rate between males and females becomes greater rather than smaller. Let me say a word about statistical methods and statistical associations. Statistical associations may means 2 or 3 different things. The fact that two happenings appear to be related may be purely on a basis of happenstance. It may be due to the fact that both have a factor in common. It is really extraordinarily rare in biology that it is a result of a cause-and-effect relationship.

Let me illustrate what I mean. First, happenstance: There are certain months of the year—November, December, January, and February—that are cold. Other months—May, June, July—are warm.

Now the cold months all have longer names than do the hot months. There are many more letters in November, December, January, and February than there are in May, June, and July. As a matter of fact one can draw up a significant statistical association between the number of letters in the month and the mean temperature. The months with longer names, more letters, being colder than the months with

shorter letters. But this, of course, is not a cause-and-effect relationship.

December is not cold because it has more letters and July is not hot because it has fewer letters. That is an association that is significant from a statistical point of view but obviously has no cause-and-effect relationship.

Now as I said, this statistical association may be due to the occurrence of a common factor.

Now many years ago it was noted that the front row in a burlesque house was almost invariably occupied by baldheaded indviduals. In fact it was known as the baldheaded row. This occurred with such frequency that one might assume on statistical evidence that the continued close observation of chorus girls in tights caused loss of hair from the top of the head and thus one could construct a nice cause-and-effect sequence relating the two.

However, subsequent investigation has shown that in effect, baldheadedness and the desire to see chorus girls at close range are part of the same constitutional diathesis. Both are due to an excess of the male hormone testosterone.

On the other hand this hormone in abundance brings about excessive virility and, on the other, causes the hair on top of the head to fall out.

The point is to be made that if cigarette smoke and lung cancer have any relationship, it may well be of this variety and without any significance whatsoever from the point of view of causation.

I hope I have made myself clear. Another illustration may help: If one questioned, in a State such as Connecticut, individuals who had lung cancer, about their political affiliations, I am sure that the vast majority would reply that they were Republicans. Now does that mean that this is purely happenstance or is it a cause-and-effect relationship? Is there something in the tenets of the Republican Party that is carcinogenic?

This might well be a subject for future investigation. But you get what I mean.

Now statistical methods are a natural adjunct in the everyday decisions of people who are outside of insane asylums. Correlations between various happenings determine most of our behavior from youth to old age. But an interpretation of the significance of the correlations is determined by judgment.

For example, one recognizes the fact that corset covers have gradually disappeared as articles of female apparel, and one also observes that during the same period of time it has become increasingly difficult to get a good molasses cookie. One's judgment immediately rejects the possibility of a cause-and-effect relationship.

The point to be made is that a determination of the nature of correlations as cause-and-effect sequences or merely phenomena associated by happenstance is a function of judgment, and judgment cannot be substituted for by statistical techniques.

Now the basis of judgment is experience, and experience with new things is necessarily limited. Accordingly, the experience essential for evaluation must be attained and in research work, at least, that is done by means of experimentation. Associated phenomena are observed and the validity of a causal relationship is tested by controlling one variable and noting the effect on the other.

This is known as the scientific method and its usage in one form or the other is responsible for the present age of medical discovery.

According to this approach, an evaluation of the possible relationship between lung cancer and smoking rests on the results of experimentation. As a matter of fact, the possibility of such a relationship has long been appreciated, long before the present statistical studies were made, and pertinent experiments were performed. They were performed by a number of different investigators utilizing various techniques, and no causal relationship was found.

One of these studies was carried out in my laboratory. We had found that embryonic tissues were much more susceptible to cancer-inducing substances than were their adult counterparts. If, for example, one killed a pregnant mouse and removed the embryos and then dissected out of the embryo a specific organ, added to that organ a known carcinogen, such as methyl cholanthrene or dibenzanthracene, and then transplanted that organ subsequently in an adult mouse, a take occurred, the organ grew and survived, and at the end of that month if the material used was a carcinogen, one found a cancer in the transplanted organ.

These embryonic transplants are much more susceptible to carcinogenic chemicals than are their adult counterparts and they offer a unique material for testing the carcinogenicity of various substances.

In these experiments we used embryonic lung, embryonic mouse lung which we infiltrated with a variety of tobacco products. Tar—a great many different brands of cigarettes, cake and cleanings from my pipe, ash from cigarette paper, and so on. We used literally hundreds of animals and kept them for long periods of time, but not in one single, solitary instance did we find anything remotely resembling a cancer.

I emphasize again that here we used a much more susceptible material than adult material and we used lung rather than skin.

Now, despite such results we obtained, and so also did other investigators, the possibility of a causal relationship between lung cancer and cigarette products lingered in some minds, and the recent statistical studies together with the experiments of Wynder and Graham, have appeared to reopen the subject.

The statistical data were obtained by a different method but the enumeration of individuals dying of lung cancer was based largely on death certificates without post mortem examination in the vast majority of cases. The words were the words of Esau but the voice was Jacob's voice and the conclusions derived from analysis were the same.

Accordingly, there has been call for more experimentation. It is of some interest in this connection that the use of statistical methods in the prevalence of statisticians have shown an increase that corresponds as well with the assumed rise in lung cancer as does the consumption of tobacco. Yet the alternative conclusion has not been drawn. Why has cigarette smoking rather than the prevalence of statisticians been suggested as a cause of lung cancer? Perhaps some of us would have shown more enthusiasm in pertinent experimentation had that been the case.

The coincidence of the reports of Wynder's experiments in the tobacco statistics is probably a major factor in the extent of the present

scare. Wynder extracted a tar from cigarettes smoked by machinery at a high temperature and painted this material, three times a week, on the skin of mice of a certain strain known as the CAF strain. Of the 62 mice so painted who survived for 12 months, 59 percent developed cancer of the skin at the site of painting, and these results have been considered by some as sufficient investigative experience to validate the statistical suggestion. However, such a conclusion is untenable from a critical point of view. The experiments demonstrated that tobacco tar extracted by a special technique induces cancer in the skin of CAF mice, and nothing more.

This point is of little significance to workers in the field who have found that under certain conditions a multiplicity of substances in everyday use will induce cancer in mice of highly susceptible strains.

In certain animals, cancer has followed the injection of such material as glucose, sugar, and olive oil, but the interpretation of the experiments has been that something was wrong with the animals rather than that the materials were cancer-producing. The pertinence of this fact to Wynder's experiments is brought out in a recent publication reporting more recent work in which he duplicated the technique but used other mouse strains. In one strain, Swiss, 14 percent of 86 animals developed cancer, while in another, C57, only 2 percent of 89 were so affected.

It is apparent from this that the mice used in Wynder's original experiment were not comparable to mice in general, but, on the contrary, represented a strain bearing a constitutional factor which rendered the animals different from others in relation to cancer susceptibility. The occurrence of such a high differential susceptibility among mouse strains would suggest the existence of an even more pronounced difference between species, and an extrapolation of the findings to man would be absurd.

It should be noted also that the skin of the mouse rather than the lung of man was the object of study in these experiments. There is no surety that a substance capable of inducing cancer in mouse skin would also induce cancer in mouse lung—to say nothing of human lung. As a matter of fact, in our hands, the same tar failed to produce cancer in transplants of embryonic mouse lung, a material known to be highly susceptible to cancer-inducing substances.

In any case, the human statistics purport to relate tobacco smoking with lung cancer not with skin cancer. It is not claimed that the incidence of skin cancer in man has increased despite the fact that the contact between the fingers and the burning cigarette is sufficiently intimate and prolonged to result in a yellow discoloration of the skin in that area.

In summary, the methods employed in the statistical inquiry under question, particularly the type of data used for analysis, raise doubts that the results obtained can be interpreted as conveying a suggestion of a causal relationship between tobacco smoking and lung cancer. However, the results have been accepted by some investigators as sufficiently suggestive to warrant a direct experimental approach. The investigation has been reported as it progressed and, from my own point of view, has not succeeded in supplying confirmatory evidence.

Thank you.

Mr. BLATNIK. Dr. Greene, thank you. Let me start off with just a few questions.

Doctor, you state that the rather substantial increase in number of deaths caused by lung cancer are due largely to better diagnosis?

Dr. GREENE. I think that may be one of the important factors.

Mr. BLATNIK. Why wouldn't increase be uniform for all people whether they smoke or not or whether they are male or female? Let's leave out the female. Why wouldn't it be uniform for all men, regardless of whether they are heavy smokers or nonsmokers?

Dr. GREENE. Well, it is, isn't it? There has been an increase in nonsmokers over what it was when I was a student in pathology. When I was a student in pathology, if I saw a lung cancer I called everybody around to see it. It was a rare occurrence in the autopsy, at least from my knowledge. But there is an increase over that in nonsmokers at the present time.

Mr. BLATNIK. But a greater increase in smokers?

Dr. GREENE. But we don't know, because we don't know what the incidence was 25 years ago and we have nothing upon which to form a base.

Mr. BLATNIK. Not that I am an expert at all, but I did have 4 years of mathematics, I majored in chemistry and mathematics—not that I don't have some questions, and in fact quite some questions on the statistical material we have gone through. But the disproportionate ratio, running to hundreds of times more—in the bar group, this high rate [indicating] of lung-cancer deaths in heavy smokers, and a relatively low one for the others. How do you explain that? Repeatedly, over and over again, in the big city, in intermediate cities, in the village, on the farm, over and over and over again the pattern repeats.

Would you say offhand there is at least something here to attract a second look at it or would you just ignore it?

Dr. GREENE. We have got to examine the external environment for a possible agent in the increase in lung cancer. I think that is quite obvious. But I don ot believe that our investigation should be limited to tobacco smoke. As I say, many things have increased, significantly. X-ray of the lung is carried on routinely and it is known that X-rays are carcinogenical. Yet I haven't heard any great furor about it as we have about smoking.

Mr. BLATNIK. I agree, Doctor, and I have nothing but the highest respect for your profession and you personally, but what concerns me is that repeatedly the same pattern, wherever it has been tried out, statistically, in England, Sweden, Switzerland, Germany, France—I hope it will be tried in other countries around the world—I can't help but have a feeling that wherever you try it that that same pattern, with some variations, of course, will show up—that overwhelmingly the higher percentage of those dying of lung cancer are heavy and prolonged smokers.

Dr. GREENE. Yes. I would like to make a few points there. It might well be that here we are dealing with the operation of a common factor. There is much to suggest that, as a matter of fact, that people who have an unusual hunger for cigarette smoke, are people who are also susceptible to lung cancer. Do you get my point?

Mr. BLATNIK. Yes, I do.

Dr. GREENE. And that they are not causally related but one follows along with the other. In fact, the statistics brought out an incidence

of many other diseases completely unrelated to lung cancer among constant smokers and that might well be a factor. That is a constitutional affair, rather than restricting it to smokers.

Mr. BLATNIK. Again I will be frank and say I am in no position to challenge your statements, but I do have a little larger than the normal question mark.

At the bottom of page 7 and page 8, you treated a little too quickly the discrepancy in smoking between females and males. You passed it off with a statement at the top of page 8 that the consumption of cigarettes by many females equals or surpasses that by many males.

That is true, but that is a limited statement. May I ask what percentage of the male population smokes?

Dr. GREENE. I can't give you percentages like that. This is the basis of my own observation.

Mr. BLATNIK. Would you say more than half of the male population?

Dr. GREENE. I couldn't give you any data. Most of my friends smoke.

Mr. BLATNIK. Do as many women smoke as men smoke?

Dr. GREENE. Oh, I think so, certainly among my associates.

Mr. BLATNIK. Many more?

Dr. GREENE. Yes, and they smoke all day long.

Mr. BLATNIK. My memory is not clear on this, and we can check it. As I recall, I think it is stated that over 70 percent of the men smoke. Over 70 percent, and perhaps around 33 percent of the women smoke. That means over twice as many men smoke, and most of the men have smoked for a much longer time than the women.

You say there are many females who smoke more than some males. That is true, but why do you put that in testimony when it is so limited?

Dr. GREENE. In my experience, and my experience is the only thing I have to talk about. The experience of others is before you and has been put before you. I am giving my own.

The point I wanted to bring out is, despite the fact that more and more smoking females are reaching at the present time the lung-cancer age, the disparity in incidence between the males and females is getting greater instead of smaller.

Mr. BLATNIK. Getting back to the need for more direct medical evidence that death was due to lung cancer, I agree with you on that.

I wonder if there wasn't some misunderstanding, however—and this is just to clear up the record, Doctor—on page 10 in the middle of the page you refer to statistical data, that the enumeration of individuals dying of lung cancer was based on death certificates without post-mortem verification in the vast majority of the cases. I wanted to clarify that.

Dr. Hammond testified last week, and I quote him:

> In 79 percent of the cases of cancer reported on death certificates, it was proved microscopically that of the 79 percent, the remaining 21 percent were not microscopically tested.

Is there a conflict there between the two statements?

Dr. GREENE. May I make a point there? Dr. Hammond is not a pathologist. He did not examine the microscopic tissues of these patients, and the question is, what does he mean by microscopic con-

firmation? Does he mean these doctors examined adequate sections of the tissue, or does he mean that they looked at cells in sputum? Now, the latter is not acceptable to dying of lung cancer, and as I pointed out, epidermoid carcinoma of other parts of the body frequently mastacizes to the lung, and microscopic confirmation is not sufficient. One has to have a thorough physical examination and an X–ray study to determine that the lung tumor is not a mastacyst or some other tumor.

Further, there is a great discrepancy among pathologists as to what is called a lung cancer. I mean under the microscope.

Mr. BLATNIK. Mr. Meader asked:

> And in these cases where lung cancer existed a relationship between an existence of lung cancer and a death was determined by a pathologist, was it?
> Dr. HAMMOND. I can't give you the exact number. About 10 percent of those were verified by autopsy, which is as good evidence as you can get. In the other cases it was the opinion of the doctors and in 79 percent of the cases it was also the opinion of the pathologist.

Would that answer it?

Dr. GREENE. No, it doesn't. They would all have to be looked at by the same pathologist. Let me give an example of that sort of thing. Before I left to come to Washington yesterday, a pathologist from an adjacent hospital came to show me a section. This section had been obtained from the cirvex of a woman in still another hospital, and a diagnosis of epidermoid carcinoma had been made, with a recommendation that the patient return to her home and be treated in that hospital. This pathologist came from that hospital. He had looked at the section and could find no cancer in it. He sent this section to a pathologist in an adjacent town. There had been three diagnoses, epidermoid carcinoma, carcinoma in situ, and nothing wrong with the cervix. He therefore brought it to me for examination. I looked at it and it showed a condition known as epidermoidization, a common occurrence in the cervix due to chronic inflammatory action. There were four different diagnoses.

Mr. BLATNIK. The same errors in diagnosis would also be made in nonsmokers who had died of lung cancer which would equalize it; but the overwhelming cases of lung cancer deaths are those who have been smoking. A 50 percent increase wouldn't be bad but it is a 200 and 300 percent increase.

Just to comment, I agree with you on statistics that you would attempt to prove that January and February were colder months because they are longer words. The late Dr. Graham who worked with Dr. Wynder was a chain smoker himself, by the way. He earlier expressed in conversation to Dr. Wynder and others the same thought. In fact, he ridiculed the statistical approach. At the outset, as I recall, around 1950, he went to Dr. Wynder, close friends, with great regard for each other, but with completely different points of view. He said, "I prove that with the increase in the use of nylon stockings, there is an increase in lung cancer deaths in women," and he did. He did prove it in actual tables. But the more he got into these other statistics with Dr. Wynder, the more he became impressed until he finally collaborated with him on it.

Dr. GREENE. I am sorry to say that I have little faith in statistics. I was intimately concerned in statistical work for 7 long years of my life and as a result of that study, I have far less faith in statistics than I have in the tenets of the Buddhist religion.

Mr. BLATNIK. Let's get back to this. Here is a book entitled "Science Looks at Smoking," by Eric Northrup, a writer of some repute, and you wrote an excellent introduction which I read which is essentially the same as the testimony presented here this morning. Did you read the rest of the book?

Dr. GREENE. Yes.

Mr. BLATNIK. I am getting to the pertinent point of the committee's inquiry on the effectiveness of filters, Doctor. Do you have any opinions on whether filters on cigarettes are necessary? Do they protect? Do they serve any function as regards or concerns the health of the smoker?

Dr. GREENE. Well, my feeling in this business is that there isn't anything in tobacco that is carcinogenic, that is going to do the individual any harm and, therefore, it is difficult for me to see how a filter could take out something that doesn't exist.

Mr. BLATNIK. I share that hope because I have always smoked nonfilter cigarettes.

Dr. GREENE. I don't know why they are used, they make a convenient mouthpiece and they perhaps remove some tobacco dust and inhaled material.

Mr. BLATNICK. In Mr. Northrup's book on page 183 he states that—

there is no significant evidence that filter tips provide a special safeguard in smoking. They merely reduce the smoke particle intake, but not particularly selectively.

There is nothing you believe that is cancinogenic in a cigarette that would harm a person, or anything else that is harmful.

May I ask, is there anything beneficial about a cigarette?

Dr. GREENE. I wouldn't know. I haven't seen studies on that, but I would like to see them. There is no question that the incidence of many diseases has dropped during the past 30 or 40 years when the consumption of cigarettes increased. That, of course, applies to upper respiratory disease which has dropped way down.

I have heard no arguments on that side. I think it would be extremely interesting if they were drawn up. I am sure they would be as significant as the present statistics purporting to show a causative relationship between lung cancer and cigarette smoke.

Mr. BLATNIK. Getting back to filters, now, you don't see any purpose they serve outside of being more convenient and keeping tobacco particles out of the mouth?

This book to which you write a very impressive introduction states the same thing.

May I ask, Doctor, and I do this not with any personal embarrassment, but did the tobacco committee or any tobacco people ask you to come down here to present testimony?

Dr. GREENE. I don't know, somebody called up, I think it was a Mr. Johnson, and asked me to come down.

Mr. BLATNIK. Mr. Curt Johnson?

Dr. GREENE. I think that is who it was. I was glad to come down but I insisted that it be today, because I am going fishing tomorrow.

Mr. BLATNIK. He is the staff director of our committee. Was he the only person who asked you to come down?

Dr. GREENE. Yes; so far as I am aware. There were a number of telephone calls, Science Service, and things that day, but they didn't specifically ask me to come down here.

Mr. BLATNIK. As I understand, representatives of the tobacco industry asked Mr. Johnson if you couldn't appear. I don't know if they contacted you.

Dr. GREENE. It probably comes from the introduction to that book.

Mr. BLATNIK. The reason I am interested is that they have failed to produce any witnesses or themselves come here to explain the function of a filter, or what would justify the expenditure of millions of dollars a year in merely advertising and promoting the sale of a filter.

We want to know if they are misleading the people, if testimony such as you have presented—and we have had several other eminent colleagues of your profession join you in your point of view to different degrees. We would like to know if there is so little or no danger in smoking, why the filters? Why this great promotional campaign?

Why this great exepense to the manufacturer, to the advertising and to the consumer who spends as much as $15 a month to buy filters. And somehow or other we have not yet to date received any evidence that the filters are necessary or that they serve any function as concerns the health of the smoker.

Dr. GREENE. I have heard comments from people who have used filters on their cigarettes, people who have been troubled by cough from regular cigarettes. They have said that the cough has disappeared after using the filter, but I have nothing to say about it. I don't know anything about it.

Mr. BLATNIK. You have already said that smoking is not harmful, but that you would be interested in knowing what good it may do.

In short, you don't know how smoking affects the health of an individual, either for the worse or for the better?

Dr. GREENE. Only personal experience. If I have a bad cold coming on, I smoke a lot of cigarettes and usually wake up the next morning without the cold.

Mr. BLATNIK. Before the cancer concern arose, years ago the tobacco people advertised that smoking would help in many ways, give you more energy, make you a more active athlete, it aids the T zone, and in fact it helps the health.

Now they have switched and say it doesn't hurt the health. I wonder whether they are entitled to go in this field of health unless they present a case, either one way or the other.

Dr. GREENE. I agree with you there.

Mr. BLATNIK. That is where we are trying to narrow down the findings in our hearings.

Thank you, Dr. Greene. I am sorry I took so much time. Mrs. Griffiths?

Mrs. GRIFFITHS. I have no questions to ask, but did you say, Doctor, where you did your statistical work?

Dr. GREENE. The Rockefeller Institute.

We were trying to correlate various constitutional differences in animals with their susceptibility to tumor transplantations.

Mrs. GRIFFITHS. What did you find out?

Dr. GREENE. I found out in the majority of cases when we relied on statistics we were throwing the baby away with the bath water.

Mrs. GRIFFITHS. How many cases did you study?

Dr. GREENE. Oh, gracious, innumerable ones. In this study we followed rabbits from birth to death over a period of some 10 years, and

during that period there are a great many rabbits. I couldn't tell you how many cases.

Mrs. GRIFFITHS. But at least the rabbits didn't prove anything?

Dr. GREENE. Not to me.

Mr. MEADER. Dr. Greene, I was quite interested in your discussion of philosophy and logic, on cause and effect relationship and so on.

You appear to require a higher degree of proof than even Dr. Little who appeared here as our witness. Dr. Little seems to concede that the statistical association at least indicated areas where research might well be undertaken.

I would like to quote from page 64 of the transcript of Dr. Little's testimony.

> To establish a cause and effect relationship on statistical associaation without experimental evidence is not safe. It cannot be done. You may get an indication of something to look for, but to say that the case is finished, the evidence is all in and that you can satisfy experimental scientists, all of them, that is not possible because too many of us have seen too many statistical relationships which have not helped the cause and effect relationship.

Do I see some distinction between your attitude and his?

Dr. GREENE. I think not. I think that I feel about the same way about it. One has to use statistical associations in research work. One observes a series of happenings in mice-bearing tumors and on a basis of that observation you do experiments.

For example one might find that a tumor was occurring with greater frequency in males than in females. That is a statistical observation. Then to find out whether or not it was significant, you would subject that to experimentation.

Mr. MEADER. Now we have in law a phrase known as a non sequitur. I was interested in your association of bald men and the burlesque show as a very picturesque example of a non sequitur, but I would like to ask you if you are contending that any of these other scientists who appear on the opposite side of this controversy have claimed that the association has proved a watertight cause and effect relationship.

Dr. GREENE. Now, they haven't, but they have suggested that it was not beyond a reasonable doubt that such a cause and effect relationship existed, if my understanding of the Public Health Service statement was correct.

Mr. MEADER. No one, I assume, could claim that a cause and effect relationship between lung cancer and smoking had been established until the cause of lung cancer, itself had been established, and I don't think anyone in these hearings has claimed that they know the cause of cancer.

Dr. GREENE. That is right.

Mr. MEADER. I have gathered from your statement that until such a discovery is made, that we ought not to do anything. Am I to conclude that from your statement?

Dr. GREENE. I don't know whether you are concluding that or not. I shouldn't think so. If there was evidence, for example, that tobacco products produced cancer in transplants of embryonic human lung, I would be concerned. But there is no such evidence. I should perhaps emphasize the fact that I have tried that experiment. One can transplant embryonic human tissues to laboratory animals, where they survive and grow.

If one obtains embryonic human organs or tissues before the fourth month of gestation and transplants them in certain sites—the eye or the brain, of laboratory animals—these organs survive and grow. They, therefore, offer an excellent material for the study of disease that man has, or for carcinogenic agents that might be effective in man.

Now, we have infiltrated fragments of embryonic human lung with these tobacco products, including Wynder's tar, transplanted them to adult animals, and let them go on, and there have been no cancers formed in them at all.

Mr. MEADER. I am interested that you brought that up, because I had a question as you were reading your prepared statement, on page 9 where you say pertinent experiments were carried out and performed by a number of different investigators utilizing various techniques and no causal relationship was found.

Now, I am wondering if you are not, perhaps, at least apparently, guilty of another fallacy of human reasoning, in leaving the impression that, because a particular experiment didn't establish the casual relationship, therefore, there was no casual connection.

Dr. GREENE. No; I don't mean that at all, of course. Experiments have to be continued; there is no question about it. The fact that a thousand experiments have been carried out and nothing has been shown doesn't mean that No. 1,001 won't show it.

Mr. MEADER. Now, I wanted to draw your attention to Dr. Wynder's comment on this point. I asked him about his difference in point of view from Dr. Little, and this is what he had to say, at page 170 of our transcript of Friday, July 19.

Dr. Little has often said on the association:

"Now, you demonstrate association but not causation." I thought that, by showing you all the evidence to find how it fits with the presumptive evidence, the causative relationship is the more direct one to assume. But, from my point of view, it makes very little difference whether we argue about the words "contributing to, initiating, causing, association, relating to," the important factor is that the person who doesn't smoke rarely gets lung cancer, and the more you smoke the more lung cancer you get. That is the important issue.

I certainly admit there are numerous intrinsic factors of lung cancer about which we know very little, but if we were to apply the same argument of Dr. Little to the point of radiation, and I think it is a good example to use, we can rule out that radiation couldn't possibly cause cancer in men. Yet I am sure that you will not find a single scientist in this country who will say that radiation cannot cause cancer in men. We have the evidence in men. We have the evidence in the experimental animals. The relationship is identical.

Then, again, Dr. Wynder said:

One of my hobbies is history, and one of the things in history of medicine that interested me most was in the last century when many women, particularly in Europe, died from childbed fever. Dr. Semelweiss, in particular, demonstrated this was due to the fact that doctors didn't wash their hands.

Then he gave the example of bubonic plague in London.

At that time Dr. Snow, who did the first plague or study on epidemic controlling, got the idea that this might have to do with a Broad Street pump where there was water consumed. He did a study and found that people who drank water from that pump got cholera. At that time they didn't have the vaguest idea what the basic cause of cholera was. Yet he went to the board of governors one day and said, "Let's try to remove the handle of that pump just on the basis of epidemiological evidence." They removed the handle and the cholera epidemic ceased.

I am reading these statements of Dr. Wynder because it seems to me he took a different attitude toward this question than you have, and that you and Dr. Little seem to require an almost perfect, logical indication of proof before you believe anything should be done, and Dr. Wynder says we can't wait that long, that something can be done before we find the basic cause of cancer. And he used these historical examples on which I would like your comment.

Dr. GREENE. Of course, Dr. Wynder has selected his examples. There have been other supposed cause-and-effect relationships derived from statistics that haven't held. I think, actually, one of them was cited in one of the reports, and that was due to the statistical studies on pellagra in France, where it was found that the French population ate a great amount of corn, particularly among those people who had pellagra and, therefore, they assumed that the pellagra was due to the eating of corn.

Well, that is known not to be true. It is not a cause-and-effect relationship. The occurrence of pellagra in those people is not because they ate corn but because they didn't eat anything else but corn, and corn doesn't contain enough tryptophan to manufacture an essential vitamin.

He has selected instances there very carefully.

Mr. MEADER. Let me see if I can clarify this: You do agree that the statistical studies and the association shown by the studies of the Cancer Society and others, and what experimentation there has been, do indicate areas where further experimentation and research should be conducted and in that sense you are in line with Dr. Little?

Dr. GREENE. Well, I think those areas were indicated long before the present statistical studies or the experimentation. Just from simple observation, that the surface of the lung was exposed to the environment and, therefore, environmental factors should be investigated as possible causes of cancer. I don't think the present statistical studies, to my mind, have added much to that.

Mr. MEADER. You don't take the position, I take it from the answers to my questions, although I got the impression from your statement that you did take that position, that nothing can be done until the basic cause of cancer is discovered?

Dr. GREENE. No; the treatment for tuberculosis was effective, or comparatively effective, before we knew the cause. You don't have to know the intimate cause of a disease in order to treat it effectively, or to treat it.

Mr. MEADER. That is all, Mr. Chairman.

Mr. BLATNIK. Mr. Minshall.

Mr. MINSHALL. Who requested or asked you to write the introduction for this book, Science Looks at Smoking, by Eric Northrup?

Dr. GREENE. Eric Northrup.

Mr. MINSHALL. Were you paid for that in any way?

Dr. GREENE. Yes.

Mr. MINSHALL And who paid you?

Dr. GREENE. Eric Northrup.

Mr. MINSHALL. Do you know anyone at the Hill & Knowlton public relations firm?

Dr. GREENE. No.

Mr. MINSHALL. Have you ever talked with anyone over there?

Dr. GREENE. I must have, but I don't know their name.

Mr. MINSHALL. What do you mean, you must have?

Dr. GREENE. People are continually calling me up on the telephone asking me questions.

Mr. MINSHALL. Somebody called you from Hill & Knowlton; is that correct?

Dr. GREENE. Not to my knowledge. I couldn't tell you.

Mr. MINSHALL. You said they must have. Now, have they?

Dr. GREENE. I said they must have, but I don't know that they have.

Mr. MINSHALL. You are not at all consistent.

Dr. GREENE. Well, I don't know, then; let's say that.

Mr. MINSHALL. You have many acquaintances, don't you, in the tobacco industry?

Dr. GREENE. No; not many.

Mr. MINSHALL. Have you discussed your testimony with anyone in the tobacco industry, or Hill & Knowlton, prior to appearing here today?

Dr. GREENE. I tell you I don't know anybody at Hill & Knowlton I have not been concerned with the tobacco companies, except in one instance where I acted as consultant in the case of some proposed litigation that was coming up.

Mr. MINSHALL. When was that?

Dr. GREENE. That was back several months ago.

Mr. MINSHALL. And what was that in relation to?

Dr. GREENE. Some patient, some individual who had died of lung cancer and the wife I believe was considering suing the tobacco company or something like that.

Mr. MINSHALL. And you were going to appear on behalf of the tobacco company?

Dr. GREENE. Yes.

Mr. MINSHALL. Did you appear?

Dr. GREENE. No.

Mr. MINSHALL. Hasn't it come to trial yet?

Dr. GREENE. No; the case was dismissed.

Mr. MINSHALL. Settled out of court?

Dr. GREENE. No; it was dismissed. Let me emphasize a point with regard to this book that you are talking about. One time several years ago, Mr. Northrup called my laboratory about something entirely different, I have forgotten what it was, and we had a long conversation. During the course of this conversation the subject of tobacco smoking came up and that day I had seen a number of people who had given up smoking because of the tobacco scare and I was contacted and we discussed the question as to whether or not the public was sufficiently informed as to the basis on which these conclusions were drawn.

Mr. MINSHALL. How long have you know Eric Northrup?

Dr. GREENE. I think that was his first visit.

Mr. MINSHALL. When was that?

Dr. GREENE. Approximately 2 years ago.

Mr. MINSHALL. What was the occasion for him visiting your laboratory, or your hospital, wherever it was?

Dr. GREENE. I think it had something to do with transplants of embryonic organs that he wanted to write about. I don't really remem-

ber. Or the transplantation of human cancer, or some such thing as that. In any case, after discussing this business, I told him that I thought it would be a great service to the American public if someone who was a good writer wrote a book about it, and he said he was going to do it and asked if he did, would I write an introduction and I consented to do it.

Mr. MINSHALL. Do you know if anyone paid Eric Northrup for writing this book and, if so, whom?

Dr. GREENE. I assume the publishers.

Mr. MINSHALL. Do you know who the publishers were?

Dr. GREENE. Coward-McCann, I believe.

Mr. MINSHALL. Have you received any grants or other financial assistance from anyone associated with the tobacco industry?

Dr. GREENE. None whatsoever.

Mr. MINSHALL. You were paid for your testimony, or your advice to the tobacco people several months ago?

Dr. GREENE. Yes; they paid for my time.

Mr. MINSHALL. That is the only time they have ever used your services?

Dr. GREENE. Yes; let me emphasize——

Mr. MINSHALL. Let me ask you now, have you discussed this at all with anyone prior to testifying here today?

Dr. GREENE. I discussed it with my associates at Yale, I discussed it with Dr. Macdonald, who is to testify next. I can't tell you but I have been discussing this for a great many years.

Mr. MINSHALL. When were you asked to testify here today, how long ago?

Dr. GREENE. Some day last week.

Mr. MINSHALL. And who have you discussed your testimony with since that time?

Dr. GREENE. Everybody that I came in contact with.

Mr. MINSHALL. Did anybody get in touch with you?

Dr. GREENE. Yes.

Mr. MINSHALL. Concerning who?

Dr. GREENE. Science News called me up the other day and asked for a transcript of my testimony.

Mr. MINSHALL. Did anyone connected with the tobacco industry or any publishing firm or public-relations house get in touch with you?

Dr. GREENE. No.

Mr. MINSHALL. Did anyone assist you in writing your statement?

Dr. GREENE. There was someone who called me up and asked if I was invited to come here to talk, would I accept. I said I would first have to be asked. Now I don't know who that individual was. He might have been from the tobacco committee, I don't know. I didn't catch the name over the telephone.

Mr. MINSHALL. When was that call made?

Dr. GREENE. A week or so ago.

Let me emphasize the fact that I am an investor. The funds for my research come from the American Cancer Society, the Public Health Association, the Jane Hoffman Childs' Fund and they haven't got any ax to grind in this business and neither have I—except my personal prejudice and I am prejudiced about tobacco.

Mr. MINSHALL. You see no reason then to use filters on cigarettes?

Dr. GREENE. I don't know anything about filters on cigarettes.

Mr. MINSHALL. You have already testified to that effect that you see no reason for putting them on them?

Dr. GREENE. I see no reason for putting them on, if they are supposed to withdraw carcinogenic materials which according to my point of view are not present.

Mr. MEADER. Wouldn't it be better to say, Dr. Greene, not that the carcinogenic materials according to your point of view are not present, but there is no proof that they are present?

Dr. GREENE. There is no proof that they are present. And whether or not these filters take them out or not, I am in no position to say.

Mr. MEADER. If they are not there they can't take them out.

Dr. GREENE. If they are present, I couldn't tell you whether they will take them out.

Mr. MINSHALL. Just one other question, Doctor: You say you don't believe in statistics. You made that pointblank statement.

Dr. GREENE. I don't believe that a distinction can be used to draw out cause and effect relationships.

Mr. MINSHALL. Just for my own information, weren't some statistics used in resolving Dr. Salk's now famous discovery?

Dr. GREENE. Yes, but aided by experimentation.

Mr. MINSHALL. Then statistics aided by experimentation are of some use?

Dr. GREENE. Oh, of course, all experimental work, most all experimental work is based on statistical observation. Oh, yes; of course.

Mr. MINSHALL. Then statistics are of some value?

Dr. GREENE. Why, of course. I didn't say they weren't. I said statistics could not be used to form judgments. The judgment has to be based on experience or experimentation.

Mr. MINSHALL. That is all I have.

Mr. PLAPINGER. When was the introduction to the Northrup book written?

Dr. GREENE. I would say I completed it about a year and a half ago. A long time ago.

Mr. PLAPINGER. That was before the American Cancer Society recently published its statistical information?

Dr. GREENE. Yes.

Mr. PLAPINGER. Incidentally, in your testimony you pay particular reference to Dr. Wynder. I am interested in that because in the joint Report of the Study Group on Smoking and Health (see appendix, exhibit 6, p. 421). Under epidemilogic evidence as supported by laboratory studies on animals—it states that "at last five independent investigators had produced malignant neoplasms by tobacco-smoke condensate." That would indicate that there were investigations other than Dr. Wynder's alone that confirmed the fact that there were carcinogenic agents.

Dr. GREENE. Let me emphasize, this doesn't say anything about the negative experiments.

Mr. PLAPINGER. I understand that. Now, sir, on the negative experiments. You refer to the fact that there were transplants—unsuccessful as far as producing cancer were concerned.

Dr. GREENE. Yes.

Mr. PLAPINGER. Has this data been published?

Dr. GREENE. It has not been published. If I attempted to publish all my negative results I wouldn't be able to do any further research. I don't publish negative results and I expect that has happened throughout the country. If people have tried this stuff and gotten negative results they have therefore not published it.

Mr. PLAPINGER. Except that within an area that is literally immersed in such public interest, it might be to the benefit of the public and the scientific world at large, if this negative data were published.

Dr. GREENE. I expect that could happen.

Mr. PLAPINGER. When Dr. Little appeared here I questioned him concerning a quotation attributed to him as follows:

Any possible role of smoking in the ideology of lung cancer remains an unresolved question. It cannot be said that smoking has been absolved from suspicion. Neither have the charges been proven.

Would you comment on that statement?

Dr. GREENE. I would agree with that statement.

Mr. PLAPINGER. I quote another statement attributed to him:

Although no definite evidence exists concerning the relation between use of tobacco and the instance of lung cancer, it would seem unwise to film the lungs repeatedly with the suspension of foreign particles of tobacco products of which smoke consists.

Dr. GREENE. I don't think it is unwise.

Mr. PLAPINGER. You would disagree with Dr. Little as far as that statement goes?

Dr. GREENE. As far as that statement goes.

Mr. PLAPINGER. Incidentally, he did not limit it only to tobacco smoke later on. This was a 1944 excerpt from an article that he had written. In his testimony, Dr. Little said this statement would apply not only to tobacco smoke, but to any irritant.

Dr. GREENE. Your question is, Do I think it unwise to fill my lungs with tobacco smoke?

Mr. PLAPINGER. You answered that question. I was just explaining Dr. Little's further statement before the subcommittee.

Mrs. GRIFFITHS (presiding). Thank you very much for your testimony, Dr. Greene.

Dr. Macdonald.

STATEMENT OF IAN G. MACDONALD, M. D., UNIVERSITY OF SOUTHERN CALIFORNIA

Mr. MACDONALD. I am Ian G. Macdonald, and I am a doctor of medicine. I received my doctor of medicine degree from McGill University in 1928, and am clinical professor of surgery at the University of Southern California, School of Medicine, where I have also been coordinator of cancer teaching for the past 10 years.

In addition to a number of national surgical and other medical society memberships I am a member of the American Association for Cancer Research. I have been engaged in the treatment of cancer and allied diseases since 1935, and am a surgeon slightly gone wrong in that I am certified by the American Board of Radiology in the area of radiation therapy.

I also happen to be a member of the National Board of Directors of the American Cancer Society and am a past president of the Cali-

fornia division of that society. I am also chairman of the subcommittee on cancer of the committee on research of the American Medical Association.

The constant reiteration of the claim that cigarette smoking is one of the important causes, if not the most important cause of lung cancer, has certainly persuaded many that a cause and effect relationship has actually been established. And I am sure I do not need to recite the results of a Gallup poll reported in the public prints of yesterday to the effect that some 50 percent of the general public now entertains at least a suspicion that cigarette smoking may actually be one of the causes of lung cancer. It seems to me many of those who so fervently advocate this relationship have an attitude that is almost evangelical and they are oblivious of the fact that virtually the entire basis on which this belief rests is statistical.

One must say that there is an apparent association between cigarette smoking and lung cancer, but when one reviews the total evidence, I think it is not possible to establish a cause and effect relationship.

I should like to offer some remarks made by Berkson, the biometrician at the Mayo Clinic, as follows. He said:

> I am not affected by the considerable number of statistical studies published, showing an association between smoking and cancer of the lungs. On the contrary, undeviating consistency of statistical results all in support of the same conclusion is in some circumstances the hallmark of spurious statistical correlation. If correlation is produced by some elements of the statistical procedure itself, it is almost inevitable that the correlation will appear whenever the statistical procedure is used.

It really shouldn't be necessary but it probably is, to say that things which are connected by a cause and effect relationship will, of course, show a high degree of correlation satistically, but a high degree of correlation between two things certainly does not necessarily mean a causal relationship.

Now, even at the risk of being repetitious, I should like to offer a few very general remarks about the nature of cancer as an extremely complex group of diseases of extremely disparate manifestations of abnormal growth of tissues of the individual in whom this disease arises. One should not refer to it as "this disease." It is in fact a whole galaxy of diseases.

A fundamental definition of cancer is that it is an abnormality of growth of cells resulting from a disturbance in an extraordinarily delicate system of check and balance which, under normal conditions, allows the body to replace worn out cells or to repair effectively the result of injury of various sorts. The possibility of a given individual developing cancer depends on two basic factors—this may be a little oversimplification:

One: The capacity of the individual to respond to unfavorable influences by developing cancer, or the lack of the capacity, is now accepted by many as being genetically determined, that is, the ability of the individual's tissues to respond or not respond to unfavorable influences was laid down at the time of fertilization.

Mrs. GRIFFITHS. May I ask you at that point: Isn't that statistically determined?

Dr. MACDONALD. The genetic potential to develop cancer in most humans cannot be statistically demonstrated.

Mrs. GRIFFITHS. Cancer occurs, does it not, in families? That is, if your grandmother had it, you could have it, too? If one sister had it at 45, wouldn't another sister possibly develop it at 45?

Dr. MACDONALD. That is true, and there are certain cancers such as breast cancer, for example, where there is a distinct traceable hereditary pattern by study of family groups. But referring to the larger problem of all cancer, it is the conviction of many and I am certainly one, that there has to be within the individual the capacity to respond abnormally and that in its ultimate expression is cancer.

The second very general factor is the exposure of the individual to environmental factors which, in the susceptible individual, may eventually result in the degree of abnormal growth finally resulting in cancer.

Now, we don't have too much information about some of the genetic factors. We do have considerable information about predisposing environmental influences which may lead to cancer, but we are still ignorant of the actual trigger mechanism that sets off the cancerous process in any individual.

Now, in the next few sentences are offered a commonplace illustration of this fact in that individuals who have sensitive skins and who are what I refer to as heliophobic—that is who are intensively and abnormally sensitive to sunshine—are candidates for the development of skin cancer, unless they exercise due care and caution in exposure to sun.

At the same time, people who have a sort of average toleration of sunshine, if they overexpose to sunshine, they, too, will develop cancer of the skin. In either of these groups for every hundred people, either the sensitive variety or those who have overexposed themselves to solar radiation, there will develop the stigmata of skin damage by sunshine. The little erosions of the skin that break and heal and bleed a little occasionally. And in our part of the country where we rejoice in seven times more hours of sunshine than Chicago——

Mr. PLAPINGER. This is a statistical analysis?

Dr. MACDONALD. Yes, and in California we happen to have about 8 times more skin cancer than in Illinois. And yet of the hundreds of people one sees in their 60's and 70's in this area in which I live, with these areas of damaged skin, there are only 2 or 3 or perhaps 5 at the most out of every hundred in whom these changes actually go on to be skin cancer.

And we haven't the slightest idea why two individuals with comparable damage, one will go on to develop skin cancer, the other will live to be 92, not having developed any such change.

Mr. MEADER. Madam Chairman, may I ask a question?

Dr. Macdonald, I wasn't quite satisfied with the answer you gave the chairman a minute ago. It seems to me that these generalizations about cancer, as long as you do not know the trigger mechanism or the causation of cancer, must necessarily be based upon statistical studies. Isn't that true?

Dr. MACDONALD. In a way sir, it ties into some studies in which I have been interested for many years, having to do with what I call the biologic potential of cancer.

Not only is the individual either capable or not capable of developing cancer but having developed it, he develops a type of cancer which

is either going to be highly unfavorable or very favorable type of growth, or any other spectrum of cancers in between.

I do not have, sir, any statistical proof, I have no scientific proof that my concept of the capacity to develop cancer is correct. I believe it thoroughly.

Mr. MEADER. There are two factors, heredity and environment which have a bearing upon the person becoming cancerous, that seems to be based only upon a number of specific cases in which these two factors appear, isn't that correct?

Dr. MACDONALD. No, it is a little more complicated than the word "heredity" implies. It has to do with the capacities of the cells of the individual.

Mr. MEADER. What I am saying is that unless you have the definite scientific causative relationship, a foolproof perfect case showing the causation of cancer, what else do you have to rely upon except cases that have arisen and the statistical association about these factors and the occurrence of cancer?

Dr. MACDONALD. Do you mean what do I rely on upon which to base this concept?

Mr. MEADER. What does anyone have to rely upon except a collection of figures showing where these cases are present and a person has contracted cancer?

Dr. MACDONALD. I base this on my study of and judgment concerning the behavior of cancer in the individual. I think it is so highly specific a process that it must be of genetic determination.

Mr. MEADER. What I am getting at, to be very plain about it, is that you and the witness who preceded you and Dr. Little have discounted this statistical association. Yet it seems to me that unless you have the actual cause of cancer, almost everything we know about cancer is based upon statistical association and I want to know whether that is correct or not in your view?

Dr. MACDONALD. I am sorry, sir, it is not, and again I will say that my belief about this genetic capacity is not provable by statistics. It is provable, if it is proof, and I believe it is, by a study of the behavior of cancer in individuals and not on a statistical, mass basis.

Mr. MEADER. It may not be a survey such as conducted by the Cancer Society of 187,000 men but it is based upon the observations of medical people over the course of the years and essentially it is based upon the facts found in individual cases from which you deduce that the occurrence of cancer has a relationship to these factors which you have stated, is it not?

Dr. MACDONALD. In some respects. My belief and my understanding of the cancerous process come from data which I have analyzed in a series of 800 to 1,200 patients with cancer of certain types, yes.

Mr. MEADER. Is that not a statistical association, whether done by survey or questionnaire method or whether done by the collection of cases of cancer?

Dr. MACDONALD. The kind of work I was doing was not an association, it was determining behavior within the cancer, itself.

Mrs. GRIFFITHS. If 200,000 people move out to California and are exposed to sunlight over a certain period if time, can you predict the number of people who will have cancer?

Dr. MACDONALD. I could if I had the table to refer to that shows the figure.

Mrs. GRIFFITHS. If those 200,000 people had remained in Iowa and not been subjected to that sunlight can you predict how many fewer people would have had cancer of the skin?

Dr. MACDONALD. There are figures to indicate that.

Mrs. GRIFFITHS. Then are you saying to us that while it may be true that the sunlight will cause cancer in some people, that some people who are exposed to a given amount of sunlight will have cancer, many others will not have, and that this is also true of smoking and, therefore, there is no relation between sunlight and cancer, and smoking and cancer?

Dr. MACDONALD. No, I was not making that intellectual jump.

Mrs. GRIFFITHS. Are you saying there is some relation between sunlight and cancer and smoking and cancer?

Dr. MACDONALD. I am saying, Madam, that sunlight is one of a number of factors which may act as predisposing factors to the development of skin cancer, but that we still have no fundamental understanding of the trigger mechanism, the real, basic determinative cause of cancer.

Mrs. GRIFFITHS. Because you don't understand what causes it, are you saying that sunlight is not the cause and smoking is not the cause?

Dr. MACDONALD. Sunlight is a predisposing factor. My attitude toward smoking, if I may, I would sooner develop as we go along.

Mrs. GRIFFITHS. Very well, but we will remember it. Proceed.

Dr. MACDONALD. One further generalization I would like to offer, which I think is worth mentioning, to establish a general understanding of the cancerous process; factors which are found rather consistently in the backgrounds of certain individuals with a certain type of cancer may contribute directly to the initiation of the cancerous change or there may be evidence of some other factor which is actually and really the predisposing agent. For example, women who have their first child by the age of 18 and who complete childbearing by age 25 have a distinctly greater hazard of developing cancer of the uterine cervix but these factors are far more common in girls who grow up in less favorable economic situations.

And so the increased incidence of these cancers of the neck of the uterus may well be a reflection of poor nutrition rather than early childbearing and as of this moment we do not know which is the significant factor.

In other words, the factor which is first discovered by statistical studies as being in excess, in the background of the individual who develops cancer, may simply indicate the presence or even the absence of some other factor entirely, which is actually of predisposing or causative significance.

Now there is an increasing amount of evidence in the last 10 or more years to show that multiple factors are operative in setting the stage for a considerable variety of cancers. Again we are talking about predisposing factors.

Commonly there are two or more separate factors frequently of entirely different nature which combine to increase the likelihood of cancer developing in an individual and this is referred to as cocarcinogenesis or the inability of a single agent to produce cancer by itself, but when combined with another, or other agents, it may lead to the development of cancer.

Some of these factors are extremely weak carcinogens or predisposing agents to cancer. In experimental animals, some agents administered in large excess cannot produce any semblance of cancer but their combination in a relatively modest degree with other agents may produce cancer in one who is susceptible.

I would like to remind us all of prior errors concerning causation of disease due to statistical association. During the second or even the third decade of this century when I was a medical student, for example, medical textbooks referred to a certain form of cirrhosis of the liver as being "alcoholic cirrhosis," simply due to the fact that some 9 out of 10 individuals with this type of liver disease could be demonstrated on history-taking, or an accurate knowledge of their background, as having been excessive users of alcohol over long periods of years.

It was also noted, however, even by those most convinced of the causative role of alcohol in this disease, that it did occur at times in individuals who apparently had never been addicted, even in representatives of the clergy in whom a nonalcoholic history could be established.

Well, for years now it has been established that what was formerly regarded as being alcoholic cirrhosis has nothing to do with the alcohol, per se, but to the nutritional deficiency, which is very frequently a concommitant of the unwise use of alcohol.

A high incidence of primary cancer of the liver—that is cancer having its origin in the liver, in some natives of South Africa and in inhabitants of the Malayan Peninsula—was originally thought to be due to environmental exposure. Eventually it was shown with little room for doubt that the causative background or predisposing situation was of dietary deficiency rather than from unfavorable environmental agents.

In the later part of the last century there were dire warnings being issued about the rising incidence of cancer of the mouth and statistical evidence was being cited for tobacco as the principal offender and was, therefore, a causative agent, indicating nothing new, much, under the sun ever comes.

Some of these individuals who correspond to the present evangelistic statistical exponents concerning lung cancer were advocating at that time a severe restrictive program on the use of tobacco in order that cancer of the mouth could be diminished. In the years that have followed it has been demonstrated that cancer of the mouth is attended by a severe nutritional deficiency in the great majority of individuals with this group of cancers. Tobacco has now been relegated—that is except by the most feverish antitobacco apostles—to an extremely minor role, if indeed it occupies any position of causative significance.

Mrs. GRIFFITHS. Does that mean you have discovered what causes cancer of the mouth?

Dr. MACDONALD. Dietary deficiencies, particularly of protein and vitamin B.

Mr. GRIFFITHS. How do you know about that?

Dr. MACDONALD. By a very thorough and excellent study of a large group of clinical patients done by Hays Martin & Associates and published in 1940. Work has been repeated by other observers, including our own group.

Mrs. GRIFFITHS. And you accept that?

Dr. MACDONALD. I accept it, and I accept it, too, on the basis of now almost 20 years of seeing patients with cancer of the mouth.

Mrs. GRIFFITHS. You accept the information, then, on smoking and lung cancer?

Dr. MACDONALD. I accept the information that the important background situation in mouth cancer is nutritional deficiency. Second, if you should be interested, oral sepsis, or an unhealthy mouth, often with dental infection.

Mrs. GRIFFITHS. What do you consider to be the distinguishing feature between that work that you have cited and the work on smoking and lung cancer?

Dr. MACDONALD. The work on lung cancer—I am sorry, I am afraid I did not grasp your question correctly.

Mrs. GRIFFITHS. If you agree with the work that says cancer of the mouth is due to a nutritional deficiency, then why do you not agree with those people—or maybe you do agree—with those people who say that smoking is causing lung cancer?

Dr. MACDONALD. Because I think there are far too many contributory factors in relation to the association of smoking and lung cancer.

Mrs. GRIFFITHS. How did they isolate the factors in cancer of the mouth?

Dr. MACDONALD. One is the clinical recognition by the trained individual of abnormal changes in the mucus membrane of the mouth, throat, gums, and palate so often present. And a very considerable percentage of these people have never smoked. And investigation as to their history concerning diet, objective evidence in the form of blood tests as to the level of serum proteins and the various types of serum proteins in the blood—all of these form a contributory mass of evidence which makes this situation to me entirely valid.

In particular I must say that I develop a little intellectual nausea when we are told nowadays in 1957 that pipes cause cancer of the tongue, cigars cause cancer of the larnyx and cigarettes cause cancer of the lung. This is a degree of specificity that just leaves me slightly staggered.

Some years ago there was a rather fervent discussion in our teaching hospital which happens to be Los Angeles County Hospital, between several who were deeply impressed with the frequency of tobacco smoking in patients with cancer of the mouth, and some of us who were convinced that it was a matter of association, only.

Mrs. GRIFFITHS. Excuse me. There has just been a quorum call. Could you come back at 2 o'clock?

Dr. MACDONALD. I can.

Mrs. GRIFFITHS. Thank you very much and we are very sorry to interrupt you, but we have to answer the roll.

(Whereupon, at 12:05 p. m., the committee adjourned to reconvene at 2 p. m. of the same day.)

AFTERNOON SESSION

Mr. BLATNIK. The Legal and Monetary Affiairs Subcommittee of the Government Operations Committee will resume public hearings regarding misleading advertising regulated by the Federal Trade Commission, with specific reference to filter-tip cigarettes.

We shall continue with the interrupted testimony being presented by Dr. I. G. Macdonald, clinical professor of surgery, University of Southern California, School of Medicine, Los Angeles, Calif.

I believe this morning, Doctor, you were on page 6. We had considerable questioning at that time which interrupted your testimony. Would you proceed from where you left off so that there wil be no break in your testimony?

Dr. MACDONALD. I was about to comment at that time, sir, that some years ago at our teaching hospital, the Los Angeles County Hospital, because of some interest in the subject, consecutive histories of some 100 patients with cancer of the mouth and throat were obtained, along with similar histories of patients in the medical wards without cancer of any sort.

Now, this survey revealed that 82 percent of the patients with mouth cancer had a history of steady smoking, while 80 percent of the patients on the medical services had the same sort of smoking history.

What was much more striking was the fact that 92 out of 100 patients with cancer of the mouth and throat had in their background various degrees of nutritional deficiency, some of them as a concomitant to addiction to alcohol, some of them because of a past history of ulcer of the stomach or restricted diets, people who were food faddists, and so forth.

One of the principal arguments that smoking is responsible for an assorted increasing mortality from cancer of the lung is based on the gradually arising mortality statistics for lung cancer generally quoted. I think it is more than proper, even though I am here repetitious, to offer some inquiry as to the validity of this alleged increase. It is certainly a striking fact that from the year 1900 to 1956 there has been a reduction in crude death rates from the principal respiratory diseases from 430 to 57 per hundred thousand, and there are, of course, United States Public Health Service, vital statistics.

When you consider now that reasonably exact measures for diagnosis of the lung cancer have become available only recently, it is obvious that many of the deaths in the earlier decades of this century were actually due to lung cancer were recorded in vital statistics as pneumonia, influenza, tuberculosis, and so forth.

If you accept the fact that this error of past decades was as little as 5 percent, which in my opinion is reasonable, this adjustment would show that there has been no real increase in lung cancer during this century. There has been some degree of increase simply because of the survival of larger segments of our population to an older age at which time they become more apt to develop lung cancer.

Now, there have been a number of investigators who have reviewed evidence of this sort and one I would quote is the investigation of Dr. Paul E. Steiner, who is professor of pathology at the University of Chicago. He is regarded generally in our profession as being one of the outstanding authorities in cancer research, he is a former president of the American Association for Cancer Research.

Dr. Steiner says that an increase in the frequency of lung cancer is reported in most clinical studies and some mortality surveys, but in

very few autopsy studies. His observations I believe deserve some quotation as follows:

> Great improvement has occurred in recent decades in the clinical diagnosis of lung cancer but accuracy in the autopsy recognition of the natural types has undergone little change, having started from a much higher level. These facts cast doubt on whether the increase is disproportionate to other cancers. Even in the autopsy studies, reported increases may not be real but represent merely a shift in the type of material that the clinical departments send to the autopsy room.

Steiner remarks that in St. Louis in 1935 to 1950, in 12,400-plus autopsies, lung cancer had apparently increased from 1.5 percent to 1.9 percent, to 2.5 percent of all autopsies in 3 successive 5-year periods, but there were 2 other types of cancer, those of the pancreas and breast that had increased nearly as much as that of the lung and that all cancers combined had increased from 16.8 to 20.6 percent during this same period under study.

Summarizing this phase of the problem then I think it is proper to say that while most of the reports of studies made on clinical diagnosis of lung cancer made during life do show an increase; that studies that are based on autopsies vary. Some of them do show an apparent increase, others show none at all. The accuracy of diagnosis at post morten examination has changed very little since 1900, but the accuracy of clinical diagnosis has tremendously improved.

In view of these facts, then, an increase in lung cancer disproportionate to that for all or selected types of cancer in my opinion has not been demonstrated in those geographical regions and perhaps even in none.

Now, as to the evidence claiming that smoking causes lung cancer on this basis of statistical survey. There are a number of geographical disparities which I would call to your attention which in my opinion seem to suggest invalidation of the thesis of cigarettes as a causative factor in lung cancer.

Australia, for example, 1949 to 1951, tobacco consumption averaged 4.7 and then 4.9 pounds per capita, and this compared with 5 pounds per head in the United Kingdom. In Australia the incidence of lung cancer was 13.3 per hundred thousand as compared to 55.5 for the United Kingdom.

Taking the age group 55 to 64, the Australians had 30.5 cases per hundred thousand, the United Kingdom, 111 plus.

Let's take an example closer to home. In a recent year in Idaho, there were 2,003 tax-paid cigarettes sold per person and, in New York, 2,319 tax-paid cigarettes, but the lung cancer rate in New York was 4 times greater than that in Idaho. The difference in industrialization in New York and Idaho is too well known to require any mention.

It seems appropriate here, perhaps, to inject a note concerning an experience in East Pakistan reported by the medical college hospital in that country. During an 18-month study involving 362 cases of pulmonary disease, 20 cases of cancer of the lung were found. The sex, age, and occupational distribution corresponded to reports elsewhere. That is the predominance in males, occurrence in adult lift, and a preponderance of manual workers.

To repeat, the findings emphasize the characteristics which I think are essential in cancer of the lung, and that is maleness, age, and urbanization. All of these cases but two came from rural areas where

alleged carcinogens from tarred roads and automobiles were conspicuously absent. Even more striking, of the 20 cases there were 14 who had never smoked, 4 were occasional smokers, and only 2 were heavy smokers.

This simply helps to add to the confusion of our lack of real knowledge about etiology.

Now, the cause of lung cancer must be much more complicated than the oversimplified thesis of cigarette smoking. The data advanced by those who are mesmerized by statistics include the English findings offered by Doll and Hill. Their controls—that is patients without lung cancer, actually contained more subjects in the moderate smoking group than there were lung-cancer patients in that moderate smoking group. Their figures would just indicate that moderate smoking is actually commoner—speaking of the English sample now—in persons without lung cancer. Even these data may indicate smoking to be a harmless pastime up to 24 cigarettes per day. One could modify an old slogan: A pack a day keeps lung cancer away.

Still another interesting fact in the data collected by English investigators concerns the individual habits of inhalation of cigarette smoke. As you know, the proponents of the theory of cause and effect, cigarettes to lung cancer, have talked about the increase in inhalation by cigarette smokers as compared to the users of pipes and cigars. But Doll and Hill report that cancer of the lung was more common in cigarette smokers who did not inhale than in those who did.

We have had to my knowledge no data on this subject in United States samples.

Still another curious finding advanced by the advocates of cigarette smoking in relation to lung cancer is the fact that the dilution of cigarette smoking by some use of cigars and pipes materially decreases the probability or the risk of lung cancer. This seems to be without reference to the degree of moderation or excessiveness with which the individual uses cigarettes along with cigar and/or pipe. If cigarettes are carcinogenic they surely should be so whether accompanied by tobacco in other forms or not. It is an insult, I think, to reason, to believe that two men who indulge equally in cigarettes will have a different chance of lung cancer simply because one periodically adds to his cigarette smoking the use of tobacco in another form.

Another fallacy in the theory that cigarettes have a casual relationship to cancer of the lung is shown by the differences in the relative incidence of lung cancer in males and females. Long before cigarettes were suspect, the ratio of lung-cancer males to females was in the ratio of 1.5 to 1 or in some parts of the country almost unity, or almost 1 to 1. In the intervening period, disparity in incidence of lung cancer has constantly increased until for the United States at large it is approximately 5 males to 1 female. In some areas as upstate New York it is a ratio of 1 female to 7 males.

During this period, cigarette smoking by females has constantly increased, as a matter of common observation. In a recent study of patterns of smoking by the United States Public Health Service, it is indicated that slightly over 40 percent of women smoke or have smoked since 1930. Thus, there has been at least a 25-year period during which women have been exposed to the possible causative effects of cigarette smoking, if there be any such effects, and nevertheless the disparity

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of incidence of lung cancer as compared with males continues to widen instead of closing.

An interesting pattern of incidence of lung cancer is shown by some racial groups and one outstanding example is the high frequency of lung cancer in Mexican women dying in the Los Angeles area as reported by Steiner. In autopsies performed at the Los Angeles County Hospital from 1918 to 1947, over 40,000 in number, parenthetically, the Caucasoid and Mexican were the largest of the various racial groups involved. Mexican men had only slightly more lung cancer than Caucasoid males, but Mexican females had significantly more lung cancer than Caucasoid females and nearly as much as their male counterparts.

Nevertheless, when we did a rough survey of smoking habits among older Mexican women in Los Angeles, or those in the lung-cancer age, we found their use of cigarettes to be less than that of the corresponding Caucasoid, or actually non-Mexican groups.

If cigarette smoking were a real and distinct factor in lung cancer, the relative incidence in men and women should be approaching parity rather than being more disparate as is the actual situation. Knowing that lung cancer is predominantly a male disease, there should be a correlation in consumption of cigarettes with increase in incidence, if there be any, in lung cancer.

However, much of the increase in cigarette consumption since 1930 has been due to the increasing use of cigarettes by women, and although there are no accurate figures available, to my knowledge, we have tried as best we can to evaluate this part of the problem and our semi-informed guess is that about one-half of the United States increase in cigarette sales has been due to their increased use by women.

In addition, the actual increased use of cigarettes by men allowing for increased use by women is nowhere nearly proportionate to the alleged increase in lung-cancer rates in men.

Now, I think it is entirely proper to compare cigarette consumption and lung cancer in the United States with the same data in England and Wales. From 1920 to 1950, the consumption of cigarettes increased in pounds per capita, in England and Wales, from 1.6 to 3.6 pounds, while lung cancer was increasing from 17 plus to 72 plus per hundred thousand.

In the United States a vastly greater increase in cigarette consumption took place in this same 30-year period, or from 1.6 to 6.3 pounds per capita, but our lung-cancer rate increased only from 6.2 to 31.5 per hundred thousand.

Dr. Richard H. Sweet, who is one of the country's outstanding chest surgeons, recently commented that the concentration of effort being put forth to prove a causal relationship of cigarettes to lung cancer has resulted in neglect of other factors of equal or greater importance which have been studied and reported as showing an association with increased incidence in death rate from lung cancer.

Hammond has published data on this subject showing increase in mileage of State asphalt highways and national consumption of motor fuel, both of which are considerably greater in relative increase than cigarettes. Fuel-oil sales, motor-vehicle registrations also parallel the increase in lung cancer.

There have also been similar sharp increases in the production or consumption of cancer-related industrial chemicals over the same

period of time, such as carbon black, petroleum, coal tar, asbestos, arsenic, and chromite. All of these factors contribute to the greater degree of air pollution in urban areas and constitute, to me, the most reasonable explanation for the difference in the incidence of lung cancer in urban and rural areas.

Some other associations of more estoeric nature can be offered. One is the fact developed by the English investigator, Dr. Percy Stocks, as quoted by Steiner, which shows a negative correlation between the mortality from cancer of the lung and the number of hours of sunshine. According to that criterion, we should have less lung cancer in southern California than we do.

Several studies have demonstrated a relationship in social and economic status and lung cancer and I am inclined to think that this factor is of some real importance.

In New Haven, Conn., for example, Cohart reported the incidence of lung cancer was more than 40 percent greater among the poor than among the more fortunate economic groups. Cohart concluded that unless one could assume that cigarette smoking is inversely related to economic status—and this seems to be an unjustifiable assumption—it is reasonable to conclude that important environmental factors other than cigarettes are contributory.

Clemmenssen and Nielsen reported that there was a significant excess of lung cancer in the male population of poorer classes in Copenhagen. There have been several investigators who ascribe importance to the coexistence of old healed tuberculosis and lung cancer. Some observers suggest that as treatment of tuberculosis improved, those who were enabled to survive are now, years later, the persons highly susceptible to the development of cancer of the lungs as they reach the cancer age.

On this theory one such investigator, Woodruff of Detroit, made the prediction in 1951, that the increasing incidence of lung cancer should reach a plateau, and within a few years actually begin to decline. The present figures indicate that the prediction is proving sound.

Stocks also showed a positive correlation of beer drinking with increased rates of lung cancer, and a negative association between milk drinking and cancer. This may not be nearly as humorous as it sounds, because it may be a reflection of nutritional deficiency or nutritional advantages, respectively.

Differences in lung cancer rates in the country and in the city are consistently found. Several investigators have concluded that residence and employment in urban areas with constantly increasing industrialization constitute more significant and reasonable associations with lung cancer than smoking.

The adjusted mortality rate for cancer of the lung for white males in the United States, 1948 to 1949, was 22.3 per 100,000 in urban areas, and only 12.3 in rural areas.

In the United States, individual cities offer confirmatory evidence of the importance of industrialization. For example, it needs no statistician or expert in sociology to realize that the smoking habits of the residents of Charlotte, N. C., are little different from those of other comparable communities in the eastern part of the country. Yet Charlotte has a lung cancer standardized mortality rate of only

32 per 100,000. The national average on this SMR basis is 100, and this compares to various heavily industrialized areas that run as high as 137. It has been estimated that 6 tons of tarry material fall on each square mile of Manhattan every year, and walking down Fifth Avenue or Madison Avenue can well convince one of that fact. In English towns, various studies have shown that the number of lung-cancers deaths increases in proportion to the number of chimneys per acre in the towns studied.

Now, to take up the experimental problem and in particular the results reported by Wynder, which have already been described here today. Before I offer some attempt of my own to comment on this doubtful activity, I think it is proper to emphasize that mouse skin cancer bears no relationship to human cancer and this is admitted even by those who have done the experiments. The lungs and the skin have different origins in development of the animal and it cannot be assumed they will react to the same irritant.

Still further, mice and men are completely unlike in their response or lack of response to many agents. For example, cancer of the breast can be produced in a high percentage of mice of certain strains by the use of estrogens, or female sex hormone. But the intensive use of the same hormone over a long period of years in female monkeys, has failed to produce a single cancer of the breast, or any other cancer.

I think it is proper, therefore, to make these generalizations on this subject.

(1) The demonstration of the production of cancer by an agent in a mouse cannot be accepted as valid evidence of a similar property in men, particularly if the experiment cannot be repeated successfully in the higher mammals, which has certainly not been done in the instance of tobacco tars.

(2) No satisfactory approach to an equivalent of human lung cancer has been produced in animals after exposure to concentrated doses of tobacco smoke produced by smoking machines. In spite of the fallacies of this particular experimental approach, then, there are still other reasons for denying any real analogy to the situation of smoking in the human, as follows:

(*a*) A certain English investigator exposed 160 mice for 18 months to cigarette smoke. That is for about half of their life span. The smoking mice actually lived longer than the nonsmoking mice.

(*b*) Another British investigator, by name Campbell, observed an increase in lung-tumor rate in mice from 8 percent to 80 percent when they were subjected to prolonged inhalation of dust from roads with a 2 percent content of tar.

An attempt to compare the amount of cigarette smoking by humans, which would be required to equal the exposure of the skin of mice in the Wynder experiments, I think points up the absurdity of the comparison.

Extrapolating the concentration of tar on the small area of the mouse's skin to an equivalent concentration on the large area of the lung of men, and with the help, gentlemen, of one of our statisticians—we also use statisticians—I estimated that a man would have to smoke over 100,000 cigarettes a day to equal the dose that Wynder gave his mice.

Now Wynder has told this committee that when his dose is cut in half no tumors result and thus on this further admittedly absurd

extrapolation, a man could smoke over 50,000 cigarettes a day if he had the time and energy and not be in danger of developing cancer from smoking.

Extrapolations such as this show the dangers of predicting results in man from results in mice and of predicting cancer-producing doses in men from mouse experience. To make an even further attempt at humorous comment, it would seem to me that all the mouse experiments indicate is that mice should smoke their cigarettes and not put the stuff on their backs.

Wynder also said before this committee in his statement that he produced about 50 grams of tar per thousand cigarettes, or 50 milligrams per cigarette. This raises a question whether the tar was produced under conditions that really simulated human smoking, since none of the figures presented to you Wednesday indicated that much tar content to be in the smoke of a cigarette.

Finally, I offer with respect to experimental evidence, the work done by Kotin and his associates at our school of medicine in Los Angeles, in exposing mice to artificial and natural samples of atmosphere, to show how potent the pollutants in air can be in their production of tumors—and, of course, the pollutants in urbanized, highly industrialized areas. Kotin applied various substances that contribute to air pollution on a quantitative basis and he used a lesser dosage in 1 year, weight for weight, than Wynder used in 1 week.

When the material obtained from diesel exhaust fumes was so applied, 17 out of 20 animals so exposed bore tumors or 85 percent and 11 of these 17 bore cancerous tumors.

When a similar experiment was conducted again with a pure strain of mice using a concentrate from gasoline internal combustion engines, 38 out of 86 mice developed tumors. There were multiple tumors in over 60 percent of the animals, or 44 percent and 22 out of the 38 animals had tumors which were cancerous.

Finally, when products simply filtered out of Los Angeles air were similarly applied, 13 out of 35 mice, or 42 percent developed tumors, and 9 of these 13 animals showed malignant tumors on microscopic examination.

None of this makes very consoling reading for the residents of our own Pueblo. It does, however, make obvious the fact that duplicating the Wynder type of experiment not only with diesel and gasoline engine exhaust concentrates, but also with samples of Los Angeles atmosphere, produce much more convincing evidence of carcinogenic ability in mice—for whatever this may be worth—than did the Wynder experiment.

It should be noted, too, that Kotin's observations have been duplicated readily by several other competent investigators. Wynder's results to my knowledge at least have been rather difficult of reproduction by other scientists.

I would finally state then that the total evidence I have tried to review fails to establish any sound basis on which a causative influence can be assigned to cigarette smoking in the production of cancer of the lung.

As in the majority of human cancers, we have at hand an imposing list of predisposing factors, no one of which is of more than ephemeral status at present. The total problem of cancer in man, to me, remains about as much a mystery as it did hundreds of years ago.

Mr. BLATNIK. Doctor, thank you for your statement. I commend you for what is, obviously, a very carefully and well-thought-out statement, prepared with considerable special effort. Regardless of the point of view, we appreciate what, we are sure, was a very serious attempt to submit before the committee a very well-thought-out statement.

Doctor, you make a rather convincing case here in allaying any fears one may have as to any substance in cigaretter smoking being carcinogenic. That brings us to the specific problem that we have, as to why the filters on cigarettes and the terrific effort and expenditure of time and money to promote them. In your opinion, is there anything that is or may be carcinogenic in tobacco smoke?

Dr. MACDONALD. I do not believe there is any evidence to so indicate, at present.

Mr. BLATNIK. Could there be any other substances, to your knowledge, that you think might be harmful to the human body?

Dr. MACDONALD. From a standpoint of production of cancer, I do not believe so.

Mr. BLATNIK. I am thinking of not only cancer, but other effects on the vascular system—carbon monoxide, nicotine?

Dr. MACDONALD. I would not venture to express an opinion on other diseases, sir. You know the person who specializes in cancer always fears heart disease, and the heart specialist is always dying of cancer.

Mr. BLATNIK. Did I understand you to say that, in normal quantities of cigarette smoke—let's say a pack a day—the amount of tar collected would be so insignificant that it is very unlikely that it would induce any cancer, even in mice—that your dose would have to be many times larger or heavier?

Dr. MACDONALD. Yes, sir, based on the Wynder experiment, at least. The concentration which he had to employ was so enormous that it was out of all resemblance to smoking habits.

Mr. BLATNIK. In recent months there has been more and more mention made of a substance called 3,4-benzpyrene. Is that a very carcinogenic substance?

Dr. MACDONALD. 3,4-benzpyrene does have a distinct carcinogenic capacity for certain experimental animals. However, under the circumstances of combustion, I am told by Dr. Kotin—and here I must quote his work—that the dissipation of benzpyrene during a process of combustion is so nearly complete that only an infinitesimal part of it is emitted to the stream of smoke emerging from the tip of the cigarette.

Mr. BLATNIK. Mention was made the other day about research being made by Latarjet and Cusin in France, that benzyprene was definitely present in cigarettes, and carcinogenic. You see, in France the Government owns a monopoly on tobacco. They have been carrying on research on benzyprene. Are you familiar with their research work, Doctor?

Dr. MACDONALD. Only in a general way. Not sufficiently to offer any critical comment.

Mr. BLATNIK. On the basis of your testimony, would it be fair for the committee to feel that, as far as health is concerned, the filters serve little, if any, purpose or function in cigarettes?

Dr. MACDONALD. I have no opinion about filters, Mr. Chairman.

Mr. BLATNIK. If there is nothing to remove in a cigarette to begin with—I am talking from the standpoint of health now——

Mr. MACDONALD. My interests, sir, I have been concentrating in one area. My intense curiosity and desire is to know, is there some relationship between smoking, in particular, cigarette smoking, and neoplasms, tumors, benign or malignant.

Mr. BLATNIK. What is your opinion on it?

Dr. MACDONALD. I do not think there is any sound basis for such belief.

Mr. BLATNIK. So, with or without a filter, a cigarette is not very likely to give you any tumor or cancer?

Dr. MACDONALD. In my opinion, no.

Mr. BLATNIK. That is all. Mr. Meader——

Mr. MEADER. I have no questions.

Mr. BLATNIK. Mr. Minshall——

Mr. MINSHALL. Doctor, you have been specializing for how many years in the cancer field?

Dr. MACDONALD. Since 1936.

Mr. MINSHALL. Have you come to any conclusions of any kind as to what might be the cause of cancer?

Dr. MACDONALD. Not as to the trigger mechanism—the fundamental basic factor that upsets an abnormal cell and starts it on its way to becoming a cancer cell. No. I have learned a great deal over the years, sir, I hope, as to predisposing factors, and how many of them may be avoided, but nothing as to the actual——

Mr. MINSHALL. I don't quite understand what you mean by how they may be avoided—how cancer may be avoided?

Dr. MACDONALD. Yes, sir. For example, it is a curiosity to see cancer of the mouth in a person with a clean mouth and healthy mucous membranes and a good dietary background. That is one way of avoiding a particular group of cancers. But, again to be repetitious, one may have the same factors that lead to cancer of the mouth—namely, a dirty mouth and a bad diet, and one person will develop cancer and another will not, with equally bad situations.

Mr. MINSHALL. Your specialty, then, is in skin cancer?

Dr. MACDONALD. Oh, no, sir.

Mr. MINSHALL. You specialize in all forms of cancer?

Dr. MACDONALD. I specialize in the treatment of cancer, except that of the central nervous system and chest.

Mr. MINSHALL. What is the treatment that you use today for cancer? What is your recommended procedure to your patients, depending, of course, on the degree and the stage of it?

Dr. MACDONALD. It is highly individualized, sir? Surgery in some, radiation in others, and, added to that, some of the newer chemicals and isotopes that are now available.

Mr. MINSHALL. From your studies, have you found out, Doctor, if any agents—say some of the tars; do they have any casual relationship to cancer?

Mr. MACDONALD. Not that I can be sure of.

Mr. MINSHALL. Are you familiar with the British Government studies in allowing for compensation claims to their chimney sweeps over there for the past several hundred years—they have had studies

and found out that it caused cancer of the skin on various parts of the body.

Dr. MACDONALD. Yes, sir. I am. That, however, if I may suggest, is not an analogous situation.

Mr. MINSHALL. What is that situation? I have just heard briefly about it. Could you explain it to us, please?

Dr. MACDONALD. I will try, sir. This is an example of direct contact of a known carcinogen-containing substance—that is, soot—on the skin.

Mr. MINSHALL. Soot is a tar, is it not?

Dr. MACDONALD. It is a tarry substance containing——

Mr. MINSHALL. In other words, this caused the cancer. You admit that, then?

Dr. MACDONALD. Yes, by contact on the exposed skin, just as Wynder produced cancer of the skin by——

Mr. MINSHALL. Then tar has caused cancer; but a moment ago you said it hadn't.

Dr. MACDONALD. Yes; it does, but not in the lung, to my knowledge. I am sorry. I thought you were asking me, sir about the lung. In the skin, yes.

Mr. MINSHALL. Please tell us a little bit more about this situation we started to discuss.

Dr. MACDONALD. About chimneysweeps

Mr. MINSHALL. Yes, please.

Dr. MACDONALD. The cancer-producing substances in soot are several in number. There is 3,4-benzpyrene, and several other hydrocarbons, such as dibezanthyacene and these are distinctly carcinogenic when placed upon the skin.

Mr. MINSHALL. On the human skin?

Dr. MACDONALD. Yes, sir.

Mr. MINSHALL. In other words, there are tars that cause cancer on human beings?

Dr. MACDONALD. Yes, on skin.

Mr. MINSHALL. That is all the questions I have.

Mr. BLATNIK. If there are no further questions, again Dr. Macdonald, we thank you most sincerely.

The next witnesses we have are from the James Rand Development Corp. of Cleveland, Ohio. We have with us Mr. James Rand and Drs. Burhan and Cardon.

STATEMENT OF JAMES RAND; ACCOMPANIED BY DR. AHMED BURHAN; AND DR. S. CARDON, REPRESENTING RAND DEVELOPMENT CORP.

Mr. BLATNIK. Please, first identify the men with you, Mr. Rand.

Mr. RAND. On my left is Dr. Burhan, a medical doctor of the Rand Development Corp. On my right is Dr. Cardon, a doctor of philosophy in chemistry who is with Rand Development Corp. I am president of the corporation. My corporation is a corporation for profit engaged in research and development work. (See appendix, exhibit 24, p. 730.) About 20 percent of our entire work is in the medical field and is directed toward cancer. We have spent over a quarter of a million dollars, 6 years of time and done literally thousands of experi-

ments, consulted with the foremost authorities in this field and had our work checked by competent people.

I would like first to have Dr. Cardon give the factual, chemical analyses that he has completed that have been confirmed by the French Government and others around the world.

Mr. BLATNIK. Dr. Cardon, will you give your full name and a brief summary of your technical experience and the work you are in and then proceed with your written statement which the committee has?

Dr. CARDON. Mr. Chairman, my name is Samuel Z. Cardon. I have a bachelor's degree from the University of Chicago and a master's degree, 1941, University of Chicago, and doctor of philosophy, 1950, from Western Reserve University. My experience in this field has all been with the Rand Development Corp. in the last 4 years.

Recent statistical studies suggest a relationship between the increasing incidence of lung cancer and smoking. This implies carcinogenic activity by the smoke. Accordingly, for several years the Rand Development Corp. has been investigating the nonvolatile fractions of the smokes of cigarettes, cigarette paper, and tobacco for possible known carcinogens. The statistical studies on the relation of lung cancer and smoking pointed especially at cigarette smoking and indicated little or no relation to cigar and pipe smoking. One major difference between these types of smoking is, of course, the paper. Initially, it was thought the carcinogenic activity might be wholly due to the paper, and we were thus stimulated to start with the paper alone.

Indications of fluorescence, characteristic of the benzanthracene derivatives, was first noted by Carroll and Rand in the tars of cigarette paper smoke. The fluorescence was found by us to be due to a known carcinogen 3,4-benzpyrene, and related substances. The identification of 3,4-benzpyrene was based on 4 lines of evidence: The fluorescence spectrum, the ultraviolet absorption spectrum, indication of the presence of the iodine derivative, 6-iodo-3,4-benzpyrene in iodinated purified fractions of the tars, and the recovery of added quantities of 3,4-benzpyrene from the tars. In this method, the tar is condensed from the smoke, a known amount of benzpyrene is added to the tar and the analysis carried out. The amount of benzpyrene actually found was equal to the amount added plus that normally present.

Subsequently, we found 3,4-benzpyrene in the condensed tars of smoke of tobacco, cigarettes, and cigars as well as from the paper. The cigarettes were smoked in a smoke sampling apparatus designed by the research laboratory of the American Tobacco Co., Inc., and manufactured by Phipps & Bird, Inc., of Richmond, Va. Cigarette paper and tobacco were smoldered in a glass tube and the smoke drawn through a plug of glass wool where the tars condensed. Our procedures and techniques for smoking, collecting the tars, and chemical analysis are described in the British Journal of Cancer (vol. 10, p. 485 (1956)), in an article by Cardon, Alvord, Rand, and Hitchcock. (See appendix, exhibit 21, p. 696.)

We found the following amounts of 3,4-benzpyrene:

Cigarette paper: 1 microgram in 1.6 grams of paper

Tobacco: 1 microgram in 8 grams of tobacco

Cigarettes: 40 to 50 micrograms—

that is two different brands of cigarettes—from 400 regular cigarettes, 32 micrograms from 400 filter-tip cigarettes.

The drop in benzpyrene from regular cigarettes to filter tip cigarettes of the same manufacturer was 25 percent, that is, from 40 micrograms to 32 micrograms for 400 cigarettes.

The interesting point here is that according to the Readers' Digest article, the filter tip in question produced more tar than did the regular cigarettes and yet the benzpyrene has been reduced. I would have expected that. Despite the comments that have been made here by previous witnesses that the amount of tar would be expected—that is the quantity of tar would directly influence the amount of biological activity of the tar, that that isn't necessarily true. The tar is a complex mixture and the active constituents of the tar are probably only 1 percent or less of that mixture. It is easily conceivable that a proper filter tip will remove a certain proportion of that 1 percent and still leave the total amount of tar almost the same as it was before, or maybe even increase the tar in some way. The filter tip won't increase the tar but maybe a change in tobacco would increase the tar and still, as in this case, leave the benzpyrene which is the active ingredient, in smaller quantity.

Therefore, while the tar might be the same or more than what it was before, the active ingredient would be less. That is what we find here. Actually it is analogous in some ways to our method of analysis of the tars. Our method of analysis that is in our paper depends to a considerable extent upon chromatographic absorption. In chromatographic absorption techniques we pass fractions of the tars through powders which are absorbents and we can see that the absorbents selectively remove one material and let the other materials pass through. That is the basis for our separation.

In effect, the filter tip, itself, is an absorbent and could do the same thing. It can allow the active ingredient to pass through while removing most of the tar and, conversely, it can remove most of the active ingredient and allow most of the tar to go through, so that there is this distinct possibility that the filter tip could remove a part of the active ingredient of the cigarette smoke as the active biological ingredient.

The smoke of a popular denicotinized cigarette contained about half the benzpyrene of other regular cigarettes with a 40-percent drop in the corresponding filter-tip cigarettes. These results are based on only a few experiments and are to be considered somewhat tentative.

We do have much more thorough experimental work planned under consideration and, once we get done with that, we will know a little bit more about the story of filter tips and cigarettes in general.

Cigars varied greatly with the brand, giving from 1 microgram from 2 grams to 1 microgram from 8 grams of the cigar, based on the weight of the cigars smoked.

We found no corresponding difference in the amounts of tar, which indicates that the amounts of time, while they might be the same, the active ingredient could be materially different in different cigars and different tobacco products.

Considering that cigarette smoke might be carcinogenic due to the 3,4-benzpyrene in the smoke, we went to work to attempt to eliminate this well-known carcinogen from the smoke. Our initial effort was with cigarette paper, and we added substances to the paper which would affect the combustion products so that less benzpyrene would be formed.

Using a rapid-screening method described in a second article by Alvord and Cardon in the British Journal of Cancer, volume 10, page 498 (1956) (see appendix, exhibit 22, p. 710), we were able to test many compounds and found one class, the ammonium salts of strong mineral acids, sharply reduced the production of benzpyrene in cigarette-paper tars. This reduction is 95 percent or more for a 5-percent addition of ammonium sulfamate to the paper. The total tars are reduced and the characteristic fluorescence almost entirely eliminated from the oil-soluble fractions of the tar. This is especially significant, since any carcinogenic compounds of the benzanthracene type—benzypyrene is a benzanthracene compound; we would almost call it a benzpyrene complex of compounds—would be expected to fluoresce in this spectral region, 395 to 465 millimicrons, and no fluorescence in this region is a good indication that not only has benzpyrene been almost completely eliminated but other similar compounds, possibly carcinogenic, have also been greatly reduced or eliminated from the tars.

The treatment was next applied to tobacco, and an 80-percent reduction in 3,4-benzpyrene was effected in laboratory tests. Cigarettes made with treated paper gave 60 percent less benzpyrene than did the same cigarettes made with untreated paper. In preliminary tests, using treated paper and treated tobacco, only the 60-percent reduction was obtained. As yet unexplained is the low effect the treated tobacco had on cigarettes made with it as compared to the large effects of the treated paper.

Our work has been repeated in France by Latarjet, Cusin, and their coworkers at the Pasteur Institute and the laboratories of the French tobacco monopoly, with verification of our results. Their work was reported in the French Bulletin of Cancer, volume 43, page 180 (1956). (See appendix, exhibit 23, p. 716.) Benzpyrene in cigarettes and in other tobacco products has also been found by Cooper and Lindsey in England.

As a chemist of 16 years' experience, I want to make this concrete statement. A known cancer-producing agent has been found in the smoke from cigarette paper, and a lesser amount from tobacco alone and in the composite smoke of cigarette paper and tobacco. This compound is known as 3,4-benzpyrene. It typifies a group of related compounds which has been demonstrated by Wynder, of the Sloan-Kettering Institute, and many others as being strongly carcinogenic. We have found that additives releasing ammonia at the approximate combustion temperatures of cigarettes can reduce by a large factor, or eliminate entirely, these compounds from the smoke of cigarettes.

That concludes my formal statement.

Mr. BLATNIK. Dr. Cardon, you have made a very striking statement, which is the first testimony we have received indicating not only the positive identification of a carcinogenic substance in tobacco smoke—in this case you identify it as benzpyrene, which was indicated to exist in extremely minute quantities. Apparently, from your testimony here, you identify this substance as a carcinogenic substance and, also, you have measured it.

I am not quite clear what you mean. What is a microgram; a millionth of a gram?

Dr. CARDON. Yes.

Mr. BLATNIK. In talking about tars, we measure them in terms of milligrams; that is, a thousand?

Dr. CARDON. Yes.

Mr. BLATNIK. Other witnesses have testified that there was, or possibly was, 3,4-benzpyrene in cigarette smoke, but it was in such extremely minute amounts that it could not and has not induced cancer. Is that correct?

Mr. RAND. Dr. Burban can testify on the medical end.

Mr. BLATNIK. The important thing is that we have identified a specific carcinogenic substance, and you have measured it.

Dr. CARDON. That is right. The previous witness mentioned this cocarcinogenic effect, and I understand there is recent evidence that nicotine itself might be a cocarcinogenic agent. The statement that there isn't enough benzpyrene here to cause cancer, which would depend on using a pure benzpyrene solution, in the presence of these possible cocarcinogen materials, like nicotine and other materials in the tar; I don't think that is valid any more. We don't know how much benzpyrene—we don't know what the minimum amount of benzpyrene is that would produce a cancer in the presence of these cocarcinogenic agents.

Mr. BLATNIK. I have no further questions for the moment.

Mr. MEADER. I wanted to ask Mr. Rand—I notice in the second paragraph of your mimeographed statement it says part of the cost of your cigarette research was underwritten by a small tobacco company.

Mr. RAND. Yes, sir.

Mr. MEADER. That sounds as though it was prior to 1952. Are you still being financed in your research activities with funds from the tobacco industry?

Mr. RAND. No; after we found 3,4-benzpyrene in the smoke, shortly after that, the funds from the tobacco company dropped out.

Mr. MEADER. I understand that yours is a profitmaking institution. From what funds are your present research activities being financed?

Mr. RAND. From our own funds.

Mr. MEADER. You mean you are not being——

Mr. RAND. We have a number of proprietary projects. By proprietary means, we have bought the project, or we initiated it and we financed it ourselves.

Mr. BLATNIK. And then market it?

Mr. RAND. Yes. License people to use our findings.

Mr. MEADER. If there is something here that is carcinogenic you will remove it?

Mr. RAND. That is right, and license the industry to use it. We bought the tobacco company's interest out.

Mr. MEADER. You have no funds from anyone, either a charitable foundation or from an industry fund?

Mr. RAND. Not to date. Ours is a publicly owned corporation.

Mr. BLATNIK. Mr. Minshall.

Mr. MINSHALL. Mr. Rand, I believe you have said that your organization was the first organization at least in this country, to separate and identify 3,4-benzpyrene; is that correct?

Mr. RAND. That is correct. It was developed in Argentina in the middle thirties but at that time equipment didn't exist to identify it

properly and everybody criticized that, but we were the first in this country, certainly, to find 3,4-benzpyrene in products of cigarette smoke, and there is a very simple straightforward method of doing it now with proper scientific equipment.

Mr. MINSHALL. How soon after you made known your discovery to the tobacco people did the funds from the tobacco people disappear?

Mr. RAND. About a year.

Mr. MINSHALL. You have spent how much altogether on this particular project?

Mr. RAND. Over $250,000.

Mr. MINSHALL. How does that compare to funds spent by other concerns either public or private, at least in this country on similar projects?

Mr. RAND. Tobacco industry altogether so far has spent $1,800,000. Whether it is on cancer or not, I don't know.

Mr. MINSHALL. The amount of money you have spent thus far is far in excess, is it not, of any other company of similar size?

Mr. RAND. As far as we know, ours is the most concerted effort.

Mr. MINSHALL. I have no other questions.

Mr. MEADER. Mr. Minshall's question has suggested another to me. Do you know to what extent tobacco companies themselves are seeking information on possible carcinogenic substances in tobacco smoke and are seeking to find ways of reducing or eliminating them if they exist?

Mr. RAND. No, sir; I do not. The only tobacco group that I know of that is seriously interested in our work is the French Government, the tobacco group, and there the Director of Research of the French Government tobacco group was the coauthor of the paper substantiating our work.

Mr. MEADER. If there were any activities on the part of tobacco companies in the field in which you are working, would there be any reason why you should know about it?

Mr. RAND. I don't think so. I think if they did, if they repeated our experiments they would know they were true and would come around looking for a license.

Mr. MEADER. And there hasn't been any activity in that direction, I take it?

Mr. RAND. None.

Mr. BLATNIK. Has the tobacco industry research committee—are they aware of your work?

Mr. RAND. Oh, yes.

Mr. BLATNIK. Have they contacted you for any further information?

Mr. RAND. Oh, yes; we have had several meetings with them and privately they admit that a 3,4 benzpyrene is a dangerous substance and should be removed, if possible, if it doesn't ruin the cigarette—the treatment to remove it. But they say they have no influence with the tobacco industry whatsoever and they have made a report on our findings. They questioned the way be burned our cigarettes, they questioned temperature. We answered every objection they have but they still won't come out and admit——

Mr. BLATNIK. You answered whose objections?

Mr. RAND. All the objections that were raised at the various visits. The first thing they questioned was whether we could find 3,4-benzpyrene.

Mr. MEADER. You don't mean Dr. Little?

Mr. RAND. Yes, Dr. Little and his group.

Mr. BLATNIK. Mr. Rand, we have a copy, and we are certain this is correct and we will recheck it, a news item in the Cleveland News in Cleveland, Ohio, dated February 17, 1956, and it is a short news story report. "A visit made by Dr. Clarence Cook Little, Chairman of the tobacco industry's advisory committee on its visit to Cleveland, made a visit to the James H. Rand Laboratories."

[Article in Cleveland News, February 17, 1956, by S. Severino]

STUDY RAND PAPER FOR CIGARETTES

Cigarette paper made at the James H. Rand Labratories here impressed Dr. Clarence Cook Little, chairman of the Tobacco Industries Advisory Committee, on his visit to Cleveland.

Dr. Little, who spent several hours at Rand Laboratories, said he will send two of his committee to Cleveland for a more thorough study of the work.

Rand, Cleveland inventor and medical scientist, has developed research which showed that cigarettes when smoked give off a substance called benzpyrene, a known cancer-producing agent.

A second research project by the Rand Laboratories was the development of a cigarette paper, treated with an ammonia compound, which prevents the formation of benzpyrene.

Dr. Little said he would ask Dr. Paul Kotin, chemical scientist from the University of California, and Dr. McKeen Catell, professor of pharmacology at Cornell University, to study the Rand work.

Dr. Little admitted being quite impressed by the research in the Bratenahl Laboratories.

Would you recall if that visit took place?

Mr. RAND. That was the first visit that he had made since he was chairman. He followed our work long before he became chairman of the Tobacco Advisory Committee. He encouraged it.

Mr. BLATNIK. He encouraged your work before he came is as chairman of the Tobacco Industry's Research Committee?

Mr. RAND. I am a trustee on his memorial laboratory and I know him very well.

Mr. BLATNIK. You are a trustee at his Jackson Memorial Laboratory in Bar Harbor, Maine?

Mr. RAND. Yes.

Mr. BLATNIK. We have this clipping which will be put in this record and it is something that can be checked if it is to be used as an authoritative document, but it shows the visit was made.

The news item states:

Dr. Little, who spent several hours at Rand Laboratories, said he will send two of his committee to Cleveland for a more thorough study of the work.

Rand, Cleveland inventor and medical scientist, has developed research which showed that cigarettes when smoked give off a substance called benzpyrene, a known cancer-producing agent.

A second research project by the Rand Laboratories was the development of a cigarette paper, treated with an ammonia compound, which prevents the formation of benzpyrene.

Dr. Little said he would ask Dr. Paul Kotin, chemical scientist from the University of California, and Dr. McKeen Catell, professor of pharmacology at Cornell University, to study the Rand work.

Dr. Little admitted being quite impressed by the research in the Bratenahl laboratories.

Were Drs. Kotin and Catell ever sent to further observe and discuss your research?

Mr. RAND. Dr. Catell never came out. He was supposed to come out with a group of five people. They never came out but wrote a report anyway criticizing our work.

Finally I induced Dr. Little to come out and he subsequently sent Dr. Kotin, and Dr. Kotin said the same thing Dr. Little did. He said, "I am not sure 3,4-benzpyrene is carcinogenic but is a suspicious thing and should be taken out."

We are happy they would say that but in a subsequent report on Dr. Little he questioned our methods and the thing was dropped.

Mr. BLATNIK. Are there any further questions?

Mr. Rand, will you later on give us more details of the work that you have been doing with the French Government?

Mr. RAND. Yes.

Mr. MINSHALL. When was it Dr. Little was at your laboratory?

Mr. RAND. It is the date of that newspaper clipping, about a year ago.

Mr. MINSHALL. February 17, 1956, is the date of the newspaper article.

Mr. RAND. That is the correct date.

Mr. MINSHALL. How long was he in your laboratory?

Mr. RAND. Well, he was in the laboratory I would say 3 or 4 hours.

Mr. MINSHALL. And during that length of time you had many conversations with him and he asked many questions?

Mr. RAND. That is right.

Mr. MINSHALL. Did you ever discuss the efficacy of filter cigarettes with him at that time?

Mr. RAND. No, I never did.

Mr. MINSHALL. You merely discussed the work you were doing, about 3,4-benzpyrene?

Mr. RAND. That is right.

Mr. MINSHALL. And explained how you derived it from cigarettes?

Mr. RAND. Yes. We put on a complete presentation with everybody involved in the project giving their own part, slides and pictures and diagrams and charts and the whole presentation.

Mr. MINSHALL. What was his general comment again?

Did he give one at that time?

Mr. RAND. He said, "This is very valuable work." He said, "I think this should be pursued, and I think the tobacco industry should do something about it, because 3,4-benzyprene shouldn't be in the smoke if you can get rid of it."

Mr. MINSHALL. Have you head anything from him since that time or had any discussions with him since that time?

Mr. RAND. I haven't had any report except he sent me a copy of a letter that Dr. Kotin wrote me—I mean wrote him.

Mr. BLATNIK. Could we proceed now to get the medical end of the testimony?

If there are no further questions, we will proceed with Dr. Burhan. Doctor, will you give your full name and position with the Rand Development Corp. and a brief summary of your professional training and experience and the work with the Rand Development Corp.

Dr. BURHAN. My name is A. S. Burhan and I hold bachelor of science and doctor of medicine degrees from the University of Istan-

bul and a master in medical science degree from the Northwestern University Medical School. I am primarily a cytologist, which deals with physiology morphology, and pathology of cells.

I have been associated with the University of Utah in Salt Lake City, the University of Oregon in Portland, Oreg., Northwestern University Medical School and just before joining the staff of the Rand Development Corp. I was associated with the Cleveland Clinic Foundation and Frank E. Bunts Educational Institute.

The following is my official statement on this subject.

The successful isolation of a fluorescing polycyclic hydrocarbon from the cigarette smoke, in this country and abroad, and its identification as 3,4-benzyprene brings the efforts of correlating the increased incidence of lung cancer with heavy smoking toward a finite conclusion.

Upon identification and approximate quantitation of a universally recognized potent carcinogenic agent, like 3,4-benzpyrene and its possible cogeners, and upon developing of a rather simple technique for the inhibition of its formation by the chemists of the Rand Development Corp., we were confronted with the problem of translating into, and reevaluating by means of biological criteria. Based on the initial promising findings by some outside consultants, a new department for biology was established. The following statements are the result of experiments performed on 1,402 animals, which I believe is the largest number of animals used for a certain kind of experiment, so far as I know.

We felt that it was very fortunate to step into the problem at a stage when comparatively abundant leads were available for orientation. The multitude of techniques developed for laboratory evaluation of carcinogens and the natural tendencies of an individual investigator toward his favorite techniques is usually regarded as subject to bias. For some, this needs corroboration of validity by others.

In our case, the orienting leads were identification of 3,4-benzpyrene and the verification of it by fluorescence spectrum, ultraviolet absorption, formation of 6-iodo-3,4-benzpyrene, and finally the recovery of an added amount of chemically pure 3,4-benzpyrene at the end of the process. This way one could base his experimental approach from Percival Potts' observation on the cancer of the skin in chimney sweeps, to the systematic screening of all aromatic hydrocarbons at our times as accomplished by Japanese investigators.

In 1915, two Japanese experimentors, Yamagiwa and Ishikawa, succeeded in producing malignant tumors by application of coal tar to the ears of rabbits. Later, Tsutsui, another Japanese investigator, demonstrated a simpler method for biologic testing of carcinogenic material by merely painting tar on the back of the mice. This latter procedure finally developed into short-term application.

A supposedly carcinogenic material is applied to the skin of the mice and the effects are determined in as short periods as 4 to 5 days. Finally the isolation and identification of 1,2-benzanthracene, 1,2,5,6-dibenzanthracene, 3,4-benzpyrene, among others, in the coal tars as well as soot, et cetera, gave to this test a rather well-established place in the field of experimental carcinogenesis.

Today we have two biologically accepted techniques for evaluating the carcinogenecity of any given material. These consist of short- and long-term cutaneous application of the compound under investigation, dissolved in a suitable solvent. In fact, one is the continuation of the other. A long-term experiment requires an average minimum of 6 months latent period during which continuous close observation and fulfilling other necessary requirements are essential.

The short-term experiment, on the other hand, is more suitable for screening, and provides sufficient information for the feasibility of more detailed studies, including long-term application. Due to the wide variety of compounds tested, and physical condition existing in our laboratory at that time, we based our biological evaluations principally on short-term application. Under close observation of principles of animal experimentation in general, and those of skin testing in particular (like age, sex, state of nutrition, state of health of the skin and its appendages) we have tested the following materials: Tars of the cigarette paper and ammonium sulfamate-treated paper, tars from cigarettes made of treated paper, treated tobacco, untreated paper, treated tobacco, and untreated paper, treated tobacco and untreated paper, untreated tobacco. The dosage applied to each animal was 1 microgram per day as calculated on the basis of 3,4-benzpyrene as estimated spectroscopically. This amount corresponds to the tar obtained from 10 cigarettes. In the case of ammonium sulfamate treated components, tars derived from equivalent number of cigarettes—10- -were used to obtain a picture comparable to that of untreated cigarettes.

The results obtained were compared with those induced by known potent carcinogens like methycholanthrene and dibenzanthracene, and with those caused by chemically pure 3,4-benzpyrene.

I would like for you to refer to figure 1, which shows a very sketchy picture of the skin of a normal mouse. Here you will see that the mouse skin is made up of two parts as in human beings, the dermis and the epidermis. The dermis is followed by the fat layer.

There is shown here a large gland known as the sebaceous gland. Instead of reading from the statement I would like to show you the ektachron photomicrographs.

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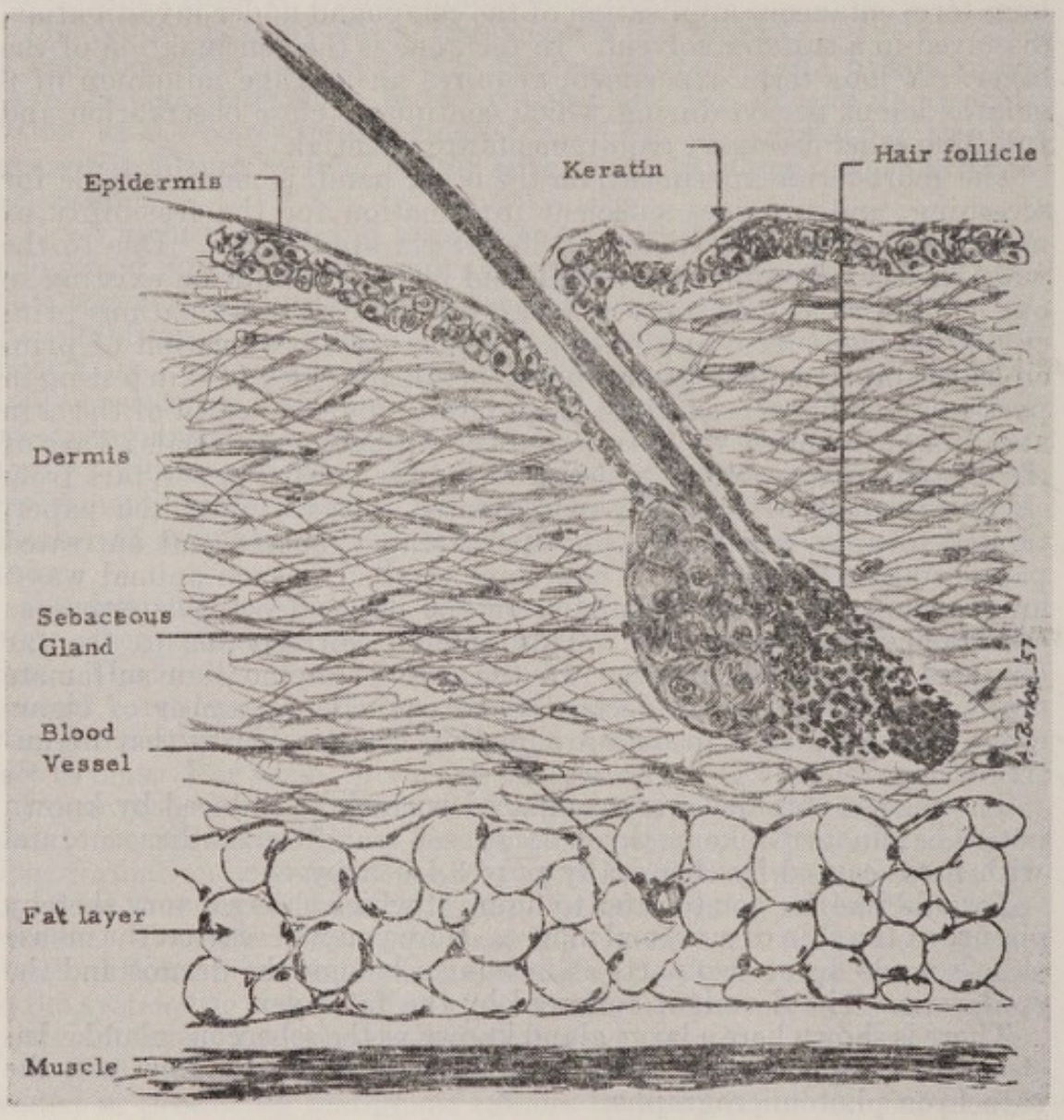


Figure 1.—Schematic drawing of the microscopic structure of the skin of normal mouse.

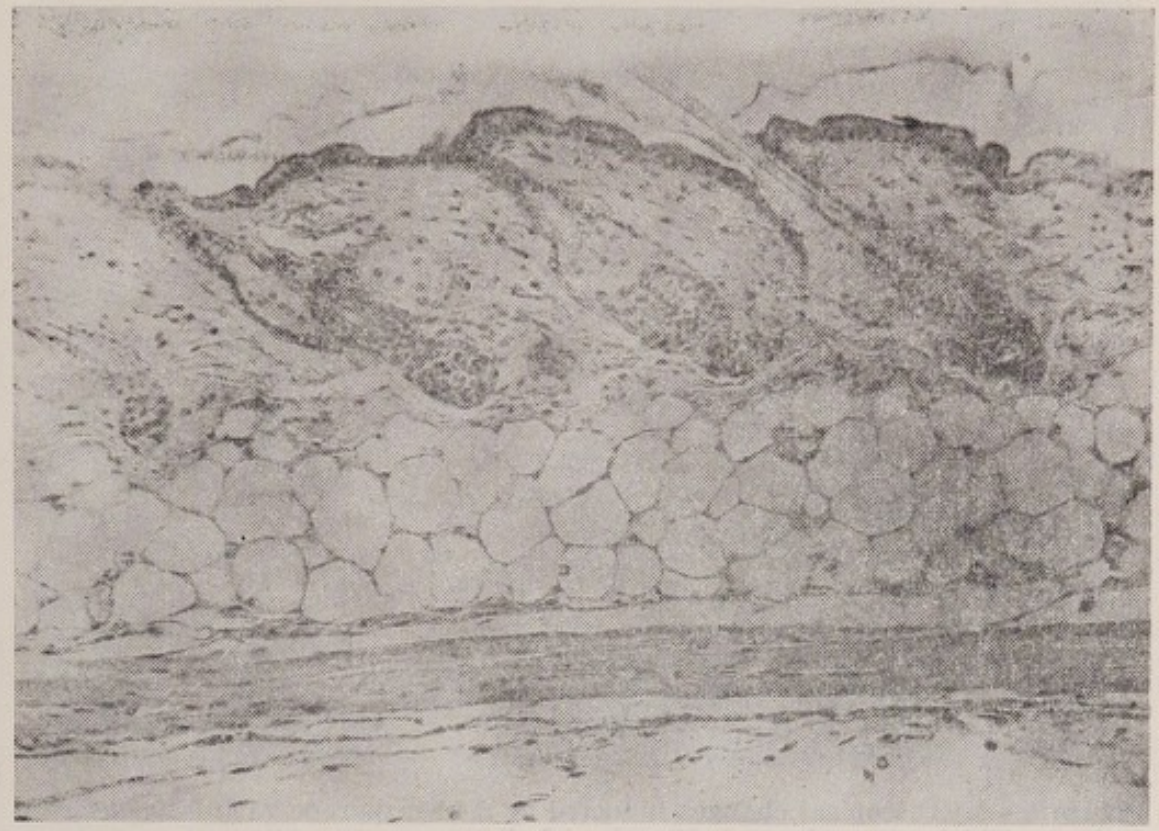


Figure 2.—Microscopic picture of the normal skin. Large sebaceous glands, hair follicles are easily seen. There is no thickening of epidermis. No infiltration in any layers of the skin.

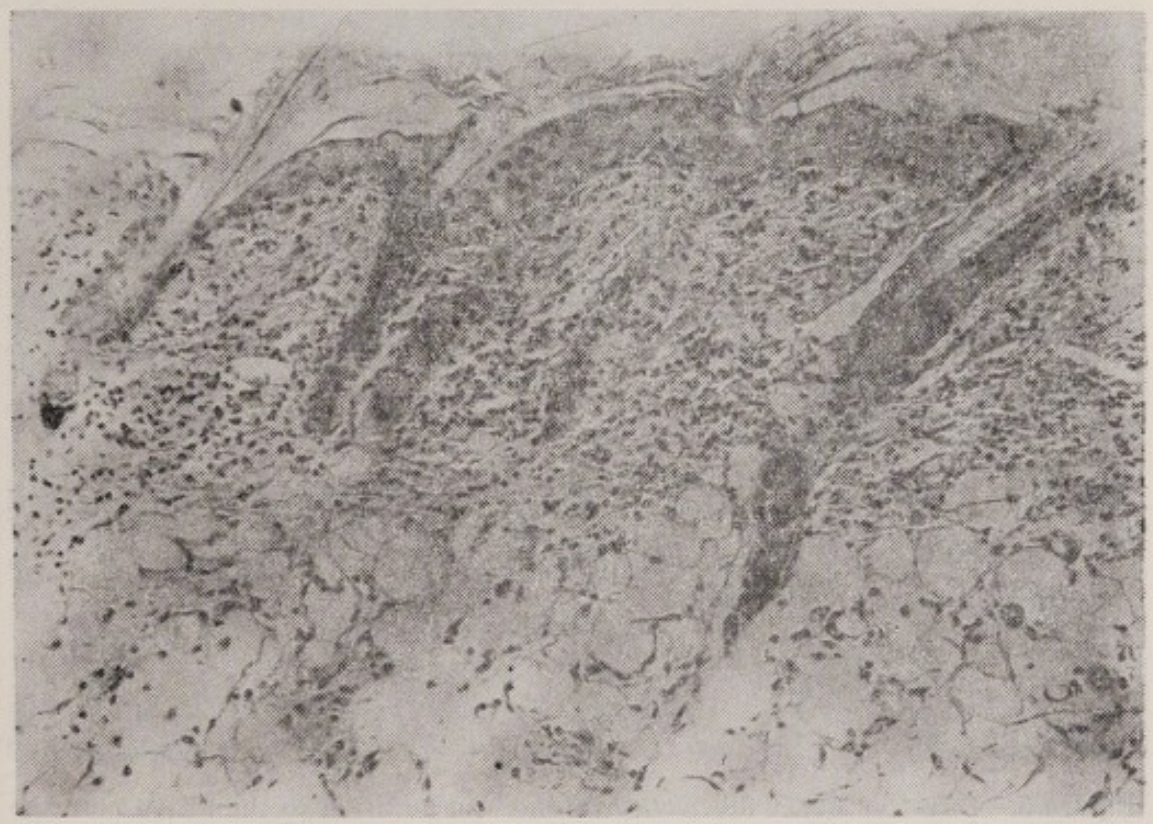


Figure 3.—Microscopical changes taking place following application of 20-methylcolanthrene. Hyperkeratinization, thickening of epidermis, infiltration, and loss of sebaceous glands are characteristic.

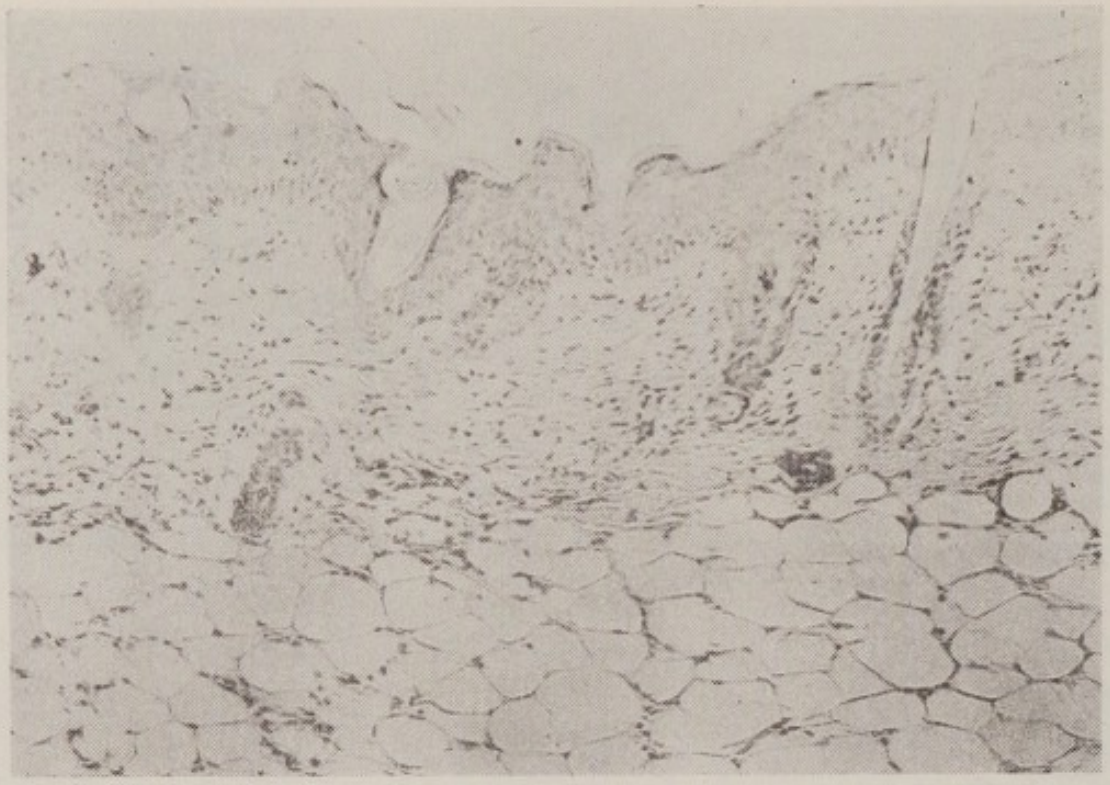


Figure 4.—Microscopical changes inducted by 3.4-benzpyrene (1.10^{-6} concentration). Thickening, edema, infiltration of all layers with obliteration of glands represent principal changes.



Figure 5.—Effect of 1,2,5 6-dibenzanthracene. In addition to the above note the necrotization and formation of keratin pearls.

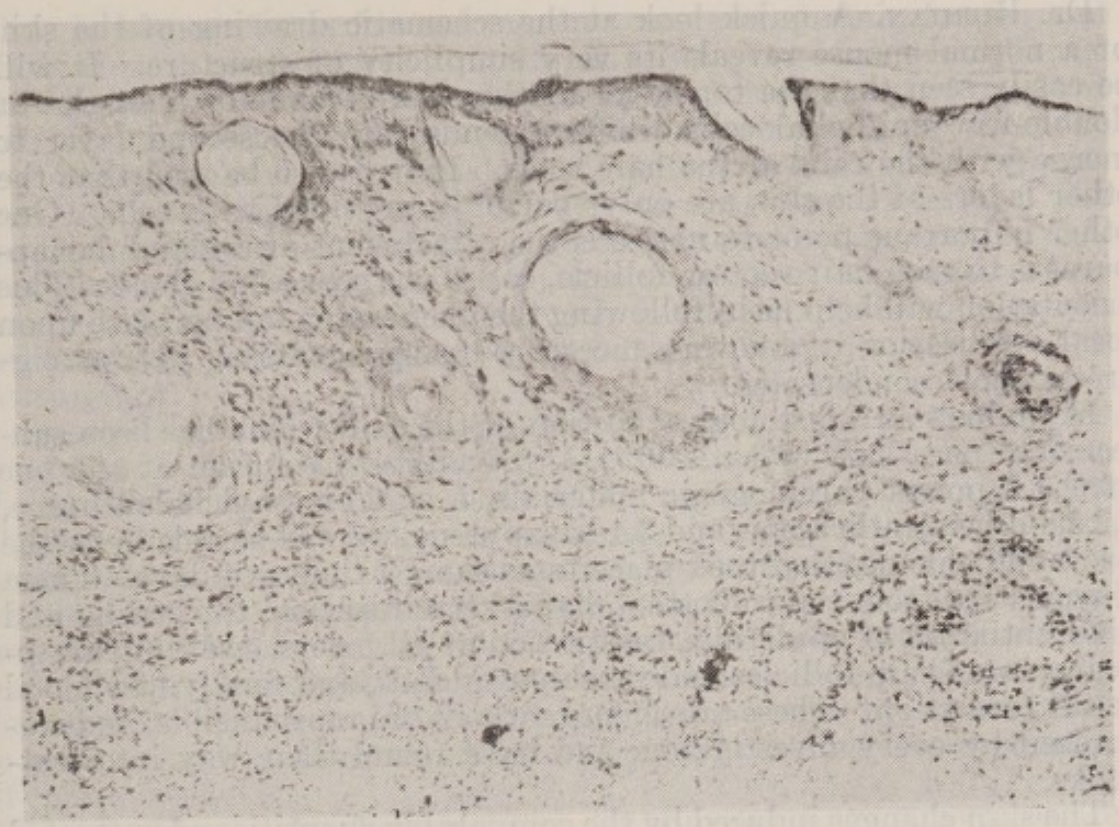


Figure 6.—Tar of untreated cigarette paper. Thickening of the epidermis, massive infiltration, complete loss of sebaceous glands and hair follicles, obliteration of subcutaneous fatty layer. The dose applied corresponds to 1 microgram of 3,4-benzpyrene.

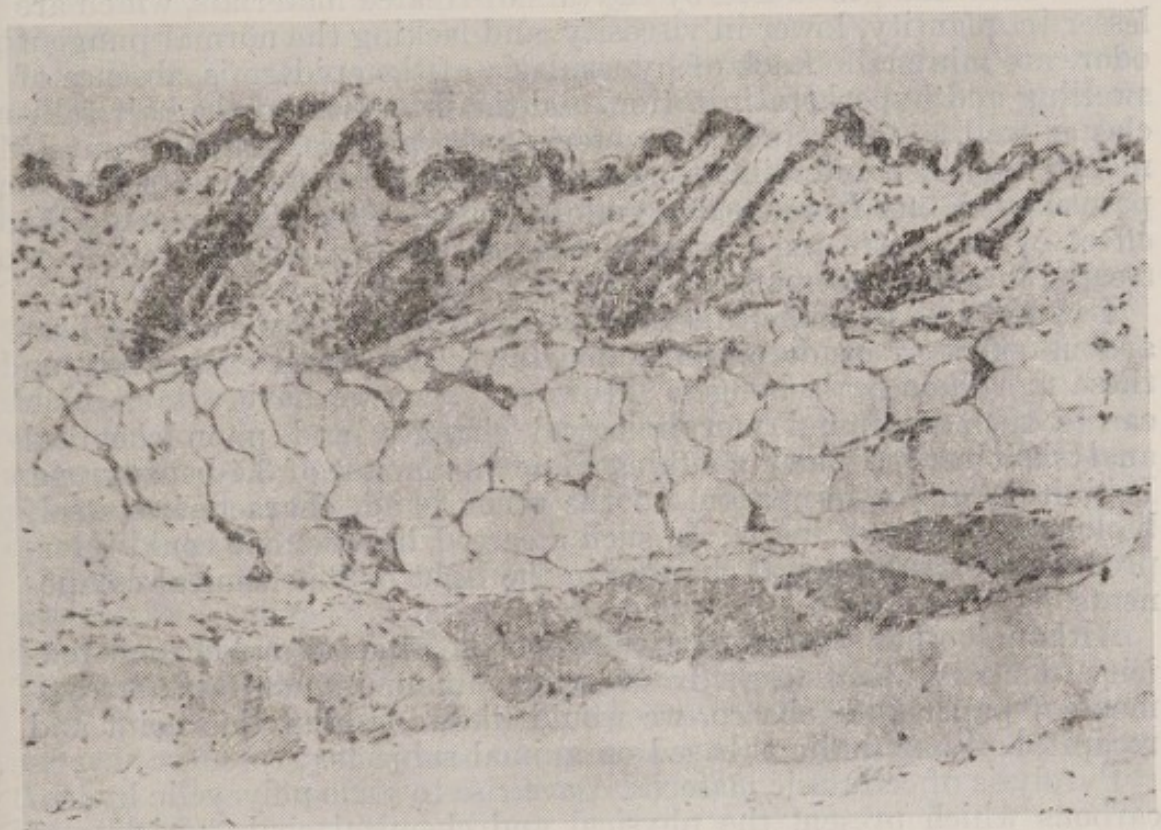


Figure 7.—Tar of treated paper. Hairfollicles, sebaceous glands, and epidermis are intact. There is no visible infiltration of the dermis or subcutaneous fatty layer. This picture is only comparable with figure 2 which represents the normal skin.

Dr. BURHAN. A quick look at the schematic drawing of the skin of a normal mouse reveals its very simplicity of structure. It will be easily seen that the top layer of the skin is made of a single- or double-row epithelial cells which extends into the second layer to merge with the cells of the hair roots. It will also be seen that the other layers of the skin are only sparingly provided with cells. One other important point to notice is the attachment of a glandular apparatus to each hair root or follicle, called the sebaceous gland. This orientation will help us in following the phenomena taking place upon local application of known cancer-producing agents as well as cigarette smoke condensates.

If we look at a histological section of the skin which has been subjected to paintings with $1—10^{-6}$ (1 to 1 million) solution of any one of the 3 potent carcinogenic materials, i. e., 20-methylcholanthrene, 1,2,5,6-dibenzanthracene, and 3,4-benzpyrene, we will be able to see the profound changes that took place in as short a time as 4 days. These changes are from top to bottom, hyperkeratinization, thickening and differentiation of epidermis, infiltration of all layers, swelling, disappearance of hair follicles and sebaceous glands, and finally new blood vessel formation. These are changes which are reproducible, and recognized by every investigator; and have found their way into textbooks.

The skin changes induced by the comparable quantities of untreated tar are even more pronounced than those brought about by chemically pure agents. These are undoubtedly due to some other irritants present in the tar.

The skin changes caused by the tars of treated materials, which are lesser in quantity, lower in viscosity, and lacking the normal pungent odor, are minimal. Lack of hyperplasia of the epidermis, absence of swelling and hyperkeratinization, and the intactness of the hair follicles as well as the glands characterizes the picture. From a purely morphological standpoint, the skin sections of animals subjected to paintings of tars from treated and untreated materials in placing the effect of the untreated tars with the carcinogens, and the effect of treated tars with normals.

I would like to make this point very clear that my claim on hazardous effect of crude or untreated tar is not based exclusively on these morphological changes. However, here we have a tar which causes such profound morphological changes, and upon chemical analysis it proves that it contains a known amount of 3,4-benzpyrene. Inhibition of its formation, on the other hand, characterizes itself biologically, almost inert. In such a case, I believe, one could interpret these morphological changes in the light of its chemical components.

Although other studies, particularly those of acute and chronic toxicity of tars, indicate the advantages of ammonium sulfamate treatment of paper and tobacco, we would like to stop at this point and recapitulate our findings based on animal subjects.

Pyrolysis of cellulosic materials gave rise to some polycyclic hydrocarbons which present the physical and chemical characteristics of 3,4-benzpyrene. The biological reaction to this material is identical with that of other known cancer-producing agents. The irritating power of this tar is obviously more pronounced than the pure chemi-

cals. It is more toxic for animals in acute and chronic forms; and finally more tar is produced from the same weight of initial substance. The addition of ammonium sulfamate in the form of pretreatment of both tobacco and paper reduces the amount of tars produced; these are not toxic. The minimal toxic dose of tar from treated material is at least 10 times larger than for tars from untreated material. The physical and chemical characteristics of a known carcinogen like 3,4-benzpyrene is reduced from more than 90 percent in treated specimens; and there is almost no reaction in the skin, inflammatory or precancerous, whichever term is more attractive to the individual investigator.

As a doctor with 17 years' experience in both applied and experimental medicine, I wish to make this statement. The investigation shows that tobacco smoke contains an agent which has the same physiological activity as known carcinogenic materials, and that this agent can be largely, if not completely, eliminated by an additive which alters the combustion to prevent the formation of 3,4-benzpyrene and similar condensed ring compounds. I believe that the morphological evidence which I have presented here should convincingly demonstrate the desirability of treating cigarettes by some means to reduce the irritation and also the potential carcinogenic properties of cigarette smoke.

Mr. BLATNIK. Are these pictures, Dr. Burhan, hand drawn, or are they actual photographs?

Dr. BURHAN. Yes, sir; they are photomicrographs taken directly from the microscope on Ektachrome films.

Mr. BLATNIK. Mr. Rand, would you proceed with your statement, please?

Mr. RAND. Your committee has been hearing much about nicotine and tar and the need of eliminating them from tobacco smoke. Nicotine and tar are condensed smoke. To get tobacco smoke is the purpose of smoking. Therefore, to remove the nicotine and tar would be the same as removing alcohol from whisky or taking the substance out of milk.

The job of Rand Development Corp. was to determine if there were any harmful chemicals in the smoke and, if so, to determine how they could be removed without changing the quality of the smoke in other respects. As one of our attorneys expressed it, "You want to do the same thing for tobacco smoke that pasteurization accomplished for milk. You want to remove the chemicals that appear to open the door for cancer much as a cut on your foot might open the door for bacteria."

You have heard my associates in Rand Development Corp. tell you how we isolated the harmful chemicals and, when we knew what they were, how we added a neutralizer to prevent their formation. You have seen the results of comparative tests before and after.

We had that solution in 1954. However, the attack on smoking seemed to run everybody into the ground. One gained the impression that we were attacking the tobacco industry instead of seeking to help it. Therefore, although our findings were substantiated here by other research people who went to the trouble to make the same tests, we could get no recognition from the industry. As a result, we ultimately went to England and France to obtain acceptance accompanied by action.

I wish to make it clear to this committee what we and others found as a result of our experiments with mice is not conclusive evidence that we are right. We cannot, as Walter Reed did, use human beings as guinea pigs, even if the industry were to support such a project. Unless Dr. Little and some of the others want to be guinea pigs.

However, if our neutralizer had been put on trial in 1954, the results would have shown us right or wrong in a matter of a year or two by the statistical methods employed by the American Cancer Society. In research, it is just as important to show wrong as it is to show right. The sooner we make the trial the more quickly we will know.

We do not see anything wrong with smoking, and that goes for the tobacco industry, which includes the tobacco farmer. As a matter of fact, it is our belief, although we have made no tests in that direction, that smoking is beneficial.

I might recall to some of the medical people who testified that the Public Health Service, Dr. Hueper, exposed animals to smoke and found that the respiratory-infection rate went down by about 50 percent.

I say that to emphasize that we have not been showing sick mice with the intention of condemning smoking and we do not think that the others have shown them for that purpose. We merely desire to draw your attention to the only apparent cause—the less than 1 percent of harmful cigarette tar as testified by Dr. Wynder, as to which correction action can be taken.

Mr. BLATNIK. Mr. Rand, in a new scientific development such as this, what is the usual procedure to verify, by having other independent researchers in the same field, using the same techniques, using other techniques to check on that—in other words, to verify or to bring to light if there may be any of your own bias in your own research?

Dr. BURHAN. We follow routine procedure in any scientific work. We leave out the bias. You repeat again and again until you reach a point where you have developed a firm opinion and then you expose your results to anyone who is desirous to do them. At that stage we put our cards down and say, "Here are our results and here is the material. If you would like, we will send the material. If you want to see, we will send you the mice and you do it according to your criteria."

Mr. RAND. We actually sent the boys to Paris to put on a demonstration for the French Government and they went right down the thing and they said we were conservative in our findings.

Mr. BLATNIK. They carried out research and came to the same conclusion?

Mr. RAND. Yes.

Mr. BLATNIK. You say, though, your findings were substantiated here by other research people who made the same tests—does that mean you have had others in the United States?

Mr. RAND. There was a chemist sent to see us by one firm. Sir Ernest Kent, who first found 3,4-benzpyrene in coal tar and soot, put his boys to work on it and they confirmed the presence of it.

Mr. BLATNIK. Did you attempt to get the Tobacco Industry Research Committee interested in checking on the validity of your findings?

Mr. RAND. Yes, sir; I suggested they duplicate our work but all they did was to criticize it.

Mr. BLATNIK. To your knowledge, have they made any——

Mr. RAND. Not to my knowledge. These are facts. It is like counting pennies. There are so many pennies there and if you can count them you will always come out with the same number.

Mr. BLATNIK. Did you call this to the attention of Dr. Wynder, of the Sloane Kettering Institute?

Mr. RAND. Oh, yes.

Mr. BLATNIK. What was his reaction?

Mr. RAND. He is doing some work along the same line now.

Mr. BLAKNIK. He is interested in checking?

Mr. RAND. Yes; he is checking the treatment.

Mr. BLATNIK. How did you happen to go to France to look for further verification?

Mr. RAND. Because we couldn't get any interest of the cigarette people at all in this country and I went to most of the big ones. They just didn't want to discuss it and that was that. I went to Mr. Kent, for example, and asked him to smoke one of the cigarettes and try it. He said his doctor had taken him off smoking.

We just couldn't get anywhere and I felt that perhaps a government that controlled cigarettes would certainly be interested in these findings so I went to see the director of research in Paris and he financed the whole works through Government funds and picked out the best chemists and physiologist he could find, the codirector of the Institute of Radium, and they have confirmed our findings and now they are working on making experimental cigarettes. It has a black ash instead of a light ash which isn't desirable apparently in the cigarette industry and they want to be sure it doesn't change the taste.

Mr. BLATNIK. This research work carried on by the French, to what part does the French Government participate?

Mr. RAND. They paid for it all. It says right at the bottom, "Underwritten by the Society"; that is the French term, the Government monopoly.

The second man on the paper is the director of research of the Government monopoly.

Mr. BLATNIK. This brief you give us is the report of the French findings?

Mr. RAND. Yes. In this report they confirm our chemical findings completely. I asked them why they wouldn't do some animal experiments and Dr. Latarjet said, "My goodness, why monkey with it, everybody knows 3,4-benzpyrene is carcinogenic for animals and probably with men." They weren't interested. They just wanted to do away with the 3,4-benzpyrene.

Mr. BLATNIK. They felt it was serious enough to remove it?

Mr. RAND. And members of the National Cancer Institute visited our laboratories and they felt the same way, that this was a good piece of work.

Mr. BLATNIK. We have another publication, a reprint from the British Journal of Cancer, 1956, volume 10, pages 498. (See appendix, exhibit 22, p. 710.)

Mr. RAND. The reason we published it over there is because we couldn't get publication anyplace in this country. As a matter of fact,

our work was confirmed by Cooper and Lindsay in England before it was ever published in this country. I don't know why we couldn't get publication in this country but we were turned down by several of the major publications.

Mr. BLATNIK. Was this called to the attention of the National Cancer Institute and Dr. Heller?

Mr. RAND. Yes; and he was very helpful to us.

Mr. BLATNIK. Has he done any work on this?

Mr. RAND. They are doing some work, I believe, now.

Mr. BLATNIK. They are doing work at the present time?

Mr. RAND. Yes.

Mr. BLATNIK. This is very striking testimony. You identify the carcinogenic substance, you measure it, you get sufficient amounts out of cigarettes to produce cancer in animals. At that point it has been repeatedly stated that it is not conclusive——

Mr. RAND. May I correct your statement. We haven't produced cancer in animals from 10 cigarettes, but this is a short-term quick test. The long-term test—we use approximately the same dosage per day but continue it longer. Carcinoma of the skin can be produced in this way from cigarette smoke. Dr. Sigura at the Sloan-Kettering Institute produced 60 percent cancer from just the paper tar alone.

Mr. BLATNIK. It has been repeatedly stated here by other medical witnesses that tests on animals would not be sufficient, or conclusive of the same results being obtained on human beings. What would be your thinking on that?

Mr. RAND. We have a toxicity test on animals and we don't use humans. If you are worried about arsenic, lead poisoning, radiation or anything, you test on animals. If it kills the animals you don't use it. This is the first time I have ever heard anybody challenge an animal as a toxic test material. There has been so much evidence, anyway, on human experience when faced with polycyclic compounds, of which 3,4-benzpyrene is typical, that I don't think any sincere scientist would question the fact that 3,4-benzpyrene has carcinogenic effects on man as well as animals. As far as the site is concerned, when you inject carcinogenic agents into the blood stream they can cause cancer all over the body. In the lung or any other place. If you inject it under the skin you get it very often. If you put it into the spleen and the bone morrow and so on, you often get leukemia. A carcinogenic material really just opens the door for cancer, where it is supplied.

Mr. BLATNIK. Mr. Rand, may I get your opinion as well as those of the scientists with you. Is there any carcinogenic substance in tobacco smoke in sufficient quantities to be a very likely or possible source of—is it very likely to be cancer inducing?

Mr. RAND. We wouldn't have continued this work as long as we have if we all didn't believe it.

Mr. BLATNIK. Did you do any work in trying to remove it with selective filtration?

Mr. RAND. Just by accident. When we heard we were coming down here we went through our notes on filters and Dr. Cardon has some very definite indication that one cigarette filter has a selective capacity for removing 3,4-benzpyrene. That is a very important lead because if that is true, you should be able to develop a filter that will

remove it. Another lead is that treatment of tobacco influences the amount of 3,4-benzpyrene because one cigar will have 10 times as much 3,4-benzpyrene as another. One big cigar company sent us a number of cigars and finally they had a cigar with no 3,4-benzpyrene. They didn't tell us what they did with it, but they were able to do that.

Dr. BURHAN. The tar from the filter-tip cigarettes causes less skin changes in the mice as compared to those of nonfilter cigarettes.

Mr. MEADER. This is the first time anybody has said filters were any good.

Mr. RAND. If you face the fact that smoke tar contains irritating substances and you cut down the amount of tar, then filter tips are of value and I think Dr. Wynder brought that out very clearly.

Mr. MEADER. Now, I want to understand whether that is what Mr. Burhan is saying. That is why I want to go into this a little bit.

Are you saying that where some of the tar is taken out by a filter and that there is less tar produced at the end of a filter cigarette than the regular cigarette that has this characteristic of less irritation to the mouse skin?

Dr. BURHAN. As was pointed out the amount of tars produced might change from brand to brand depending upon the composition. However, it is our experience, although not so extensive as the other aspect of the whole project, that the filter cuts down the amount of tar in conjunction with the lessening of 3,4-benzpyrene.

Mr. MEADER. Suppose the same amount of tar comes out of a filter cigarette as comes out of a regular cigarette. You claim still there would be less carcinogenic results.

Dr. BURHAN. In some instances. We have made only a preliminary investigation. We do not have extensive experience in the filter cigarettes as in the case of the nonfilters.

Dr. CARDON. In the specific example I had in the paper, here, the filter cigarette, according to Reader's Digest produced more tar than the regular cigarette of the same manufacture. It was Camel and Winston. Camel produces less than Winston, according to Reader's Digest.

According to the two experiments we made, Winston produces less benzpyrene by about 25 percent. Now, whether that is a function of the filter type directly or a function of the tobacco that is in Winston we are not sure. It would take a lot more extensive work on filter types used with the same tobacco before we could say definitely that the filter type is doing the job.

There is indication though that just the fact the two cigarettes give the same tar does not necessarily mean that they will give the same amount of benzpyrene and Dr. Burhan's experiments show they won't have the same biological effect, either. There might be a reduced biological effect even though you have the same amount of tar from two different cigarettes.

Mr. MEADER. In other words, what you are saying now is it isn't the tar but it is the benzpyrene.

Dr. CARDON. Chemically, yes. There is benzpyrene or there isn't. There is evidence that the biological activity, even, is related to the benzyprene content.

Mr. BLATNIK. Your geneticist though, Mr. Rand, as I understand it, is one to—the point is to go thoroughly into all the research work

and conclusively find out what substances in the smoke are injurious and then eliminate them?

Mr. RAND. That is right.

Mr. BLATNIK. Along the lines suggested by Dr. Meader. Have a type of tobacco that gives a lower tar yield, and nicotine yield, and improve the filter to keep those substances below the threshold of being injurious.

Mr. RAND. I think we have just scratched the surface on this investigation. I think that a tremendous amount can be accomplished in the entailing of a mixture of compounds like tobacco smoke.

Mr. MEADER. May I ask about this ammonium sulfamate, or whatever that is. Do you find any other chemical compounds that have the same effect on reduction of the amount of benzpyrene?

Mr. RAND. Yes. They always seem to be related to ammonia releasing compounds. In other words, if you put ammonia in the cigarette, normally as it is found in the tobacco, it just comes off and volatilizes in a few hours. So Dr. Cardon found this stable form of ammonia, which is ammonia sulfamate which at the combustion temperature of the cigarette volatilizes, and releases ammonia which interferes with the formation of these compounds and prevents their formation.

We found a whole series of them that will do that.

Mr. MEADER. Does your cigarette taste a little bit like ammonia, then?

Mr. RAND. No. You see, ammonia is normally found in tobacco and that accounts for the fact that only one-fifth as much benzpyrene comes in tobacco as in the paper.

We looked for something in the tobacco that prevented that cellulose from forming benzpyrene when it burned. We were much amazed to find that just the paper alone, the additional ammonia affected even the formation of the benzpyrene by the tobacco.

I think it reduced it 60 percent by treating the paper alone.

Mr. MEADER. Did you ever smoke one of these cigarettes treated that way?

Mr. RAND. Yes.

Mr. MEADER. What do they taste like?

Mr. RAND. We can't see any difference, but the French Government said the other day they did find a small change in the taste. They didn't indicate whether it was better or worse. I suppose it is worse.

Mr. BLATNIK. Mr. Minshall.

Mr. MINSHALL. I have no questions.

Mr. BLATNIK. We thank you very much.

You people have made a special effort and have given us very well-prepared testimony. We commend you for the effort—even though it is a profit enterprise, it is directed at something that concerns millions of people.

We thank you very much for your cooperation and assistance.

Our last witness for today is Mr. Max Greenhouse, certified public accountant and statistician, from Rochester, N. Y.

STATEMENT OF MAX GREENHOUSE, CERTIFIED PUBLIC ACCOUNTANT AND STATISTICIAN, ROCHESTER, N. Y.

Mr. BLATNIK. Mr. Greenhouse, before you read your statement, would you give a brief summary of your background, and whom you represent?

Mr. GREENHOUSE. That is all in the report, but I can state it separately at the outset, if you prefer.

I am a certified public accountant, and have been such since 1927, in practice in the city of Rochester. I have my own office, and I am here in the private capacity of a citizen representing no one, and paying my own expenses and I am simply appearing, as it were, pro bono publico.

Mr. BLATNIK. I am sorry. I have just been notified that there is a rollcall vote on the floor of the House, at this moment, which requires our personal presence on the floor. I know you have a 7-page statement, Mr. Greenhouse. Could you summarize it or would you prefer to come back tomorrow morning?

Mr. GREENHOUSE. I could leave the whole matter until tomorrow morning.

Mr. BLATNIK. We have scheduled a full morning with the Federal Trade Commission. That is our problem.

Mr. GREENHOUSE. Well, I could read this whole paper in about 5 minutes. It might be faster than my trying to give you an informal summary. I shall attempt to. My purpose here is to call your attention to what I consider a misleading report by the American Cancer Society, which has tended to frighten millions of people. My qualification as an expert witness here in the field of statistical study of unaudited reports, and the American Cancer Society report is such a report.

They have omitted a number of essential points that would make their study untenable scientifically and fail to stand up statistically.

Unfortunately, it was in this form of an unaudited report that it was received by the American Medical Association last month in New York at its annual meeting, and the report shows, as I say at the bottom of page 2 of my report, gives every indication of having been made to order in the preparation of which its authors set out to find something they wanted to find and came up with results.

Mr. MEADER. Mr. Greenhouse, your statement seems to criticize the statistical validity of the study made by the American Cancer Society and that is the substance of your statement as I gather.

Mr. GREENHOUSE. I wanted to call your attention especially to slide 26, this chart, where they indicate an incidence of 52 percent in coronary disease and 27 percent in cancer where the actual percentage should have been three-quarters of 1 percent and 1 percent, respectively, if those figures had been compared to the total in the study instead of to the total shown.

Mr. MEADER. Mr. Chairman, I believe the comments of Mr. Greenhouse's statement on the statistical study by the American Cancer Society would be just as useful to the committee if it was inserted in the record in full, and I rather doubt that I would be concerned with going into the mechanics of the statistical study in too much detail in connection with the purpose of these hearings, other than to have him point out what he believes are defects in the method they have used.

Mr. GREENHOUSE. My object here is twofold. One is to have the committee not rely on this report with regard to your policies and No. 2, to "unfrighten" the American people who have been scared witless by a report that to any experienced observer is misleading.

Mr. MEADER. Your statement has been available to the press and to what extent that has that second advantage you seek will remain to be seen.

Mr. MINSHALL. What prompted your interest in this matter?

Mr. GREENHOUSE. My duties as a citizen to protect his fellow Americans—a hundred million of them, primarily.

(The statement of Mr. Greenhouse is as follows:)

STATEMENT OF MAX GREENHOUSE, CERTIFIED PUBLIC ACCOUNTANT, ROCHESTER, N. Y.

My name is Max Greenhouse, I am a resident of the city of Rochester, N. Y., and my profession is that of a certified public accountant.

I came to Washington to testify before the House Government Operations Subcommittee hearings on the subject of hazards of cigarette smoking and merits of filters; my purpose in asking to be heard was the desire, in the capacity of a private citizen, to petition this Congress, as it were, in a cause of public good and welfare, the welfare of 100 million people comprising cigarette smokers and their families in the Nation. Millions were made anxiety-ridden by the American Cancer Society's statistical report of June 4 last, a report which ties in smoking habits with dire consequences of cancer, heart disease, and other illnesses. The report, in my opinion, is unwarranted statistically and untenable scientifically. Furthermore, the fear it may spread can lead to a sort of cancer and coronary phobia that may cause more suffering than the diseases themselves.

I was a voluntary witness and paid my own expenses to come here; I do not smoke, represent no tobacco-industry client, and do not own tobacco securities.

My qualification as a witness attacking the ACS report is a lifelong experience in the verification of unaudited statistical reports of various types, an experience acquired in my profession of certified public accountant.

The ACS's report, prepared by laymen, is an unaudited statistical report; its findings have not been checked as to accuracy, for example, of the state of health reported by subjects at beginning of study and of the diagnoses listed as cause of the 11,870 deaths reported. Nor is there an explanation given for the report's failure to include consideration of the adequacy of medical treatment as a factor in shortening or prolonging life, nor for failure to consider possible contributory effects of such carcinogenic agents as soot, engine exhaust fumes, asphalt, rubber products, etc., to which people are constantly exposed, despite the fact that these agents outnumber by far any of the possible carcinogens in tobacco products. The last consideration is probably the most serious; it raises the question of the reliability of the entire ACS report, because the smokers in the study outnumber the nonsmokers 146,063 to 32,392, or almost 5 to 1. Any errors and omissions are weighted heavily against the smokers.

Yet it was in its unaudited form that the report was read last month at the annual meeting of the AMA, to be followed, unfortunately, by nationwide publicity and scare headlines. It is in the same unaudited form that the report has been accorded recognition by the Public Health Service and the Surgeon General. Its consideration by the subcommittee with which it has been filed, I feel confident, will be free from that intimidating influence upon intelligence which the report seemingly has exercised elsewhere.

The ACS report gives every indication of having been made "to order"; one, in the preparation of which its authors set out to find something they wanted to find and came up with results. For example, the study upon which the report was based was limited, to begin with, to men between the ages of 50 to 70, a period in life during which resistance to all types of illness is apt to be at its lowest. It is during this age that cancer most often occurs. In this respect I quote from one of the current ACS's handout circulars:

"The chances for developing cancer increases rapidly as people grow older. Below the age of 20 fewer than 10 people in 100,000 develop cancer in a year * * *."

And as to lung cancer and age-relativity incidence, the following quote is taken from an article by Prof. Paul E. Steiner, department of pathology, University of Chicago:

"In 1950, 18,313 deaths were attributed to lung cancer in the United States among 1,452,454 from all causes in a population of nearly 160 million. * * * The risk (of lung cancer) can be expressed in another simple form. Of every 100 babies born in this country, nearly 2 will ultimately develop lung cancer, *mostly between 50 and 80 years of age.*" (My italic; Paul E. Steiner, M. D.; Etiological Problem in Human Lung Cancer, Cancer, vol. 9, 1956, p. 664.)

It is because lung cancer has been frequently mentioned in "spectacular association" with cigarette smoking that one can find considerable solace in what Dr. Steiner says above; Dr. Steiner does not mention cigarette smoking as a hazard in the etiology of lung cancer in the entire article.

The smokers of the Nation have a reason to be grateful to the Public Health Service for not following the ACS's report in its attempt to associate heart disease with cigarette smoking. Here is what the Service says about it, in part, in its statement of July 12, 1957:

"* * * there is no convincing biological or clinical evidence to date to indicate that smoking per se is one of the causative factors in heart disease."

Another audit item about the ACS report calling for corrective comment is the label frequently given it as a "massive statistical study" or a "major report" about 187,783 men "who have been traced for an average of 44 months."

It would be more accurate, in my opinion, to call the report an "enrollment" or a "registration" instead of a "study" of 187,783 men, judging by descriptive of procedure contained in the report itself. Also, "the tracing" of the men is explained to mean that the voluntary workers, once a year, reported on each man enrolled as "alive," "dead," or "don't know." Then, also, apparently once a year, "a copy or abstract of the death certificate was obtained on each death reported."

The entire ACS report, in the final analysis, once the subjects had been enrolled, was concerned only with deaths, a total of 11,870 "bodies," or about 6 percent of the total enrolled. This number was further reduced to 2,665 in order to reflect, in "slide 26" of the report, "Excess deaths among men with a history of regular cigarette smoking." The reduced number, 2,665, is less than 1½ percent of total "enrollment" of 187,783 men; this number of men and small percentage is what the whole "study" finally boils down to, a study the publication of which has frightened quite needlessly and it seems, almost wantonly millions of people.

It is a study, finally, that with properly punched IBM cards might have been completed in a matter of hours; it may take much longer to offset the harm it has done.

Slide 26, incidentally, to which I have just referred, may be called the giveaway piece of the whole ACS report. An experienced observer can spot it a mile away. It gives away what must be obviously, the ugly purpose of the report, the use of what Dr. Crile, famous Cleveland surgeon, in his book, Cancer and Common Sense, calls the weapon of fear. The chart on the slide, as the committee can observe, is in ¼-inch heavy black lines, with a heavily lined "mourner's circle," resembling a tombstone, off to the right side. The figures on the chart, with percentages, are used in such way as to blowup an infinitesimal minimum of fact into an exaggerated maximum of frightening fancy.

The "minimum of fact," as pointed out above, is that only 2,655 is the grand total of excess deaths from all causes, over a 44-month period ,or about 700 per year, equal to an annual mortality ratio of less than one-half of 1 percent of the total number of 187,000 men in the study. Yet the chart on the slide confronts us with "52.1 percent" for coronary disease and "27 percent" for cancer; it does it by using a "provocative" and meaningless comparison of fractional parts of a total to the whole total. It is as if one would report the death of 3 people, 2 of them by cancer, 1 by coronary, by stating 66⅔ percent died from cancer and 33⅓ percent from coronary, respectively, though the size of the figures in themselves are entirely insignificant.

I have made a calculation in the column on left of the chart, using the mortality figures of the report epidemiologically, that is, by comparing them with the total men in the study. You will observe the new percentages thus shown—¾ percent for "coronary artery disease" against "52.1 percent" on the slide, and ½ percent for "lung and other cancer" against "27 percent"—indicating variations of over 5000 percent between the two columns. The percentages in left

column, as indicated, have been arrived at epidemiologically; the basis used on the report, in right column may be described as "inspired hysteriology."

We should be thankful to the Public Health Service for "knocking out" the "52.1 percent coronary line" by its reassurance, quoted above, that there is "no convincing biological or clinical evidence to date" for associating heart disease with cigarette smoking.

And as to lung cancer, we have the statement by Dr. Steiner, internationally recognized cancer authority, in another part of the article previously referred to, that the lung cancer risk in the Nation is about 20,000 to 750 million or 1 to about 4,000, smokers or not.

If it is fitting to close with a prayer, I would entreat the Lord to entrust our Nation's future health to doctors, test tubes, and microscopes, not to statisticians, adding machines, and calculators. I would also pray that in any future study of human habits, whether it be concerned with smoking, eating, or drinking, that human emotions are not isolated from consideration as they have been in this ACS study. For it is generally accepted that human beings, unlike animals, have a psychic life; they do more than just eat, drink, sleep, and smoke cigarettes.

For all I know, it may be advisable for the committee, and I so recommend, that it table further consideration about filters until there is definitely established, by credible evidence, that there are hazards in cigarette smoking. Your committee may be already convinced that as yet no such evidence exists.

May I close by repeating that my entire purpose of appearing before the committee is to help unfrighten the American people, to permit them once more, and for the time being, to smoke in peace themselves, and quit worrying about their loved ones who smoke.

Mr. BLATNIK. Mr. Greenhouse, we thank you.

We will print your statement in full, and if there are more questions we will certainly get in touch with you.

The members are notified to be on the floor immediately. The hearings are adjourned until 10 o'clock tomorrow morning.

(Whereupon, at 4 p. m., the subcommittee adjourned to reconvene at 10 a. m., Friday, July 26, 1957.)

96946—57——18

FALSE AND MISLEADING ADVERTISING

(Filter-Tip Cigarettes)

FRIDAY, JULY 26, 1957

HOUSE OF REPRESENTATIVES,
LEGAL AND MONETARY AFFAIRS SUBCOMMITTEE
OF THE COMMITTEE ON GOVERNMENT OPERATIONS,
Washington, D. C.

The subcommittee met, pursuant to adjournment, at 10:10 a. m., in room 100, George Washington Inn, Hon. John A. Blatnik (chairman) presiding.

Present: Representatives Blatnik, Hardy, Meader, and Minshall.

Also present: Jerome S. Plapinger, subcommittee counsel; Curtis E. Johnson, staff director; and Elizabeth D. Heater, clerk.

Mr. BLATNIK. The Legal and Monetary Affairs Subcommittee will continue its public hearings on the regulation of false and misleading advertising by the Federal Trade Commission with particular reference to the filter cigarettes.

This morning our first witness is Mr. Roy Norr, editor of the Norr Newsletter About Smoking and Health.

Mr. Norr, will you please take the witness chair.

Mr. Norr, will you give your full name, title, and occupation to identity yourself and your connection and interest in this particular field.

Mr. NORR. I have a statement which I presume I can read in about 10 minutes. May I read that? It contains that information.

Mr. BLATNIK. You may proceed.

STATEMENT OF ROY NORR, PUBLISHER AND EDITOR, NORR NEWSLETTER ABOUT SMOKING AND HEALTH

Mr. NORR. My name is Roy Norr. For almost 30 years, as a writer and publicist, I have been a student of the medical aspects of smoking and health, the advertising practices of the cigarette industry, the relation of tobacco abuse to other drug addictions, and the regulatory problems which the sale of tobacco products raised here and abroad. I have visited and met or have been in correspondence with nearly every leading authority on these subjects here, in England, France, and other countries.

Five years ago, due to the circumstance that Reader's Digest picked up and published under the title "Cancer by the Carton," my study on the subject previously featured in the Christian Herald, the stark facts I presented crashed the smokebarrier and brought the problem for the first time to the attention of many millions of people. Cancer by the Carton was not only published in the American edition but in

10 other countries by Reader's Digest. A copy is appended herewith, should this article prove of interest to your committee.

My presentation of the mounting evidence that associated relentless cigarette smoking with atrocious lung cancer brought a flood of requests that I establish a regular service of information on the latest medical developments, so I founded the monthly Norr Newsletter About Smoking and Health, now entering upon its fifth year of publication. I am its editor and publisher.

Mr. BLATNIK. At this point, Mr. Norr, not to interrupt but just to elaborate, may this be called a business enterprise, your Norr Newsletter? (See appendix, exhibit 25, p. 730.) Is this your means of livelihood?

Mr. NORR. It is not. It is owned by my wife and myself who are out of pocket about $60,000 in the enterprise and I propose to continue it in the effort to make it self-paying. It will never be profitable because I continue to lower the publication rates. I started at $25. I reduced the rate to $15, it is now $10 and some day I hope to give it away if I can afford it.

Mr. BLATNIK. What is the circulation, Mr. Norr?

Mr. NORR. I would rather not tell that, sir, for important reasons.

In brief remarks I have to make on the issues before you, I shall confine myself to the most important conclusions that bear upon the problem of cigarette advertising.

The cigarette industry today stands with the albatross of cancer hung firmly around its neck. And cancer is the most anguished of all ways of life—anguish that is mental and physical, progressive and protracted, as Dr. Charles S. Cameron, former medical director of the American Cancer Society puts it. "We believe it to be the greatest curse levied by nature on mankind, in terms of human suffering, sorrow and economic loss," concludes the annual report of the Sloan-Kettering Institute for Cancer Research.

I quote this in stating what has moved me in starting the work and continuing it.

Some 100 constituents have so far been identified in tobacco smoke, and of these, 5 have proved to be cancer-causing substances in animal experiments. This from the latest report of the British Medical Research Council. Take it that confident scientists will be able to remove the deadly carcinogens from tobacco smoke, when or if all are discovered, what will be left of tobacco flavor? Cigarette makers are in the tobacco business, not in the business of cabbage leaves or brown paper for smoking purposes. In the self-pollution that is smoking, there is no substitute for abstinence.

The greatest discovery of the century in cancer research is the incontrovertible relation of chronic smoking to deadly lung cancer. There could be no finer crusade than the tracking down through research of the evil spirit of cancer throug the immense darkness in which it still lies. But let us gaze unflinchingly at every promise of cure. Despite the immense work being done with nitrogen mustards, hormones, contisone, radio isotopes, there is still no cure for cancer—nothing but the surgical removal of malignant growths and/or their destruction by radiation. The light that beckons is prevention—the prevention of preventable cancer.

And we know today beyond any reasonable doubt, except the doubts expressed by the hired researchers of the cigarette industry, that

smoking is a casual factor in cancer of the lung and in other cancers of the respiratory tract, not to mention the high association of smoking with cancer of the liver, gall bladder and certain other sites. Whatever alleged controversy still remains as among independent scientists is as to which of a number of inhalants or pollutants are major or minor contributors to the type of epidermoid cancer now generally known as Smoker's Cancer. The great Pasteur said:

When meditating over a disease, I never think of finding a remedy for it, but a means of preventing it.

My interest in the subject, gentlemen, is the fact that I think there is a means of preventing preventable cancer that has been largely ignored.

Incredible as these revelations seemed at the time, every laboratory determination since my article Cancer by the Carton unlocked the medical findings made before 1952, every report that has been written on the subject, every charge made in Congress fully confirmed the facts I had given (see appendix, exhibit 26, p. 737).

The most significant finding, from the standpoint of the smoking population is that even heavy smokers who give up smoking for health protection—not because of ill-health—reduce their risks substantially.

"The cessation of smoking by chronic smokers," the study group of authorities appointed by the Government health institutes, the American Cancer Society, and the American Heart Association found, "decreases the probability of such individuals developing lung cancer."

The greatest bar to cancer prevention—again, the prevention of preventable cancer—is the false, meretricious and misleading advertising of most of the Big Six of the tobacco industry—six companies that control approximately 98 percent of the business.

The industry has adopted the techniques of the oldest profession in the world. The strumpet cry of cigarette advertising today is: Pleasure for sale! "Big, big pleasure", if you smoke 'em king size. I quote from the advertisements. The myth of "flavor" and "aroma" has been effectively exposed by Dr. Hugh Lennox-Johnston, the noted authority on cigarette addiction. Smokers smoke mainly, he says:

to obtain a satisfying dose of nicotine, just as smokers of opium smoke to obtain a dose of morphine, or hashish smokers smoke to obtain a dose of cannabis indica.

The most deadly gadget which the cigarette makers use is what I have described in my article, The Greate Filter Tip Hoax, the first publication of the facts which I made almost 10 months ago—in the September 1956 issue of the Christian Herald—and which was republished in full in the Congressional Record of January 17, 1957 on the instance of Senator Neuberger, of Oregon. I submit a copy for the record of these hearings. (See appendix, exhibit 27, p. 740.)

Five years ago Pope Pius XII told a European tobacco group holding a congress in Rome that a duty of conscience bound all those involved in the tobacco enterprise to reduce to a minimum the toxic and other harmful qualities of their products. But deliberately, studiedly and with the purpose, I charge, of binding scared addicts to their addiction, the leading American cigarette companies have set about to hold the carrot of "health protection" before the scared smokers of the Nation with filter tip cigarettes, while actually adding to the hazards of smoking by degrading filtration to the least possible effectiveness to the point where plain cigarettes often carry less

nicotine and tars than filter tips—in fact, so patently ineffective that "you don't know the filter's there," to quote one of their slogans. The cigarette hucksters well know that it's harder to wean a drunkard from 100 proof whisky than from 3 percent beer, a dope addict from heroin than from marihuana.

"One reason why Americans are smoking again more or less fearlessly is that they see safety in filters", says Time magazine.

The greatest victims of the campaigns of tobacco-narcotic addiction are the youth of our Nation. I present copies of current advertising where high school boys or younger are pictured as welcoming a new filter-tip brand, where male escorts light up cigarettes for early teen-age girls.

I am in position to state to your committee that, when an investigation of smoking in our schools is completed, the fathers and mothers of the Nation will find the most shocking proof of the success of the cigarette pushers. The sample is yet too small to be "statistically significant," as statisticians say.

But, if the vast number of cigarette addicts in the 10- and 12-year age groups among our school population should appear unbelievable, look now at what investigations among schoolchildren have disclosed abroad, where the economic position makes cigarettes harder to come by for children and adolescents.

The Swedish Government has entered upon an extensive educational campaign since a survey of 5,000 schoolchildren in Stockholm showed an alarming spread of the habit. The Swedish tobacco monopoly has ceased all advertising, foreign companies have followed suit, and substantial sums have been given by the tobacconists to aid educators in their work of protecting youth from the menace of death and disease from cigarette addiction.

In Holland, following the report of a medical committee, the Government has appointed an educational committee to cope with the problem. In England, health officers are finding that some children begin smoking as early as 7 years of age—I am referring to medical reports of their investigation—and that there are many inveterate smokers in the 13 to 14 year age-group. In Australia, the state government has inaugurated a poster campaign proclaiming: "If you smoke, cut down; if you haven't taken it up yet, don't."

The apparent accomplices of the evil cigarette campaigns are certain leaders in the American television networks. I refrain from mentioning names, because they may be unwilling accomplices; the cigarette industry, with its huge advertising disbursements, may have them by the throat. On this issue, the three men who control the people's air are usually found well hidden behind the baggy trousers of their innumerable vice presidents.

The printed press advertising is relatively cold and lifeless puffery; besides, the great newspapers of the Nation have broken their former silence. What the public knows of the menace of intemperate smoking it has learned from the press. Newspaper commentators do not end their columns with a fervent appeal that you smoke their brand of cigarettes, use their underarm deodorants, or take their favorite laxatives.

But look at the television screen. The people's air has been handed over to the cigarette huckster. His weapon is the tainted testimonial. His targets are children and adolescents.

The glamor girls who just love that cigarette, the smirking announcers who blow clouds of "fragrant" smoke into the home, the baseball heroes who now train on "ciggies, not wheaties," are not directing their fire at grandpa and grandma. They're shooting at children and youth. They are even training lisping babies to repeat their singing commercials. And you can't shut off a child as you can your television set.

As I have indicated in various articles, no advertising hoax in the unwholesome puffery of cigarette advertising is so laden with evil promise to the health of the Nation as the present filter-tip fake. Here are some of the claims made for filter-tip advertising on the air during the past 2 years:

High filtration to help you keep your smoking moderate.
Effective filtration.
Real filtration.
Self-filtering action.

Note that these claims followed on previous air commercials such as these:

No other filter tip takes out so much nicotine and tar.
For the greatest health protection in cigarette history.
Filters out so much harmful smoke that it also filters out the worry in every puff.

I would like to interject something here: This is the cleverest campaign that has ever been conceived in the cigarette industry. The idea was, first, to run the series of ads that would make definite claims for the health protection through filtration, then, gradually, say nothing about health protection. In other words, the technique that the Russian, Pavlov, used with his dogs.

He would ring a bell at feeding time and give them meat. Finally, he removed the meat and just rang the bell, and the dogs would rush in, salivating for the expected food.

Mr. BLATNIK. That is called the conditioned response; is that right?

Mr. NORR. Conditioned reflex.

Thus, the smoker was brainwashed for the advertising that followed.

The value which the cigarette industry puts upon its domination of the air could be not better stated than it was by one of the Big Six presidents a few years ago:

Here is a medium—

he said about TV—

that gets inside people as no other form of communication has done before. You hear it, you see it—you almost taste it.

I am quoting from an address he made before a tobacco convention in Chicago.

He added:

The public gives us its faith that whatever words and pictures we send into their living rooms will be beneficial as well as entertaining, dependable as well as amusing. And it is our solemn obligation to keep faith with that trust.

Suffice it to say he is no longer president, or even associated with the tobacco business.

Never before in the history of broadcasting has such revolting hucksterism as the present cigarette advertising been allowed on

the TV screen, and I have a had a good deal of experience in the industry. I have been an adviser to television interests since the formation of the first television network.

But network leaders continue to accept medals, awards, citations for the public service with the greatest of ease. One, who deserves, perhaps, his honors more than most, has received nearly every award except mother of the year.

Yet in huckstering a product that carries disease and death in its wake, cigarette advertisers who bestride the TV screen with the blessing of our radio bosses utter no caution, sound no warning, give no indication of the perils of tobacco abuse in relation to cancer or sudden death from coronary disease.

The liquor industry, at least, has spent millions of dollars in saying, "Don't take one drink too many."

We are a nation of sneezers, snifflers, coughers, spitters—and the greatest consumers of tobacco products in the world. Smoker's cough has become the cough of the Nation. Time does not permit an extensive rundown of other health emergencies which medical research has exposed with regard to tobacco abuse.

How many thousands have been crushed into a pulp due to sudden failure of sight suffered by a hard-smoking driver due to nicotine amblyopia causing a sudden spasm of the blood vessels of the eye? How many mysterious air disasters have occurred because the pilot's vision and depth perception have been affected by smoking? It is only recently that aviation doctors have taken steps to warn pilots of the danger. Medical literature is full of papers on that subject.

Tobacco apologists have cried out that the industry is the victim of a propaganda campaign engineered by medical headline seekers. If so, the headline seekers include the medical advisers who told President Eisenhower, when they noted a marked pulse irregularity when he was president of Columbia University, that he ought to stop smoking. The President, then a cigarette chain smoker, quit on the instant, and many physicians believe that his fortunate recovery from an attack of coronary thrombosis was due to his self-discipline.

Franklin D. Roosevelt's physicians warned him to cut down on cigarettes and he tried bravely to do so before he died from a massive brain hemorrhage. He wrote ailing Harry Hopkins that he found it could be done. President McKinley, seriously wounded by an assassin, might have survived, according to his friend, Luther Burbank, the famous naturalist, if his strength had not been vitiated by heavy smoking.

In my view, the following steps are essential with regard to the control of cigarette advertising, if we are to do our duty to youth of the Nation. I am concerned about addicts created by the cigarette industry. Medical history is full of cases where men have lost a leg or an arm due to Berger's disease, but yet insisted upon smoking.

In my view, the following steps are essential with regard to the control of cigarette advertising, if we are to do our duty to youth of the Nation:

1. Action by Congress to declare tobacco a drug, which it inescapably is.

It disappeared from the United States Pharmacoepia under mysterious circumstances after the adoption of the Pure Food and Drug Act, with a note to the effect that it was no longer used in the treatment of humans, but that it was a very satisfactory bedbug poison.

To the extent that an extract of tobacco leaves is still used in veterinary practice, it is under the discipline of the Pure Food Act, I believe.

Thus, a hog would be protected by law from irresponsibility made tobacco preparations, but a human can be exploited without let or hindrance by the tobacco huckster.

2. A demand by Congress that broadcasters in the public interest outlaw completely testimonial cigarette advertising.

A bought-and-paid-for testimonial is inherently a lying testimonial.

More than 20 years ago the American Association of Advertising Agencies issued a statement of principles declaring:

> We believe that making a practice of paying for testimonials is an unfortunate development in advertising.

About the same time the Association of National Advertisers adopted a resolution expressing disapproval of insincere testimonials, "gratuitous or paid for."

3. A labeling act, as already suggested in testimony before your committee, in which the claims for filter-tip cigarettes with reference to the removal of nicotine and tars from the mainstream smoke could be clearly stated.

If filters should prove some protection to the smoker, such an act might reverse the present trend to see which cigarette maker could produce not the best but the worst possible filter.

4. A congressional mandate to the Federal Communications Commission to wake up and use its inherent powers to determine that advertisers and broadcasters use the people's air in the service of public interest, convenience, and necessity.

The "scientific doubt" with regard to smoking and health, that the cigarette industry can keep alive forever through its hired scientists, poses this question: "Shall poison or people, smoking or smokers, money or men, be given the benefit of their 'doubt'?" There can only be one answer.

Mr. BLATNIK. Thank you, Mr. Norr. Your statement is quite self-explanatory. Even your recommendations, which I was going to ask for, are listed very clearly. Your statement will be given most serious consideration by the committee.

Are there any questions on my right? On my left? There are no further questions. Thank you, Mr. Norr.

Our next witness, which will conclude our hearings on this first series, is the Acting Chairman of the Federal Trade Commission, the Honorable Robert T. Secrest.

Mr. Commissioner, will you please take the chair?

STATEMENT OF ROBERT T. SECREST, ACTING CHAIRMAN, FEDERAL TRADE COMMISSION

Mr. SECREST. Thank you.

Mr. BLATNIK. Mr. Commissioner, as a former highly respected colleague on both sides of the aisle and close personal friend of practically all of us who knew you during your outstanding service in the House, we welcome you back to Congress. It is a pleasure to get your thinking and your recommendations.

I notice that you have a well-prepared statement here. How long have you been with the Commission? As I recall it is 2 or 3 years.

Mr. SECREST. It will be 3 years this coming September.

Mr. BLATNIK. We welcome and appreciate your assistance and the conferences we have had with your staff assistants during these many past weeks.

Mr. HARDY. Mr. Chairman, I would like to add a word of welcome, too, and make an observation that our former colleague seems to be living higher on the hog than he used to when he was up here.

Mr. SECREST. Heavier.

John Gwynne, our chairman, would be here today except for the fact that he is out of town. He served for 14 years as a member of the Judiciary Committee and I believe him to be one of the greatest men I have ever known in my life. I am pinch-hitting for him.

The Commission has not met on this particular statement I am giving but it has been given careful consideration by our staff and by myself. We have worked on it diligently for at least 2 weeks, since we knew we were going to testify.

If permissible, I would like to read it and then I will be glad to answer any questions I can to the best of my ability.

Mr. BLATNIK. Please proceed.

Mr. SECREST. I appreciate this opportunity to discuss with the Committee the Commission's administration of the laws regulating cigarette advertising.

At the outset I would like to point out that, whenever necessary in our consideration of matters in this field, we have consulted with and received the full cooperation of the Department of Justice and the National Health Agencies, including the National Institutes of Health and the Office of the Surgeon General. We have also been in touch with other scientific groups interested in the health aspects of the problem.

The Commission's authority over cigarette advertising is governed by amended section 5 of the FTC Act, which declares unfair methods of competition and unfair or deceptive acts or practices in commerce to be unlawful. Under this act the Commission has authority to issue orders to cease and desist from specific practices it finds to violate section 5. Such orders become final after 60 days unless they are appealed, in which case the order is stayed until 30 days after its affirmation by the highest court to which appeal is taken.

The cigarette industry, as you know, is a substantial one. Its retail sales are about $5 billion a year. It is also 1 of our biggest national advertisers, spending about $85 million annually. This advertising has been characterized by frequent changes in claims and themes. In addition, it has been reported that blends and filters of cigarettes have been changed rapidly. Also, new filter-tip brands have been constantly introduced.

The Commission has proceeded in a substantial number of cases involving health claims for cigarettes. These include 7 formal complaint cases, beginning in 1934 and culminating in 4 final orders, with 1 complaint still being tried. Each of these formal cases presented unusually difficult problems of investigation and trial and required a prolonged effort to obtain a final order.

Typical of the direct and indirect representations concerning health in these formal matters were these:

That the smoking of "X cigarettes" encourages the flow of digestive fluids or increases the alkalinity of the digestive tract, or that it aids digestion in any respect (D. 4795).

That the smoking of X cigarettes relieves fatigue, or that it creates, restores, renews, gives, or releases bodily energy (D. 4795).

That X cigarettes or the smoke therefrom will not harm or irritate the throat, or will provide any defense against throat irritation (D. 4922).

That X cigarettes or the smoke therefrom contain less nicotine, or less tars and resins, than the cigarettes or the smoke therefrom of any of the six other leading selling brands of cigarettes (D. 4922).

That X cigarettes will save * * * the nose, throat or mouth; contain no irritating properties; will not irritate delicate throat tissues (D. 4981).

That X cigarettes are endosed or approved by the medical profession (D. 4981).

That X's method of processing the tobacco—eliminated harsh irritants, and that its cigarettes contain no harsh irritants (D. 4981).

In addition to its formal cases, the Commission settled 17 other cases by accepting stipulations from the cigarette companies to stop objectionable advertising. Through requiring less time than formal cases, the stipulations required protracted effort.

Some of the typical health claims stopped by these stipulations were:

Play safe or be on guard by smoking X cigarettes that said cigarettes give extra protection (stipulation No. 3486).

That X cigarettes may be smoked to the full extent of anyone's desire without irritation or ill effects (stipulation No. 8021).

That X cigarettes are safer for the throat, safer for the lungs, better for health (stipulation No. 8021).

Because of the time required before the false claims involved in these 24 cases could be stopped, the Commission sought to employ in injunctive power in a later case. This was in 1952 when it asked the United States district court under section 13 (a) of its amended act for an injunction to stop, among others, the claim, "Nose, throat, and accessory organs not adversely affected by smoking X cigarettes," pending the issuance of its complaint and its final adjudication. The Commission argued that cigarettes were a drug, under section 15 (c) of its amended act, which applies to food, drugs, devices, and cosmetics only. The district court, however, held that cigarettes were not a drug within the meaning of the statute or within the intent of Congress in enacting the amendment (108 Fed. Supp. 573). This holding was affirmed on appeal by the Second Circuit Court of Appeals (203 Fed. (2d) 956).

The Commission's experience clearly indicated that prompt policing of cigarette advertising presented unusual difficulties—which were all the more serious because of the newly raised serious health question.

The Commission's difficulties included the facts that:

(*a*) It had no facilities to test cigarettes;

(*b*) Its burden of proof almost invariably required unassailable current test data not only on the advertised brand but on all other

brands with which the advertised brand was directly or indirectly compared;

(*c*) There were no uniform procedures for testing the smoke content of cigarettes, and the Commission has no authority to establish such standards. As a result its test data were almost invariably the subject of vigorous, prolonged, and sometimes successful attack in formal cases;

(*d*) The blends of tobaccos in cigarettes or the composition of the filter used, or both, could be and were frequently changed. In addition, new brands were constantly being put on the market. Claims for them changed frequently. All of these factors increased the Commission's difficulty in obtaining adequate test data; and

(*e*) Its burden of proving that claims were false—and you must remember, we must prove it—first by competent evidence of their meaning and second by competent scientific proof in light of that established understanding, was greatly complicated by some or all of the foregoing factors.

To prevent deceptive cigarette advertising wherever possible and to obtain its prompt discontinuance—while scientific research resolved the newly raised question of serious health hazards—prompted the Commission to undertake an industrywide, unprecedented approach to the problem.

On September 14, 1954, the Commission directed its Bureau of Consultation to confer with the producers of cigarettes for the purpose of adopting standards for their advertising. Adherence to these standards would, the Commission believed, prevent deceptive claims. The Bureau of Consultation promptly sent a letter to each cigarette manufacturer and enclosed a suggested set of standards for consideration and comments. This was in 1954. That letter stated in part:

> Recent scientific developments with regard to the effects of cigarette smoking have increased the Commission's interest in advertising claims made for such products and have increased its responsibility under the law to prevent the use of false or misleading claims.
>
> In our opinion, the scientific development referred to above have likewise increased the responsibility of the industry to eliminate voluntarily from its advertising all claims and implications which are questionable in light of present day scientific knowledge.

Full details were given the press and wide publicity followed.

Then ensuing conferences were chairmaned by a representative of the Commission's Bureau of Consultation. Each was attended by representatives of cigarette produces, a representative of the legal staff of the Commission's Bureau of Investigation, and also by a representative of that Bureau's Division of Scientific Opinions.

The industry was advised to bring its advertising into conformance with the law (expressed in the original and subsequent revisions of the suggested standards) while the conferences were in progress. During this period the discontinuance of most of the questionable advertising was obtained through correspondence or personal contact with individual companies.

The conferences culminated in the Commission's adoption, on September 15, 1955, of Cigarette Advertising Guides for the use of its staff in evaluating cigarette advertising. A copy of the guides was sent to each cigarette producer, together with a request that he conform his advertising practices to the guides voluntarity. Facilities of the

Commission's Bureau of Consultation to aid them were offered. The whole procedure was fully publicized.

The guides are consistent with Commission law and decisions. They were drafted after detailed consideration by representatives of all of the Commission's staffs concerned with the problem. They were approved by the Commission's General Counsel and were thoughtfully considered by the Commission prior to their adoption.

Prior to the adoption of the guides, typical cigarette filter claims involved health and extravagant comparisons with competing filters. Typical were these:

Maximum filtration.

Effective filtration.

Superior filtering efficiency.

Much less nicotine—the filter removes one-third of the smoke, leaves you all of the satisfaction.

Filters out what you don't want in

Gain the real assurance you can only get with the greater protection of—'s filter

Just what the doctor ordered.

No other cigarette approaches such a degree of health protection.

And at the same time you'll be enjoying the greatest health protection in cigarette history.

Inhale to your heart's content.

Guarantees cleaner, milder, safer smoking.

These were standard advertising practices by cigarette companies prior to the issuance of these guides.

Mr. BLATNIK. Just at that point, Commissioner, not to interrupt you, did you discuss these individual claims? In short, did the tobacco representatives using these various claims, such as "a maximum filtration," "effective," and so forth on down the line, did they produce any testimony or evidence to substantiate those statements?

Mr. SECREST. They attempted in some cases—and the Commission was guided by what tests they had from outside sources and by the guides. And a reading of the guides—I will be glad to discuss them a little later—will show why these things were taken out.

Mr. BLATNIK. I would like to mention, Mr. Commissioner, what puzzles me, and I say this with all candor. The industry will spend millions of dollars with such statements, but for some reason won't voluntarily come before us—a free, responsible public body in a public forum—tell us what is superior about their filters, or justify, at least in some measure, the claims they are making before the entire American audience here at great expense to themselves in promotion of the filter tips.

Mr. SECREST. I assume they know more about their product than all of the other people put together, because they have their own laboratories and they certainly know what is in their own cigarettes. Some of it may be trade secrets, and undoubtedly is, but we operate within the current of all the knowledge we have, and we try to get all we can.

Our information at the time the guides were being considered and adopted indicated that the smoke of the filter cigarettes then offered to the public did, indeed, contain less tar and nicotine than was present in the smoke of the same company's nonfilter brand. Similar informa-

tion during that time and since has indicated that the filters do perform a filtering action.

I don't think anyone could question that. It would be possible to put in tobacco with twice the nicotine or tar content and the filter would not take out as much, possibly, as if the tobacco were weaker, but filters do filter.

The Commission's staff has reviewed cigarette advertising continuously during and since the adoption of the guides. When claims considered questionable have been detected, they have been considered by representatives of the Bureau of Consultation and by both legal and scientific representatives of the Bureau of Investigation. The Bureau of Consultation has brought all such claims deemed violative of the guides to the attention of the company involved, and their discontinuance has been obtained as soon as possible, excepting, of course, for those matters currently receiving staff attention. And every day they are examining cigarette advertising.

In the absence of its own continuing test data on all industry products, it has been necessary to rely upon reports of tests by private organizations. Because of the different testing methods used and the absence of regularity in the testing of all industry products by the same organizations and methods over prolonged periods of time, it has been difficult, if not impossible, to evaluate accurately the tar and nicotine content of each brand of cigarettes offered the public. These factors also made it difficult to keep abreast of changes in either the blend of tobaccos used in, or the composition and efficiency of filters used on, the same brand of cigarettes.

Notwithstanding these difficulties, the adoption and administration of the guides resulted in a marked improvement in the advertising of all cigarettes, including that of filter-tip cigarettes.

Prior to the issuance of the guides, cigarette advertising generally involved health claims. Since their issuance, the theme of all such advertising, including that for filter tips, has centered around taste and flavor.

Prior to and after the guides were adopted, the Bureau of Consultation obtained the voluntary discontinuance of over 75 objectionable claims for industry products. In the majority of those instances, the claims were discontinued within a brief time after their first appearance.

Additionally, the Bureau of Consultation, as directed by the Commission, has in its administration of the guides consulted with advertising agencies or officials of the industry member they represented on a voluntary basis whenever they were requested to do so concerning either specific claims or entire advertising programs prior to their use. And many agencies and people who propose to advertise will submit their copy to the Commission and, where that is done, it certainly gives the Commission then an opportunity to do a more effective job than if the advertising is put before the American people and we have to clip it out of the newspaper and go after it later, or get it from a television commercial.

In that way the use of a great many other objectionable claims was prevented in the first instance. The high degree of public interest in all advertising of cigarettes within the Commission's jurisdiction has thus been served to the fullest extent by its adoption and administration of the guides.

Although of lesser importance, under the circumstances, we believe your committee's interest in efficiency and economy in Government operations warrants our commenting that these affirmative accomplishments were brought about at a fraction of the cost the use of the Commission's regular procedures would have required.

On the basis of past experience, if we had issued formal complaints in each of these 75 instances where we have been able to get conformance with our guides slowly, these claims could have been used perhaps for years before a Supreme Court or a final court would have ruled on whether we were right or not.

Mr. BLATNIK. Just one question, Commissioner. You said you have obtained the voluntary discontinuance of over 75 objectionable claims. Was in necessary to institute any formal proceedings during this same period and, if so, in how many cases?

Mr. SECREST. It was not. I think there are some matters now pending but in every case other than those that are now pending, we have secured voluntary compliance. If we had not, I can't say what the Commission would have done until the complaint came before the full Commission and we discussed whether or not we could prove the allegations in the complaint.

Additionally, the prevention of some and discontinuance of other claims by this means overcame the time factor which otherwise would have been involved.

You see no doctors standing up lecturing the people. Since these guides, you see no claims that they are good for your throat or that they won't irritate your throat. Some may say that the impression in instances may be given, but even there we are trying to stop any impression that might be given indirectly that the health of a person is affected, by cigarette advertising.

Mr. BLATNIK. Commissioner, may I ask you a question there? While you may have, and most likely have, succeeded in stopping any further advertising which may give an impression relating to health, is there any carryover, or residual mental attitude or mind set in the public after years of exposure to the other type of health claims?

Mr. SECREST. I can discuss that a little later.

Mr. BLATNIK. The one word, "filter," may give them a big connotation built up in the past.

Mr. SECREST. But the Commission must prove that and the only way to prove that is to have a survey of consumers to see what connotation they get. We can't go in and say we believe the people think such and such with regard to filters. We have to prove it if we have a formal case, and I will discuss that just a little later in the statement.

The Commission believes its industry wide approach to cigarette advertising and its adoption of advertising guides have served to eliminate completely all health implications from cigarette advertising, thus achieving a marked and prompt improvement in the advertising of cigarettes.

During the interim when scientists were conducting research into the more serious health aspects of cigarette smoking, the Commission believed that to achieve cessation of all health claims for all cigarettes, including filters, was highly desirable. It is of the firm conviction that its adoption and administration of the guides did more to prevent deceptive advertising of cigarettes and to fulfill the Commission's

responsibility to the public than it could possibly have accomplished by any other means.

The headline of the Washington Daily News, September 22, 1955, issue, read: "FTC Tunes Out Health Claims in Cigarette Ads."

The annual report of the National Better Business Bureau for 1955 made this significant comment:

MANY IMPROVEMENTS EFFECTED

On the contrary, there have been many fields of national advertising which have been free of serious criticism during 1955 and others where substantial improvement has been effected.

For example, the cigarette industry has long been a whipping boy for critics of advertising. Granting that much cigarette consumer complaint, that is not true today.

In September, the Federal Trade Commission made public a set of "guides" for evaluating cigarette advertising. * * *

The Reader's Digest, in its August 1957 issue, says that after adoption of the guides "the industry fell in line." The same article, however, warns of a possible "rash" of new claims soon.

An article in the Sunday, July 21, 1957, New York Times (see appendix, exhibit No. 11, p. 579) commented on industry advertising as follows:

The advertising has shifted with the public winds over the years. Previously the emphasis was on health, with such slogans as "Guard against throat scratch" and "safe for your T-zone." Those catch phrases led to conflict with the Federal Trade Commission, which 2 years ago laid down a guide to its staff in judging cigarette advertising. The guide included cautions against claims of medical approval, references to the effect of smoking on nerves, noses, and other parts of the human body and claims on nicotine and tar content.

This guide followed the first impact of the cancer controversy. The manufacturers shifted tactics. Now the emphasis is on pleasure and taste. * * *

We have been advised that since the guides were issued there have been continuous changes in the blends of tobacco used in particular brands of cigarettes and the filters attached to them. Also, it is a fact that there have been frequent entries into the market of entirely new and different brands concerning which little or no reliable tests or other data have been available.

The Commission's staff has noted in recent months a recurrence of some objectionable copy and in some instances the introduction of new and objectionable advertising themes. Upon detection, such advertising has been challenged and in most instances actual or assured discontinuance has been obtained. The remaining instances of questioned advertisements are continuing to receive expenditious attention.

During this period the Commission has kept in touch with scientific development and reports concerning the effects of cigarette smoking. Particularly did it note the Surgeon General's July 12, 1957, announcement that:

In the light of these studies, it is clear that there is an increasing and consistent body of evidence that excessive cigarette smoking is one of the causative factors in lung cancer—

and

While there are naturally differences of opinion in interpreting the data on lung cancer and cigarette smoking, the Public Health Service feels the weight of the evidence is increasingly pointing in one direction: that excessive smoking is one of the causative factors in lung cancer.

That announcement by the public health officer, upon whom the Commission relied for a medical determination in this matter, calls for a fresh approach by the Commission in its regulation of cigarette advertising in the public interest. To this end, the Commission already has directed and given high priority to a consumer survey that would reveal the public's current understanding of the meaning of the terminology used for filter-tip cigarettes.

The results of that survey will enable the Commission to take the necessary steps to protect the public from deception in the labeling or the advertising of cigarettes to the fullest extent of the laws the Commission enforces.

The Commission also is giving serious consideration to additional steps it may take or suggest in light of this recent information from the Government's chief public health officer. The Commission also is alert to the test data published by a private publication in late May which indicated that the tar and nicotine content of the smoke of many filter-tip cigarettes is approaching the tar and nicotine content of the smoke of the company's same length non-filter-tip brand and in some instances is higher than the tar and nicotine content of the smoke of the company's regular popular non-filter-tip brands.

In speeding consideration of these serious problems, the Commission will consult, as it has in the past, with all Federal agencies concerned and with the staff of this committee. It will cooperate to the fullest extend in every effort to protect the public from all questionable advertising and labeling of industry products in the light of present and developing facts and scientific conclusions.

The Commission and its staff welcome the efforts of your committee to assist it and the other Federal authorities concerned in arriving at the proper solutions to this serious and mutual problem. We appreciate this opportunity to apprise the committee and, through it, the Congress and the American people of our concern and continuing efforts to secure truthful advertising of cigarettes, and I want to point out that under the law that is our only obligation, our only legal responsibility: to prevent misleading and false advertising. If it isn't misleading or false or if we can't prove it is, that is the end of our enforcement.

Mr. HARDY. Mr. Chairman, if I might be permitted, I would like to compliment Mr. Secrest upon a very clear and understandable statement and on the obvious good work which has been done in the past.

Since he referred to this accomplishment from an economy standpoint of working out these guidelines and assuring compliance, I would congratulate the Commission on having done that but might ask why they haven't done it sooner and whether or not they are applying that same practice to other items.

Mr. SECREST. The Commission acted, I think, as soon as there was any deceptive or false or misleading advertising. These formal cases brought against the big cigarette companies started in 1934 against claims that were made, compared with other industries—for instance, "smoke a cigarette instead of eating candy."

Mr. HARDY. We remember that, when you and I were a little younger.

Mr. SECREST. Yes, sir. The Commission discouraged that because it was disparaging on the part of another product.

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Mr. HARDY. The candy manufacturers led in that, too.

Mr. SECREST. If they say cigarettes will not let you get fat, also that is not always true.

Mr. HARDY. I do think, Mr. Chairman, Mr. Secrest has made a very fine statement and obviously the Commission has given a great deal of thought and attention to this matter.

It would seem to me that this voluntary compliance he has emphasized should work out very well and I am glad to observe that, in general at least, the cigarette companies have apparently been cooperating with the Commission in trying to clear this matter up.

Would you say that is generally true?

Mr. SECREST. I would say there are probably right now pending discussion over 4 or 5 claims of cigarette companies that have not been resolved but in at least more than 75 instances in the past year or two where we have called their attention to advertising, they have voluntarily given it up.

Now "voluntary" means they may want to clean it up themselves, or it may mean that the Commission, if they didn't clean it up, would issue a formal complaint. No one knows. I am only 1 Commissioner and there are 5 of us who would have to decide on proceeding formally.

Mr. HARDY. You are keeping current as new advertising techniques show up, I gather?

Mr. SECREST. We have a bureau in the Commission that constantly checks advertising. They go through—we get 50 magazines every month, we get about 200 newspapers every month, not only for cigarette advertising but every other kind of advertising.

The 2 major networks send to the Commission every 2 weeks out of every month, every line of advertising they use so that we can check radio and television—television.

Every radio station in the United States sends to us 4 days of its complete radio advertising each year and they don't know what they are going to send. It is spot-checked. We say "Send in the advertising that you used yesterday or that you have lined up for tomorrow." There can be no shifting around of the advertising. We get that.

In addition the Commission has a monitoring system now that was instigated, with money given to us by Congress, in which we have lawyers in every 1 of our 9 branch offices who take certain parts of their time to monitor television advertising. We do that on the theory of "what you say may be truthful but what you show the people could be a lie." And I can give you an example of it. The announcer might say, "You can buy this basket of fruit for $1 down at my store," and then he can show a basket of fruit there that would hold 2 bushels of apples. When you go to a store, there are 10 apples in a little basket. By magnifying what you have can be a lie in a picture. That is why we have the monitoring system.

There was great interest in Congress—Senator Magnuson and others were greatly interested in it and gave us money to do it and told us to do it and we are doing it. We have issued some six complaints already involving television advertising.

Mr. HARDY. I am delighted to know that the Commission is pursuing that practice of monitoring all of this advertising. I am not just thinking now of cigarettes.

I appreciate, Mr. Chairman, the very fine presentation that our former colleague has made to us.

Mr. SECREST. Thank you very much.

Mr. BLATNIK. Thank you, Commissioner, for what I consider to be a very solid, thoughtful, and, more important, a very frank and straightforward and positive statement.

I have just a few questions, Commissioner. First, on the testing you indicated that you were handicapped by lack of testing facilities. Is there any other Government agency which would be capable or adequate enough to do testing for you? Or would you require your own testing facilities?

Mr. SECREST. We have authority to ask any Government agency to test for us. We can ask them. The Bureau of Standards has made tests—none that I know of in connection with cigarettes, but we do have available practically all of the private tests made. We have those the Reader's Digest has published and many other magazines have published them. I have one here showing a very complete listing with respect to the total advertising from Printer's Ink. If we get this compliance by calling them and saying, "You violate our guides and these guides are predicated on what we think the law requires," and we say to them, "You have violated our guides," and they say, "We have not," assume the Commission went on and issued a formal complaint. That has to be tried. We had 1 case where I think there were 12 separate tests of the cigarette put into that case. We put a test in, they would come back with a test showing that ours was wrong. We would come back showing theirs was wrong and back and forth and when it finally got to the court of appeals, our own attorneys said, "That case is so confused with conflicting laboratory tests, scientific tests, that we never could win it," and the Commission on its own motion dismissed it.

But we did get a voluntary compliance with most of the things that were charged in the complaint. So the lack of standards of testing is the first thing that you face and then the second thing, of course, we can get testing done but that takes funds and the Commission's money for testing of all the things that we must test is limited.

We requested an additional amount this year, which we did not get and we asked for one more doctor this year that we thought we needed to go into these kind of claims and we did not get that doctor. Possibly next year the problem will be such that we will get additional funds but we use what we have in the testing of these cases.

Even if you tested and found out that cigarette advertising was completely wrong, you still have the formal case to go through that at best would take a good many years, if it is fought all the way through.

Mr. BLATNIK. Commissioner, do you have access to other facilities, or assistance from other governmental facilities?

Mr. SECREST. We do in any kind of testing and we also have private testing done. We are having a test made at the University of Virginia through arrangement with their facilities there. They seem to be in a position to make a very—and our tests must be accurate and complete, we don't want any other kind, if they exonerate the respondent, we want that just as much as if we convict the respondent. We want the truth, that is all.

Through private laboratories with any group we can arrange for testing of any of our products that involve scientific claims.

Mr. BLATNIK. You feel getting voluntary compliance or instituting formal procedures for enforcement aimed at a cease-and-desist order, the advertising can still continue?

Mr. SECREST. Yes, sir. The advertising can continue until that case is completely settled.

Mr. BLATNIK. Give us some idea of how long the average case would take.

Mr. SECREST. It would be impossible even to get a consent order in less than 150 days, if the respondents used all their time. We file a complaint, they are given 60 days to answer. If they have an answer in 10 days the case is hurried. If they want to wait the full 60, they can. At the end of 60 days they can ask for a 30-day extension. If we deny that 30-day extension and that case ultimately goes to a court of appeals, they may say, "You denied them due process and didn't give them time to make their brief."

Our rules provide for so many days in every step of the way and then in addition, we grant other days if it is such a case that it might prejudice due process or might call for a claim of lack of due process.

Mr. BLATNIK. How long would it be before you got through with the court action?

Mr. SECREST. It would depend on the amount of evidence they put into the case.

In that one case of cigarettes where consumer evidence was put in, I think from both sides, I think probably 12 different tests, in a case like that if a company comes before a hearing examiner—and all our cases are held before him, not the Commission—if it goes before the hearing examiner and the defendant says, "I will need 6 months to adequately defend myself," the hearing examiner rules on that question.

Mr. BLATNIK. How many cases are now pending or in process or active?

Mr. SECREST. There is only one cigarette case.

Mr. BLATNIK. How long has that been underway?

Mr. SECREST. Since 1953; 4 years.

Mr. BLATNIK. Is this an unusual thing?

Mr. SECREST. Yes; 4 years is unusual for our average case, of course.

Mr. BLATNIK. What would be an average length of time for an average case?

Mr. SECREST. That would be hard to say, because the average doesn't give the picture. We have 5 or 6 cases that took years. When you average, you have to include those. For instance, a case that I don't think is finally settled yet, Carter's Little Liver Pills, has been going 11 years that I know of. It has been to the Supreme Court and back, and through the mill. When it will finally be settled, I don't know.

We had two cases involving brushes where they were putting in some, oh, maybe other fibers in the hog hair and such; those lasted for years.

These cigarette cases started in 1934. Now you get other things that intervene. During the war, the Commission practically suspended operation in many fields, especially if they involved candy and things needed overseas, or that they thought were needed, and then the Commission's personnel, itself, many of them were in the service.

We have a board down there, with 100 or 200 names on it, of lawyers who left the Commission—there were delays there; a delay maybe for 3 or 4 years, where no action was taken at all just because of the war's intervention.

I would say that 90 percent of our cases, or more than that, would be handled in 150 to 200 days, including everything, consent orders and all.

Mr. BLATNIK. No matter what the length of time is, the particular firm can keep on advertising and using the exact advertisement which you challenge and dispute until the final orders?

Mr. SECREST. If it is cigarettes or any other product under the Federal Trade Commission Act, we cannot stop the sale of that until the order is finally sustained in the final court of review. Now, if it involves foods, drugs, cosmetics, or devices under the Wheeler-Lea amendment—and I was in Congress when it was passed, and was interested in it—under that act we can go in and ask for an injunction and stop the alleged false advertising of it until the case is tried.

Mr. BLATNIK. Would you recommend that tobacco also be included?

Mr. SECREST. I can say that the Commission already tried to get an injunction, and they certainly believed that the injunction was warranted, to stop that particular cigarette advertising. If we could get an injunction, it is obvious that——

Mr. BLATNIK. You tried that in court; you requested the court?

Mr. SECREST. We took it up to the court of appeals, and the court of appeals said it was not a drug, so we didn't have the authority to get an injunction.

Mr. BLATNIK. We could amend the law.

Mr. SECREST. The law could be amended by adding, I think, perhaps, the word "tobacco" under that section that deals with drugs, cosmetics, or devices, or could be amended by an act such as we have in wool, fur, and flammable fabrics, where the Congress allowed injunction to stop advertising in the sale of the goods until the case was settled.

We can't automatically issue an injunction, ourselves; we must go to court and prove grounds for that injunction, which, I think, is proper. There should be some authority above the Commission to look over its injunctive power. If we could issue an injunction against any false or misleading advertising where we thought the situation warranted it and the court would grant the injunction, the advertising of that commodity—and I will restrict it to advertising—the advertising of that commodity would stop until the case is decided. As it is now, it is continued until it is decided.

Mr. BLATNIK. Does the Pure Food and Drug Agency have any jurisdiction over any part of the cigarettes?

Mr. SECREST. Pure Food and Drug has jurisdiction over the labeling of many things.

Mr. BLATNIK. Including cigarettes?

Mr. SECREST. No; they don't.

Mr. PLAPINGER. That was the important fact in your injunction case; that cigarettes were not drugs.

Mr. SECREST. The court said it was not a drug and we could have no injunction.

Mr. MEADER. You say it was appealed to the circuit court of appeals. Is that now on the way to the Supreme Court?

Mr. SECREST. It was not appealed.

Mr. BLATNIK. I know there are Federal regulations on such things as salt, for a salt-free-diet compound. A product may carry a label saying, "This contains not more than 0.08"—whatever it may be—"percent salt."

In the case of beer, it says, "Not more than 3.2 alcoholic content."

We have those things that are not too much concerned with the health of the public, and yet here is something that has nicotine and tar content and we have no label on it.

Mr. SECREST. I don't think anyone would deny that Congress would have the right to require labeling.

Mr. BLATNIK. We could standardize tests and merely state, without passing judgment on whether they are harmful or not—if we could just pass a law and say, "These king-size cigarettes contain 30 milligrams of tar and 3—."

Mr. SECREST. You would have to leave some tolerance, because I don't believe any testing could ever be devised that would come right to the milligram, and there would have to be some tolerance.

Mr. BLATNIK. I agree with that.

Mr. SECREST. They would say, "Not more than so much tar"; "not more than so much nicotine," and let the company, itself, figure the tolerance.

Mr. BLATNIK. Of the regular cigarettes on the market today, I know there are, perhaps, 15 milligrams of tar. That is a rough illustration. If I buy a king-size filter, there are approximately 22 milligrams of tar, and so forth. If I buy a regular filter, it is so much, and so forth. I would be able to see it on the package, and I could depend on it, rather than these statements as to "supereffective" and so forth.

Mr. SECREST. The reason I am not commenting on that, and am not even personally in a position to make a recommendation, is the fact that if you limited it to tar content, that would be predicated on medical and scientific opinion.

In the first place, does the tar cause the cancer? The Commission can't say. We are not in a position to say what happens. That is a medical question. And if the Public Health Service or the medical people eventually would reach a conclusion that there was a necessity that so much tar was dangerous and below that was not dangerous, that is purely medical, and we couldn't comment on it.

We could certainly comment on legislation introduced, to see that it was technically correct and that it would be enforcible by the Commission, and that is standard practice in Congress.

If the committee receives any bill introduced, and intends to give it any consideration, that affects the Federal Trade Commission, they always send the bill to us for comment, and we send our comments back as to the drafting of the bill and any other suggestions we have to make.

But the question as to whether or not labeling should be placed on cigarettes is one that is based on the medical requirements for it, and we are not in a position to discuss that—that particular thing. Now, the injunctive power, we certainly can.

Mr. BLATNIK. What I am getting at—we hope later on to get a pretty good overall picture. For example, you buy a cold remedy,

12 tablets in a box. It says "Take 1 every 4 hours, but do not exceed 8 in any 24-hour period," or something like that. Well you stop, automatically.

Why don't you say, "Take all you want of these, but do not exceed 20 in any 24-hour period" or 30, or 15, or something like that?

Why do we make it a point of a Federal agency enforcing a description on one article and not on the other.

Mr. SECREST. I think the law requires notice if it contains poison or something that can hurt you.

The scientific testimony would have to go further probably than it has before you could—well you could require, I guess, anything which is based on that.

Mr. BLATNIK. I prefer this voluntary, positive method.

Mr. SECREST. We have been able to get results quick. In some cases, 3 days after we ask the cigarette company to quit using an expression, within 3 days' time they have stopped that advertising.

Mr. BLATNIK. Could you give us an example without identifying the firms? We don't want that, it wouldn't be fair, but give us an example.

Mr. SECREST. Above all I don't want to mention one firm or one brand of cigarettes because without mentioning all, it might imply this one was worse than the others.

Mr. BLATNIK. Give us an example of the advertising voluntarily deleted.

Mr. SECREST. It might be general to a good many claims. "Effective filter." We don't believe that the word "effective" filter is proper.

"Superior filter, best filter, less nicotine, just what the doctor ordered"—that was stopped January 11, 1956—"protect you from tar and nicotine, lowest on nicotine and tar, full old-fashioned flavor of X cigarette may have caused you to overlook the unique efficacy of the highly selective filter—it screens the smoke effectively but let's the honest tobacco flavor flow through."

We have no objection to the reference to flavor but when you say a filter is effective, there has to be some kind of evidence in the form of tests to indicate that it is. Now every filter filters. We can't deny that.

Mr. MEADER. Mr. Chairman, would you yield to me?

Mr. BLATNIK. Mr. Meader.

Mr. MEADER. Commissioner Secrest, I assume what you are reading from is the list of 75 objectionable claims which you referred to on page 10 of your statement?

Mr. SECREST. That is true.

Mr. MEADER. Has that list been furnished to the staff?

Mr. SECREST. It has not. The words "finer filter"—the many claims that we have asked them to withdraw—advertising phrases we have asked them to withdraw, and in over 75 cases they have volunteered to do it.

Mr. MEADER. Is there any reason why that list could not be furnished and made a part of the record?

Mr. SECREST. Here is why we didn't furnish it along with the statement: This is an entirely voluntary operation. It is a highly competitive industry. If we call manufacturer A in and say "That claim we think is false and misleading," manufacturer A may say, "Well I will agree to take it out but don't tell my competitor that you

called me in." It gives him a competitive advantage. His competitor can say, "Well the Federal Trade Commission had him in and whacked him over the shoulder."

Mr. MEADER. I have no desire to embarrass the Commission or any company.

Mr. SECREST. If the members of the committee care for a copy of this, you are welcome to have it.

Mr. BLATNIK. I have gone over the list with him. We are not interested in that element. It is a voluntary, confidential matter.

Mr. SECREST. I don't think it is necessary to the question, because we have pointed out in all cases what the examples were. It would just give you the name of the brand or company. That would hurt us definitely, I think, in getting them to come in tomorrow or next week.

A lot of that advertising was never used. They come in, in advance, and say, "We plan to use this," and say, "Look, you better not."

We just felt to make it public we would certainly hurt what we are trying to do voluntarily because they could come in with advertising ahead of time and if that were made public——

Mr. MEADER. It did seem to me it would be appropriate for the Committee, whether it was published or not, to have in its files the list referred to in his testimony.

Mr. SECREST. The Commission operates on this theory.

Mr. BLATNIK. The suggestion is well taken. It will be treated confidentially and not be put in the public record, as least for the time being.

Mr. SECREST. It can be furnished.

Mr. BLATNIK. Referring to the top of page 14 of your statement, you stated here that the Commission is alert to the test data published by private publications, and there are instances in which you have a higher nicotine content in a new, filter tip, longer cigarette than you have in your old, regular, standard cigarette.

Mr. SECREST. According to the tests.

Mr. BLATNIK. These things were brought up in open hearings and I say that we do not single out any company or any brand. There is testimony from several witnesses listing the brands they tested by name and the amount of tar and nicotine they got out by the process they used, which they explained.

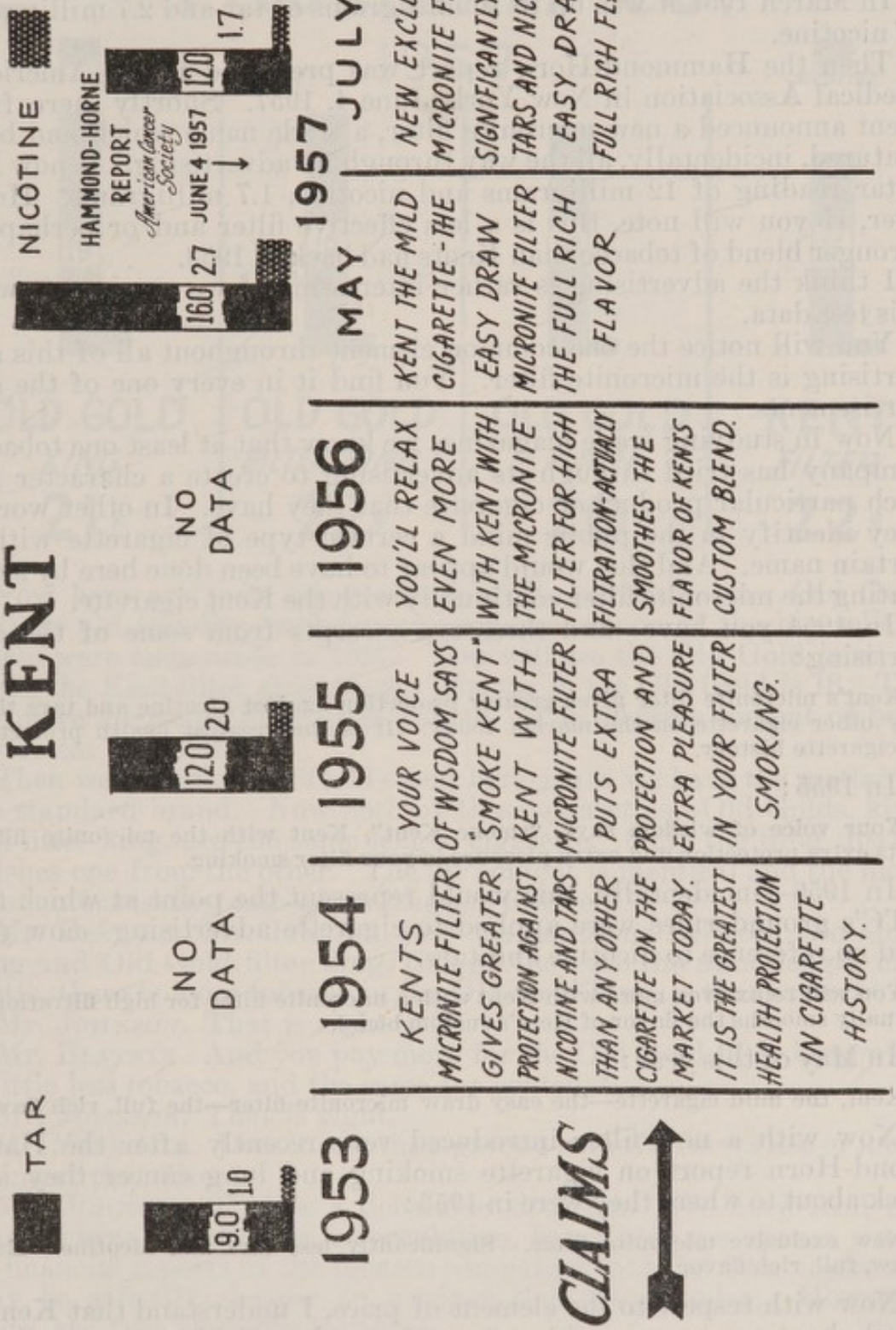
We have gone over some of these and we would like to check a few more of the current advertising—perhaps some has already been taken out—to give you an example of the thing that concerns us now.

They are statements that we don't see how they can be justified from what we know about it.

Mr. SECREST. Any advertising claim that this committee or any member of it or any Member of Congress or anyone thinks is false and misleading can be submitted to the Commission and we will welcome it.

Many of the things we investigate do come from such sources, including the public, members of the public. Any statement you have that you think may be false or misleading, if you will submit them to the Commission, they will be checked and checked expeditiously, as we do everything that we find that we think is false and misleading advertising.

Mr. BLATNIK. Could you give a few examples, Mr. Johnson? Do you have some of these charts you are working on that we have a summary of some of these claims which have been advertised before this committee in the past 3 days?



Mr. JOHNSON. Kent cigarettes has been one of the continuing advertisers of the filter cigarette. They were first introduced in 1952 and

while we don't have test data for that time, I understand that it removed such a substantial portion of the nicotine and tar that the cigarette was completely unsatisfactory commercially and was almost immediately modified.

However, in tests that were made in 1953 by Consumers Union, Kents had 9 milligrams of tar, 1 milligram of nicotine.

In 1955, this was stepped up to 12 milligrams of tar, 2 of nicotine.

In March 1957 it was up to 16 milligrams of tar and 2.7 milligrams of nicotine.

Then the Hammond-Horn report was presented to the American Medical Association in New York, June 4, 1957. Shortly thereafter, Kent announced a new micronite filter, a trade name which had been featured, incidentally, all the way through its advertising. It now has a tar reading of 12 milligrams and nicotine, 1.7 milligrams. However, if you will note, this is a less effective filter and/or perhaps a stronger blend of tobacco than Kents had back in 1953.

I think the advertising is rather interesting if we consider it with this test data.

You will notice the one common element throughout all of this advertising is the micronite filter. You find it in every one of the advertisements.

Now in studying trade magazines we know that at least one tobacco company has tried through its advertising to create a character for each particular product or cigarette that they have. In other words, they identify in the public mind a certain type of cigarette with a certain name. And this would appear to have been done here by associating the micronite filter continually with the Kent cigarette.

In 1954 you have—and these are excerpts from some of the advertising:

Kent's micronite filter gives greater protection against nicotine and tars than any other cigarette on the market today. It is the greatest health protection in cigarette history.

In 1955:

Your voice of wisdom says, "smoke Kent", Kent with the micronite filter. Puts extra protection and extra pleasure in your filter smoking.

In 1956—incidentally, this would represent the point at which the FTC's ground rules were applied to cigarette advertising—now you find no reference to nicotine and tars.

You will relax even more with Kent with a micronite filter for high filtration—actually smooths the flavor of Kent's custom blend.

In May of this year:

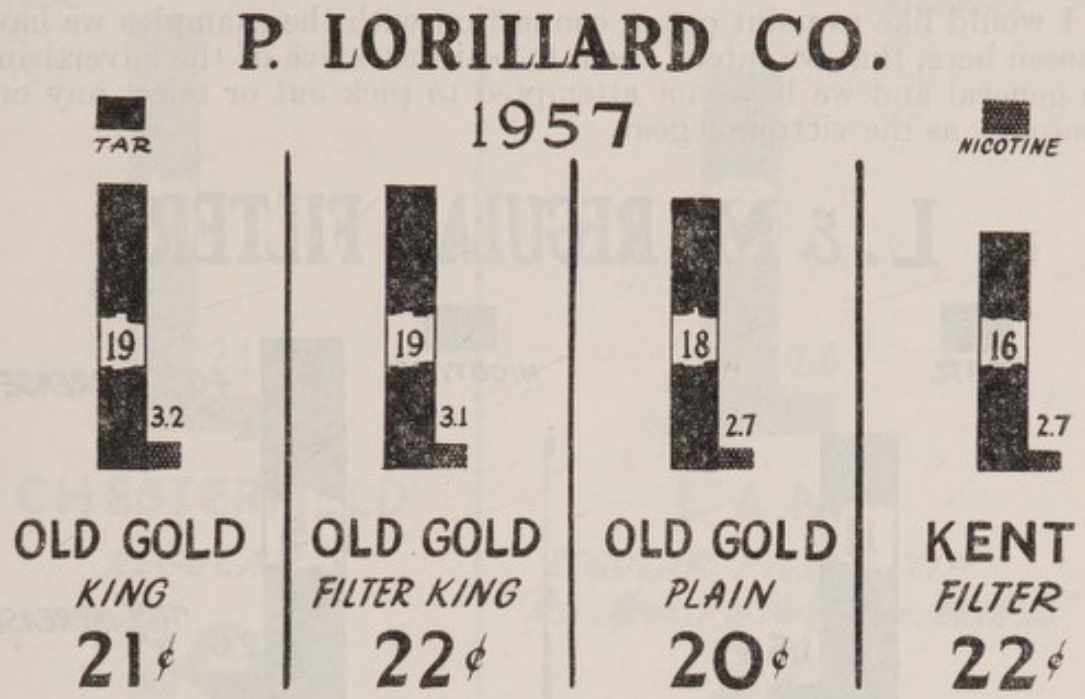
Kent, the mild cigarette—the easy draw micronite filter—the full, rich flavor.

Now with a new filter introduced very recently after the Hammond-Horn report on cigarette smoking and lung cancer they are back about to where they were in 1953:

New exclusive micronite filter. Significantly less tars and nicotine. Easy draw, full, rich flavor.

Now with respect to the element of price, I understand that Kents, until about a year ago, sold at 6 cents a pack more than the standard cigarette. The price differential now is 2 cents. I checked the prices yesterday at the House restaurant and Kents sell for 22 cents and the standard nonfilter brands at 20 cents.

But we felt there is an element of deception in this sort of advertising, because the micronite filter is still fixed in the public mind, and these changes make it quite a different product now from what it was in 1953 or 1955, or just a few months ago, and yet the public was in no way advised.



Now here are some comparisons of P. Lorillard Co. Old Gold is their old standby, sold for many years as one of the big sellers. These were tests made in 1957. You will see the Old Gold here, 20 cents, the Kent filter at 22 cents. The tar in the Old Gold is 18. The exclusive micronite filter gives you 16. The nicotine content of both cigarettes is identical.

Then we can take Old Gold—and here again we have the matter of the standard brand. Now both of these cigarettes—Old Golds, king and filter king, are the same length cigarette. Only the filter distinguishes one from the other. The tar content is identical and the nicotine content is one-tenth of 1 milligram different.

Mr. BLATNIK. Mr. Johnson, just one question: Between Old Gold king and Old Gold filter king, though they are the same length cigarette, there is more tobacco in the Old Gold king, is that correct?

Mr. JOHNSON. That is right.

Mr. BLATNIK. And you pay more for the Old Gold filter king with a little less tobacco, and the same tar content?

Mr. JOHNSON. That is right.

Mr. MINSHALL. Does it cost the cigarette manufacturers less or more to insert that filter?

Mr. JOHNSON. That is a debatable situation. We have read the trade journals and there is speculation, based perhaps upon a study of financial reports of the tobacco companies that the filter cigarettes may be actually cheaper to produce than the regular. However, there are two elements involved.

Tobaccos do account, I believe, for about 75 percent of the cost of the cigarette, but you do need an additional machine and an additional

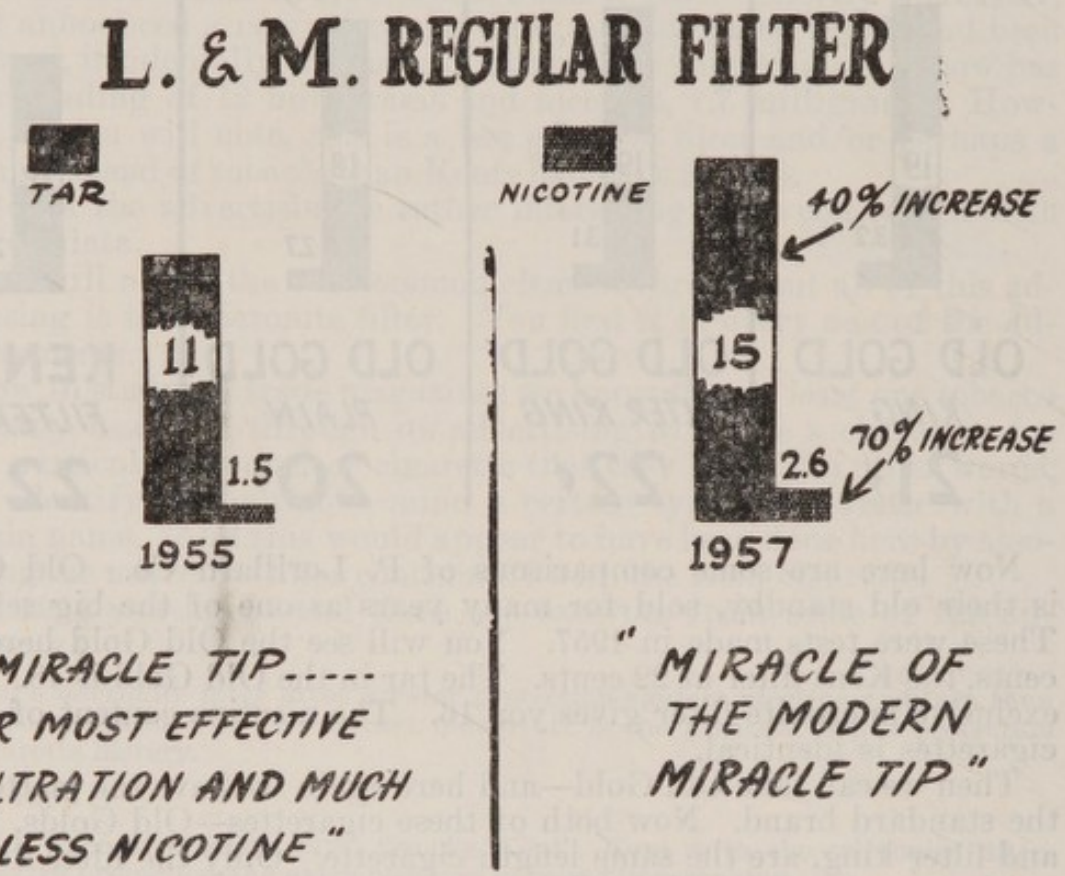
process to apply the filter. The filter may be cheaper but the extra process may result in about the same price or conceivably less.

Mr. MINSHALL. The material in the filter costs less.

(See appendix, exhibit 28, p. 751.)

Mr. JOHNSON. I think that is correct. It is acetate cellulose, I believe.

I would like to point out in connection with the examples we have chosen here, that we intend them to be illustrative of the advertising in general and we have not attempted to pick out or select any one company as the victim or goat.

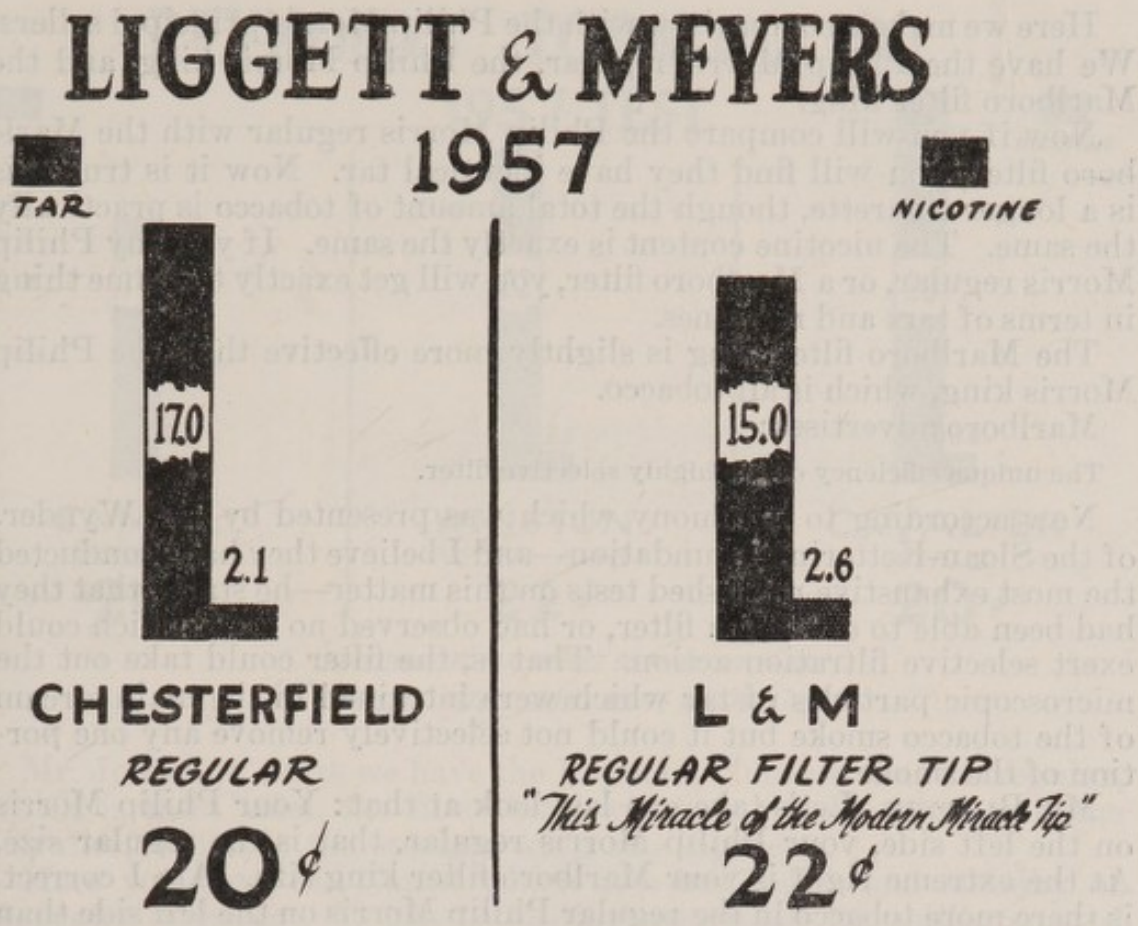


Now here we again find the application and evolution of a common advertising term—in this case, the miracle tip. The L & M regular when originally introduced had 11 milligrams of tar, 1.5 milligrams of nicotine. Now, incidentally, this is comparable to the present improved micronite filter Kent cigarette—a reasonably effective filter or combination of filter and tobacco blends to produce a cigarette low in nicotine and tar content.

The advertising in 1955: Miracle tip—for most effective filtration and much less nicotine.

There may have been some justification for such a claim at that time. In 1957 we still have the common element in the advertising—now the "miracle of the modern miracle tip."

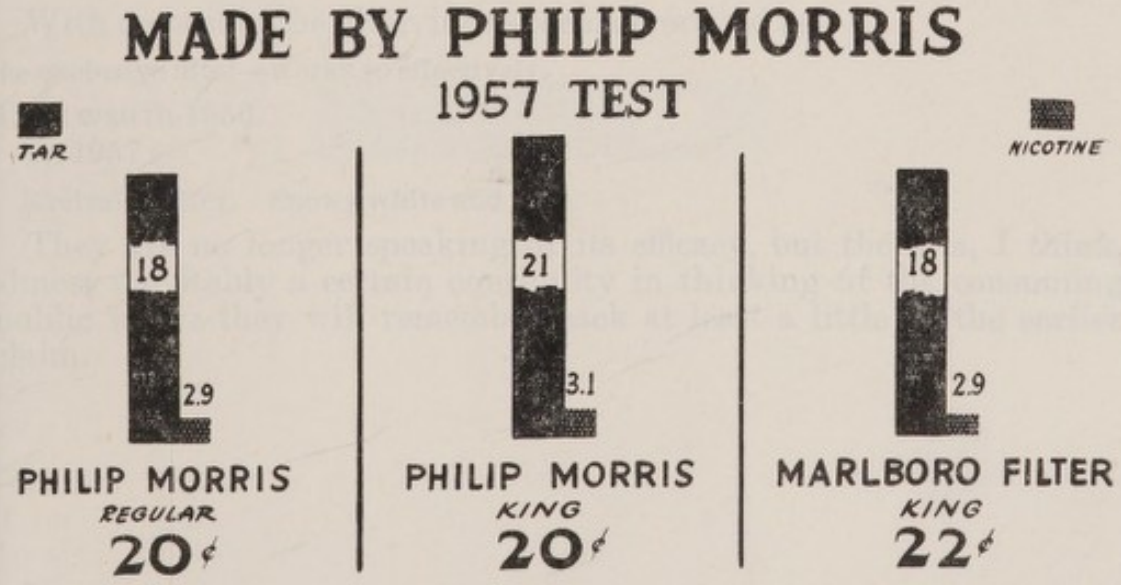
Tar however has now gone up to 15 milligrams, a 40 percent increase. Nicotine has gone up to 2.6 milligrams, a 70 percent increase and I believe the consumer could quite validly ask, "Where is the miracle of the modern miracle tip?"



Here we combine the L & M with the Chesterfield regular. The Chesterfield sells for 20 cents, one of the old popular brands, and the L & M introduced since the cancer scare or whatever we wish to call it, and along with the promotion of other filter cigarettes.

The Chesterfield has 17 milligrams of tar. You have 15 in the L & M's. Slightly less.

In the Chesterfield you will get less nicotine than you will get in the L & M filter tip, and again we have the question, "Where is the miracle of the modern miracle tip?"



MARLBORO....."THE UNIQUE EFFICIENCY OF THE HIGHLY SELECTIVE FILTER WORKS FINE BUT DOESN'T GET IN THE WAY"

Here we make a comparison with the Philip Morris principal sellers. We have the Philip Morris regular, the Philip Morris king, and the Marlboro filter king.

Now if you will compare the Philip Morris regular with the Marlboro filter, you will find they have identical tar. Now it is true this is a longer cigarette, though the total amount of tobacco is practically the same. The nicotine content is exactly the same. If you buy Philip Morris regular, or a Marlboro filter, you will get exactly the same thing in terms of tars and nicotines.

The Marlboro filter king is slightly more effective than the Philip Morris king, which is all tobacco.

Marlboro advertises:

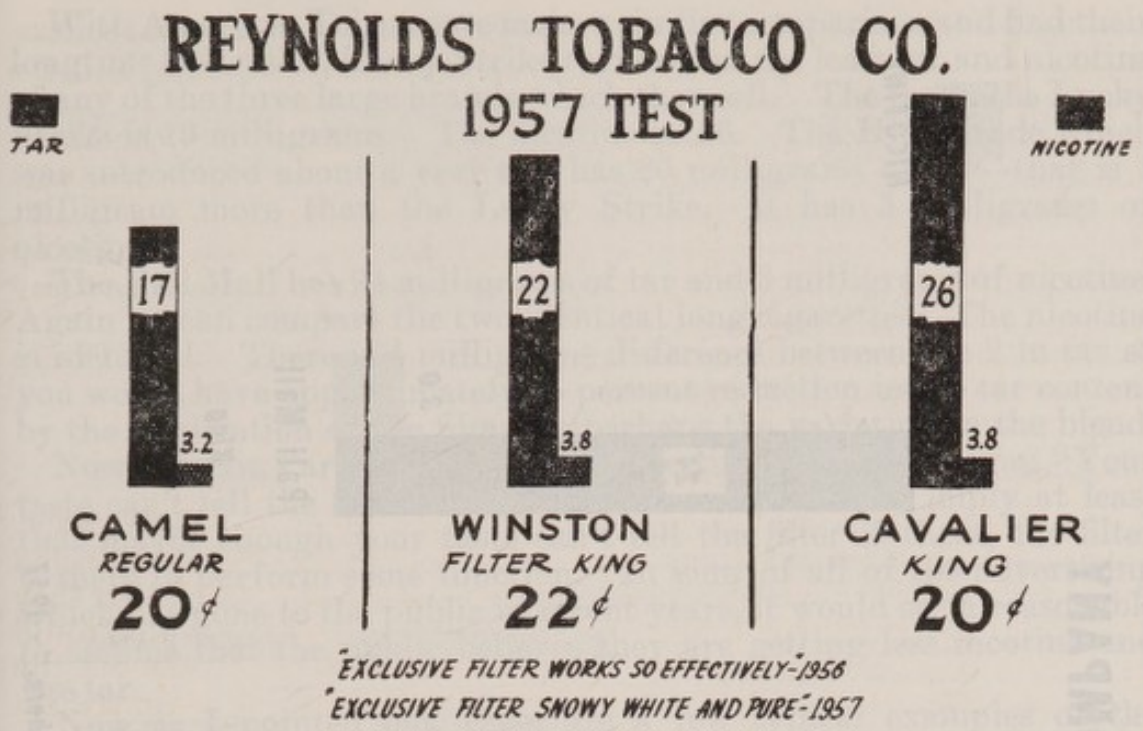
The unique efficiency of the highly selective filter.

Now according to testimony which was presented by Dr. Wynder, of the Sloan-Kettering Foundation—and I believe they have conducted the most exhaustive published tests on this matter—he stated that they had been able to develop a filter, or had observed no filter which could exert selective filtration action. That is, the filter could take out the microscopic particles of tar which were intrained in the main stream of the tobacco smoke but it could not selectively remove any one portion of the smoke.

Mr. BLATNIK. Let's take one last look at that: Your Philip Morris on the left side, your Philip Morris regular, that is the regular size. At the extreme right is your Marlboro filter king size. Am I correct, is there more tobacco in the regular Philip Morris on the left side than there is—there is less tobacco in the regular without the filter and you get just as much tar and just as much nicotine in a Marlboro king which has less tobacco and a filter?

Mr. JOHNSON. That is right.

It has been estimated that in most brands of filter kings there is about 10 percent less tobacco than in the regular size, nonfilter cigarettes. This will vary from brand to brand and we have no data as to which those brands are. I think in terms of tobacco there will be approximately the same amount in these two cigarettes.



Mr. JOHNSON. Here we have the Reynolds Tobacco Co., the regular, which is the Camel, the filter king which is the Winston, and the Cavalier, which is the straight king size cigarette.

Here you have the Camel which is a best seller and standby of Reynolds Tobacco. It has 17 milligrams of tar, 3.2 milligrams of nicotine.

In the Winston filter king you will get 22 milligrams of tar, 3.8 milligrams of nicotine.

In the Cavalier which is a straight king cigarette, you will get 26 milligrams of tar and 3.8 milligrams of nicotine.

These two cigarettes have identical lengths. The nicotine content is identical. The difference in tar content is 4 milligrams.

The Winston has been advertised as:

The cigarette which brought flavor back to filter smoking.

With respect to the filter, it has been advertised as—

the exclusive filter—works so effectively.

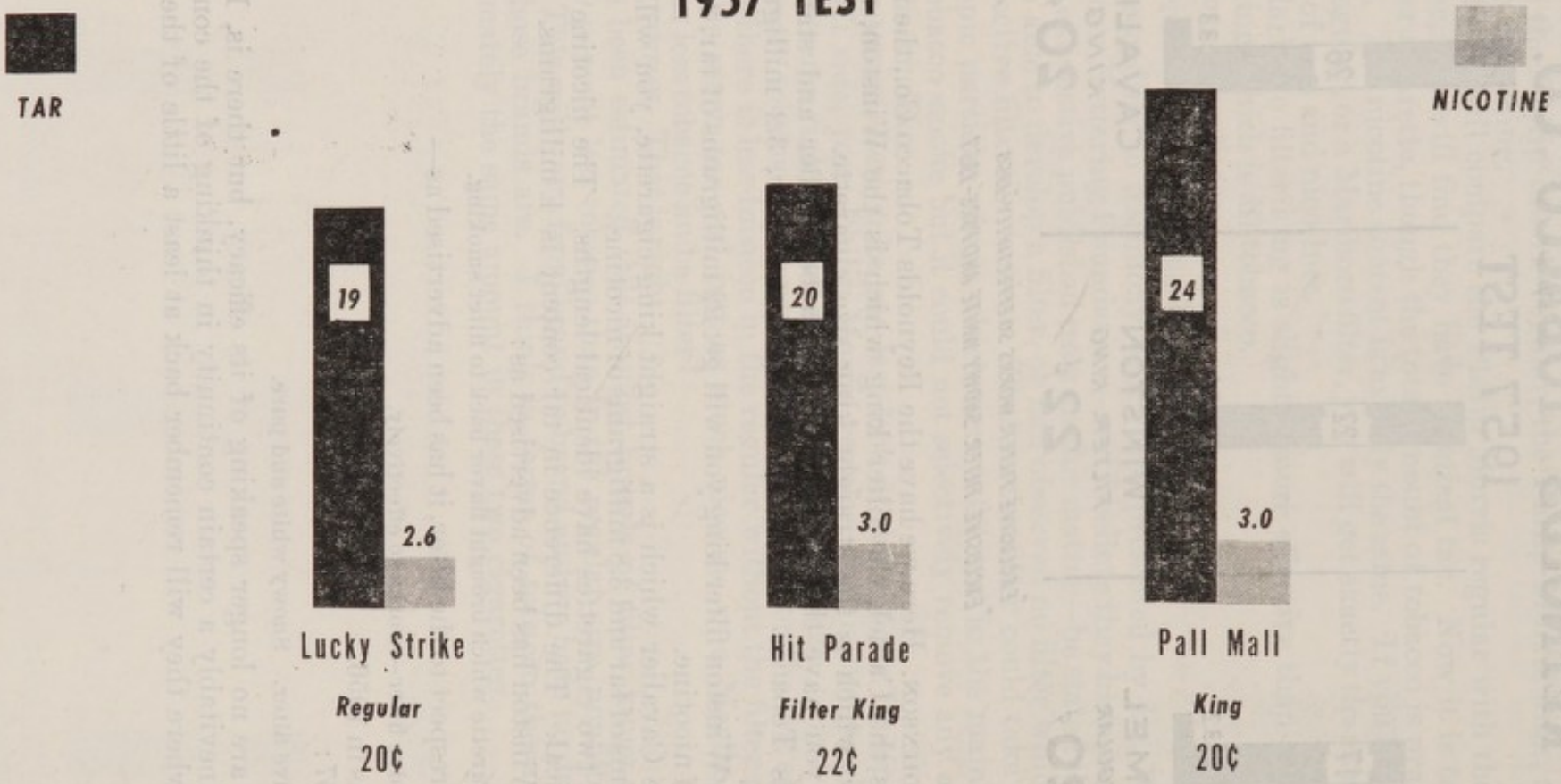
That was in 1956.

In 1957:

Exclusive filter. Snowy white and pure.

They are no longer speaking of its efficacy, but there is, I think, almost inevitably a certain continuity in thinking of the consuming public where they will remember back at least a little of the earlier claim.

AMERICAN TOBACCO COMPANY
1957 TEST



"Your taste can't tell the filter's there." - 1957

With American Tobacco we make a similar comparison and find their longtime best seller, Lucky Strike regular has the least tar and nicotine of any of the three large brands which they sell. The tar in the Lucky Strike is 19 milligrams. The nicotine is 2.6. The Hit Parade which was introduced about a year ago has 20 milligrams of tar—that is 1 milligram more than the Lucky Strike. It has 3 milligrams of nicotine.

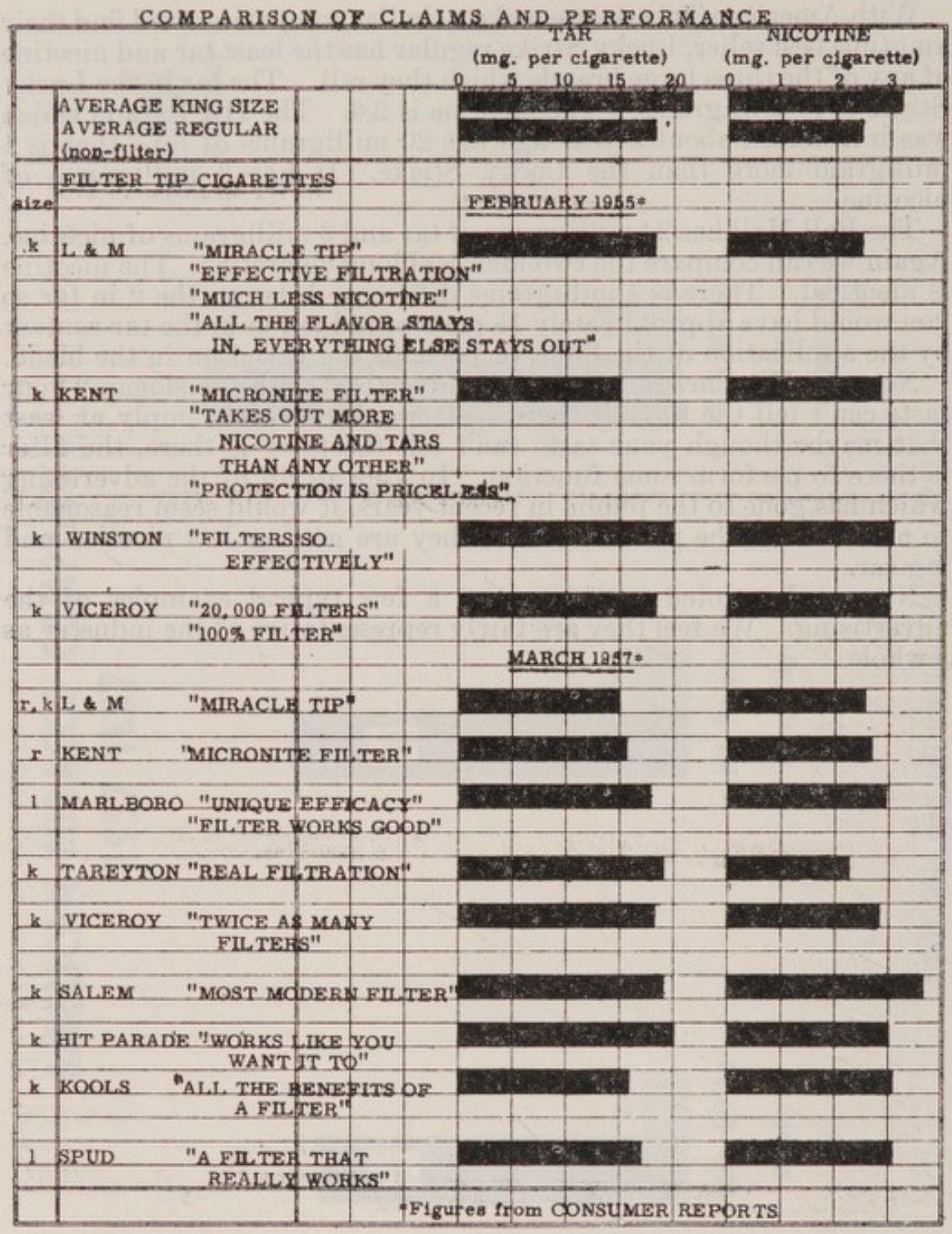
The Pall Mall has 24 milligrams of tar and 3 milligrams of nicotine. Again we can compare the two identical long cigarettes. The nicotine is identical. There is 4 milligrams difference between the 2 in tar so you would have approximately 15 percent reduction in the tar content by the application of the filter, or perhaps the variation in the blend.

Now the Hit Parade is currently advertised with the slogan "Your taste can't tell the filter is there." It would, I think, imply at least that maybe though your taste can't tell the filter is there, the filter is there to perform some function. In view of all of the advertising which has gone to the public in recent years, it would seem reasonable to assume that the public believes they are getting less nicotine and less tar.

Now as I pointed out, these are a few typical examples of the advertising. We feel they are fairly representative of the industry as a whole.

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COMPARISON OF CLAIMS AND PERFORMANCE



Mr. BLATNIK. Whose tests are used there?

Mr. JOHNSON. They are tests by Consumers Union. The reason we selected them is that theirs was the only source where we could get a series of tests over a period of time. That is, they made tests in 1953, 1955, and 1957.

Dr. Wolman, director of the American Medical Association laboratory appeared before the subcommittee a few days ago and testified that the method used by Consumers Union paralleled their own and that the results were remarkably close, so we assume that these tests for comparative purposes, should be accurate and valid.

Mr. BLATNIK. So we are not so interested in the absolute figures because the type of tests will vary. You could get another test which

is accurate and would run perhaps a few points higher, but the relation would be the same.

Mr. JOHNSON. That is right.

In Dr. Wynder's tests and also the tests performed by Foster E. Snell for Reader's Digest, used a different method in extracting tar. They used the whole smoke condensate rather than just that portion soluble in chloroform, so they got a larger reading for tar.

Mr. MEADER. I would like to ask the Commissioner a question or two on this statement.

On page 7 you refer to the cigarette advertising guides. Have you furnished a copy of those guides to the committee staff for our record?

Mr. SECREST. I think the committee staff has it and I have copies here.

Mr. MEADER. Is it anything very voluminous?

Mr. SECREST. No.

Mr. MEADER. Mr. Chairman, I would suggest we incorporate the advertising guide he referred to in the record.

(The document referred to is as follows:)

CIGARETTE ADVERTISING GUIDES

The following guides have been adopted by the Federal Trade Commission for use of its staff in the evaluation of cigarette advertising.

No representation, claim, illustration, or combination thereof, should be made or used which directly or indirectly:

1. Refers to either the presence or absence of any physical effect or effects of cigarette smoking in general or the smoking of any brand of cigarette.

NOTE.—Words, including those relating to filters or filtration, which imply the presence or absence of any physical effect or effects are considered subject to this guide.

2. Represents that any brand of cigarette or the smoke therefrom is low in nicotine or tars, or contains less nicotine, tars, acids, resins, or other substances, by virtue of its ingredients, method of manufacture, length, added filter, or for any other reason or without any assigned reason, than any other brand or brands of cigarettes when it has not been established by competent scientific proof applicable at the time of dissemination that the claim is true, and if true, that such difference or differences are significant.

NOTE.—Words, including those relating to filtration, which imply lesser substances in the smoke, through filter comparisons or otherwise, are considered subject to this guide.

3. Refers to the effects of cigarette smoking in general or the smoking of any brand of cigarette on the (*a*) nose, throat, larynx, or other part of the respiratory tract, (*b*) digestive system, (*c*) nerves, (*d*) any other part of the body, or (*e*) energy.

4. Represents medical approval of cigarette smoking in general or the smoking of any brand of cigarette.

5. Compares the volume of sales of competitive brands of cigarettes, or the purchase or use of particular types, qualities, or grades of tobacco in cigarettes, when such claim is not based on reliable information currently applicable when disseminated.

6. Relates to or contains testimonials respecting cigarette smoking or the smoking of any brand of cigarette unless (*a*) the testimonial is genuine, (*b*) the advertiser has good reason to believe it represents the current opinion of the author who currently smokes the brand named, and (*c*) it contains nothing violative of any of the other guides set forth herein.

7. Falsely or misleadingly disparages other cigarette manufacturers or their products.

NOTES.—(*a*) Nothing contained in these guides is intended to prohibit the use of any representation, claim, or illustration relating solely to taste, flavor, aroma, or enjoyment.

(*b*) Nothing contained in these guides will have the effect of modifying the provisions of any existing cease-and-desist order or stipulation or altering the responsibility of any party thereto to fully comply with the specific provisions

of such order or stipulation affecting it. They do not constitute a finding in and will not necessarily affect the disposition of any formal or informal matter now pending with the Commission.

(c) These guides will be altered, modified, or otherwise amended when and if the facts and circumstances warrant.

Mr. MEADER. Has this method of establishing advertising guides been established in other industries than the tobacco industry?

Mr. SECREST. We have what can be compared to them in what we call trade-practice rules which are guides on price discrimination, false and misleading advertising, the laws the Commission enforces for 160 industries. They are just industrywide guides. The metal awning people, the fountain pen manufacturers and 160 others. They are guides for them. That is a form of procedure. We hold public hearings and such and we did not do that in this case because that was time-consuming and we wanted guides as quickly as we could get them to put into operation.

The other would have taken maybe a year or two. This was considered in the Commission and we had the case law back of us in what we used in the guides. We went into nothing but advertising. In the other rules we go into every discrimination of prices and the other things that the Federal Trade Commission has to deal with besides advertising. We have many other duties.

Mr. MEADER. Let me see if I am clear about this: Your jurisdiction would be limited to the tobacco industry as contrasted to the press or to the television and radio industries. You can't issue any orders to the television and radio industry?

Mr. SECREST. I think the Commission might be able to do so, but it is the advertising we are after. I don't know if the radio station would ever today publish nothing but false and misleading advertising and keep it up for a year, one after the other—I don't know. I think we can proceed against the radio station, but as a policy we don't. We are after the advertiser.

Mr. MEADER. I am thinking of the possible conflict in jurisdiction between the Federal Communications Commission and the Federal Trade Commission.

Mr. SECREST. Every order we issue, every stipulation we take, every proceeding we have against any manufacturer or against any advertiser, that is all sent to the FCC, together with the call name of the station that put it out and the FCC then has that for its own guidance. I don't know of any station that has ever been refused a license on false and misleading advertising or probably ever will, but they in turn send that to the station that is involved.

If we issue a complaint against X company for false and misleading advertising, that complaint goes under our arrangements with FCC direct to them. They immediately send that complaint to the local radio station or to the television station.

Mr. MEADER. In your formal proceedings your complaint is issued against a particular company?

Mr. SECREST. Yes, for particular acts.

Mr. MEADER. But others that may have had a part in distributing the false claims, if they are false, are not parties to your proceeding. For instance, you don't enjoin all the newspapers?

Mr. SECREST. We go to the source of the ads.

Mr. MEADER. You go to the manufacturer and don't attempt to govern the media of distribution.

Mr. SECREST. We do not. We go to the man who initiates the advertisement, who has something to sell, a commodity or service to give.

Mr. MEADER. You wouldn't enjoin an advertising agency representing the manufacturer of the product?

Mr. SECREST. We have in rare instances. It would just depend if the advertising agency drew up all the advertising and was responsible for it, I assume we could, but it is a rare instance. We always go to the man who is going to benefit from that advertising and that is the fellow with something to sell.

Twelve claims on this list here have been discontinued and four of them within the last few weeks by our voluntary process.

Mr. MEADER. In the consultations that you had prior to the establishment of the guides, was there any record maintained of those conferences?

Mr. SECREST. Just memorandum records. No stenographic record. Because it is all voluntary. Every one of these 75 claims that have been discontinued were discontinued voluntarily by the companies at our request.

Mr. MEADER. Now, on the whole from your statement I gather that you feel that the tobacco industry has cooperated rather well with you on a voluntary basis.

Mr. SECREST. I would say it has worked exceptionally well considering the conflict that exists in the medical testimony with respect to tar and nicotine.

Now, if tomorrow morning they could say that "This tar, or this in tar causes cancer," then Congress would be faced possibly with a national health problem and the Commission would have something upon which to pinpoint more definitely complaints or require advertising, but I think our proper procedure under these guides has been a miracle of achievement in stopping what might be false and misleading advertising in the cigarette industry.

Mr. BLATNIK. If the Commission had the voluntary injunctive power to order them to cease and desist immediately but on a temporary basis, would that further encourage this good start on proper policing of advertising?

Mr. SECREST. The injunctive power, as we have it now in drugs, on the theory if you keep on selling a medicine that might harm someone, you better stop it now. We go to the court and can get an injunction. If it is false and misleading advertising—drugs, cosmetics, food—any false and misleading food advertisement, we can ask for an injunction to stop it and, if the court agrees with us, we get the injunction. If they say, "You don't need an injunction; that is too close a question," you can't have it. If that were given to the Commission, it would certainly make more effective, quicker, the regulation of false and misleading advertising in the cigarette field, or any field, for that matter.

Mr. MINSHALL. At the outset, I would like to commend you on a very excellent, forthright, and, above all, a candid statement. I wish I could say the same thing for some of the other witnesses who have testified before this committee, but, unfortunately—I believe the rest of the committee agrees with me—I cannot. I notice on page 13 of your statement that you said, in view of the Public Health Service's statement, that it calls for a fresh approach by the Commission

in its regulation of cigarette advertising in the public interest. Now, what do you intend doing in the way of a fresh approach?

Mr. SECREST. I can say this: that we considered, months ago, a consumer survey, and reached the conclusion that, until there was a more definite pronouncement on the subject of the relationship of cancer to cigarette smoking, we should hold it in abeyance.

Immediately after this statement by the Surgeon General, the Commission authorized our staff to prepare the questions—and that is very important; those questions have to be used as evidence to be used in a legal proceeding—to prepare the questions, to prepare the plans for a consumer survey. The whole staff, including our General Counsel, the Chief of our Bureau of Consultation, the Chief of our Bureau of Investigation, all of them will work the plans out for a consumer survey. That will be presented to the Commission. That action then would give us some basis in the approach to the question of filter alone in cigarettes. What does a man think about when he buys a filter cigarette? Does he want to keep loose tobacco out of his mouth? Does he want to protect his health, or does he think he is protecting his health? Those are things the Commission needs to know to put in proof in the case of false and misleading advertising.

That is not all the proof, but that is a part of it, and an essential part. So we are laying the groundwork for any future formal action that we might be compelled to take in the field of cigarette advertising.

Mr. MINSHALL. How long do you contemplate it will take to make a survey like that?

Mr. SECREST. I should think that, at most, 2 or 3 months; by then they should have the questions. They have to be considered. There is no use to take a survey and leave one question out that is essential or find one question wasn't worded so it could be used legally in the formal trial of the case. That will take some time. The survey itself must be broad; it must be broad enough so that it can be considered a cross section of what all the people think when they buy a filter cigarette.

Mr. PLAPINGER. Are you familiar with past surveys that have been made——

Mr. SECREST. Not exactly on this question, but we have made numerous surveys in the past.

Mr. PLAPINGER. ——by other agencies. I understand the Bureau of the Census made one. There was reference during our testimony to one conducted by the Institute of Public Opinion, and also reference made to one by Sloan-Kettering.

Mr. SECREST. You get certain delays. Now, in any surveys such as this, questions we send out we have to clear with the Bureau of the Budget under an act of Congress. That is so we won't send out a questionnaire at the same time another agency does and pester businessmen to death. It is a good law. We clear it with the Bureau of the Budget. Mr. Babcock will be responsible for that. What would you estimate?

Mr. BABCOCK. Four to six months.

Mr. SECREST. Four to six months for the actual survey itself.

Mr. MINSHALL. Would you consider it accomplishing any useful purpose to put the contents of tars and nicotine on the package of the cigarettes?

Mr. SECREST. I don't want to comment on the advisability of it, because the advisability of it rests solely upon medical opinion. If tar doesn't hurt you, there is no need for it. If medical opinion says tar

is a causative factor and thinks that the health of the Nation requires it, it could be done. It certainly would be easy for us to enforce it. If the wording said, "Not more than so much tar," all we would have to do would be to get some samples of cigarettes, test them by what I hope would be some standard procedure. If it is above that "not more than," it is then false and misleading—substantially above.

The law would have to outline the standard of testing procedures, but I think I have mentioned that several times. That law would be enforcible.

Mr. MINSHALL. Would you consider an alternative of that; to have the cigarette manufacturers submit to you, based on a standard method of testing at periodic intervals, the ratings of their cigarettes as to tar and nicotine?

Mr. SECREST. You get, then, back to the health question. Of course, the Commission hasn't considered the answers I am giving, but I am giving them to you because I think they are right; that whether you required that to be put on a pack of cigarettes is a health matter. We are not the health agency, and we are not in a position to say what effect this has and, I think, from some of the material—I noted a recent publication of the American Cancer Society, whose officials reviewed the various scientific data on the problem states:

> It would be gratifying if the Cancer Society could produce a neat, final answer, but it cannot.

When the question of marking the package of cigarettes and such comes up, if tar doesn't hurt you a bit—assuming that; that there is no connection between cigarettes and cancer—then there can be no false and misleading advertising with respect to it.

Mr. BLATNIK. But would it be helpful just to know how much tar is in there? For example, there is no question about the alcohol in beer hurting you, yet they mark it 3.2.

Mr. SECREST. I don't know how it would work out and, since it is a medical question, my comments are merely incidental to that particular part of the question. It might be, if cigarette companies had to put on each package how much tar was in it, that there would then be competition in the industry to reduce the tar content of the smoke. The public might, "I want the one with the least tar." If they wanted the one with the least tar, then the competition would see who could get the least tar in the smoke of their product to sell it. It might have that effect and it might not, depending on the test of the consumer.

Mr. MINSHALL. It doesn't prove anything in alcoholic beverages when they put the proof on the bottle.

Mr. SECREST. I don't know of anyone the proof ever stopped.

Mr. BLATNIK. With regard to alcoholic beverages, what Government agency is responsible for supervising?

Mr. SECREST. The Alcohol Tax Unit in the Bureau of Internal Revenue, and here is what they do, as I told you: We get these newspapers and magazines, different ones every month, we get half of all the commercial broadcasting that is used by the two major networks sent to us voluntarily by them. We get one week a month from one of the smaller networks. We go through those advertisements. Every one that advertises alcohol that we see, we clip out and send over to this Alcohol Tax Unit. I think in the last weekly report I saw we sent them in a week's time about 370 or 380 advertisements dealing with

liquor. They have the control over it, and we send those to them and it saves them setting up a bureau—we have to look these papers through and get the ads anyhow, and it saves them setting up a bureau to duplicate the work. Every ad we get dealing with liquor we send over to the people who enforce it.

Our liaison with Justice and the other agencies of the Government, I think, has been very well perfect to the point where they get the most good out of what we do and there is the least duplication or waste of the taxpayers' money. And, on this whole subject, I feel that we made the only approach that could be made on it, to get the best results in the quickest time, and I think that the fact that the Commission even issued guides as far back as 1955 indicates that we were well ahead of the field in this business of regulating advertising in cigarettes.

Mr. BLATNIK. Now here you have two different agencies handling advertising, one alcohol and the other in tobacco and the cigarette industry.

My knowledge, such as it is, on the alcohol competition, it is a very competitive field.

Mr. SECREST. They have done much in the way of self-regulation.

Mr. BLATNIK. There is a voluntary aspect about it, it is quiet, not much fuss or fanfare. I think the industry has done a good job and the governmental agencies have done a good job. They are not overballyhooing the liquor and what it is going to do to or for you.

I wonder if we could find some lessons here for the cigarette industry where a cooperative, positive joint effort might be worked out to have a little bit more of a realistic and a little better taste in all this advertising.

Mr. SECREST. The greatest hope of getting results in any advertising field is the sincerity of the advertiser to try to advertise his product truthfully. If every advertiser in the country wasn't trying to control his advertising, you couldn't appropriate us enough money to do the job that we are asked to do. Ninety-nine percent of all industry and maybe more advertising just as honestly as they know how, and there is a difference here between the people who manufacture cigarettes and the Federal Trade Commission on many of these claims, because there is a difference in the scientific evidence that is before anyone, at this time. I think we moved well ahead of the field in our efforts to try to get this advertising. We prohibited health claims in 1955, the month of September, in these guides and that has been now almost 2 years ago—before we had anything like the tests that we now have available to us with respect to tar and nicotine content.

Mr. BLATNIK. Commissioner, you have done an excellent job. You have made great strides here with your guide and with your ground rules, so to speak, and the voluntary cooperation with the cigarette industry. But yet it is my feeling with the literally hundreds of millions of dollars that have been spent by the cigarette industry in advertising in the last 4 years, I don't think the average consumer has much of an idea of just what he is getting—whether he is buying a "miracle tip," or an "effective filter" or "superfilter," in whatever brand he is buying. Why so little knowledge on a thing that has been so widely advertised is the thing that puzzles me. I think we are en-

titled to know. The consumer knows about many other products which are far less important from a health standpoint. He doesn't know whether 18 milligrams is bad for him, but I would like to know whether one has 30 milligrams or 18 milligrams. I know that an 18-milligram cigarette isn't any safer than a 30-milligram cigarette if you are smoking 3 packs of one and 1 of the other.

I am directing these comments more to the industry people, and you have contact with them.

I think this thing can be resolved in a fair and a positive and constructive way.

As someone said, I think smoking is here to stay until something drastic comes up. But it has to be done in moderation. It is not the use of a bad thing, if you want to say cigarettes are bad. It may not be so much the use of a bad thing as the abuse of a good thing.

I can only speak for myself, but I think we would make far better progress in reaching the people, by advertising in good taste and even going out in honesty and saying that you should use this in moderation. On certain occasions, the liquor industry says, "If you must have that last drink for the road, make it a cup of coffee."

It is a good feeling between the industry and the people and the industry and the Government agency, and I wonder if something along that line couldn't be developed here with the cigarette industry.

Mr. SECREST. In the guides we attempted to do it and we certainly would be happy if they could advertise in such a way that we would never be compelled to question it. It would save our time and our money for many other things that the Commission is supposed to be doing, and I hope they will.

Our responsibility under the law, and our only responsibility as of right now, is to see that advertising in any field within our jurisdiction is not false and not misleading, and we will continue to question every claim of any advertiser—the cigarette people included—that we think is false and misleading. There are honest differences of opinion and above all we want to be fair to any segment of industry in the country. We have tried to do that, I think, as well as we could, but we haven't stopped or rested just because we have guides.

Every day we are trying to get more and more compliance with those guides. When we get consumer testimony we will have some ideas of what a man thinks when he buys a filter cigarette or when he says the word "filter" by itself, and if this committee has any advertisement that they deem to be false or misleading, just let us have it and it will be corrected. Maybe it will be found false and misleading and maybe it won't. That depends upon the facts in the case.

Mr. BLATNIK. Thank you, Commissioner. Any further questions?

Thank you, Commissioner, for the splendid help, information and cooperation which you have given the committee.

We have a letter from our colleague, Congressman John Watts of Kentucky, and enclosed is a copy of a letter by Dr. Milton Rosenblatt of New York, addressed to Dr. Leroy E. Burney, the Surgeon General, which we will include in the record. (See appendix, exhibit 29, p. 752.)

This closes the hearings as far as the Federal Trade Commission is concerned on the cigarettes, particularly the filter cigarettes, except

that the Chair would like to ask unanimous consent or agreement with the members of the committee that he hold the record open for a week to complete the record with many exhibits we have asked for and give us time to insert them in the record. I direct counsel to include in the record at the appropriate places material that is necessary to complete the record. (See appendix, exhibit 30, p. 754.)

The only important group we have not yet heard from is the cigarette industry, itself. We do not want to be arbitrary, nor do we want to be impulsive in this matter. Although we are closing the hearings we would be glad to hear from the cigarette people if upon further consultation amongst themselves or within their own industry they feel that in all fairness and to make the record complete they should be heard.

Our main objective here in getting the medical background was to collect together in one reliable volume, all the points of view in testimony without imposing upon ourselves the responsibility of making a determination which is not within our jurisdiction to make.

We hope that in a fair and positive and comprehensive open discussion of all points of view on something that is unquestionably of great concern to literally millions of American people we have made a little contribution to a better understanding of the problem and specifically we hope a more effective program of advertising of these particular products.

In closing I want to commend the members of the subcommittee, particularly the gentlemen here. Knowing what an extremely heavy floor schedule we have had, the Chair is deeply grateful for the special effort made to be here in afternoon sessions as well as the many morning sessions we have had.

If there are no further comments or questions, the Chair adjourns the——

Mr. GREENHOUSE. Mr. Chairman, may I——

Mr. BLATNIK. Mr. Greenhouse, I am sorry——

Mr. GREENHOUSE. May I respectfully ask permission of the Chair to make a statement to the committee that will take about 5 minutes? You will recall that I was interrupted in my testimony yesterday by a rollcall. I came here a thousand miles at my own expense. I believe that what I have to say is very pertinent to your issue here, and of interest to 100 million Americans. May I have that permission, please?

Mr. BLATNIK. We do give you the permission. Will you please take the chair, Mr. Greenhouse?

Mr. GREENHOUSE. Thank you.

Mr. BLATNIK. I do want to emphasize this: We appreciate the special effort you made to be here——

Mr. GREENHOUSE. Thank you.

Mr. BLATNIK. But I hope you do not feel or indicate here that the statement was not given or will be given the same consideration given to all testimony.

Mr. GREENHOUSE. I realize that.

Mr. BLATNIK. It is already properly and officially included in the official record of these hearings. We will be glad to hear whatever supplementary or additional information you may have to present orally.

Mr. GREENHOUSE. I qualify as a witness and I attach my comments entirely against the American Cancer Society report which has been put into your record and which has received nationwide publicity in the press and has appeared in scare headlines to an extent that it has filled the American public with anxiety about the dire consequences of smoking. I am addressing my remarks exclusively to the statistical validity of that report because I am an expert on statistics and have spent my life analyzing all types of reports.

I say to you that the American Cancer Society report is in many respects a phony from cover to cover; that the report was designed especially to impress upon the American people a certain point of view and in doing so it has violated all scientific procedures in obtaining reports.

To start with, they selected only men between the ages of 50 and 70 who would have died anyway 25 years ago from natural causes; they have extended even today with the health improvements the age on that report to 70, when 67 by actuarial science is the limit of a man's life.

No. 2, they have sent out 22,000 volunteer workers and with the best of intentions were told not to take any men who were sick, but it was left to the research workers themselves to determine whether or not a man was sick. They could have very well gotten a doctor's certificate for each case.

No. 3, they did not look into the question of medical treatment in the case of a death. They dealt only with dead bodies, although they call it a study of 187,000 men. It is not a study, it is merely a census or a registration, an enrollment of 187,000 men who after they were enrolled were just checked up once a year by these workers. They turned in the question on each one, "alive or dead or change of address," and if they died, they followed the certificate.

That, I submit, is not a study, it is merely an enrollment.

Now, they overlooked the fact that cancer in many cases is hereditary, and they took no pains to determine whether any of the deaths resulted from hereditary traits.

I am skipping a lot of other factors which should have been considered by them in an honest study. I am taking slide No. 26 of their own report. (See p. 265, supra.) In this slide they show that 52.1 percent, in a great big line, die of coronary-artery diseases. The first impression of that as a man looks at it is that smokers will die to the extent of 52 percent from smoking excessively, which would not have happened if they did not smoke.

Now, I examined this chart. A statistician, a person who is experienced in studying statistics can spot a chart of this sort a mile away.

I drew comparative figures to the side to show what the chart really should have expressed if they desired a logical statistical report.

Instead of showing 52 percent, my chart shows three-quarters of 1 percent, and down below instead of showing 27 percent dying from cancer, when you compare the total to the total men in the study, 187,000, the percentage is less than 1 percent. So there is a difference of 5,000 percent in 1 method of figuring which is more logical than the arbitrary method used here. It is as if they took 100 men who died in any locality and say 70 of those men died from coronary-artery disease and 30 from cancer or other causes. They draw a blank line.

Seventy percent died from cancer; but they do not consider that the total deaths with which that part is compared, the total is only 1½ percent of the entire 187,000 cases.

Mr. BLATNIK. Just straighten me out in my thinking: Dr. Hammond's testimony was not that this smoking caused 70 percent of all deaths. I thought the testimony was that of those who do die of lung cancer, or who have lung cancer when they die, that a high percentage of them are heavy smokers. Isn't that what they were trying to tell us?

Mr. GREENHOUSE. What they were trying to tell the committee is that there is a high intimacy of association between cigarette smoking and lung cancer. What they overlook is the fact——

Mr. BLATNIK. No; what they were saying, as I understood it, and I want you to correct me on it, was that of all of those who die of lung cancer, the overwhelming number of those people were smokers of long duration and only a relatively small percentage of those people did not smoke. Isn't that correct?

Mr. GREENHOUSE. That is correct, but——

Mr. BLATNIK. I am lost now. I didn't think they said 70 percent of all the people who die each year die of lung cancer.

Mr. GREENHOUSE. I say this chart is misleading and I tell you why: You can have this insignificant number of 3 people, 2 of them die of cancer and 1 of heart disease.

Mr. BLATNIK. How many people die a year of cancer?

Mr. GREENHOUSE. Of cancer altogether, I would say about 200,000. Lung cancer is about 20,000. But Dr. Steiner, who was quoted here yesterday by Dr. Macdonald, states in his article on lung cancer which appears in Cancer magazine, a magazine published by the American Cancer Society, Dr. Steiner says that lung cancer is such that 2 out of every 100 babies born will develop lung cancer between the ages of 50 and 80, smoking or no smoking. That number will be almost exactly, or approximately what they are giving you here. But why do they say that lung cancer is produced by cigarettes when it is a natural trait for persons to have it anyway?

I say that 100 million American citizens have been scared to death by this type of reporting which is inspired, statistical maneuvering to create an impression about a situation which does not exist and I am attacking this as a statistician against another statistician. I say as a statistician they are twisting figures to create a case.

Mr. BLATNIK. As the statistician, then, do you have figures that will prove that a given percentage of babies born will have cancer? Are there statistics collected on that?

Mr. GREENHOUSE. I only quote Dr. Steiner, who is an international authority on cancer.

Mr. BLATNIK. Is he a statistician? I thought you were interpreting statistics.

Mr. GREENHOUSE. He is a statistician to the extent that he says 2 out of 100 babies born every year—that is statistics——

Mr. BLATNIK. Was that based on a survey?

Mr. GREENHOUSE. That is based on Dr. Steiner's survey which was in part subsidized by the American Cancer Society, itself, which is the sponsor of this report. They knew of Dr. Steiner's statistics when they came out with this.

Now, I am not employed by the tobacco interests. I don't smoke. I have no other interest than to say 100 million American people have a right to be protected from this kind of specialized, what you might call inspired type of work—they wanted to show, I suppose, that they are doing something for the millions they checked. God bless them, I subscribe to them, but they ought to wait for laboratory findings. The adding machine and calculator should not replace the microscope and test tubes. It never can.

Mr. BLATNIK. Thank you very much, Mr. Greenhouse.

The committee is adjourned.

(Whereupon, at 12:25 p. m., the subcommittee adjourned to reconvene at the call of the chairman.)

APPENDIX

EXHIBIT 1

SMOKING IN RELATION TO DEATH RATES[1]

By E. Cuyler Hammond, Sc. D. and Daniel Horn, Ph. D.

Today we will present an analysis of death rates in relation to the smoking habits of 187,783 men who have been traced for an average of 44 months.

The first results of the study were presented when the subjects had been traced for 20 months. The major findings at that time were:

1. The death rate of cigarette smokers was far higher than the death rate of men who had never smoked cigarettes.
2. Deaths ascribed to cancer accounted for about one-quarter of the excess deaths among cigarette smokers; and deaths ascribed to coronary artery disease accounted for over one-half of the excess.

The study was continued for another 2 years as a check on the earlier findings and to obtain more detailed information on many points of interest, such as the effect of giving up smoking.

The new information fully confirms the earlier findings as outlined above.

We will first describe the procedures; then the findings in relation to the total death rates; then discuss some of the checks we have made on the accuracy of the data; and then describe the findings in relation to specific causes of death.

PROCEDURES

After designing and pretesting a smoking questionnaire, we trained over 22,000 American Cancer Society volunteers as researchers for the study. Each researcher was asked to get a smoking questionnaire filled out by about 10 white men between the ages of 50 and 69 whom she knew well and would be able to trace. The researchers were told not to enroll a man if he was seriously ill or if they knew he had lung cancer. Once a year thereafter, they reported on each man as "alive," "dead," or "don't know" and recorded all changes of address. A copy or abstract of the death certificate was obtained on each death reported. Whenever cancer was mentioned on a death certificate, further information was sought from the doctor, hospital, or tumor registry. The study area included 394 counties in 9 States: California, Illinois, Iowa, Michigan, Minnesota, New Jersey, New York, Pennsylvania, and Wisconsin.

POPULATION STUDIED

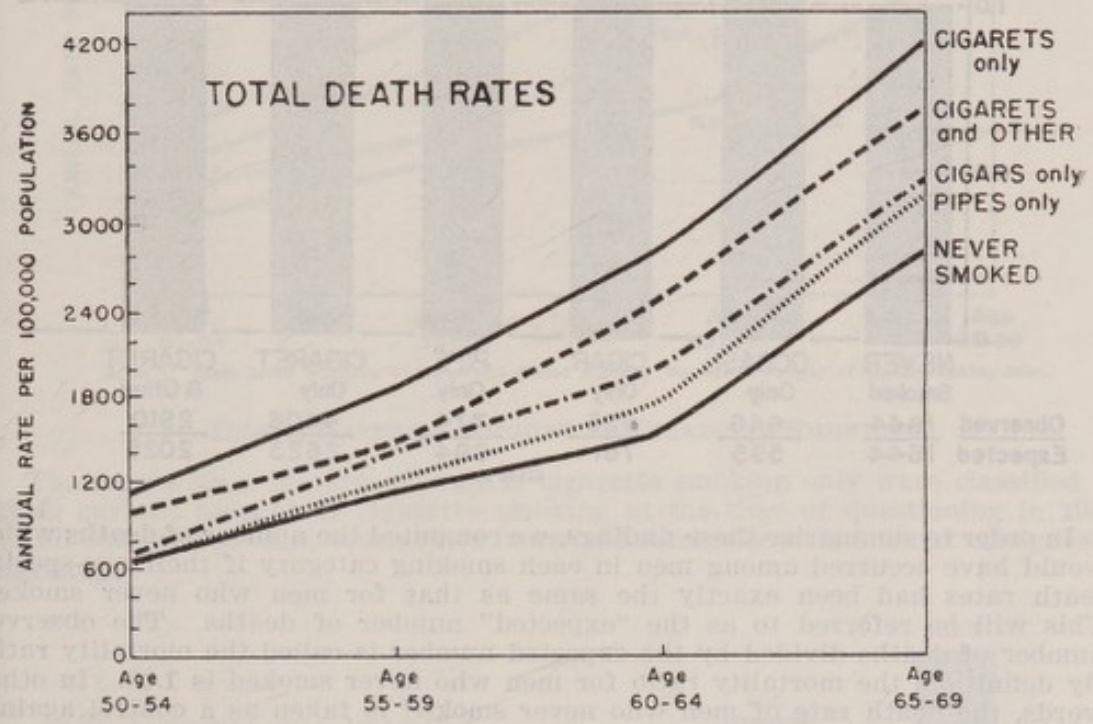
The questioning of subjects began on January 1, 1952, and continued until the summer of that year. The follow-up procedures were started on November 1 of each year from 1952 through 1955. The subjects still alive at the end of this time had been traced for an average of 44 months.

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[1] A paper read at the annual meeting of the American Medical Association, New York City, June 4, 1957.

A total of 204,547 smoking questionnaires were obtained, but we decided to exclude the 6,288 men enrolled after May 31, 1952, and an additional 8,405 questionnaires were excluded because of incompleteness of information, duplication, or administrative difficulties. This left a net total of 189,854 subjects; 187,783 of them were traced by the volunteers through October 1955 and 11,870 deaths were reported. The total experience covered 667,753 man-years.

Sixty and five-tenths percent of the men in age 55 to 59 had a history of regular cigarette smoking. As reported by Haenszel, Shimkin, and Miller, 58 percent of the men in age group 55 to 64 were found to have a history of regular cigarette smoking in a survey conducted by the Bureau of the Census in February 1955.

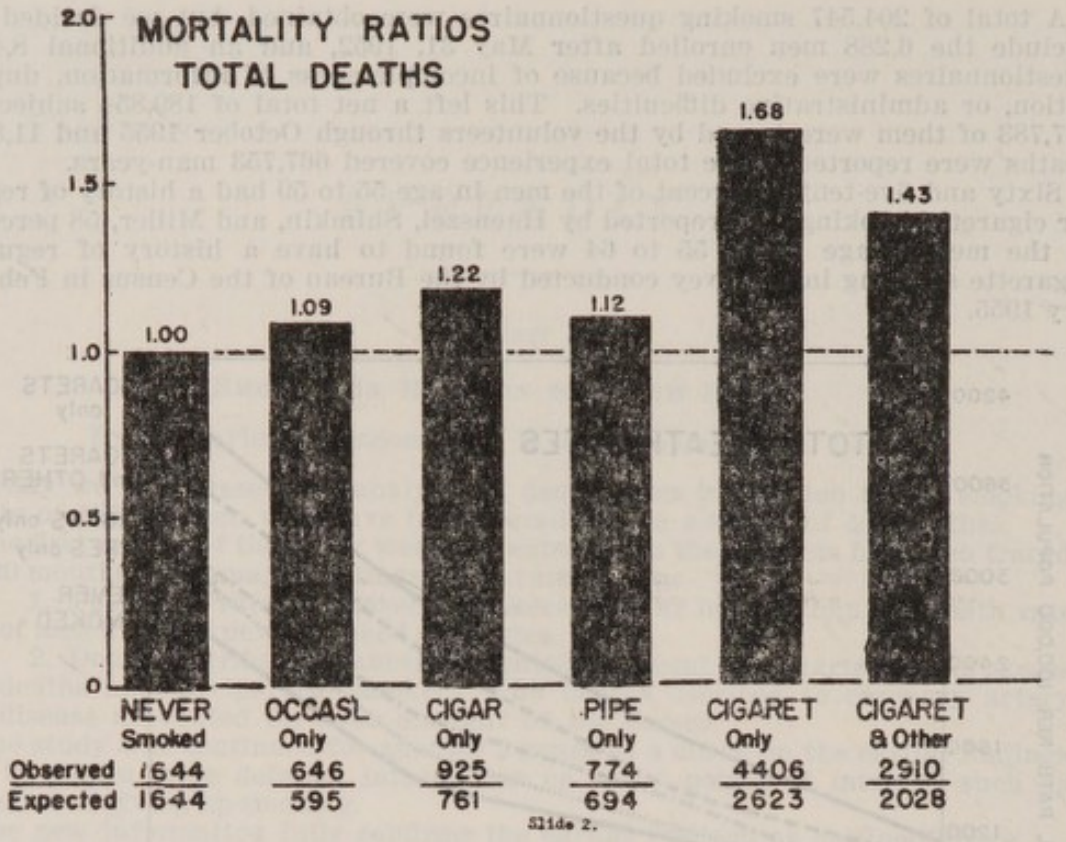


Slide 1. Total Death Rates by Type of Smoking (Lifetime History) and by Age at Start of Study

DEATH RATES BY TYPE OF SMOKING

This slide shows death rates per 100,000 man-years by type of smoking for each of 4 age groups. Age, as shown here and elsewhere in this report, refers to the ages of the men at the time they were questioned in 1952.

Note that, in all four age groups, by far the highest death rate was that for men with a history of regular cigarette smoking only. Men who never smoked had the lowest death rate. Men with a history of regular cigarette smoking who also smoked cigars and pipes had death rates somewhat lower than the death rates of men who smoked cigarettes only. The death rates of men who had only smoked pipes were just slightly above the rates for men who never smoked. The death rates of cigar smokers were slightly higher than those for pipe smokers.



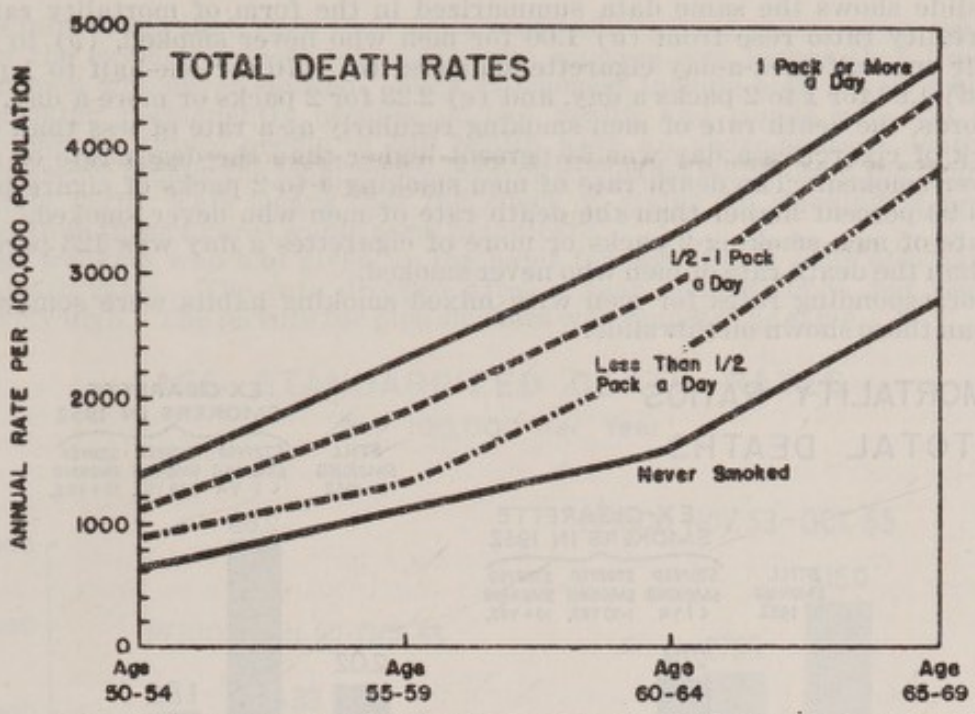
In order to summarize these findings, we computed the number of deaths which would have occurred among men in each smoking category if their age-specific death rates had been exactly the same as that for men who never smoked. This will be referred to as the "expected" number of deaths. The observed number of deaths divided by the expected number is called the mortality ratio. By definition, the mortality ratio for men who never smoked is 1.00. In other words, the death rate of men who never smoked is taken as a control against which the death rate of men in various smoking categories is compared.

Four thousand four hundred and six deaths occurred among men with a history of regular cigarette smoking only. Just 2,623 of these men would have died between January 1952 and October 1955 had their age-specific death rates been the same as for men who never smoked. The mortality ratio is 1.68. In other words, the death rate of these cigarette smokers was 68 percent higher than the death rate of a comparable group of men who never smoked, age being taken into consideration.

The mortality ratio was 1.43 for men with a history of regular cigarette smoking who also smoked cigars or pipes. It is interesting that these men with mixed smoking habits had somewhat lower death rates than men who smoked cigarettes only. This is partly due to the fact that there were fewer heavy cigarette smokers among those with mixed habits than among those who smoked cigarettes only. However, this does not entirely account for the difference.

The mortality ratios were 1.22 and 1.12, respectively, for men with a history of cigar smoking only and for men with a history of pipe smoking only. In both instances, the difference between the observed and expected number of deaths is statistically significant. However, the effect of pipe smoking seems to be small as compared with the effect of cigarette smoking.

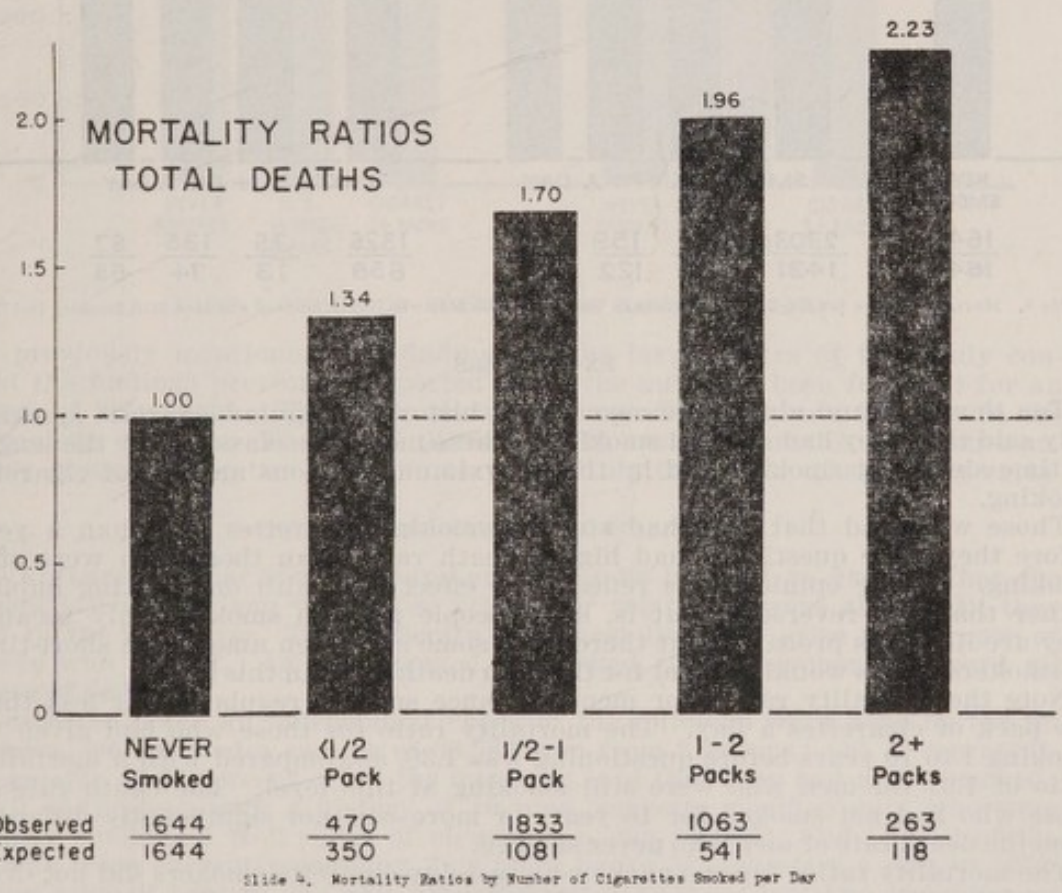
Six hundred and forty-six deaths occurred among men with a history of occasional smoking only as compared with 595 expected. This difference is not statistically significant. Therefore, it appears that occasional smoking has little or no effect on death rates.



Slide 3. Death Rates by Number of Cigarettes Smoked per Day. Men with a History of Regular Smoking Only.

Death Rates by Amount of Cigarette Smoking

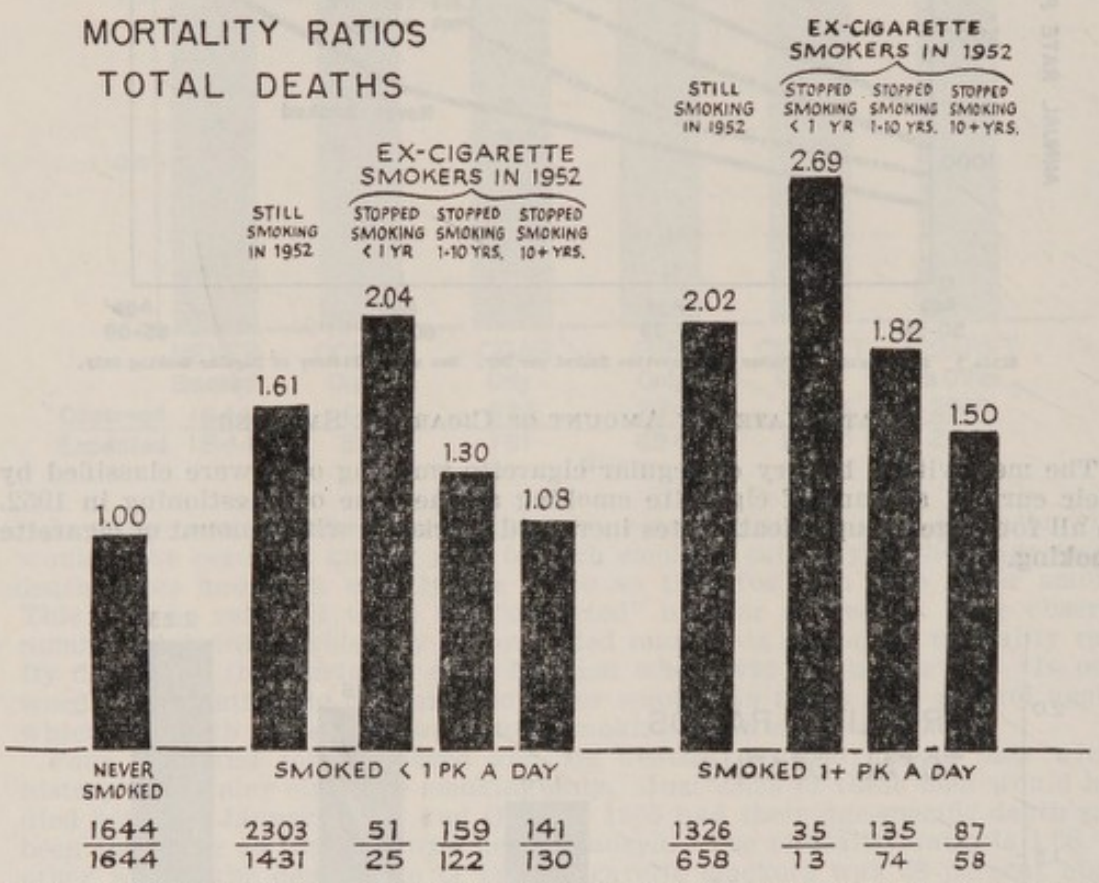
The men with a history of regular cigarette smoking only were classified by their current amount of cigarette smoking at the time of questioning in 1952. In all four age groups, death rates increased markedly with amount of cigarette smoking.



Slide 4. Mortality Ratios by Number of Cigarettes Smoked per Day

This slide shows the same data summarized in the form of mortality ratios. The mortality ratio rose from (*a*) 1.00 for men who never smoked, (*b*) to 1.34 for under one-half-pack-a-day cigarette smokers, (*c*) 1.70 for one-half to 1 pack a day, (*d*) 1.96 for 1 to 2 packs a day, and (*e*) 2.23 for 2 packs or more a day. In other words, the death rate of men smoking regularly at a rate of less than one-half pack of cigarettes a day was 34 percent higher than the death rate of men who never smoked. The death rate of men smoking 1 to 2 packs of cigarettes a day was 96 percent higher than the death rate of men who never smoked. The death rate of men smoking 2 packs or more of cigarettes a day was 123 percent higher than the death rate of men who never smoked.

The corresponding rates for men with mixed smoking habits were somewhat lower than those shown on this slide.



Slide 4. Mortality Ratios for Ex-Cigarette Smokers Compared with Men who Never Smoked and Men Still Smoking in 1952

EX-SMOKERS

Ten thousand and ninety-five men with a history of regular cigarette smoking only said that they had stopped smoking. These men were classified by the length of time since last smoking and by their maximum previous amount of cigarette smoking.

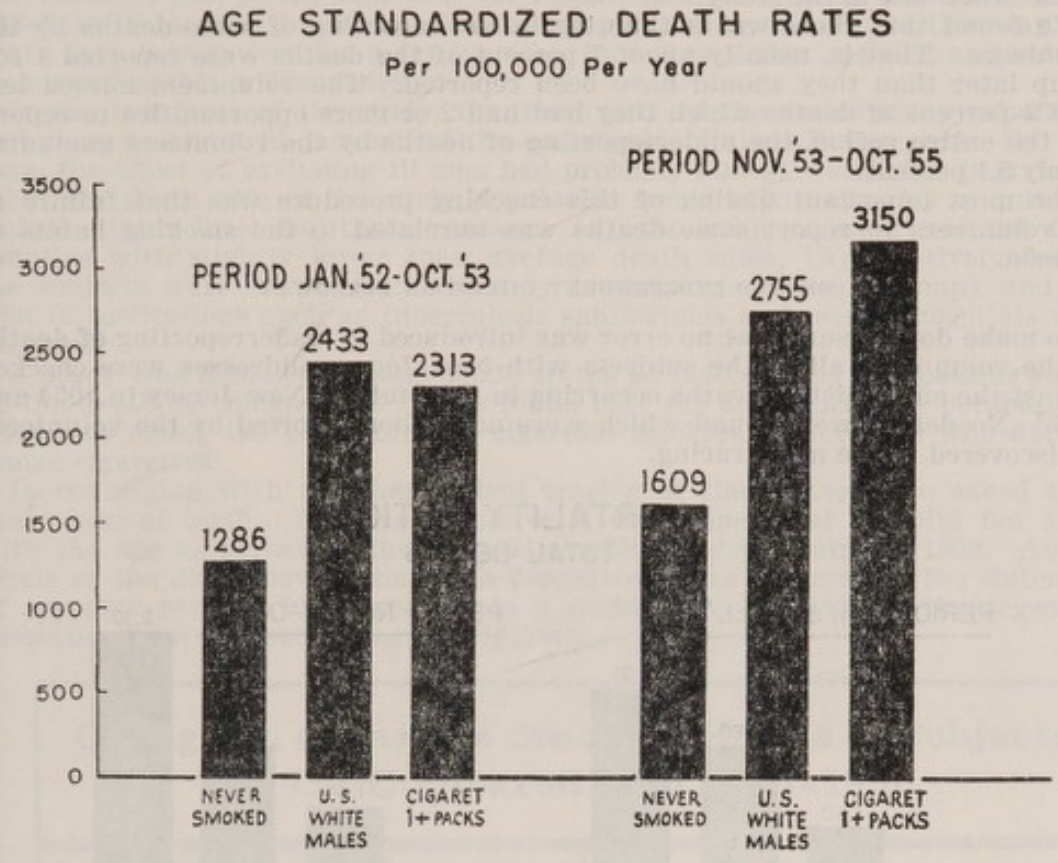
Those who said that they had stopped smoking cigarettes less than a year before they were questioned had higher death rates than those who were still smoking. In our opinion, this reflects the effect of health on smoking habits, rather than the reverse. That is, some people give up smoking only because they are ill. It is probable that there were some such men among the short-time ex-smokers. This would account for the high death rates in this group.

Note the mortality ratios for men who once smoked regularly but less than one pack of cigarettes a day. The mortality ratio for those who had given up smoking 1 to 10 years before questioning was 1.30, as compared with a mortality ratio of 1.61 for men who were still smoking at this level. The death rate of those who had not smoked for 10 years or more was not significantly different from the death rate of men who never smoked.

The mortality ratios for one-pack-or-more-a-day cigarette smokers did not drop so rapidly after giving up smoking. Nevertheless, the mortality ratio of those who had not smoked for 10 years or longer was only 1.50, as compared with a mortality ratio of 2.02 for men still smoking a pack or more of cigarettes a day.

It is interesting that, even 10 or more years after giving up smoking, the death rate of the ex-pack-or-more-a-day cigarette smokers was higher than the death rate of men who never smoked. Such ex-smokers include both people who stopped smoking permanently because of bad health and people who did so for other reasons. Since people in bad health have an above-average death rate, their presence among the longtime ex-cigarette smokers would keep the death rate relatively high. This means that the effect of giving up smoking is probably greater than these figures would seem to indicate.

Cigar smokers who had given up the habit for less than a year also had very high death rates. The rate dropped after a year of abstinence, but remained relatively high. The picture for pipe smokers was roughly the same.



LAST 2 YEARS OF STUDY VERUS EARLIER PERIOD

As previously mentioned, the findings in the last 2 years of the study confirmed the findings previously reported when the men had been followed for an average of 20 months. This slide shows a comparison between findings in the two periods of time. The small differences in mortality ratios between comparable groups in the two periods are well within the limits of sampling variation.

INDEPENDENT CHECK ON FOLLOWUP

We anticipated that some researchers might be inclined to assume that the subjects (most of whom were close friends or relatives) were alive until they heard to the contrary. This difficulty was most likely to arise in the case of subjects who moved. As a safeguard, we asked the researcher to record all changes of address.

In order to make an independent check of the followup work done by the researchers, we selected a sample of 38,583 men from 3 groups: (*a*) A representative sample of 60 percent of all the men who said that they had never smoked; (*b*) all of the men with a history of regular cigarette smoking only who were currently smoking 1 to 2 packs of cigarettes a day in 1952; and (*c*) all of the men who were currently smoking 2 or more packs of cigarettes a day in 1952. For the sake of brevity, we will hereafter refer to these last two groups combined as "heavy cigarette smokers." In the first week of November 1955, we mailed a brief questionnaire (asking current cigarette-smoking habits and date of birth) to all of these subjects except those whom we knew to be dead. The

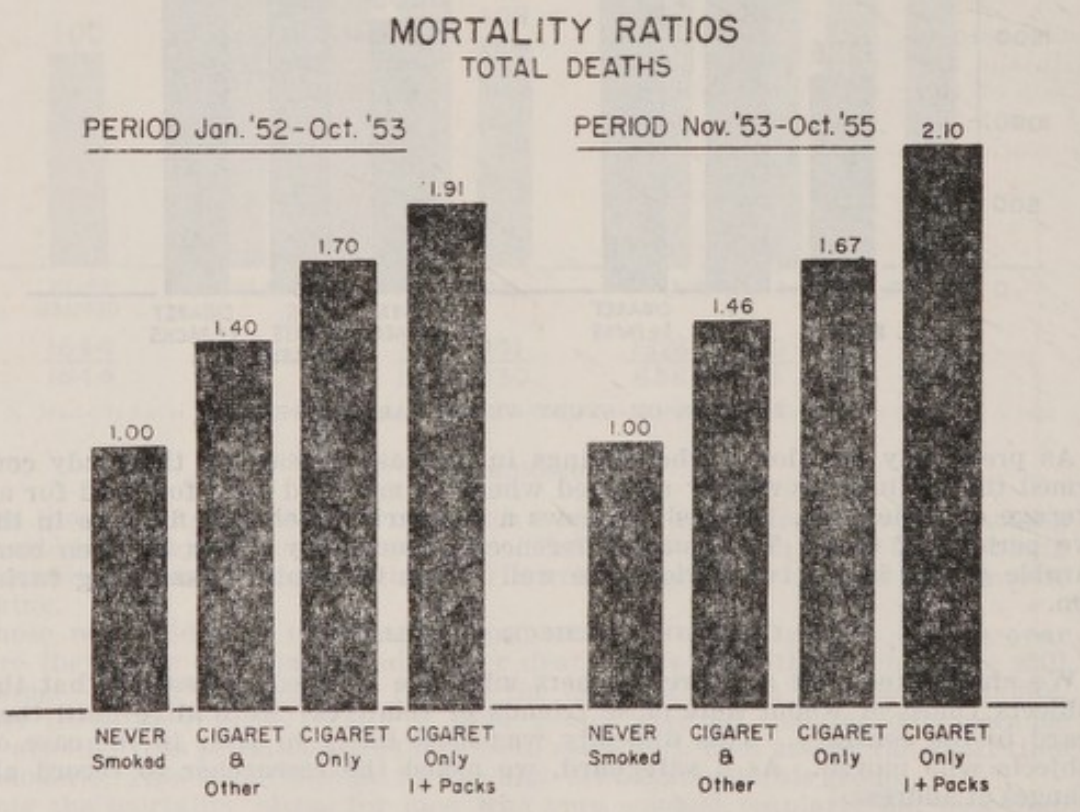
following request was printed on the face of the questionnaire: "If the man whose name appears on the other side of this card is dead, check here and return this card." In order to obtain as complete coverage as possible, five successive mailings were used, the last being certified mail requiring that the addressee sign a receipt card, which was returned to us by the post office. By the end of this mailing process, we had obtained information on all but 2,135 of the 38,583 men in the sample. Records on these 2,135 men were sent to the respective divisions of the American Cancer Society for further checking, and 1,428 of them were found. Thus, 37,876 of the men were successfully traced, this being 98.2 percent of the sample. Entirely independently of this, the volunteers sent us routine followup reports on these men, together with reports on all the other men in the study.

We found that there was a time lag in the reporting of some deaths by the volunteers. That is, usually about 7 percent of the deaths were reported 1 followup later than they should have been reported. The volunteers missed less than 3 percent of deaths which they had had 2 or more opportunities to report. For the entire period, the underreporting of deaths by the volunteers amounted to only 5.1 percent.

The most important finding of this checking procedure was that failure of the volunteers to report some deaths was unrelated to the smoking habits of the men.

SECOND INDEPENDENT CHECK ON FOLLOWUP

To make doubly sure that no error was introduced by underreporting of deaths by the volunteers, all of the subjects with New Jersey addresses were checked against the entire list of deaths occurring in the State of New Jersey in 1954 and 1955.[2] No deaths were found which were not either reported by the volunteers or discovered in the mail-tracing.



Slide 7.

This slide shows the age standardized death rates of the men who never smoked and the heavy cigarette smokers included in the special sample traced by mail as previously described. The period from January 1952 through October 1953 is compared with the period November 1953 through October 1955. Also

[2] This was accomplished through the cooperation of Dr. Marguerite F. Hall, director, Division of Vital Statistics and Administration, New Jersey State Department of Health.

shown are United States white male death rates in the same periods of time suitably adjusted for the aging of our study population.

In both periods, the death rate of the men who never smoked was about one-half the death rate of the heavy cigarette smokers. Actually, the mortality ratio—heavy cigarette smokers compared with men who never smoked—was somewhat higher in the last 2 years of the study than in the earlier period.

In the first period the death rate of the heavy cigarette smokers in the study was 4.9 percent lower than the United States average. However, in the last 2 years the death rate of the heavy cigarette smokers was 14.3 percent higher than the United States average. The death rates of both the smokers and the non-smokers in the study rose, with time, relative to the United States rates. We attribute this to the fact that the researchers were instructed not to enroll seriously ill men in the study. When a population is selected in this way, the death rate is very low for a few months, increases rather rapidly, and returns to normal within a few years.

In the last year of the study, the death rate of the entire study population had risen to about 81 percent of the comparable United States rate. By that time, the effect of excluding ill men had probably almost worn off. It is doubtful if the death rate of the study population would eventually rise to quite the United States level for three reasons: (*a*) the subjects were selected from counties with slightly lower than average death rates, (*b*) relatively few of the subjects were drawn from the very lowest socioeconomic groups, and (*c*) men in institutions such as tuberculosis sanitoriums and mental hospitals were excluded.

A study was made of socioeconomic status as indicated by occupations stated on the death certificates and it was found that the distribution by occupational level was about the same for the cigarette smokers as for men who did not smoke cigarettes.

In connection with the independent tracing of the subjects, we asked them their date of birth. In some instances it was found that this did not agree with the age as stated on the original smoking questionnaire in 1952. An analysis of the data showed that such discrepancies as occurred in the statement of age may perhaps have resulted in a slight under-estimate of the degree of asociation between death rates and cigarette smoking.

Change in Cigarette Smoking Habits of Subjects 1952 Compared with 1955-1956

Current Cigarette Smoking in 1952	% Smoking Cigarettes Regularly 1955-1956	% Not Smoking Cigarettes Regularly 1955-1956
Never Smoked	0.9%	99.1%
Smoked, But Not Cigarettes	2.3%	97.7%
None (Ex-Smoker)	7.2%	92.8%
Occasional (Ex-Regular)	26.4%	73.6%
Less than 1/2 a Day	63.8%	36.2%
1/2 to 1 Pack a Day	78.4%	21.6%
1 plus Packs a Day	86.2%	13.8%

Slide 8. Cigarette Smoking in 1955-1956 Compared with Cigarette Smoking of Same Men As Reported in 1952.

REQUESTIONING OF SUBJECTS

Between November 1955 and January 1956, we sent a mail questionnaire on current cigarette smoking habits to a sample of over 45,000 of the subjects (including all those traced by mail plus several other groups).

Less than 1 percent of the men who in 1952 said that they had never smoked said that they were smoking cigarettes regularly in 1955–56. On the other hand, 7.2 percent of the ex-cigarette smokers who had stopped altogether and 26.4 percent who had cut down to occassional smoking were again smoking cigarettes regularly in 1955–56; 36.2 percent of the men who previously had said that they smoked less than one-half pack a day of cigarettes in 1952; 21.6 percent of those who previously smoked one-half to 1 pack a day; and 13.8 percent of those who previously smoked a pack or more a day said that they were not smoking cigarettes regularly in 1955–56.

Of those who were smoking cigarettes regularly in 1955–56, 28 percent said that they smoked filter tip cigarettes.

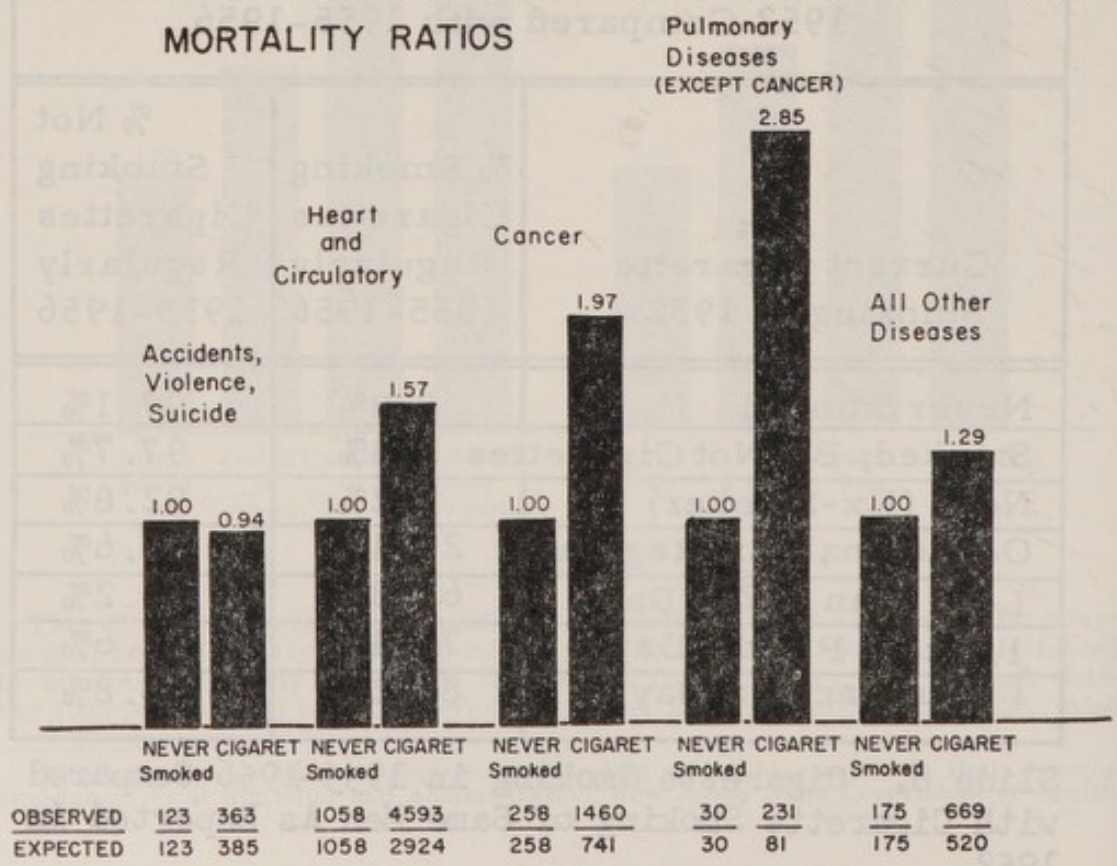
It is possible that being subjects for this study had some influence on the smoking habits of these men.

OTHER CHECKS

In retrospective studies of habits in relation to disease, the subjects are not questioned until after they become ill. Conceivably, illness could bias the way in which a person answers questions about his habits. In designing this prospective study, we sought to avoid this possibility. It was for this reaosn that we instructed the volunteers not to enroll a man if they knew he had lung cancer or if he was seriously ill from any disease. As previously described, the trends in death rates indicate that seriously ill men were largely excluded in the selection of the subjects.

Obviously, if a researcher enrolled, let us say, seven or more men with lung cancer or other fatal diseases, it is highly probable that at least 1 of them would have died within the following 2 years. Conversely, if a researcher enrolled 7 or more men and none of them died within the next 2 years, then it is improbable that she selected seriously ill men.

This fact gives us a means of eliminating men enrolled by researchers who might possibly have misunderstood instruction or deliberately selected sick men. Therefore, we made an analysis restricted to men enrolled by researchers who almost certainly did not select seriously ill subjects. The results were almost identical with the results for the study as a whole as previously described.



Slide 9. Mortality Ratios by Major Causes of Death. Cigarette Smokers Compared with Men who Never Smoked.

CAUSE OF DEATH

Having found a high degree of association between cigarette smoking and the total death rate, as well as some association between cigar and pipe smoking and the total death rate, we next sought to determine what diseases were involved. The 11,870 deaths were divided into 5 broad categories as shown on this slide.

The death rate from accidents, violence, and suicide was almost exactly the same for men with a history of regular cigarette smoking as for men who never smoked.

In contrast, 1,460 cigarette smokers died of cancer compared with an expected of only 741 deaths had their age-specific cancer death rates been the same as for men who never smoked. The mortality ratio was 1.97.

The deaths of 4,593 cigarette smokers were attributed to diseases of the heart and circulatory system as compared with 2,924 expected; a difference of 1,669 deaths. The mortality ratio was 1.57.

Only 338 of the 11,870 deaths were attributed to pulmonary diseases other than lung cancer. They showed a very high degree of association with cigarette smoking. Two hundred and thirty one deaths of cigarette smokers were attributed to these pulmonary diseases as compared with only 81 expected. The mortality ratio is 2.85.

The 338 deaths included in this category consisted of 124 attributed to pneumonia or influenza, 41 to pulmonary tuberculosis, 76 to asthma, and 97 to other pulmonary diseases including bronchitis, abscess of lung, pneumoconiosis, and bronchiectasis.

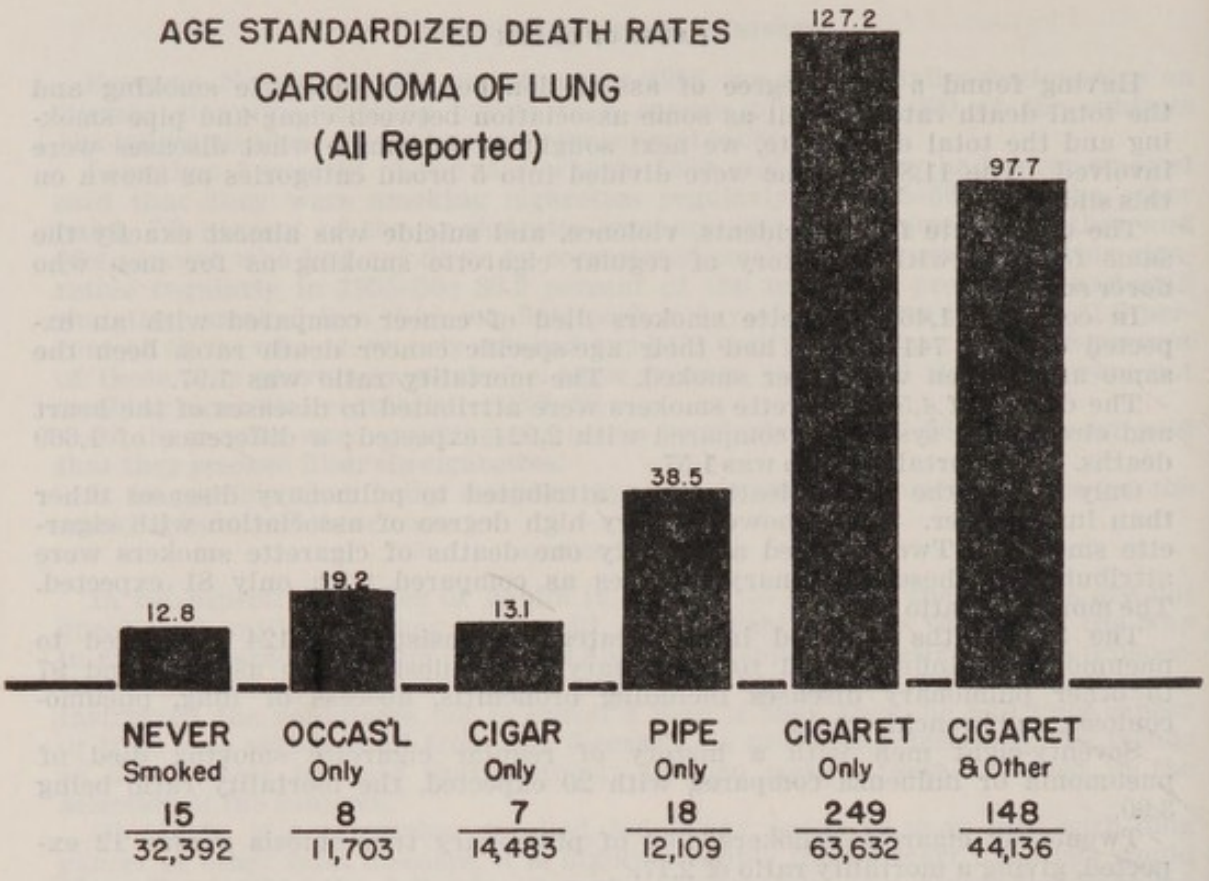
Seventy-eight men with a history of regular cigarette smoking died of pneumonia or influenza compared with 20 expected, the mortality ratio being 3.90.

Twenty-six cigarette smokers died of pulmonary tuberculosis versus 12 expected, giving a mortality ratio of 2.17.

Fifty-one cigarette smokers died of asthma versus 29 expected, giving a mortality ratio of 1.76.

Other pulmonary diseases accounted for the death of 76 cigarette smokers compared with 21 expected, the mortality ratio being 3.62.

Deaths attributed to all other causes combined (including cause of death uncertain or unknown) accounted for less than 10 percent of the 11,870 deaths. Taken together, this group showed some association with cigarette smoking as shown by the mortality ratio of 1.29. As will be shown later, a few diseases in this category account of most of this relationship.



Slide 10.

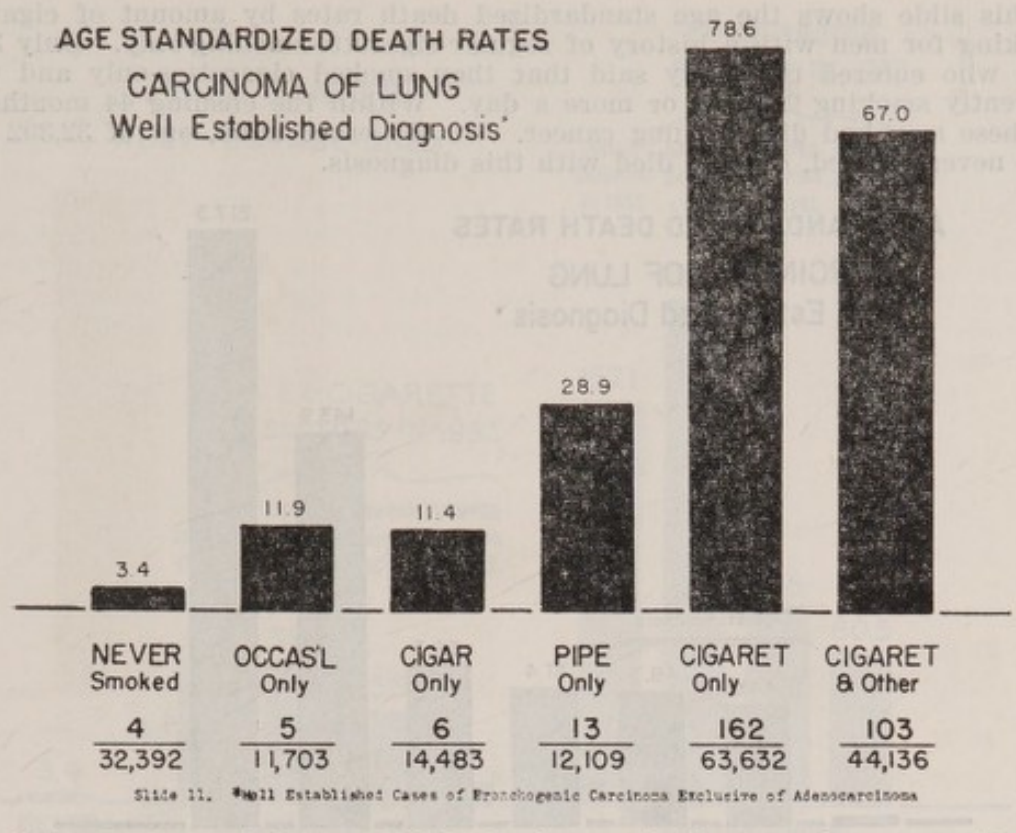
LUNG CANCER

Four hundred and forty eight deaths were attributed to primary cancer of the lung. Only 15 of these were men who never smoked. Including these 15, only 51 had never smoked cigarettes regularly whereas 397 had a history of regular cigarette smoking.

This slide [3] shows the age standardized lung cancer death rate by type of smoking. The figures at the bottom of the slide indicate the number of men enrolled in the study and the number of lung cancer deaths. The rates were very low indeed for men who never smoked, occasional smokers, and cigar smokers. Pipe smokers had an appreciably higher rate. The rate for men with a history of regular-cigarette-smoking-only was nearly 10 times as high as the rate for men who never smoked.

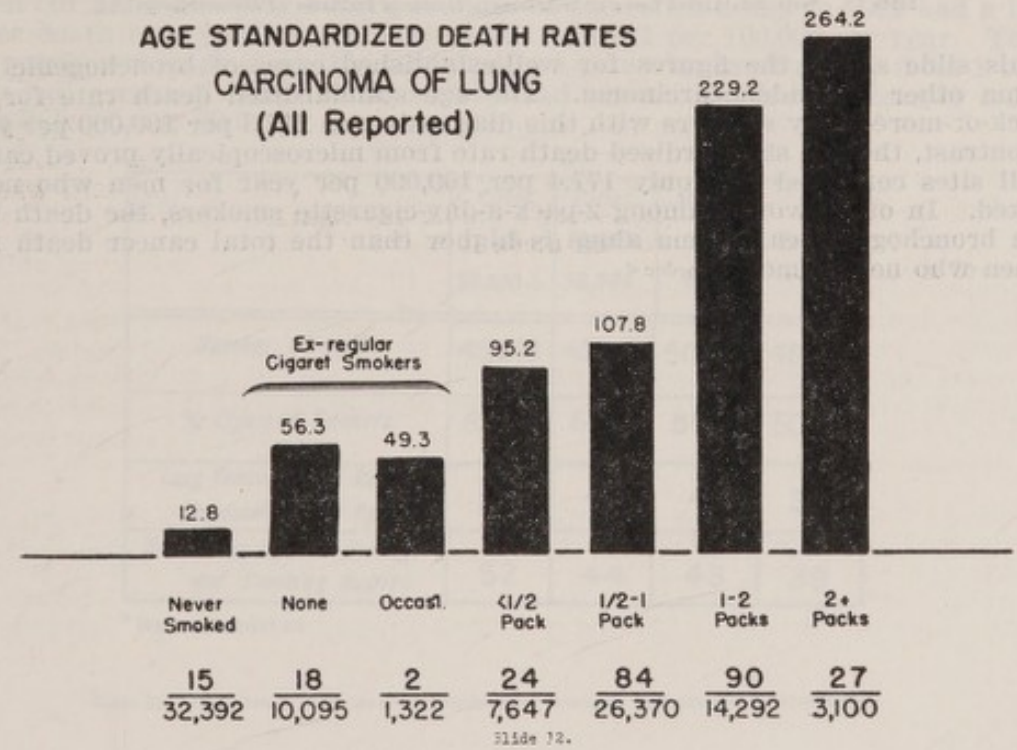
Of the 448 deaths, 32 were microscopically proved adenocarcinomas of the bronchus. These were considered separately since some investigators have expressed the opinion that this form of cancer may not be related to smoking habits; 26 of the 32 cases had a history of regular cigarette smoking, 2 never smoked, 1 was an occasional smoker, 2 were pipe smokers, and 1 a cigar smoker. The mortality ratio for the cigarette smokers was 4.33.

[3] The three lung cancer deaths of men with a history of both pipe and cigar smoking are not shown on this lantern slide.



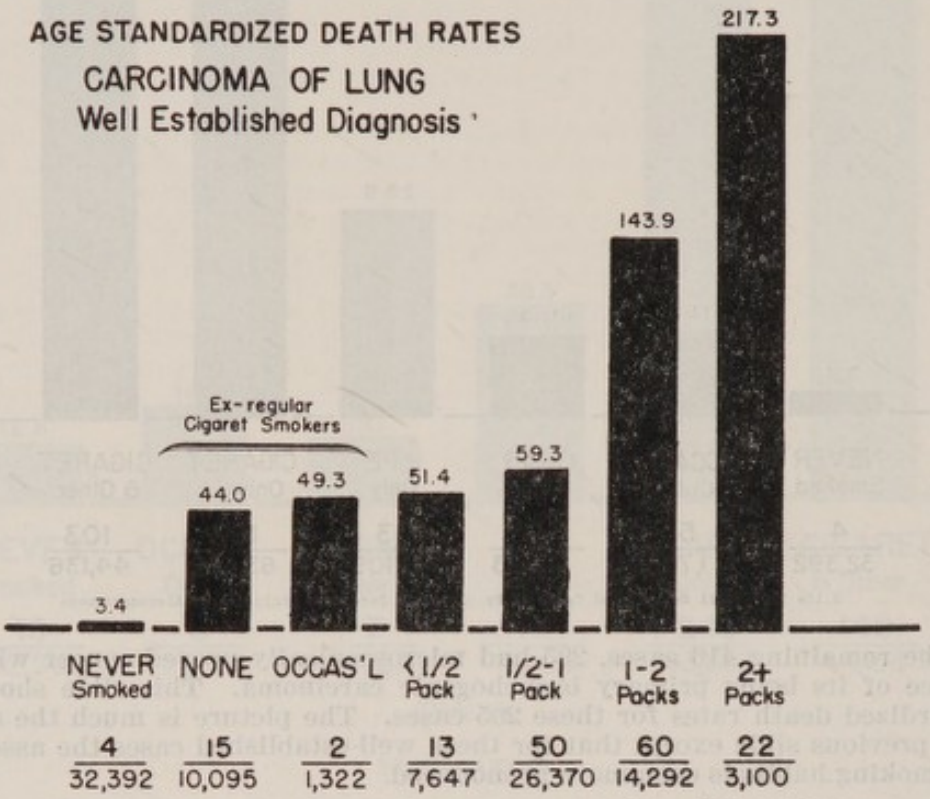
Slide 11. *Well Established Cases of Bronchogenic Carcinoma Exclusive of Adenocarcinoma

Of the remaining 416 cases, 295 had microscopically proved cancer with good evidence of its being primary bronchogenic carcinoma. This slide shows age-standardized death rates for these 295 cases. The picture is much the same as in the previous slide except that for these well-established cases the association with smoking habits is even more pronounced.



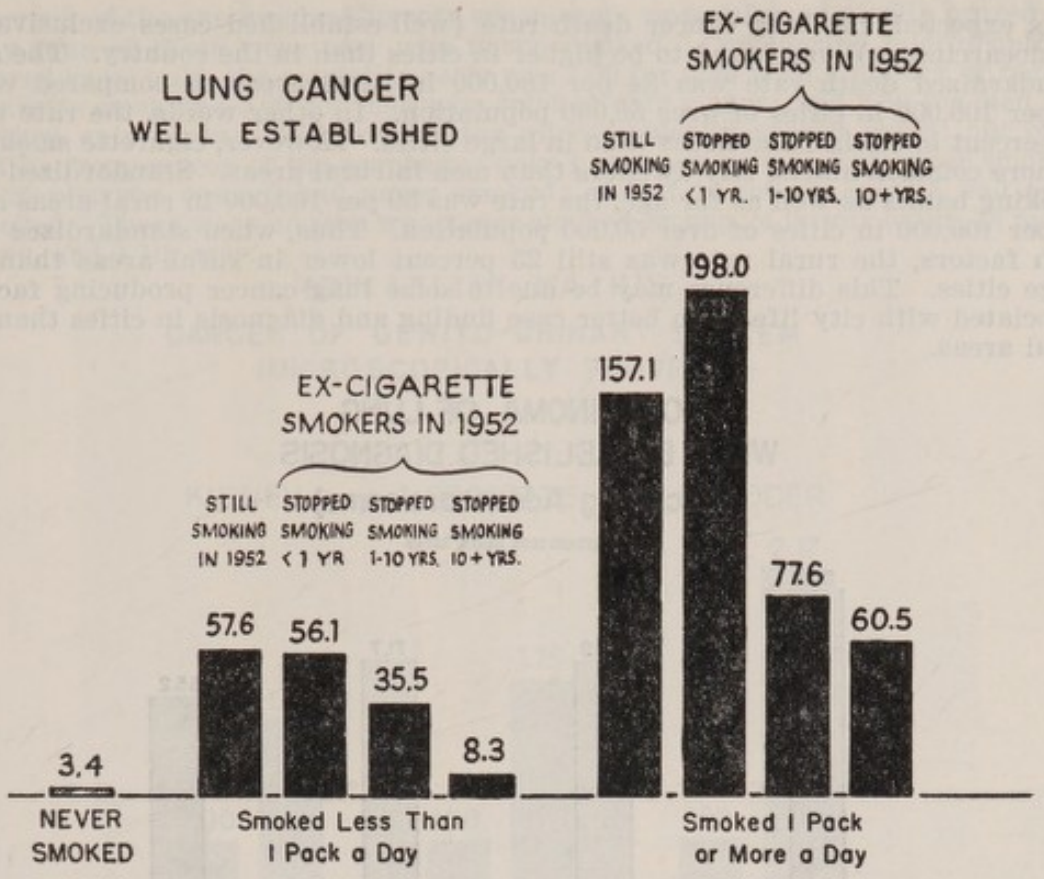
Slide 12.

This slide shows the age standardized death rates by amount of cigarette smoking for men with a history of regular-cigarette-smoking-only. Only 3,100 men who entered the study said that they smoked cigarettes-only and were currently smoking 2 packs or more a day. Within the ensuing 44 months, 27 of these men had died of lung cancer. On the other hand, out of 32,392 men who never smoked, only 15 died with this diagnosis.



Slide 13. *Well Established Cases of Bronchogenic Carcinoma Exclusive of Adenocarcinoma

This slide shows the figures for well-established cases of bronchogenic carcinoma other han adenocarcinoma. The age standardized death rate for the 2-pack-or-more-a-day smokers with this diagnosis was 217.3 per 100,000 per year. In contrast, the age standardized death rate from microscopically proved cancer of all sites combined was only 177.4 per 100,000 per year for men who never smoked. In other words, among 2-pack-a-day-cigarette smokers, the death rate from bronchogenic carcinoma alone is higher than the total cancer death rate of men who never smoked.



Those of us who are ex-very-heavy-cigarette-smokers have something of a personal interest in the lung cancer death rate of men who stopped smoking cigarettes.

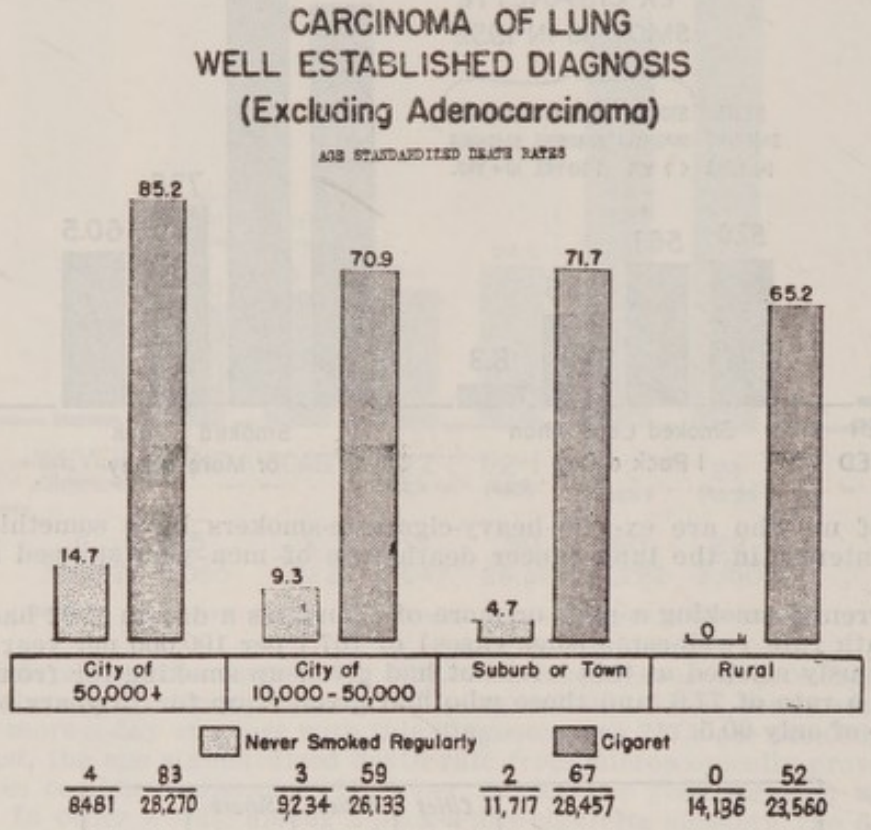
Men currently smoking a pack or more of cigarettes a day in 1952 had a lung cancer death rate (well-established cases) of 157.1 per 100,000 per year. Those who previously smoked at this level but had given up smoking for from 1 to 10 years had a rate of 77.6, and those who had given it up for 10 years or longer had a rate of only 60.5.

	Cities of 50,000+	*Cities 10,000-50,000*	*Suburb or Town*	*Rural*
Number of Men	45,218	43,502	50,039	46,783
% Cigarette Smokers	62.5%	60.1%	56.9%	50.4%
*Lung Cancer*Death Rate Standardized for Age*	56	46	43	34
Rate Standardized for Age and Smoking Habits	52	44	43	39

*Well established

Slide 15. *Well Established Cases of Bronchogenic Carcinoma Exclusive of Adenocarcinoma

As expected, the lung cancer death rate (well-established cases exclusive of adenocarcinoma) was found to be higher in cities than in the country. The age standardized death rate was 34 per 100,000 in rural areas as compared with 56 per 100,000 in cities of over 50,000 population. In other words, the rate was 39 percent lower in rural areas than in large cities. However, cigarette smoking is more common among city dwellers than men in rural areas. Standardized for smoking habits as well as for age, the rate was 39 per 100,000 in rural areas and 52 per 100,000 in cities of over 50,000 population. Thus, when standardized for both factors, the rural rate was still 25 percent lower in rural areas than in large cities. This difference may be due to some lung cancer producing factor associated with city life or to better case finding and diagnosis in cities than in rural areas.



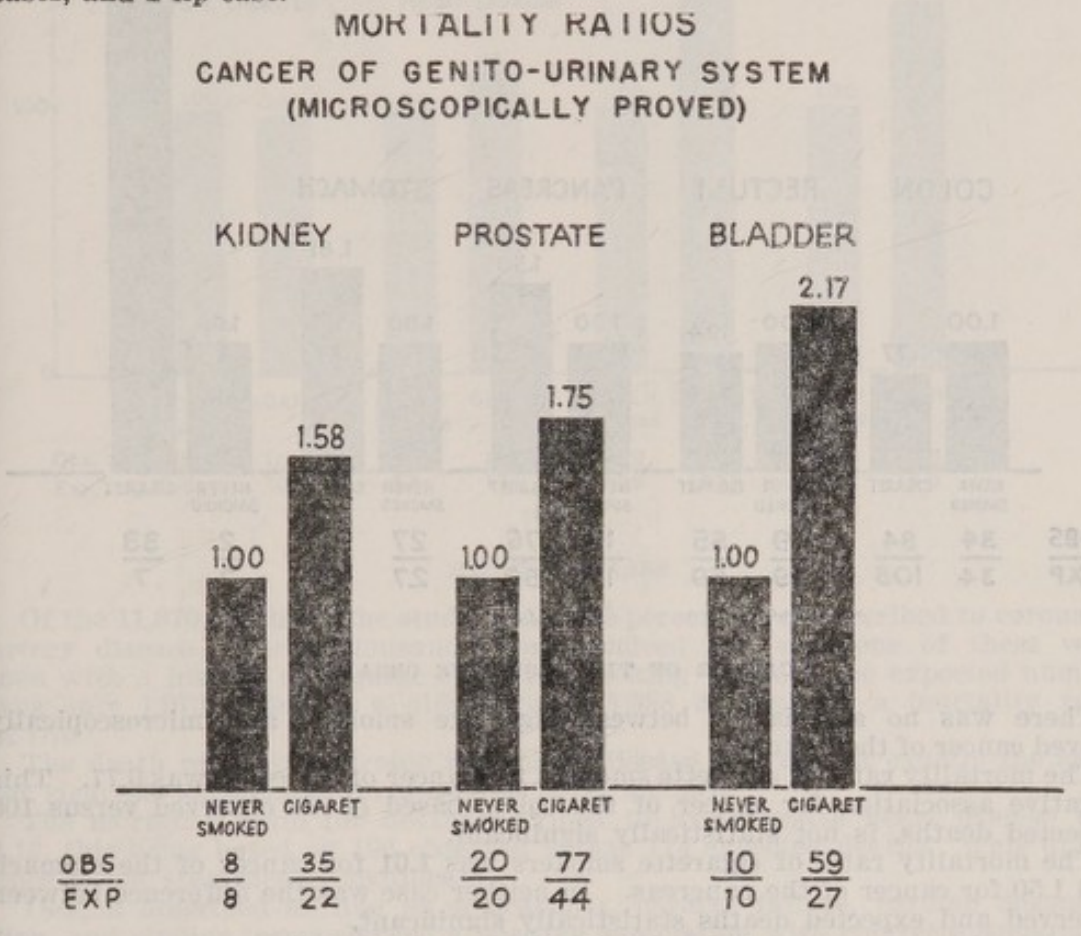
Slide 16.

However, that may be, the lung cancer death rate was low among men who never smoked cigarettes regularly and high among cigarette smokers in large cities, small cities, suburbs and towns, and rural areas. Whatever the urban factor may be, its effect on lung cancer death rates is small as compared with the effect of cigarettes as shown by the relative heights of the bars on this slide.

CANCER OF THE ESOPHAGUS, LARYNX, PHARYNX, MOUTH, TONGUE, AND LIP

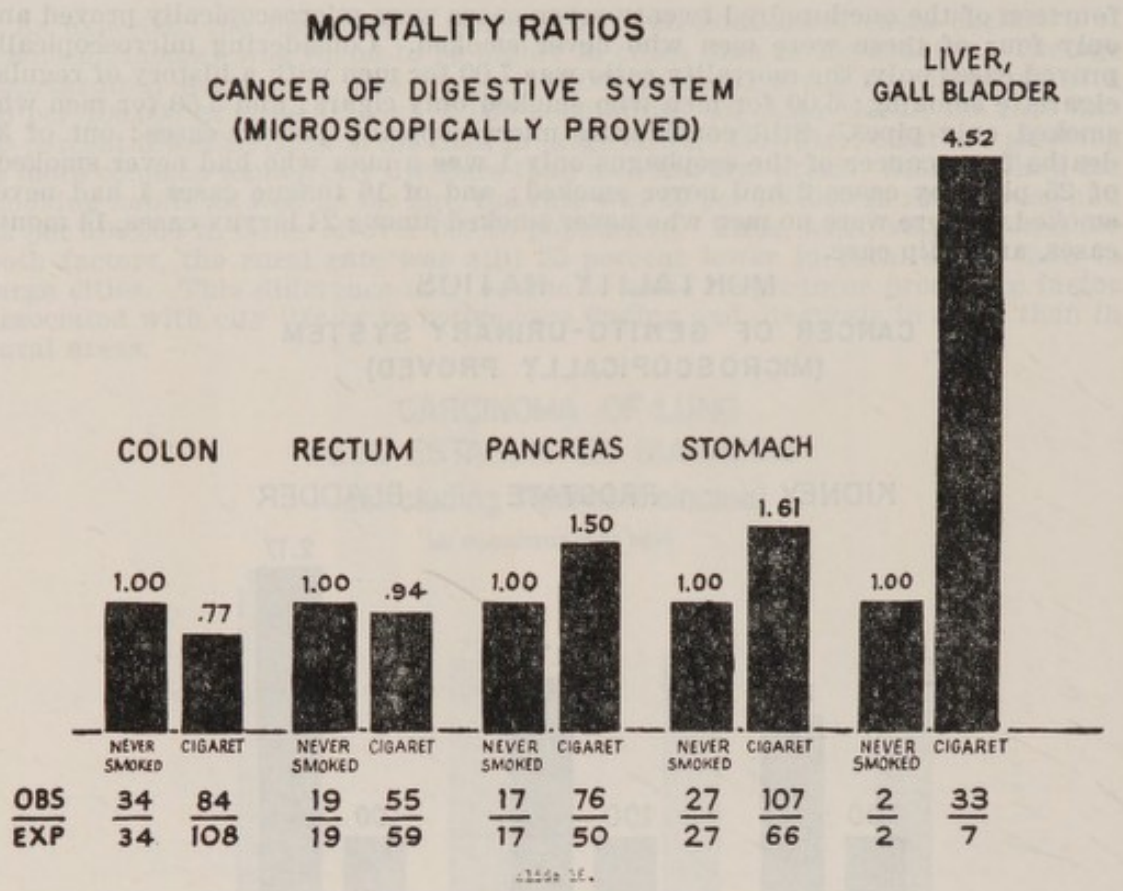
Tobacco smoke (or saliva and bronchial secretions containing material from tobacco smoke) comes into direct contact with the lips, mouth, tongue, pharynx, larynx, and esophagus. The death of 127 subjects was attributed to primary cancer of these sites. Only 6 of these men had never smoked and 3 were occasional smokers. The other 118 had a history of regular smoking. One hundred

fourteen of the one hundred twenty seven cases were microscopically proved and only four of these were men who never smoked. Considering microscopically proved cases only, the mortality ratio was 7.00 for men with a history of regular cigarette smoking; 5.00 for men who smoked only cigars; and 3.50 for men who smoked only pipes. Still considering microscopically proved cases: out of 34 deaths from cancer of the esophagus only 1 was a man who had never smoked; of 25 pharynx cases 2 had never smoked; and of 16 tongue cases 1 had never smoked. There were no men who never smoked among 24 larynx cases, 14 mouth cases, and 1 lip case.



CANCER OF THE GENITOURINARY SYSTEM

This slide shows mortality ratios for microscopically proved cancer diagnosed as primary in the genitourinary organs. The mortality ratio of cigarette smokers was 2.17 for cancer of the bladder, 1.75 for cancer of the prostate, and 1.58 for cancer of the kidney. In most of these cases, cancer was present at death in two or more of these sites, as well as in other parts of the body. While cancer was proved microscopically, the evidence as to the exact primary site was far from conclusive in many instances.



CANCER OF THE DIGESTIVE ORGANS

There was no association between cigarette smoking and microscopically proved cancer of the rectum.

The mortality ratio of cigarette smokers for cancer of the colon was 0.77. This negative association for cancer of the colon, based on 84 observed versus 108 expected deaths, is not statistically significant.

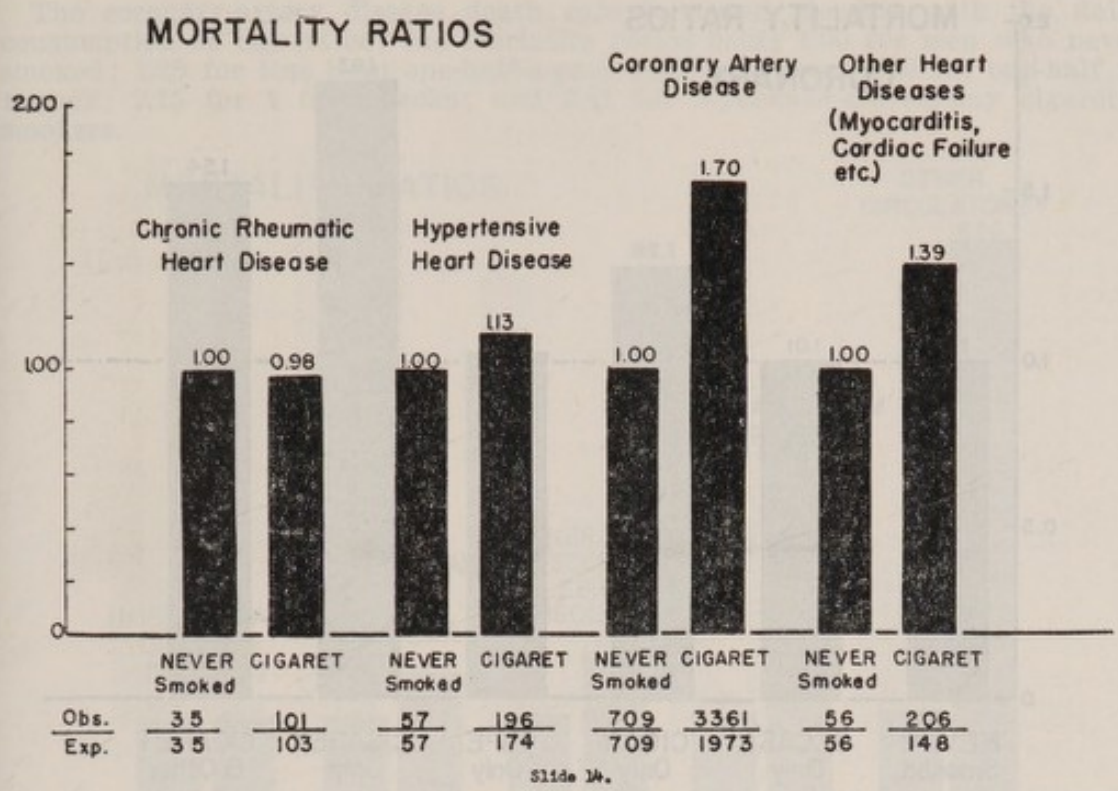
The mortality ratio of cigarette smokers was 1.61 for cancer of the stomach and 1.50 for cancer of the pancreas. In neither case was the difference between observed and expected deaths statistically significant.

Deaths from cancer of the liver, gall bladder, and biliary passages appeared to be highly associated with cigarette smoking. However, there was reasonable doubt as to the primary site in many of these cases.

CANCER OF THE LYMPHATIC AND HAEMATOPOIETIC SYSTEM

Leukemia showed no indication of an association with cigarette smoking.

Hodgkin's disease as well as lymphosarcoma and reticulosarcoma appeared to be associated with cigarette smoking, but not to a statistically significant degree.



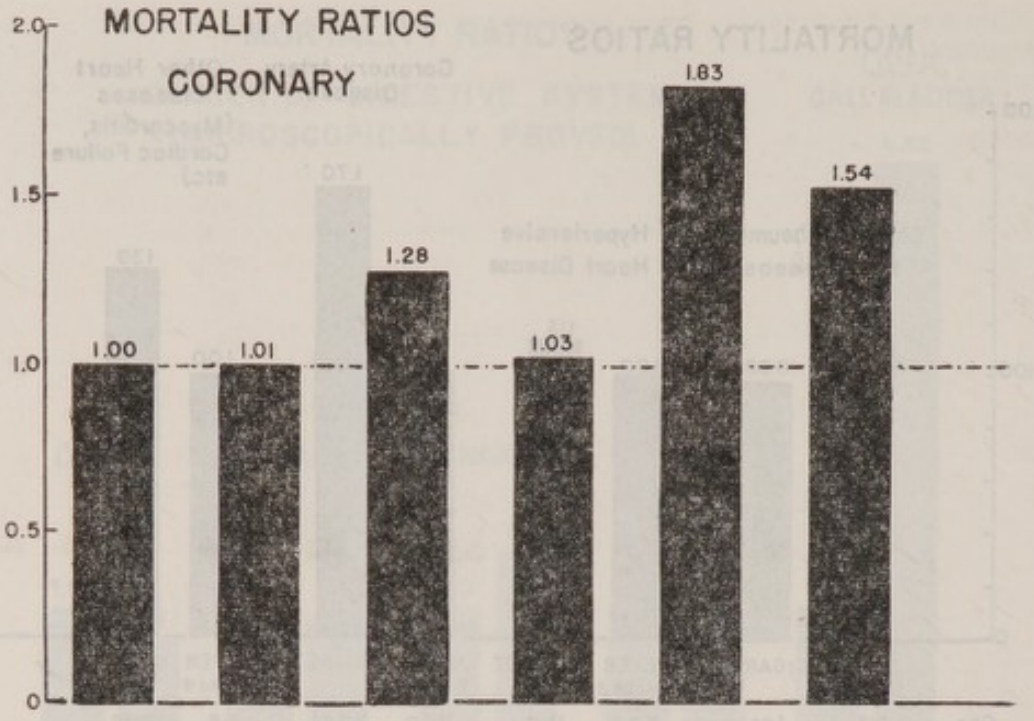
HEART DISEASES

Of the 11,870 deaths in the study, 5,297 (45 percent) were ascribed to coronary-artery disease. Three thousand three hundred and sixty-one of these were men with a history of regular cigarette smoking whereas the expected number was only 1,973. This is a difference of 1,388 deaths and a mortality ratio of 1.70.

The death rate from chronic rheumatic disease was almost exactly the same for cigarette smokers as for men who never smoked.

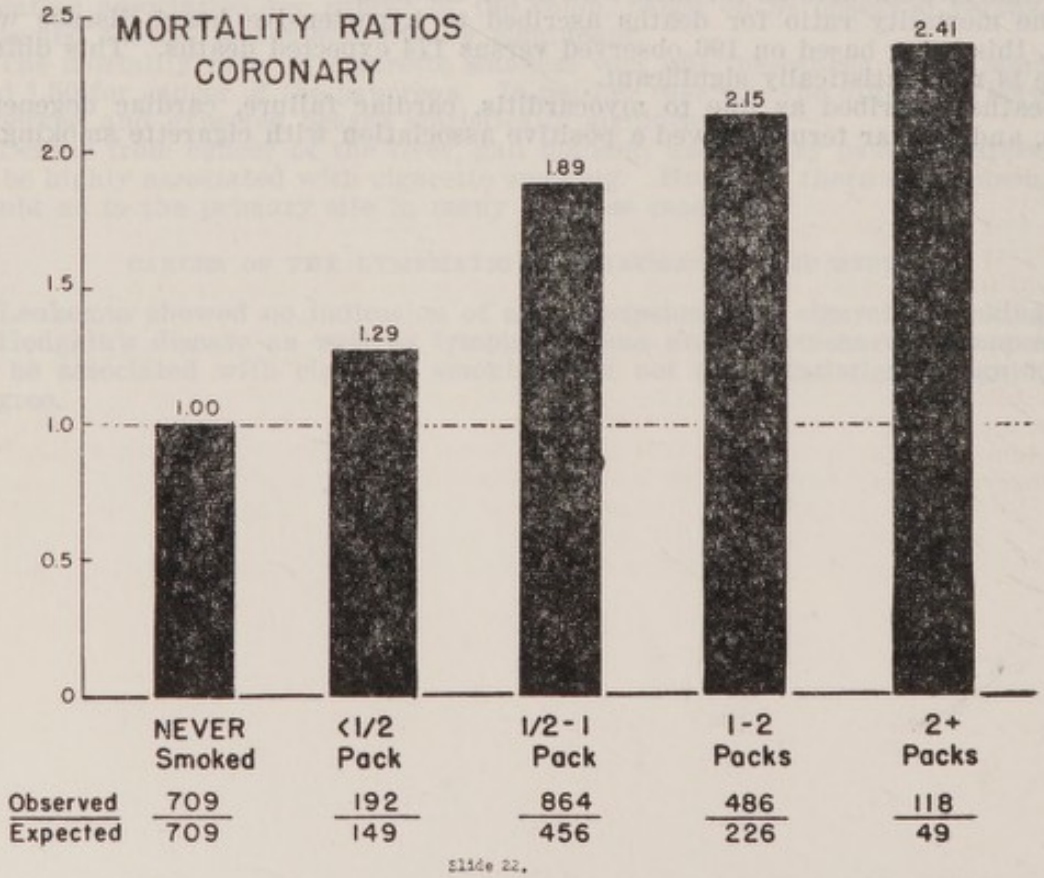
The mortality ratio for deaths ascribed to hypertensive heart disease was 1.13, this being based on 196 observed versus 174 expected deaths. This difference is not statistically significant.

Deaths described as due to myocarditis, cardiac failure, cardiac degeneration, and similar terms showed a positive association with cigarette smoking.



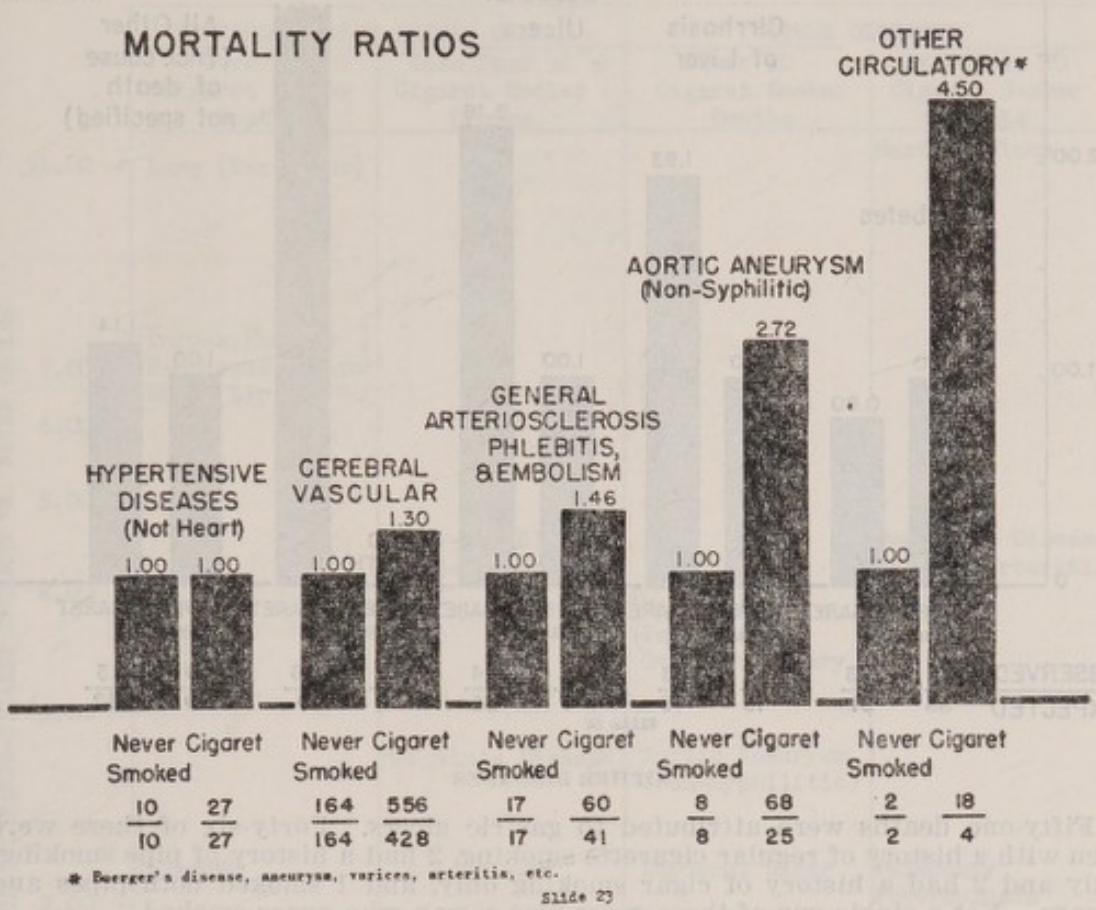
Slide 21.

The coronary artery disease death rate of pipe smokers was just about the same as for men who never smoked. However, the mortality ratio for cigar smokers was 1.28. This association is statistically significant. Men who smoked only cigarettes had by far the highest mortality ratio.



Slide 22.

The coronary-artery disease death rate increased steadily with the daily consumption of cigarettes; the mortality ratios being 1.00 for men who never smoked; 1.29 for less than one-half-a-pack-a-day smokers; 1.89 for one-half to 1 pack; 2.15 for 1 to 2 packs; and 2.41 for 2-packs-or-more-a-day cigarette smokers.



CIRCULATORY DISEASES

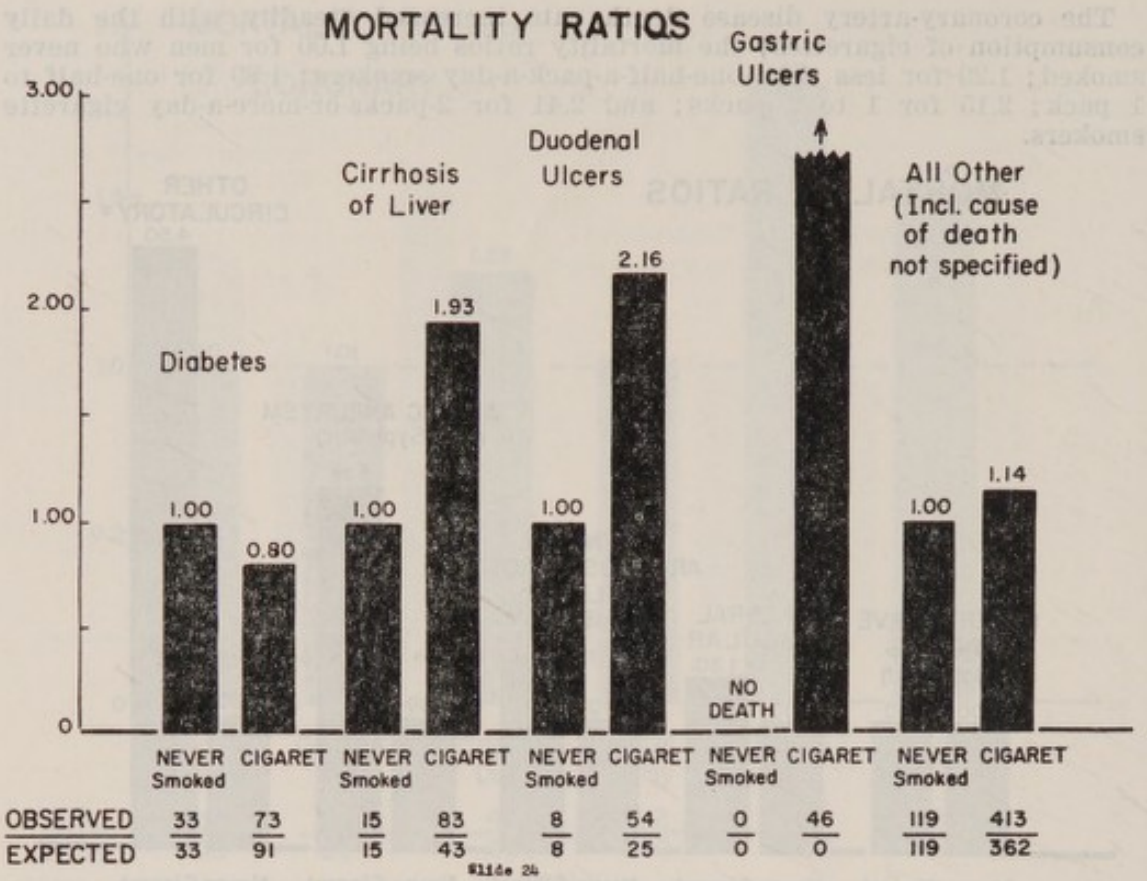
On the basis of a small number of cases, hypertensive diseases showed no indication of an association with cigarette smoking.

General arteriosclerosis, phlebitis, and embolism have been grouped together because of the small number of deaths ascribed to each. Although the mortality ratio was 1.46 for these deaths grouped together, no single 1 of these 3 diseases showed a statistically significant degree of association with cigarette smoking.

Sixty-eight cigarette smokers died of aortic aneurysm (described as non-syphilitic in origin) as compared with only 25 expected, a mortality ratio of 2.72.

A small number of deaths from Buerger's disease, aneurysm, varices, and arteritis were grouped together. They showed a high degree of association with cigarette smoking, the mortality ratio being 4.50.

A total of 1,050 deaths were ascribed to vascular lesions of the central nervous system. Five hundred and fifty-six of these deaths occurred among cigarette smokers versus 428 expected, a mortality ratio of 1.30.



OTHER DISEASES

Fifty-one deaths were attributed to gastric ulcers. Forty-six of these were men with a history of regular cigarette smoking, 2 had a history of pipe smoking only and 2 had a history of cigar smoking only, and 1 smoked both pipes and cigars. Not a single one of these cases was a man who never smoked.

Deaths attributed to duodenal ulcers were also associated with cigarette smoking but not to such a marked degree as gastric ulcers.

Eighty-three deaths to cirrhosis of the liver occurred among cigarette smokers compared with 43 expected, a mortality ratio of 1.93.

Deaths from nephritis and nephrosis showed no association with smoking.

Diabetes deaths were negatively associated with cigarette smoking but not to a statistically significant degree.

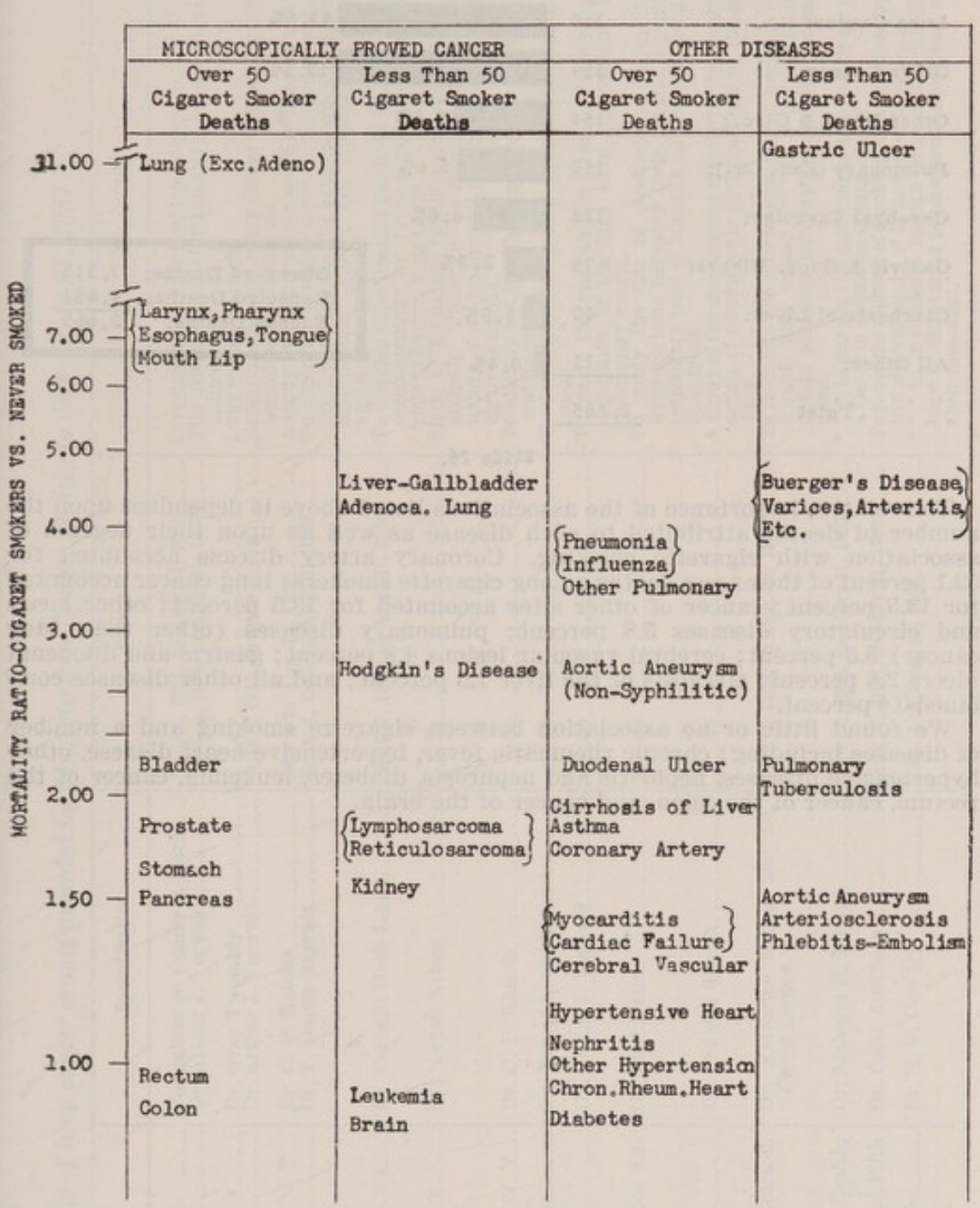
The limitations of time do not permit us to further discuss these findings. Moreover, we greatly regret that we cannot review the excellent work which has been done on this subject in this country and abroad.

SUMMARY

Our first concern was to study total death rates in relation to smoking habits. We found a high degree of association between total death rates and cigarette smoking; a far lower degree of association between total death rates and cigar smoking; and a small degree of association between total death rates and pipe smoking.

Having found a considerable relationship between cigarette smoking and total death rates, we then sought to determine what diseases were involved. Our available source of information was cause of death as recorded on death certificates supplemented by more detailed medical information on cases where cancer was mentioned.

DEGREE OF ASSOCIATION FOUND BETWEEN CIGARET SMOKING AND DEATHS FROM VARIOUS DISEASES AS INDICATED BY MORTALITY RATIOS



Slide 25

An analysis of the data showed the following relationships with cigarette smoking:

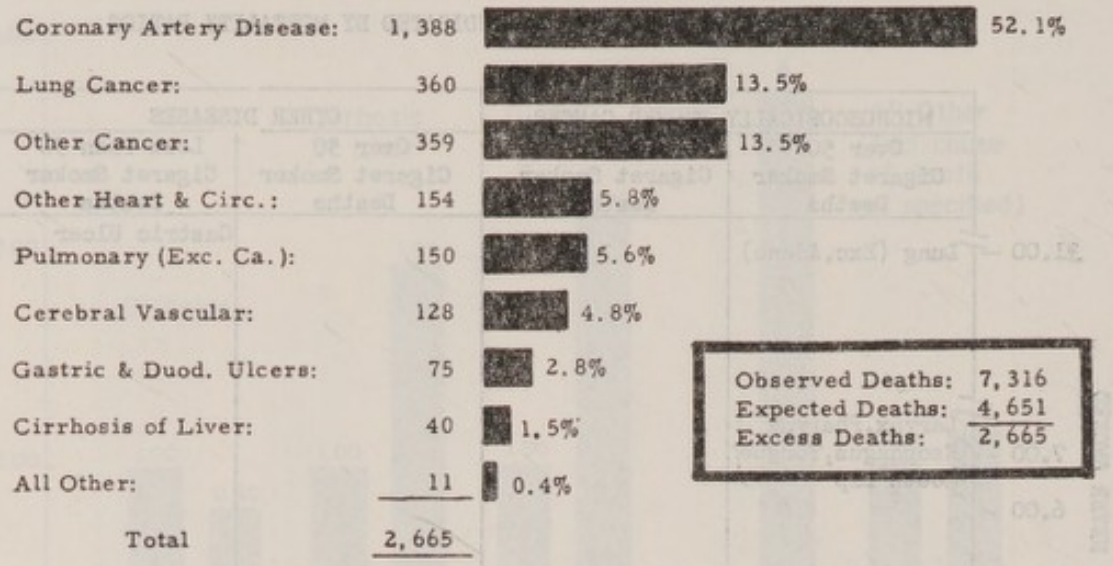
(1) An extremely high association for a few diseases such as cancer of the lung, cancer of the larynx, cancer of the esophagus, and gastric ulcers;

(2) A very high association for a few diseases such as pneumonia and influenza, duodenal ulcer, aortic aneurysm, and cancer of the bladder;

(3) A high association for a number of diseases such as coronary artery disease, cirrhosis of the liver, and cancer of several sites;

(4) A moderate association for cerebral vascular lesions.

EXCESS DEATHS AMONG MEN WITH A HISTORY OF REGULAR CIGARETTE SMOKING



Slide 26.

The relative importance of the associations listed above is dependent upon the number of deaths attributed to each disease as well as upon their degrees of association with cigarette smoking. Coronary artery disease accounted for 52.1 percent of the excess deaths among cigarette smokers; lung cancer accounted for 13.5 percent; cancer of other sites accounted for 13.5 percent; other heart and circulatory diseases 5.8 percent; pulmonary diseases (other than lung cancer) 5.6 percent; cerebral vascular lesions 4.8 percent; gastric and duodenal ulcers 2.8 percent; cirrhosis of the liver 1.5 percent; and all other diseases combined 0.4 percent.

We found little or no association between cigarette smoking and a number of diseases including: chronic rheumatic fever, hypertensive heart disaese, other hypertensive diseases, nephritis and nephrosis, diabetes, leukemia, cancer of the rectum, cancer of the colon, and cancer of the brain.

EXHIBIT 2

Schedule of lung cancer grants awarded by the American Cancer Society to date—July 23, 1957 [1]

Institution and address	Investigator	Period	Title
Washington University, St. Louis, Mo	Committee on Cancer Research	June 1, 1954, to Aug. 31, 1955	Investigation on the problem of growth.
Wayne University, Detroit, Mich	Dr. Arthur J. Vorwald	Sept. 1, 1954, to Aug. 31, 1955	The pathogenesis of pulmonary cancer in experimental animals.
New York University, New York, N. Y	Dr. Gray Twombly	do	Lung cancer research.
Wayne University, Detroit, Mich	Dr. Arthur J. Vorwald	July 1, 1955, to June 30, 1956	Experimental pulmonary cancer induced by compounds of beryllium in albino rats.
Sloan-Kettering Institute, New York, N. Y	Dr. C. P. Rhoads	Jan. 1 to Aug. 31, 1955	A study to identify carcinogenic substances in tobacco.
Yale University, New Haven, Conn	Dr. F. Duran-Reynals	Sept. 1, 1955, to Aug. 31, 1958	To ascertain whether some or all types of lung cancer, apparently induced by external specific irritants or other noninfectious causes, are fundamentally induced by viruses.
University of Virginia, Charlottesville, Va	Dr. Carnelia Hoch-Ligeti	Sept. 1, 1955, to Aug. 31, 1956	To develop experimental methods to allow for assaying of carcinogenicity by direct application of suspected materials to human lung tissue.
New York University, New York, N. Y	Dr. Norton Nelson	do	Phase I—To induce bronchogenic cancer in experimental animals. Phase II—To elucidate the specific chemical nature of environmental carcinogens, including those in cigarette smoke.
Sloan-Kettering Institute, New York, N. Y	Dr. C. P. Rhoads	do	To identify the specific carcinogenic substances in tobacco and its smoke
Washington University, St. Louis, Mo	Dr. Evarts A. Graham	do	To determine the specific carcinogenic agents in tobacco smoke.
Wayne University, Detroit, Mich	Dr. Arthur J. Vorwald	do	To study the mechanisms whereby certain agents upon deposition in the lung have the capacity to produce pulmonary cancer.
University of Southern California, Los Angeles, Calif.	Dr. Paul Kotin	do	To study the fate of carcinogenic polycyclic aromatic hydrocarbons following their natural deposition in the respiratory tract of humans, and experimental deposition in animals.
Chicago Medical School, Chicago, Ill	Dr. Philippe Shubik	do	A study of the mechanisms of penetration, localization, and action of carcinogenic hydrocarbons in the mammalian lung.
Philadelphia Pulmonary Neoplasm Research Project, Philadelphia, Pa.	Dr. Katharine Boucot and Dr. David Cooper.	Jan. 1, 1956, to Dec. 31, 1956	To delineate the characteristics and methods of detecting curable lung cancer * * * in a survey study, and to obtain accessory information essential for proper treatment.
California State Department of Public Health, Berkeley, Calif.	Dr. Malcolm H. Merrill	Apr. 1, 1956, to Mar. 31, 1957	* * * to answer the question as to whether air pollution is a factor in causation of lung cancer.
New York Medical College, Flower and Fifth Avenue Hospitals, New York, N. Y.	Dr. Oscar Auerbach	Aug. 1, 1955, to July 31, 1956	To show the relation of smoking to lung cancer by a detailed histological study of bronchial epithelium.
Washington University, St. Louis, Mo	Dr. E. V. Cowdry	Jan. 1 to Dec. 31, 1956	* * * to investigate human bronchial epithelium in the hope of discovering precancerous conditions therein and their causation.

See footnote at end of table.

Schedule of lung cancer grants awarded by the American Cancer Society to date—July 23, 1957 [1]—Continued

Institution and address	Investigator	Period	Title
Massachusetts Memorial Hospital, Boston, Mass.	Dr. Sheldon C. Sommers	Jan. 1 to Dec. 31, 1956	A pathologic study of host factors in human lung cancer.
Western Reserve University, Cleveland, Ohio.	Dr. Charles N. Loeser	Apr. 1, 1956, to Aug. 31, 1957	In vivo localization of the effects of tobacco tars in lung of experimental animals.
Johns Hopkins University, Baltimore, Md.	Dr. George Gey	do	Cell growth and virus studies of lung cancer with special reference to (1) isolation and characterization of isogenic normal versus malignant lung cells; (2) effect of pollutants on target normal (?) cells; (3) attempts at virus isolations directly from lung (and cancer) * * *.
Saranac Laboratory and Trudeau-Saranac Institute, Saranac Lake, N. Y.	G. W. H. Schepers	July 1, 1956, to June 30, 1957	To study environmental cancer of the lung: An investigation of the cocarcinogenic activity of inhaled asbestos dust in experimental beryllium neoplasia.
Western Reserve University, Cleveland, Ohio.	Dr. James W. Reagan	Apr. 1, 1956, to Aug. 31, 1957	The proposed study will be made for the purpose of investigating carcinogenesis in the lung of experimental animals.
New York Medical College, New York, N. Y.	Dr. Oscar Auerbach	Aug. 1, 1956, to July 31, 1957	To show the relation of smoke to lung cancer by a detailed histological study of bronchial epithelium.
Sloan-Kettering Institute, New York, N. Y.	Dr. C. P. Rhoads	Sept. 1, 1956, to Aug. 31, 1957	To investigate the possible effects of tobacco smoke on the respiratory tract mucosa of humans by cytologic and pathologic studies of heavy smokers and a control group of nonsmokers.
Do	do	do	To identify the specific carcinogenic substance in tobacco smoke.
Washington University, St. Louis, Mo.	Dr. Evarts A. Graham	do	A continuation of the investigation of possible causal relationship of tobacco smoking and lung cancer.
Chicago Medical School, Chicago, Ill.	Dr. Philippe Shubik	do	A study of the mechanisms and factors concerned in the penetration, localization, and biological action of carcinogenic hydrocarbons in the mammalian lung.
University of Virginia, Charlottesville, Va.	Dr. Cornelia Hoch-Ligeti	do	* * * to study carcinogenesis in human lung tissue by means of heterotransplantation and tissue culture.
New York University, New York, N. Y.	Dr. Norton Nelson	do	Phase I—Experimental lung cancer. Phase II—A search of carcinogenic substances in tobacco "tars."
University of Southern California, Los Angeles, Calif.	Dr. Paul Kotin	do	The study of the fate of carcinogenic polycyclic aromatic hydrocarbons following their natural deposition in the respiratory tract of humans and experimental deposition in animal species.
Wayne University, Detroit, Mich.	Dr. Arthur J. Vorwald	July 1, 1956, to Aug. 31, 1957	Experimental pulmonary cancer induced by compounds of beryllium in albino rats.
Sloan-Kettering Institute, New York, N. Y.	Dr. Robert C. Mellors	July 1, 1956, to June 30, 1957	* * * to study by fluorescence microscopy of the cellular localization of materials derived from tobacco smoke.
Columbia University, New York, N. Y.	Alvin L. Barach	do	Production, elimination, and examination of bronchial secretions in apparently normal subjects and in patients with miscellaneous disease.
Johns Hopkins University, Baltimore, Md.	Dr. Warde B. Allen	June 1, 1956, to Aug. 31, 1957	Investigation of new methods for cell studies in screen cases for lung cancer.

Cornell University, New York, N. Y.	Dr. G. Papanicolaou	do	Study of certain degenerative changes affecting the ciliated cells of the bronchial mucosa and their relation to cancer of the lung.
Rockefeller Institute for Medical Research, New York, N. Y.	Dr. James G. Hirsch	do	Study of certain relationships between sputum cytology, pulmonary cancer, and respiratory infections.
Philadelphia Tuberculosis and Health Association, Philadelphia, Pa.	Dr. Katharine Boucot and Dr. David Cooper.	Jan. 1 to Dec. 31, 1957	To delineate the characteristics and methods of detecting curable lung cancer.
Columbia University, New York, N. Y.	Dr. Alvin L. Barach	do	The stimulation of bronchial secretions in apparently healthy subjects and in patients with miscellaneous diseases; the elimination of these secretions and their examination by cytologic technic.
California State Department of Public Health, Berkeley, Calif.	Dr. Malcolm H. Merrill	Apr. 1, 1957, to Mar. 31, 1958	To determine whether persons exposed in Los Angeles air pollution for many years experience in higher mortality from lung cancer than persons exposed thereto only a short period of time.
Washington University, St. Louis, Mo.	Dr. E. V. Cowdry	Jan. 1 to Dec. 31, 1957	To investigate human bronchial epithelium in the hope of discovering precancerous conditions therein and causations.
University of Southern California, Los Angeles, Calif.	Dr. Irving Gordon	do	To investigate the effect of virus infection in the pathogenesis of pulmonary cancer.
St. Louis University, St. Louis, Mo.	Dr. Walter J. Burdette	do	To establish a biological screening method for testing carcinogenic agents of bronchial epithelium and to test the effect of tobacco tar on this type of epithelium.
New York Medical College, New York, N. Y.	Dr. Oscar Auerbach	Aug. 1, 1957, to Aug. 31, 1958	To show the relation of smoking and air pollution to lung cancer by a detailed histology study of the tracheobronchial epithelium.
Sloan-Kettering Institute, New York, N. Y.	Dr. Robert C. Mellors	July 1, 1957, to June 30, 1958	To study by fluorescence microscopy the cellular localization of materials derived from tobacco smoke.
Columbia University, New York, N. Y.	Dr. Alvan L. Barach	Jan. 1, 1958, to Feb. 28, 1959	Workshop conference on lung cancer research. The production and elimination of bronchial secretions for the diagnosis of lung cancer.
Philadelphia Tuberculosis and Health Association, Philadelphia, Pa.	Dr. K. R. Boucot and Dr. D. A. Cooper.	do	To delineate the methods of detecting curable lung cancer * * * in a survey study, and to obtain accessory information essential for proper treatment.
St. Louis University, St. Louis, Mo.	Dr. W. J. Burdette	Jan. 1, 1958, to Feb. 28, 1959	To establish a biological screening method for testing carcinogenic agents on bronchial epithelium and to test the effect of tobacco tar on this type of epithelium.
Rockefeller Institute, New York City	Dr. J. C. Hirsch	Sept. 1, 1957, to Aug. 31, 1958	To establish the diagnostic and prognostic significance of the presence of ciliocytophthoria (CCP) in sputum specimens stained by the Papanicolaou technique.
University of Southern California, Los Angeles, Calif.	Dr. I. Gordon	Jan. 1, 1958, to Feb. 28, 1959	To study the effects of virus infections on the pathogenesis of pulmonary cancer.
Washington University, St. Louis, Mo.	Dr. E. V. Cowdry	Sept. 1, 1957, to June 30, 1958	Experimental production of cancer by tobacco products.
Wayne State University, Detroit, Mich.	Dr. A. J. Vorwald	Sept. 1, 1957, to Aug. 31, 1958	Experimental pulmonary cancer induced by compounds of beryllium in albino rats.
Cornell University Medical College, New York City.	Dr. G. N. Papanicolaou	Sept. 1, 1957, to June 30, 1958,,	Study of certain degenerative changes affecting the ciliated cells of the bronchial mucosa and their relation to cancer of the lung.
Chicago Medical School, Chicago, Ill.	Dr. P. Shubik	Sept. 1, 1957, to Feb. 28, 1959	Factors involved in the action of carcinogenic hydrocarbons in the lung and bronchial mucosa.

See footnote at end of table.

Schedule of lung cancer grants awarded by the American Cancer Society to date—July 23, 1957 [1]—Continued

Institution and address	Investigator	Period	Title
University of Southern California, Los Angeles, Calif.	Dr. P. Kotin	Sept. 1, 1957, to Aug. 31, 1958	The fate of carcinogenic polycyclic hydrocarbons following their natural deposition in the respiratory tract of humans and experimental deposition in animal species.
Sloan-Kettering Institute, New York City	Dr. E. L. Wynder	Sept. 1, 1957, to June 30, 1958	A study to identify the specific carcinogenic substances in tobacco.
University of Virginia, Charlottesville, Va	Dr. C. Hoch-Ligeti	do	Study of cellular changes effected by carcinogens in human bronchial epithelium grown in tissue culture or as a heterotransplant in rodents.
New York University-Bellevue Medical Center, New York City.	Dr. N. Nelson	Sept. 1, 1957, to Aug. 31, 1958	Phase I—The experimental induction of lung cancer. Phase II—Studies on cigarette smoke condensates.
Western Reserve University, Cleveland, Ohio.	Dr. C. C. Loeser	Sept. 1, 1957, to June 30, 1958	In vivo, localization of the effects of carcinogenic agents in lungs of experimental animals.
Washington University, St. Louis, Mo	Dr. E. V. Cowdry	Jan. 1, 1958, to Feb. 28, 1959	Tracheal and bronchial epithelium in smokers and nonsmokers.
Johns Hopkins University, Baltimore, Md	Dr. G. O. Gey	Sept. 1, 1957, to Aug. 31, 1958	Cell growth and virus studies of lung cancer with special reference to: (1) Isolation and characterization of isogenic normal versus malignant lung cells; (2) attempts at virus isolations from the lung and from malignant lesions, and to study the effects of viruses on cells; (3) effects of pollutants on target normal (?) cells.
Albany Medical College, Albany, N. Y	Dr. K. B. Olsen	Sept. 1, 1957, to Feb. 28, 1959	Comparison of routine cytological examination of sputum with chest radiographs in the detection of lung cancer.
University of Chicago, Chicago, Ill	Dr. W. W. Adams	Sept. 1, 1957, to Aug. 31, 1958	To study acute chronic corpulmonali following pulmonary resection.

[1] Supported in part ($10,000) from a gift to the society, in memory of J. J. Hopkins.

NOTE.—These awards total $1,870,875.

EXHIBIT 3

TOBACCO INDUSTRY RESEARCH COMMITTEE,
New York, N. Y., August 1, 1957.

Hon. JOHN A. BLATNIK,
Chairman, Subcommittee on Local and Monetary Affairs,
House Committee on Government Operations,
Washington, D. C.

DEAR MR. CHAIRMAN: When Dr. Little appeared before your subcomimttee on Thursday, July 18, 1957, he agreed to provide the committee with certain pertinent information concerning the research program of the Tobacco Industry Research Committee. Dr. Little has asked me to forward you this material. You will find it enclosed.

The Tobacco Industry Research Committee has appropriated for research grants $500,000 in each of the following years: 1954, 1955, 1956, and 1957. In 1956, a supplementary $200,000 was made available at the request of the scientific advisory board. Thus, the total available funds thus far amount to $2,200,000.

These funds have not been allocated exactly as appropriated, that is, on an annual basis. To date the total grant awards made by the scientific advisory board to independent scientists in accredited institutions amount to $1,832,591.51.

The additional material, which Dr. Little agreed to supply the committee and which is appended, is:

1. A statement concerning the origin and purpose of the committee, issued at its inception.
2. A statement of policy adopted in 1954 concerning conditions and terms under which the scientific advisory board awards grants-in-aid.
3. A statement of the research program as adopted by the scientific advisory board in 1954.
4. An interim informal report of progress issued by Dr. Little, May 16, 1955.
5. The first formal report of the scientific director issued midyear 1956.
6. A list of the original grants showing the recipient of each grant, his institution, the value of the grant, and the date the project was initiated. Also, a similar list of renewed grants. This information is current as of July 1.
7. Thirteen abstracts of papers published in accepted scientific journals reporting on research work supported in whole or in part by grants approved by the scientific advisory board. These abstracts are for inclusion in the 1954 report of the scientific director, as yet unpublished, and have been approved by the investigator working on each grant.

Dr. Little has asked me to thank you for your courtesy in providing him the opportunity to appear before your subcommittee.

If you desire any further information, please let us know.

Sincerely,

W. T. HOYT, *Executive Secretary.*

ATTACHMENT A

STATEMENT CONCERNING THE ORIGIN AND PURPOSE OF THE TOBACCO INDUSTRY RESEARCH COMMITTEE AND ITS PROPOSED FUNCTIONS

The responsible chief officers of 9 of the cigarette and tobacco products manufacturing companies in America, and 5 organizations of growers of leaf tobacco and tobacco warehouse associations have formed the Tobacco Industry Research Committee in the interest of the public as well as of the industry to meet the challenge raised by widely publicized reports in the press, purporting to link tobacco smoking with the cause of lung cancer.

To avoid possible confusion and misunderstanding concerning the origin, purpose, and function of this committee, the committee makes the following statement:

I. EVENTS JUSTIFYING FORMATION OF COMMITTEE

The formation of the committee was prompted by the appearance of certain publications claiming an established relationship between cigarette smoking and lung cancer.

Typical of these reports is an article appearing in the Journal of the American Medical Association (143 JAMA 329, May 27, 1950) wherein Drs. E. L. Wynder and E. A Graham reported that applications of cigarette tars to the backs of mice had produced skin cancers.

In an address before the annual fall clinical conference of the Kansas City Southwest Clinical Society, held in Kansas City, Mo., on September 28, 1953, Dr. Alton Ochsner said: "This unprecedented increase in the incidence of bronchogenic cancer, we are convinced, is due to the carcinogenic effect of cigarette smoking" (Kansas City Medical Journal, vol. XXIX, No. 6, November-December, 1953, at p. 6).

At a recent meeting of the Greater New York Dental Association, these same doctors reiterated charges, based on statistical and other analyses, that cigarettes contain a cancer-producing factor. These assertions, and others to the same effect, have been given extensive publicity in magazines of national circulation, such as Time (November 30, 1953), Life (December 21, 1953), and Reader's Digest (December 1953), and in countless national, regional, and local newspapers.

Examination of all recent reports and publications, however, reveals that many factors, such as various types of air pollution as well as tobacco smoke, have been suspected as causes of lung cancer. Although much has been written concerning the incidence of lung cancer, there is still a dearth of authoritative findings on the subject. It is safe to say that no persuasive and definitive conclusion respecting the cause of this disease, or the relation of smoking thereto, has been established.

In the light of the foregoing agitation and in the absence of authoritative findings, there is a responsibility on the part of the management of the tobacco manufacturers and others engaged in the tobacco industry to aid in the final determination of this controversy. It is the earnest wish of the industry to encourage competent scientific authority to find ultimate facts which will dispel the present confusion and to communicate authoritative factual information on the subject to the public.

II. PLAN OF ACTION

The committee announced in a full page advertisement appearing in 448 newspapers circulated throughout the United States on Monday, January 4, 1954, that it would meet the challenge of these reports. The signing members of the Tobacco Industry Research Committee pledged to support by financial aid, in addition to that already contributed by individual companies, research under the charge and direction of a scientist of unimpeachable integrity and national repute. To guide and advise in this field, scientists disinterested in the cigarette industry and of recognized ability and professional standing in medicine, education, and associated sciences will be invited to act as an advisory board.

This statement was subscribed to by the following companies, which now make up the committee:

The American Tobacco Co., Inc., by Paul M. Hahn, president
Benson & Hedges, by Joseph F. Cullman, Jr., president
Bright Belt Warehouse Association, by F. S. Royster, president
Brown & Williamson Tobacco Corp., by Timothy V. Hartnett, president
Burley Auction Warehouse Association, by Albert Clay, president
Burley Tobacco Growers Cooperative Association, by John W. Jones, president
Larus & Bro. Co., Inc., by W. T. Reed, Jr., president
P. Lorillard & Co., by Herbert A. Kent, chairman
Maryland Tobacco Growers Association, by Samuel C. Linton, general manager
Philip Morris & Co., Ltd., Inc., by O. Parker McComas, president
R. J. Reynolds Tobacco Co., by E. A. Darr, president
Stephano Bros., Inc., by C. S. Stephano, Sc. D., director of research
Tobacco Associates, Inc., an organization of flue-cured tobacco growers, by J. B. Hutson, president
United States Tobacco Co., by J. W. Peterson, president

III. FORMATION OF THE COMMITTEE

Mr. Paul M. Hahn, president of the American Tobacco Co., on December 10 and 11, 1953, took the initial steps in the formation of the committee in a telegram sent to the following people:

Joseph F. Cullman, Jr., president, Benson & Hedges
E. A. Darr, president, R. J. Reynolds Tobacco Co.
B. F. Few, president, Liggett & Myers Tobacco Co.
William J. Halley, president, P. Lorillard Co.
Timothy V. Hartnett, president, Brown & Williamson Tobacco Co.

J. B. Hutson, president, Tobacco Associates, Inc.
O. Parker McComas, president, Philip Morris & Co., Ltd., Inc.
J. Whitney Peterson, president, United States Tobacco Co.

Mr. Hahn suggested that these heads of the leading tobacco units meet to consider some action in response to these published reports. Thereafter, those invited, with one exception,[1] met on December 14 and 15, 1953, and December 28, 1953, in New York City.

At these meetings the conclusion was reached that the tobacco industry must take public action to meet these widely publicized claims. The committee was formed, and the firm of Hill & Knowlton, Inc., of 350 Fifth Avenue, New York, N. Y., was engaged to assist the committee in effectuating its purpose.

The officers of the committee selected were Paul M. Hahn, chairman; J. Whitney Peterson, vice chairman; Joseph F. Cullman, Jr., treasurer; and Wilson T. Hoyt, of Hill & Knowlton, Inc., secretary. It was the expressed intention of the committee to have the chairmanship of the committee rotated every 3 months. It was agreed that expenses for the committee's activities, including expenditures for research, the advertising, the employment of Hill & Knowlton, and other similar expenses, would be defrayed by donations from the member firms based on their volume of business and by contributions to be agreed upon by other members. Arrangements were then perfected for insertion of the advertisement referred to in the newspapers throughout the country.

The Bright Belt Warehouse Association, the Burley Auction Warehouse Association, the Burley Tobacco Growers Cooperative Association, Larus & Bro. Co., Inc., the Maryland Tobacco Growers Association, and Stephano Bros., Inc., were invited to join with the original group and became members of the committee.

IV. LIMIT OF POWERS

The purposes and objectives of the committee are to aid and assist research into tobacco use and health, and particularly into the alleged relationship between the use of tobacco and lung cancer, and to make available to the public factual information on this subject. It is the considered judgment of the committee that its activities shall be confined to the purposes set forth above, and that it is in nowise to be considered or to operate as a trade association or to participate in any activity or give consideration to any matters affecting the business conduct or activities of its members, and that its activities in every respect shall conform to law and all decrees or judgments of courts affecting or relating to the tobacco industry. To this end the committee is proceeding under the advice of legal counsel selected from among the counsel or nominees of its members.

TOBACCO INDUSTRY RESEARCH COMMITTEE,
PAUL M. HAHN, *Chairman.*

NEW YORK, N. Y., *January 25, 1954.*

ATTACHMENT B

TOBACCO INDUSTRY RESEARCH COMMITTEE STATEMENT OF POLICY CONTAINING CONDITIONS AND TERMS UNDER WHICH PROJECT GRANTS ARE MADE

(Adopted by the Scientific Advisory Board)

I. General policy

The Tobacco Industry Research Committee is dedicated to the support of the investigation of fundamental matters relating to a connection between tobacco use and human health. In so doing the Tobacco Industry Research Committee recognizes the importance of independent research by competent investigators. Research policy and programming are the responsibility of the Scientific Advisory Board. Grants are made only after careful consideration by the Scientific Advisory Board of the merits of proposals and of the qualifications of the individual and his institution undertaking the work.

The Committee desires to have scientists work with the greatest freedom and without domination of any kind. It will make no attempt to direct the administration of the project once started, to influence its course or to control its results other than to be assured that the funds are properly expended for the purposes of the grant and that all findings are reported in accordance with the best scientific practice.

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[1] Liggett & Myers Tobacco Co.

II. Payments and budgets

Unless otherwise requested at the time of initiating a specific grant, payments will be made quarterly in advance to the institution at which research is being conducted.

Grants may not be transferred from one institution to another due to a change in affiliation by the principal investigator without express permission.

The contract for a grant may be terminated prior to normal expiration date by the grantee upon notification to the Executive Secretary of the Tobacco Industry Research Committee with a statement of the reasons for termination.

Budgets are presumed to be accurate at the time of issuance of a grant. However, if for unforeseen reasons, additional funds or reapportion of funds are required such request will be considered upon receipt of a complete statement of reasons for such change.

At the time of expiration of a grant or in the event of its termination, unexpended funds shall be returned to the Tobacco Industry Research Committee. If, at such expiration or termination, additional projects are anticipated and are approved such funds may, upon request, be applied against the new grant at the time of its issuance.

III. Reports

Grantees are to furnish a report of activities semiannually. These need not be extensive but should be sufficiently informative to permit the Scientific Advisory Board of the Tobacco Industry Research Committee to know who is being accomplished. At the conclusion of a project a detailed report is expected which shall be given in writing to the Scientific Advisory Board.

An expenditure report should be made by the grantee semiannually.

IV. Exchange of information

With the consent of the grantee, the Scientific Advisory Board may recommend the exchange of interim information between investigators working on different projects if the interim results indicate a relationship between projects. This would only be done with the object of assisting and expediting work in process.

V. Publication and public information

The Tobacco Industry Research Committee approves the initial presentation by the investigator of research results only in accepted medical and scientific journals or before accepted medical or scientific societies. It has no objection to dissemination to the public of any or all final conclusions from projects in these ways.

Information from semiannual or final reports will be released publicly only with the permission of the investigator.

When a journal or society schedules the presentation of any findings from a project the Scientific Advisory Board will expect a statement or abstract from the grantee covering the date and general subject matter of his presentation.

While no special funds are provided to assist publication of results covering costs of illustrating, typesetting or other expenditures, the Tobacco Industry Research Committee will consider a request for such funds upon presentation of the manuscript to be published.

ATTACHMENT C

THE RESEARCH PROGRAM OF THE SCIENTIFIC ADVISORY BOARD TO THE TOBACCO INDUSTRY RESEARCH COMMITTEE

THE SCIENTIFIC ADVISORY BOARD

Members of the Scientific Advisory Board to the Tobacco Industry Research Committee are:

Chairman and Scientific Director: Clarence Cook Little, Sc. D., LL. D., Lit. D., Director, the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine

McKeen Cattell, Ph. D., M. D., Professor and Head of the Department of Pharmacology, Cornell University Medical College, New York, N. Y.

Julius H. Comroe, Jr., M. D., Chairman and Professor, Department of Physiology and Pharmacology, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa.

Leon O. Jacobson, M. D., Professor of Medicine, University of Chicago, Director, the Argonne Cancer Research Hospital, Chicago, Ill.
Paul Kotin, M. D., Assistant Professor of Pathology, University of Southern California Medical School, Los Angeles, Calif.
Kenneth Merrill Lynch, M. D., Sc. D., LL. D., President, Dean of Faculty, and Professor of Pathology, Medical College of South Carolina, Charleston, S. C.
Stanley P. Reimann, M. D., Sc. D., Scientific Director, the Institute for Cancer Research, Director, the Lankenau Hospital Research Institute, Philadelphia, Pa.
William F. Rienhoff, Jr., M. D., Associate Professor of Surgery, Johns Hopkins School of Medicine, Baltimore, Md.
Edwin B. Wilson, Ph. D., Professor Emeritus of Vital Statistics, Harvard University, Cambridge, Mass.
Associate Scientific Director: Robert C. Hockett, Ph. D.

THE RESEARCH PROGRAM

The Tobacco Industry Research Committee, formed in January 1954, to sponsor independent research into tobacco use and health, put into the hands of a Scientific Advisory Board the development and continuing supervision of a research policy and program. Many doctors, educators, and scientists want to know about the Committee, the Scientific Advisory Board, the purposes and policies, and the research program. This booklet is intended to answer these questions.

"The Committee's approach has given the Scientific Advisory Board an unusual, if not unique, opportunity to foster and guide the widest possible range of research by outstanding scientists and doctors under conditions of utmost freedom," according to Dr. Clarence Cook Little.

Board determines activities

Because of the broad responsibility of the Scientific Advisory Board, scientists whose competence is securely established in their respective fields of knowledge are asked to serve as its members. They are given assurance of complete scientific freedom in their work.

The Scientific Advisory Board does not contemplate conducting specific laboratory investigations as a Board. This does not rule out the possibility, however, that individual members may seek and, in competition with other applicants, obtain a research project under a Tobacco Industry Research Committee grant. This is the policy common to similar bodies such as the Committee on Growth and the National Advisory Cancer Council.

The Board members retain completely their association with their institutions and, except for the Scientific Director, are reimbursed only for time and expenses involved in their services to the research program.

The Board determines the scope and direction of the research program; reviews and solicits requests for research grants from universities, hospitals and other recognized research organizations or from individuals there situated. Regular monthly meetings are held by the Board to carry out its scheduled work. In addition, members of the Board frequently undertake special assignments in connection with the development of the research program.

Scope of interests defined

Before undertaking a large-scale program of recommending grants to finance research projects, the Scientific Advisory Board laid the foundations on which a series of research projects could be developed and coordinated. First was the delineation of the scope of interests within which investigations should be sponsored. Second was the organization of a program to assure that necessary investigation was undertaken without unnecessary duplication of work.

Scope covers three main areas

The outline of interest sets out three main areas of investigation:

1. The physical and chemical composition of tobacco and accompanying products, such as cigarette papers and additives. This covers the preparation, fractionation and analysis of tobacco and of added substances.

2. Tissue changes in humans as well as in animals, in normal life or under laboratory conditions, subjected to various types, duration, and intensity of exposure to various tobaccos and derivatives, and other potential irritants. Tissues of special interest are those of the mouth, lungs, glands, heart, and other organs of subjects of various ages, sex, and strains.

3. Smoking and other tobacco habits, and the emotional and physical makeup of smokers, with respect to establishment, duration, and intensity of tobacco use, and correlation of these data with metabolic, glandular, and nervous types under various degrees of stress and challenge.

It is recognized that work has been done and is being done here and abroad in these fields. The Scientific Advisory Board plans to avoid repeating work that has produced accepted results, as in the field of the constituents of tobacco. Where results have been inconclusive, however, considerable further careful study and well-planned research is indicated.

The program takes shape

In its second step, the Board developed a coordinated research program intended not only to ascertain facts with respect to the questions raised concerning tobacco use and health but also to contribute to further understanding of cancer, heart disease, and other public-health problems. The program is intended to produce clinical and experimental findings necessary before valid conclusions can be made regarding possible relationships between tobacco use and human health.

Among research projects being sponsored is the study of human lung cancer in several major medical centers in the United States. A uniform method of study will allow for a comparison of the frequency and types of lung cancer in the geographic areas investigated. Such related factors as length of residence, occupational history, personal habits and associated diseases will be reviewed for possible relation to the causes and onset of lung cancer. It is expected that this research will furnish clues for still further research to be supported by the Committee.

Research projects

Research projects, to be supported at sites to be selected, will be encouraged in the following general subjects (primary responsibility for overall advice and informal guidance has been assigned to individual Board members, as indicated in parentheses) :

Inhalation of smoker.—Carefully planned and regulated experiments will be conducted with mammals subjected to inhalation of tobacco smoke under controlled conditions. (Dr. Kotin.)

Direct application of smoke tars.—Projects will test the effect of identified smoke tars, derived under controlled conditions, on tissues of animals with varying degrees of susceptibility to cancer or other ailments. (Dr. Reimann.)

Study of lung tissues.—Projects to study lung tissues in living animals and man will be undertaken. (Dr. Reinhoff, Dr. Lynch, and Dr. Reimann.)

Malignant changes in tissues.—Studies of malignant changes of tissues will be made under laboratory condition. (Dr. Jacobson.)

Study of cardiovascular tissues.—Studies will be made of changes involving degeneration or unbalance in cardiovascular tissues. (Dr. Comroe.)

Habits and characteristics of smokers.—A series of surveys of selected populations in various parts of the country will be undertaken concerning the characteristics of human smokers and nonsmokers, including age, sex, emotional habits, environmental factors, and exposure, as well as smoking habits. (Dr. Wilson.)

Preparation and analysis of tobacco derivatives.—Sources will be developed for a continuous supply of tars from tobacco smoke and its derivatives that can be used in various experiments. Efforts will be made to duplicate human smoking in machines. Production of the derivatives will be regulated so as to have full knowledge of the conditions under which smoking is simulated. (Dr. Cattell.)

Initial research grants under the approved program went to scientists at such institutions as the University of Southern California, the University of Texas, the Temple University School of Pharmacy, Mount Zion Hospital at San Francisco, the Medical College of Virginia, Fordham University, Harvard University, Roswell Park Memorial Institute at Buffalo, New York University-Bellevue Medical Center, Tufts College Medical School, University of Pennsylvania Department of Surgery, the Medical College of South Carolina, and the College of Physicians and Surgeons at Columbia University.

Many new research projects are being considered continually by the Scientific Advisory Board, and approval of grants are announced from time to time.

ADMINISTRATION OF GRANTS

The Scientific Advisory Board welcomes proposals from qualified research groups or organizations for specific research projects. The Board also is authorized to originate proposals which it believes will contribute to the program and to seek appropriate individuals and institutions to carry out such projects under a Tobacco Industry Research Committee grant.

Each applicant and prospective recipient of a grant receives a statement of policy approved by the Scientific Advisory Board.

The statement of policy declares that the Tobacco Industry Research Committee "desires to have scientists work with the greatest freedom and without domination of any kind. It will make no attempt to direct the administration of the project, once started, to influence its course or to control its results other than to be assured that the funds are properly expended for the purposes of the grant and that all findings are reported in accordance with the best scientific practice."

Forms of application

Standard application forms are sent to those desiring to apply for grants from the Tobacco Industry Research Committee. The applications are submitted to the Scientific Director and to each other member of the Scientific Advisory Board for study and comment. When indicated, personal communications or visits are made with the applicant in order to clarify or expand his proposal.

Each application is measured against such criteria as the following:

1. Is the proposal relevant to the scope of the program?
2. Is it made by a competent individual or institution?
3. Will it contribute to knowledge of the subject?
4. How does it rank in importance with other projects underway or under consideration?
5. Does it promise results not already contemplated from other research projects?
6. Can it be of value as a check against work already being carried on?

The Board discusses and reviews all applications in regular meetings and recommends grants for the approved applications. Grants are then made by the Tobacco Industry Research Committee.

Unless otherwise requested or agreed, payments are made quarterly in advance to the institution at which research is conducted. Grants may be transferred from one institution to another, where there is a change in the affiliation of the principal investigator, if permission is obtained.

Grantees are to furnish a report of activities semiannually. These should be sufficiently informative ot permit the Board to know what is being accomplished. A detailed report is expected by the Scientific Advisory Board at the conclusion of a project.

EXCHANGE OF INFORMATION AND PUBLICATION

With the consent of grantees, the Scientific Advisory Board may recommend the exchange of interim information between investigators working on different projects if interim results indicate a relationship between projects. The only purpose of this exchange of information is to assist and expedite work in progress.

The Tobacco Industry Research Committee approves the initial presentation of research results by the investigator only in accepted medical and scientific journals or before accepted medical or scientific societies. It has no objection to dissemination to the public in these ways of any or all final conclusions of the investigators.

When a journal or society schedules the presentation of any findings from a project, the Scientific Advisory Board expects the grantee to furnish a statement or an abstract covering the date and general subject matter of his presentation.

Information derived from semiannual or final reports will be made public by the Committee only with the permission of the investigator.

While no special funds are provided to assist publication of results covering costs of illustrating, typesetting, or other expenditures, the Tobacco Industry Research Committee will consider requests for such funds upon presentation of the manuscript to be published.

THE TOBACCO INDUSTRY RESEARCH COMMITTEE

The Tobacco Industry Research Committee was organized early in 1954 and is comprised of six tobacco manufacturing companies and eight associations of tobacco growers and warehousemen. Timothy V. Hartnett, former president of Brown & Williamson Tobacco Corp., is chairman.

The purposes and objectives of the Committee are to aid and assist research into tobacco use and health and to make available to the public factual information on this subject.

In the light of wide publicity given to some statistical studies attempting to link tobacco use and lung cancer, the Tobacco Industry Research Committee believes it has a public responsibility to aid in the further search for conclusive answers to cancer and other public health problems. It accepts a lasting interest in people's health as a basic premise in offering tobacco products for sale.

Examination of recent reports and publications and of standard literature on the disease indicate that many factors, such as various types of air pollution, are suspects in lung cancer and other ailments. Medical and research scientists agree that no persuasive, definitive, and final conclusion has been established with respect to the cause or causes of this disease.

The fact that cigarette smoking today should even be suspected as a cause of serious disease is a matter of deep concern to the tobacco industry. Accordingly, the Committee pledges aid and assistance to the research effort into all phases of tobacco use and health.

STATEMENT BY DR. LITTLE

"The Scientific Advisory Board of the Tobacco Industry Research Committee has certain opportunities and obligations of tremendous importance to the future development of scientific research in the United States.

"First, it can recommend financial support of basic or pioneer research on its own evaluation of individuals or institutions on the basis of promise or faith rather than on a purely factual and materialistic standard. Pioneer research is the creative 'idea' phase of discovery. It is the absolutely essential forerunner of progress to the 'project' stage!

"Second, the Board can itself plan and initiate research in fields and for purposes not now being covered. This is, I believe, an entirely new departure for any industry. Granting a group of scientists the power of creating new research activity is a great and exciting development.

"If we on the Board have the wisdom and vision to plan creatively, we may be able to justify this confidence placed in us. If we do justify it, the tobacco industry will have made its greatest contribution of service to mankind and may well establish a precedent and pattern which other industries will follow in support of research."

C. C. LITTLE.

ATTACHMENT D

A REPORT OF PROGRESS

Statement of Dr. Clarence Cook Little, Scientific Director, the Tobacco Industry Research Committee, and Chairman, the Scientific Advisory Board. Made at discussion with science writers, Monday, May 16, 1955, Tobacco Industry Research Committee offices, 5320 Empire State Building, New York, N. Y.

A year has gone by since the Scientific Advisory Board to the Tobacco Industry Research Committee first met to discuss the course of scientific research to be financed by grants from this industry group.

At that time, many of us were strangers personally, though not scientifically. As members of a newly created Scientific Advisory Board, however, we had at least three essential things in common.

First, we had been given a free rein to spend an industry's research fund as we thought best. That in itself was a challenge and responsibility.

Secondly, we shared a firm belief that sound scientific research would continue to unfold, however gradually, the solutions to perplexing health problems.

Thirdly, and importantly, we were in full agreement that the arena of public controversy was to be avoided in the interest of constructive science and genuine progress.

These common bonds have brought us a long way and we still adhere to them. Also, we now share a sense of progress and, at the same time, a realization that our work has really just begun.

Measurable progress made

Some steps taken are measurable strides such as the research projects already under way. Other accomplishments are harder to gage by visible standards but, in my opinion, they will have a profound effect on the future of basic science in this country.

Let me give you a few examples:

1. The Scientific Advisory Board has been given a unique opportunity to foster and guide the widest possible range of research by scientists and doctors under conditions of absolute freedom. The Board not only recommends financial support for basic pioneer research, but it can itself plan and initiate research into fields not now covered. This, I believe, is an entirely new research concept for any industry.

If we on the Board have the wisdom and vision to plan creatively, the tobacco industry will have made a notable contribution in support of research and may well establish a pattern and precedent for other industries to follow.

2. There has been continuing financial support of the Board's research program. In January, I reported to a meeting of the Tobacco Industry Research Committee that the original $500,000 appropriation was then being rapidly allocated and that, for proper forward planning, it would be helpful to be assured of additional funds to obligate. The Committee promptly doubled the fund to $1,000,000, and assurance of continuing support was renewed.

3. Upon recommendation of the Board, nearly half a million dollars has already been granted to research scientists at 32 recognized hospitals and educational and research institutions in 14 States. In addition, research fellowships for 1955 have been extended to 50 medical colleges for assisting medical students in pursuit of research. These fellowships are made directly on the recommendation of college officials. The work done under them need have no bearing on the problem of tobacco in relation to health. They are honest efforts to encourage promising young research workers in any field of medical science.

4. Additional applications for grants-in-aid for research projects are being approved as rapidly as the Board can carefully study them. The Board has followed a sound policy. We are not interested in making a showing on how fast money can be spent. Instead, we have concentrated on approving grants for projects which, in our combined opinion, will contribute most to our knowledge in the shortest possible time.

Essential areas of research developed

The Board's research policy is to stimulate investigation into basic health problems and, at the same time, into specific questions relating to tobacco and health.

A primary objective is to further the search for the cause or causes of cancer, particularly lung cancer, and of cardiovascular disease, and for the control of those diseases. In addition, particular attention is given to research projects that will improve our understanding of tobacco smoke and its constituents, and of tobacco use by humans.

We recognize that substantial work has been under way for some years by or under support from such other groups as the National Cancer Institute, the American Cancer Society, the Sloan-Kettering Research Foundation, and the Damon Runyon Fund. They continue to be importantly engaged in accelerated studies in these or related fields. Thus it is necessary that the Board should carefully define its areas of inquiry so as to pass intelligently on the nature of research applications and not unwisely duplicate or confuse other research efforts.

I want to touch specifically upon some areas of study being developed under the close advice of and contact with members of the Scientific Advisory Board, noting again that the actual investigation is done entirely by other independent scientists working under TIRC grants.

1. Drs. Reimann, Lynch, and Kotin have helped organize a coordinated program for pathological studies of human lung ailments. A major problem was to agree upon a procedure for collecting specimens for study and for standardizing the analysis of them. There are now pathologists at twelve major hospitals cooperating in this effort. While some of these individuals and hospitals have

been engaged in similar studies of their own, this project represents, we believe, the first effort to coordinate and analyze findings on a "cross-country" basis.

2. Dr. Jacobson and I have been particularly engaged in stimulating efforts to develop methods of growing, observing, and experimenting with human and other mammalian lung tissues in cultures. Most tissue culture work has been with relatively primitive types of cells. Lung tissues, especially when adult, pose technical problems. If we can get them out into the open where we can see them and work with them, we shall have overcome a major obstacle toward better study of the lung cancer problem.

3. From the outset, an obvious and immediate need was the standardization of methods and materials used in dealing with tobacco smoke constituents and with animals used in experimentation. Drs. Cattell and Kotin went to work on this problem which logically broke into three parts.

One phase was to provide investigators for their experiments with a supply of mice of known and, as far as possible, unvarying genetic characteristics. Only with such animals can there be proper evaluation of the significance and successful repetition of experiments with mice. We all know the extreme variations in results obtained by some investigators when they repeat their experiments with different strains of mice.

The Board, therefore, has taken steps to insure that strains of mice with known and controlled genetic characteristics are available to investigators so that this essential component of many experiments can be standardized.

Secondly, careful study has been made of smoking machines for producing substances used in experiments. It was well known that many experiments were subjecting cigarettes to a smoking process that in no way even remotely resembled what happens when a man smokes a cigarette. One such machine, for example, burned up a cigarette in one steady, all-consuming suction in a matter of seconds This is as absurd as a man trying to warm his cold, bare feet by putting them directly on burning coals. At any rate, the need for knowing, recording and duplicating conditions under which cigarettes are smoked by machines is essential. Our study has led to recommending standards for smoking machines to simulate more closely conditions of human smoking.

This leads to the third element in the need for standardization—the tar preparations made from tobacco smoke. Efforts to standardize further the process of preparation and storage of tars are being continued, but we have already made significant progress toward establishing standard methods of production and storage of these tars for use by investigators.

4. Drs. Comroe and Cattell are continuing their study of organization of research into coronary diseases. These diseases have been with us for a long time and present a very difficult problem. A number of grants have already been made and other applications are being studied. We are correlating information concerning research in this field to use as a basis for planning new and creative experimentation. There will be many opportunities to make clinical studies on various physiological processes affected by tobacco derivatives.

5. Dr. Jacobson is head of a subcommittee engaged in studying available sources of tobacco grown in the atmosphere of radioactive carbon. The use of this "tagged" carbon in the detailed analysis of tobacco derivatives is one of the best prospects for study. A major concern will be to supply the increasing demand for this experimental tobacco.

6. Drs. Wilson and Rienhoff are developing two areas of study into the relationship of human characteristics and disease. One field will explore available data relating marked physical types to incidence and causes of disease. Another area is the effect of stress in relation to physical ailments. Since stress is believed by many authorities to be a factor in such ailments as stomach ulcers, it seems logical to question whether continued stress may not also upset the physiology of some individuals so that normal functions, such as cell growth and cardiovascular activity, become abnormal. Statistical studies based on these considerations, rather than on any geographic or conventional methods, are being developed and should open up worthwhile avenues to better understanding of public health problems.

These are some of the more significant areas in which I feel we are making real progress. And, while subcommittees are given responsibility for seeing that we move forward, I want to emphasize that all Board members take part in discussions and decisions, so that we have the benefit of combined experience and knowledge in all our work.

The situation now and ahead

When we began in this endeavor a year ago, we said we would avoid entering into public controversy, particularly concerning charges associating smoking and lung cancer.

This we have done and will continue to do.

The wisdom of this decision has been reaffirmed frequently during the past year.

Scientific evidence does not offer proof that smoking or air pollution or any other single factor is the cause of lung cancer. It's no good to argue about this theory or that theory. Time is better spent getting at the facts.

The facts today make clear that there is no single or simple answer to the lung cancer problem. Science has not yet found the cause or causes for lung cancer, nor indeed for any type of human cancer.

The time for positive statements has not yet arrived. We hope that it may at the earliest possible moment consistent with proven facts.

Important advances have been made in some sectors of the complex cancer front. But at this stage, our increasing knowledge of lung cancer only serves, paradoxically, to emphasize how little medical science really knows about the causes of this disease.

Recently, experienced researchers admitted that they have not been able to isolate or identify any specific substance in tobacco smoke derivatives that can be accused of causing cancers, even on the skins of mice.

Other scientific reports tell of similar lack of success in efforts to produce laboratory evidence to support statistical suspicion of tobacco smoke in relation to lung cancer.

This does not mean the question is closed. It emphasizes more than ever that the question remains wide open and unanswered.

The problem will not be solved by any repetition or extension of charges of statistical associations. Scientists generally agree that statistical associations alone do not provide proof of cause and effect.

Goethe once said that when an erroneous hypothesis becomes entrenched and generally accepted, it is transformed into a kind of tenet that no one is allowed to question and investigate; and it then becomes an evil which endures for centuries.

During the past year, some of the nation's outstanding cancer investigators openly expressed their fear that the so-called smoking-lung cancer theory would become just such a tenet—shutting off all questions and halting further investigation.

This danger is now passd. Full credit for its passing rests with the nation's courageous and independent research scientists, with members of the medical profession, and with the informed public who insist that a hypothesis must be proved before it can be accepted.

The history of cancer research is a record of slow but steady progress. There is every reason to expect marked advances in the prevention, treatment and cure of cancer.

This is also true of the other constitutional diseases, such as heart ailments, which are our greatest present and future health challenges.

But this will take time, and we cannot count on shortcuts. Neither the generating of unnecessary fears, nor the arousing of unfounded hopes, will hasten the coming of significant discoveries.

Progress in the battle against these great health problems has depended and will depend on solidly planned and well-executed scientific research.

The Scientific Advisory Board to the Tobacco Industry Research Committee will continue to devote its energies to this course.

SCIENTIFIC ADVISORY BOARD TO THE TOBACCO INDUSTRY RESEARCH COMMITTEE

Chairman and Scientific Director: Clarence Cook Little, Sc. D., LL. D., Lit. D., Founder and Director, the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine

McKeen Cattell, Ph. D., M. D., Professor and Head of the Department of Pharmacology, Cornell University Medical College, New York, N. Y.

Julius H. Comroe, Jr., M. D., Chairman of the Department and Professor of Physiology and Pharmacology, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa.

Leon O. Jacobson, M. D., Professor of Medicine, University of Chicago, Director of the Argonne Cancer Research Hospital, Chicago, Ill.

Paul Kotin, M. D., Assistant Professor of Pathology, University of Southern California Medical School, Los Angeles, Calif.

Kenneth Merrill Lynch, M. D., Sc. D., LL. D., President, Dean of Faculty, and Professor of Pathology, Medical College of South Carolina, Charleston, S. C.
Stanley P. Reimann, M. D., Sc. D., Scientific Director, the Institute for Cancer Research, Director, the Lankenau Hospital Research Institute, Philadelphia, Pa.
William F. Rienhoff, Jr., M. D., Associate Professor of Surgery, Johns Hopkins School of Medicine, Baltimore, Md.
Edwin B. Wilson, Ph. D., Professor Emeritus of Vital Statistics, Harvard University, Cambridge, Mass.

ATTACHMENT E

REPORT OF THE SCIENTIFIC DIRECTOR—1956

Clarence Cook Little, Sc.D.,
Chairman, Scientific Advisory Board

(Tobacco Industry Research Committee.,
150 East 42d Street, New York 17, N. Y.)

Scientific Advisory Board, to the Tobacco Industry Research Committee:

Clarence Cook Little, Sc. D., LL. D., Lit. D.;, Chairman, Scientific Advisory Board; Scientific Director, Tobacco Industry Research Committee; President, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine
McKeen Cattell, Ph. D., M. D., Professor of Pharmacology, Cornell University Medical College, New York, N. Y.
Julius H. Comroe, Jr., M. D., Chairman and Professor, Department of Physiology and Pharmacology, University of Pennsylvania Graduate School of Medicine, Philadelphia, Pa.
Leon O. Jacobson, M. D., Professor of Medicine, University of Chicago; Director, Argonne Cancer Research Hospital, Chicago, Ill.
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William F. Rienhoff, Jr., M. D., Associate Professor of Surgery, Johns Hopkins University School of Medicine, Baltimore, Md.
Edwin B. Wilson, Ph. D., Professor Emeritus of Vital Statistics, Harvard University, Cambridge, Mass.
Robert C. Hockett, Ph. D., Associate Scientific Director.

THE RESEARCH PROGRAM

Two years ago, the Scientific Advisory Board to the Tobacco Industry Research Committee was formally organized and undertook to create a program of research that would contribute constructively to our search for answers to fundamental health problems. Specifically, the program was to develop facts relative to the effects of tobacco use.

Today, well over $1,200,000 in TIRC funds have already been granted to more than 55 scientists who, with their colleagues, are participating in a carefully planned, well-integrated scientific endeavor to help in the solving of important health questions. The Committee has appropriated $1,500,000 for such grants and has pledged more funds as needed. The program is still developing and growing.

This report discusses the Scientific Advisory Board's approach to its assignment and the current status of the research program.

At the outset, the Board recognized that scientific knowledge concerning health aspects of tobacco use was uncertain and spotty. Tobacco use, particularly cigarette smoking, has become widespread throughout the world, especially in the more highly developed countries. Recently, there have been charges that smoking is associated with certain human ailments, among them cancer of the lung.

The Board did not set out to prove or disprove anything. Its position was and is in agreement with the vast majority of experimental scientists who concur

in the view that our knowledge of the formidable challengers of human survival—such as cancer and heart disease—is far from conclusive or complete. The sole purpose, therefore, was to encourage and support qualified research scientists in their efforts to learn more about these complex problems, meanwhile refraining from premature judgments.

This approach has proved sound and correct. The need for further study is, if anything, more compelling than it was two years ago. We have learned much in the past two years, but perhaps the most important thing we have learned is how much more must be done before definitive answers can be given.

Progress made in major areas

At this stage, therefore, the accomplishments of the research program supported by the Tobacco Industry Research Committee must be measured in terms of progress rather than in conclusive findings.

Having been given a completely free hand to consider the whole field of the use of tobacco in relation to health, the Board assumed the following responsibilities: (*a*) blocking out major subdivisions of this field which can and should be covered by scientific research; (*b*) considering personnel and facilities available, or capable of development, to meet research needs in these subdivisions; (*c*) recommending to the Tobacco Industry Research Committee the amount of financial support needed for each type of effort, and the personnel and institutions which should receive it; and (*d*) reviewing the progress of work thus supported to make it as effective as possible and to follow up new leads and problems arising from these efforts.

Among the more significant accomplishments that have come out of these efforts are:

1. The development of a program defining the range of research pertinent to our major interests, thus making possible the most efficient and effective use of research funds. This has helped to prevent a duplication of effort, has made possible supplementation of existing work, and has enabled us to give proper emphasis to fundamental and practical projects. The outline of this program will be discussed more fully in the body of this report.

2. The initiation and support of individual research projects carried out by independent, qualified scientists in some of the Nation's outstanding research and educational institutions. While these are separate and independent investigations, each fits into the coordinated program of objectives developed by the Scientific Advisory Board.

3. In the three fields of study discussed below, initiation of cooperative endeavors among scientists working on the same or similar problems in order to coordinate their efforts and compare procedures. This approach has proven extremely valuable in eliminating unnecessary duplication and in creating a large degree of unity and comparability in the work of the individual investigators. The three areas of interest covered were:

(*a*) *Human lung tissue study.*—Pathologists from various parts of the country have met several times under the sponsorship of the Scientific Advisory Board to develop and coordinate clinical studies of human lungs to learn more of the factors involved in lung ailments.

(*b*) *Heart and circulation.*—Specialists in cardiovascular and coronary research were brought together to explore what is known and not known about relationships of tobacco use, particularly of nicotine intake, to vascular and cardiac functions.

(*c*) *Tissue culture research.*—Leading investigators in the field of tissue culture conferred under the auspices of the Scientific Advisory Board to determine the best ways of speeding research into the cultivation and maintenance of lung tissue and cardiovascular tissue under conditions that would allow better observation and control than is now possible.

4. Development of acceptable standards for governing work with tobacco derivatives to meet a challenging need among investigators. The problem was that many experiments with tobacco smoke constituents had been or were being conducted under conditions bearing little, if any, relation to human use of tobacco. A defined range of methods and conditions was needed to give a greater degree of pertinency of the experiments to the experience of human smoking. Substantial progress has been made in:

(*a*) Preparation of standard tobacco derivatives for laboratory use.

(*b*) Definition of standard operating conditions for smoking machines, including control of temperature ranges in subjecting material to combustion.

(c) Further development and production of genetically controlled laboratory animals, particularly mice, with known characteristics, to be used in tobacco experiments.

5. Inauguration of a modest program of research fellowships for medical students. Encouragement of basic medical research is considered a fundamental contribution to the efforts to learn more about how to attack the constitutional diseases. Despite its necessary limitations, this program has brought extremely gratifying response from the participating medical colleges and the recipients of the fellowships.

Active projects

It is, and will continue to be, the established policy of the Scientific Advisory Board to permit each investigator to publish his findings independently when he feels he is ready to report on his work. Although the great majority of grant recipients are still engaged in their research, a few are at the stage of making public reports of progress to scientific groups or in scientific publications.

Although grants are made on a one-year basis, many have been renewed. In the field of scientific endeavor, it is often impossible to put definite time limitations on the period of study necessary for substantial accomplishment.

True scientists are naturally cautious. They are aware of the great and obvious danger for the lay public and for those in the fields of science and medicine that may result from premature and conjectural opinions.

Investigators working under TIRC grants and the areas of study undertaken are shown in the following listing of grants now in force. For convenience, the list is alphabetical by the investigator's name.

Grantee and institution	*Project title*
D. Murray Angevine, M. D., Professor of Pathology, University of Wisconsin Medical School, Madison, Wis.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Frederick W. Barnes, Jr., M. D., Ph. D., Associate Professor of Medicine and Physiological Chemistry, Johns Hopkins University School of Medicine, Baltimore 5, Md.	The Role of Hyperplasia in Tissue Response to Chronic Damage.
Richard J. Bing, M. D., Professor of Experimental Medicine and Clinical Physiology, the Medical College of Alabama, Birmingham 3, Ala. (now at Washington University School of Medicine, St. Louis, Mo.)	The Effect of Smoking on the Coronary Blood Flow and Certain Phases of Myocardial Metabolism in Patients With Arteriosclerotic or Hypertensive Cardiovascular Disease.
James F. Bonner, Ph. D., Professor of Biology, California Institute of Technology, Pasadena, Calif.	Enzymatic Study of Methylation Reactions in Plant Tissue.
Josef M. Brozek, Ph. D., Associate Professor of Physiological Hygiene, Laboratory of Physiological Hygiene, University of Minnesota School of Public Health, Minneapolis 14, Minn.	A Quantitative Study of Biological Characteristics of Man Associated with Differences in His Tendency to Adopt and Maintain Different Smoking Habits.
E. M. Butt, M. D., Professor of Pathology, University of Southern California School of Medicine; Chief Pathologist, Los Angeles County Hospital, Los Angeles 33, Calif.	Study of Trace Metal Storage of Pulmonary and Liver Tissue by Spectographic and Chemical Methods.
William H. Carnes, M. D., Professor of Pathology, Stanford University School of Medicine, San Francisco, Calif.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Leopold Cerecedo, Ph. D., Professor of Biochemistry, Fordham University, New York 58, N. Y.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Hans T. Clarke, Sc. D., *Professor of Biochemistry*, Columbia University College of Physicians and Surgeons, New York, N. Y.	A Study of Early Chemical Changes in the Lungs of Tumor-Bearing Rats and Mice.
	Proteolytic Activities of the White Blood Cells of Man and the Effect on White Blood Cell Activities of Carcinogens, Nutrition and Other Influences.

Grantee and institution	*Project title*
Philip Cooper, M.D., *Associate Professor of Clinical Surgery*, Boston University School of Medicine, Boston 18, Massachusetts; *Chief, Surgical Service and Director, Surgical Research Laboratory*, Veterans' Administration Hospital, Providence 8, R. I.	A Study of the Effects of Cigarette Smoking on Levels of Gastric Acid, Pepsin and Uropepsin.
Hans L. Falk, Ph.D., *Assistant Professor of Biochemistry*, University of Southern California School of Medicine, Los Angeles 33, Calif.	Examination of Cigarette Paper and Cigarette Smoke Condensates for Aromatic Polycyclic Hydrocarbons.
Russell L. Holman, M.D., *Professor and Head of Department of Pathology*, Louisiana State University School of Medicine, New Orleans 12, La.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Frank C. Ferguson, Jr., M.D., *Chairman of Department of Pharmacology*, Albany Medical College, Albany 8, N. Y.	Effects of Tobacco Smoke Upon the Function of the Cardiovascular System in Animals and Man.
Russell S. Fisher, M.D., *Chief Medical Examiner*, State of Maryland; *Professor of Legal Medicine*, University of Maryland Medical School, Baltimore, Md.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
B. L. Freedlander, M.D., *Director of Cancer Research*, Mount Zion Hospital, San Francisco 15, Calif.	The Effect of Tobacco Extracts on the Carcinogenic Effect of Methylcholanthrene and of Ultraviolet Light; the Effect of Tobacco Extracts on Carcinogenic Effect of Urethane-Induced Pulmonary Adenomas in Strain A Mice; the Use of Various Sulphydryl Compounds as Neutralizing or Counteracting Agents for the Possible Carcinogenic Effect of Tobacco Extracts in Mice.
Jack Freund, M.D., *Lecturer in Pharmacology, Assistant in Medicine*, Medical College of Virginia, Richmond 19, Va.	Correlation of Multitechnical Procedures Performed on the Peripheral Circulation of Normal Individuals in Recumbent and Erect Positions and After Exercise Before and After Sham and Actual Smoking.
A. Clark Griffin, Ph. D., *Head of Biochemistry Department*, M.D. Anderson Hospital and Tumor Institute, University of Texas Medical Center, Houston 25, Tex.	The Effect of Exposure to Cigarette Smoke on the Induction of Cancer by Chemical Compounds.
Carl C. Gruhzit, Ph.D., M.D., *Associate in Physiology and Pharmacology*, University of Pennsylvania Graduate School of Medicine, Philiadelphia 4, Pa.	Pharmacologic Study of Nicotine and Related Alkaloids.
H. B. Haag, M.D., Professor of Pharmacology, Medical College of Virginia, Richmond 19, Va.	Preparation for Publication of a Book on the Biologic Aspects of Tobacco and its Smoke.
Joseph H. Hafkenschiel, M. D., Director of Cardiopulmonary Unit, Lankenau Hospital, Philadelphia 31, Pa.	Measurement of Coronary Blood Flow, Cardiac Work and Cardiac Oxygen and Carbohydrate Metabolism in Normotensive Subjects Before and After Intravenous Nicotine and After Smoking Standard Cigarettes.
Herbert R. Hawthorne, M. D., Chairman of Department of Surgery, University of Pennsylvania Graduate School of Medicine, Philadelphia 4, Pa.	Attempts To Induce Pulmonary Neoplasms in Experimental Animals by Exposure of the Tracheo-Bronchial System to Tobacco Smoke.

Grantee and institution	*Project title*
Clark W. Heath, M. D., Physician, Department of Hygiene, Harvard University, Cambridge 38, Mass. (now directed by Dana L. Farnsworth, M.D., Henry K. Oliver Professor of Hygiene and Director of University Health Services)	Personality and Smoking in College Graduates: A Fifteen-Year Followup Study.
F. Homburger, M. D., Research Professor of Medicine and Director, Cancer Research and Cancer Control Unit, Tufts University School of Medicine, New England Medical Center, Boston 11, Mass.; Scientific Associate, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.	A Study of the Effects of Cigarette Tars Upon the Behavior of Transplantable Tumors in Rodents Employing a New Technique of Intrauterine Tumor Transplantation.
Jerry Hart Jacobson, M. D., Director of Electrophysiology, New York Eye and Ear Infirmary, New York 3, N. Y.	A Comparison of Electroretinography as a Means of Evaluating the Effect of Vasoconstrictor Drugs Upon Cerebral and Retinal Circulation With Other Techniques for This Determination.
Ancel Keys, Ph.D., Professor of Physiological Hygiene and Director, Laboratory of Physiological Hygiene, University of Minnesota School of Public Health, Minneapolis 14, Minn.	Characteristics of Men, Including Smoking, in Populations Differing in the Incidence of Coronary Heart Disease.
Joseph B. Kirsner, M. D., Professor of Medicine, University of Chicago School of Medicine, Chicago, Ill.	The Effect of Tobacco Smoking Upon Basal Gastric Secretions in Man.
Kenneth P. Knudtson, M. D., Assistant Chief, Laboratory Service, Veterans Administration Hospital, Seattle, Washington; Professor of Pathology, University of Washington Medical School, Seattle, Wash.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Marvin Kuschner, M. D., Professor of Pathology, New York University College of Medicine; Director of Pathology, Bellevue Hospital, New York 16, N. Y.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Thomas C. Laipply, M. D., Associate Professor of Pathology, Northwestern University Medical School, Chicago, Ill.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Paul S. Larson, Ph. D., Professor of Pharmacology, Medical College of Virginia, Richmond 19, Va.	Enzymatic Transformations of Nicotine.
Cecilie Leuchtenberger, Ph. D., Associate Professor of Cytology, Western Reserve University School of Medicine, Cleveland 6, Ohio.	Quantitative Analysis of Nucleoproteins in Tissues From Animals Subjected to Tobacco Smoke by Microspectrophotometry and Interference Microscopy Correlated with Cytological and Histological Studies.
Kelly T. McKee, M. D., Associate Professor of Medicine, Medical College of South Carolina, Charleston, S. C.	Study of Lung Function in Smokers and Nonsmokers.
David E. Mann, Jr., Ph. D., Associate Professor of Pharmacology, Temple University School of Pharmacy, Philadelphia 40, Pa.	Effect of Tobacco Smoke and Tobacco Residues on Methylcholanthrene-Induced Skin Carcinogenesis in Mice.
Hugh Montgomery, M. D., Associate Professor of Medicine, University of Pennsylvania Medical School, Philadelphia 4, Pa.	Influence of Tobacco Smoking on the Blood Flow of Skin and of Muscles of Extremities in Sympathectomized and Unsympathectomized Subjects.

Grantee and institution	*Project title*
George E. Moore, Ph. D., M. D., Director, Roswell Park Memorial Institute, Buffalo 3, N. Y.	An Investigation of the Physiological Effects of Direct Inhalation of Tobacco Smoke by Laboratory Animals and the Study of the Biological Response of Laboratory Animals to Continuous Ingestion of Diet-Tobacco Product Mixtures.
Hurley Lee Motley, M. D., Professor of Medicine and Director, Cardio-Respiratory Laboratory, University of Southern California School of Medicine, Los Angeles 17, Calif.	A Study of the Effects of Smoking on Pulmonary Function.
William S. Murray, Sc. D., Research Associate and Administration Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine	The Production of Genetically Controlled Animals and Tumors for Use in Experimental Research on Tobacco in Relation to Health by (*a*) the Expansion of Known Inbred Stocks and Sources of Tumor Supply; (*b*) the Production of Such Hybrids or Heterozygous Types as Become Necessary; and (*c*) the Relation of this Material to Specific Experimental Work at the Laboratory.
H. R. Pratt-Thomas, M. D., Professor of Pathology, Medical College of South Carolina, Charleston, S. C.	Application of a New Bio-Assay Technique in Examination of Cigarette Smoke Condensates for Possible Carcinogens.
	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Victor Richards, M. D., Professor of Surgery, Executive Head, Department of Surgery, Stanford University School of Medicine, San Francisco 15, Calif.	A Comparative Study of the Effects of Whole and Fractionated Extracts of Cigarette Smoke and Those of Known Carcinogens on (1) The Cytology and Nuclear DNA Content of Epidermis in Various Strains of Mice and/or (2) the Cytology and Nuclear DNA Content of Lung and Epithelium of the Bronchial Tree of Mice and Hamsters.
R. H. Rigdon, M. D., Professor of Pathology and Director, Laboratory of Experimental Research, University of Texas Medical Branch, Galveston, Tex.	Study of the Effect of Methylcholanthrene on the Tissues of the Duck with Emphasis on the Reaction in the Trachea When Compared With the Skin of the Body and the Web of the Foot.
William O. Russell, M. D., Pathologist-in-chief, M. D. Anderson Hospital and Tumor Institute, University of Texas Medical Center, Houston 25, Tex.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Paul D. Saltman, Ph. D., Assistant Professor of Biochemistry, University of Southern California School of Medicine, Los Angeles 7, Calif.	The Enzymatic Mechanism for the Dark Fixation of CO_2 by Tobacco.
Isaac Schour, D. D. S., Ph. D., Sc. D., Professor of Histology and Embryology and Dean, University of Illinois College of Dentistry, Chicago 12, Ill.	Histologic Changes in the Oral, Pharyngeal and Nasal Tissues of Experimental Animals Subjected to Tobacco Smoke.
Maurice E. Segal, M. D., Clinical Professor of Medicine, Tufts University School of Medicine, Boston, Mass.; Director, Department of Inhalation Therapy, Boston City Hospital, Boston, Mass.	Effects of Cigarette Smoking on Lung Function in Normal Subjects and Patients With Certain Respiratory Disease Conditions.

Grantee and institution	*Project title*
Charles E. Sherwood, M. D., Assistant Professor of Radiology, University of Rochester School of Medicine and Dentistry, Rochester, N. Y.	Investigation Into the Natural History of Carcinoma of the Lung With Particular Reference to the Radiographic Appearance of Such Processes, the Earliest Manifestation of Cancer on Chest X-ray Photographs and the Tabulation of the Relationship of Smoking Habits and Occupation With the Incidence of Lung Cancer.
David L. Simon, M. D., Instructor in Medicine and Fellow in Cardiovascular Research, University of Cincinnati College of Medicine and Cardiac Laboratory, Cincinnati General Hospital, Cincinnati 29, Ohio.	The Effects of Chewing Tobacco on the Cardiovascular System of Man.
Sam Sorof, Ph. D., Research Associate, The Institute for Cancer Research and Lankenau Hospital Research Institute, Philadelphia 11, Pa.	Chemical and Physical Studies of the Tissue Proteins Involved in Chemical Carcinogenesis.
Frederick J. Stare, Ph. D., Professor of Nutrition, Harvard School of Public Health, Boston 15, Mass.	Experimental Studies of Cancer Utilizing a New Technique To See if Various Tars Extracted From Tobacco May Incite the Formation of Lung Tumors.
Marion B. Sulzberger, M. D., Professor and Chairman, Department of Dermatology and Syphilogy, New York University Postgraduate Medical School; Director, New York Skin and Cancer Unit, New York University-Bellevue Medical Center, New York 16, N. Y.	Investigation of the Effects of Tobacco on the Human Vascular System in Living Volunteers; and in Particular of the Possibility that Certain Tobacco Effects are Based on Peculiar Allergic Susceptibility of Specific Individuals Rather Than Upon Obligatorily Toxic Products in Tobacco Smoke.
Caroline Bedell Thomas, M. D., Associate Professor of Medicine, Johns Hopkins University School of Medicine, Baltimore 5, Md.	The Significance of Different Individual Patterns of Circulatory Response to Cigarette Smoking.
Janet Travell, M. D., Associate Professor of Clinical Pharmacology, Cornell University Medical College, New York 21, N. Y.	Electrocardiographic Effects of Nicotine in the Rabbit with Experimental Coronary Arteriosclerosis.
E. D. Warner, M. D., Professor of Pathology, State University of Iowa College of Medicine, Iowa City, Iowa.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Richard L. Wechsler, M. D., Clinical Physiologist, Montefiore Hospital Institute of Research, Pittsburgh 13, Pa.	Effect of Cigarette Smoking on Cerebral Blood Flow, Cerebral Metabolism, Blood Gases, Blood pH, Arterial Pulse Pressure Curves, Electrocardiograms, and Electroencephalograms.
Russell W. Weller, M. D., Pathologist, Memorial Hospital, West Chester, Pennsylvania; Pathologist, Community Hospital, Ephrata, Pennsylvania; Associate Professor of Pathology, Hahnemann Medical College, Philadelphia 2, Pa.	Pathologic-Anatomic Study of Cellular Changes in Human Bronchi.
Simon H. Wender, Ph. D., Research Professor of Chemistry, University of Oklahoma Research Institute, Norman, Okla.	A Qualitative and Quantative Study of the Individual Polyphenol Content of Cigarette Tobacco and of the Smoke and "Tars" Resulting from Cigarette Smoking, and Also Study of the Fate of These Compounds in the Animal Respiratory System.

Grantee and institution	*Project title*
J. Edwin Wood, M. D., Instructor in Medicine, Boston University School of Medicine and Robert Dawson Evans Memorial Department of Clinical Research and Preventive Medicine, Massachusetts Memorial Hospitals, Boston 18, Mass.	The Effect of Prolonged Inhalation of Tobacco Smoke and of Prolonged Abstinence from the Use of Tobacco on the Peripheral Vascular Response to Acute Inhalation of Tobacco Smoke in Man.

Principles and conditions of operation

Before discussing in detail the research program, it will first be wise to outline briefly the conditions under which the Scientific Advisory Board operates. These involve the following three relationships:

1. *Relation to the Tobacco Industry Research Committee.*—When the Scientific Advisory Board was organized and its participants accepted membership, it was clearly stated by the Tobacco Industry Research Committee that the Board would have complete freedom in all of its activities. The Committee must, of course, determine the total amount of funds which will be made available for research activities. The Board, however, was not to be restricted or in any way influenced in preparing or making its recommendations, either general or detailed. These policies have been observed and all of the Board's recommendations have been accepted by the TIRC.

2. *Relation to recipients of support.*—In preparing guiding principles for grantees, the Scientific Advisory Board and Tobacco Industry Research Committee followed the most liberal examples they could find recorded by other granting agencies.

A copy of "Statement of Conditions and Terms Under Which Project Grants Are Made" is given to all grant recipients, as well as anyone else interested. This statement includes these points:

"Research policy and programming are the responsibility of the Scientific Advisory Board * * *

"The Committee desires to have scientists work with the greatest freedom and without domination of any kind. It will make no attempt to direct the administration of the project once started, to influence its course or to control its results other than to be assured that the funds are properly expended for the purposes of the grant and that all findings are reported in accordance with the best scientific practice * * *

"Grantees are to furnish a report of activities semiannually. These need not be extensive but should be sufficiently informative to permit the Scientific Advisory Board to the Tobacco Industry Research Committee to know what is being accomplished * * *

"The Tobacco Industry Research Committee approves the initial presentation by the investigator of research results only in accepted medical and scientific journals or before accepted medical or scientific societies. It has no objection to dissemination to the public of any or all final conclusions from projects in these ways * * *

3. *Relation of Scientific Advisory Board members to each other.*—The Board recognized that its size must be kept small to insure the great but intangible creative value of intimate mutual friendship and understanding between its members. Such a relationship guarantees the highest degree of attendance, interest, interchange of ideas and conservation of time and effort.

However, a small board also requires free and unrestricted use of consultant, either individually or in groups, in order to have access to all available information and to obtain much necessary advice in planning new lines of research. Later in the report it will be seen how helpful these consultants have been and will continue to be.

Another important factor in the activities of the Scientific Advisory Board is the division of labor according to the special interests and aptitudes of the individual members. Preliminary review of applications for grants-in-aid, interviews with prospective grantees, and contacts with medical and scientific groups are examples of the sort of work accomplished by Board members between meetings.

Recommendations based on such contacts are discussed and acted upon by the full Board. In all of this review function, the services of Dr. Robert C. Hockett, Associate Scientific Director of the Committee, have been invaluable. The Board maintains personal contact with each major grantee, for only by so doing can the Board develop and maintain the type of "front line" information so necessary in such a complex and uncharted field.

The research program

The first responsibility of an advisory board is to take time to think about all phases and aspects of the responsibility with which it is charged.

The broad definition of "tobacco in relation to health" involved far more scope than any one person or small board could hope to cover in detail by expert knowledge.

Out of discussion by the Scientific Advisory Board evolved a broad "master plan" which included three main areas of investigation as follows:

1. *The physical and chemical composition of tobacco* and accompanying products, such as cigarette papers and additives, and of their combustion and pyrolysis products.

2. *Tissue changes in humans and in animals*, in normal life and under laboratory conditions; subjected to various types, duration and intensity of exposure to various tobacco and derivatives, and other potential irritants. Tissues of special interest are those of the lungs, cardiovascular system, mouth, throat, endocrine glands, and other organs in subjects of various ages, sex and strains.

3. *Smoking and other tobacco habits, and the emotional and physical makeup of smokers*, with respect to establishment, duration and intensity of tobacco use, and correlation of these data with metabolic, glandular and nervous types under various degrees of stress and challenge and of other environmental factors.

Broad and inclusive as this three-area program is, it defined the field of general interest and, from this, the Board could determine the pertinency of grant applications and create the more definitive research program that has since guided our efforts.

While the Board operates on policy matters and grant applications as a whole, each member has accepted special responsibilities to help guide and develop the program in one or more of the nine specific fields of research into which the $1,500,000 so far appropriated by the TIRC are now being channeled.

A discussion of these nine fields follows:

I. Preparation and analysis of tobacco derivatives (Drs. Cattell, Jacobson, and Kotin)

Definition and establishment of experimental conditions which simulate those under which tobacco is normally smoked or otherwise used was needed since the products of partial combustion and distillation of any organic material will vary in composition with conditions of the process itself.

If the smoke condensates, tars or derivatives have been prepared or stored under abnormal conditions, experimentation with them has no bearing on the practical evaluation of tobacco products in relation to health.

The Scientific Advisory Board, with the help of scientists at the various laboratories of the tobacco industry itself, developed a detailed description of the best available smoking machines. This material will be published so that investigators anywhere can reproduce the smoking techniques and increase the significance, applicability and comparability of their work.

Much more research needs to be carried out on the isolation, identification and analysis of the great number of chemical substances derived from the fractionation of tobacco and cigarette paper products. Such research is being encouraged among independent scientists as well as in the tobacco industry's own laboratories.

In planning research on substances in tobacco, the Board received valuable advice and aid from Dr. A. J. Haagen-Smit, of the California Institute of Technology. Dr. Haagen-Smit pointed out that isolation and chemical identification of tobacco fractions could proceed successfully, but he foresaw a "bottleneck" in the absence or inadequacy of methods for assaying in humans or animals the possible effects of substances so obtained.

A considerabel number of investigators in many laboratories will probably continue to concern themselves with isolation and identification of various tobacco fractions. Such efforts may be helpful, but until much more effective methods of assaying their effects on lung and other tissues are devised, their contributions to experimental analysis of the problem will be incomplete and inadequate.

One of the Board's efforts to deal with the deficiency are discussed under Section V dealing with tissue culture.

II. Use of carbon ^{14} as a tracer element in tobacco (Dr. Jacobson)

Application of our recently acquired knowledge of using induced radioactive isotopes as tracer elements has been given serious study. For conducting experiments on the analysis of tobacco, plants grown in a carbon dioxide atmosphere containing C^{14}, one of the radioactive isotopes, are potentially valuable.

The Board has surveyed existing facilities for growing such plants and has considered several steps to be taken should any extension or increase in such facilities become essential.

Because of the technical difficulties in "processing" tobacco so grown and because of the small amount of material that would eventually be available, any active program of increased production has been postponed until the need becomes more evident.

III. Pathologic-anatomic research (Drs. Reimann, Lynch, Kotin)

The Board early recognized a need for greatly increased knowledge of the pathology of the human lung. Study of human lung tissues was desired to observe the kinds and extent of abnormal changes and their possible relation to occupation, place of residence, past ailments and the like. No one institution had sufficient data for a wide coverage of this field, nor did it seem probable that any one laboratory could alone obtain a sufficient mass of data.

The Board, therefore, planned a cooperative study to include twelve clinical-pathological laboratories. It called together the following pathologists for a conference to discuss the feasibility of the plan, and, if approved, to put it into operation:

Dr. D. Murray Angevine, University of Wisconsin Medical School, Madison, Wis.
Dr. E. M. Butt, Los Angeles County Hospital and University of Southern California School of Medicine, Los Angeles, Calif.
Dr. William H. Carnes, Stanford University School of Medicine, San Francisco, Calif.
Dr. Russell S. Fisher, Office of the Chief Medical Examiner, State of Maryland and University of Maryland School of Medicine, Baltimore, Md.
Dr. Russell L. Holman, Louisiana State University School of Medicine, New Orleans, La.
Dr. Kenneth P. Knudtson, Veterans Administration Hospital and University of Washington Medical School, Seattle, Wash.
Dr. Marvin Kuschner, New York University-Bellevue Medical Center, New York, N. Y.
Dr. H. R. Pratt-Thomas, Medical College of South Carolina, Charleston, S. C.
Dr. William O. Russell, M. D., Anderson Hospital, the University of Texas Medical Center, Houston, Tex.
Dr. E. D. Warner, State University of Iowa, College of Medicine, Iowa City, Iowa
Dr. William B. Wartman, Wesley Memorial Hospital and Northwestern University Medical School, Chicago, Ill.
Dr. Russell W. Weller, the Hahnemann Medical College and Hospital of Philadelphia, Pa. (now at Ephrata, Pa., Community Hospital)

A preliminary conference enabled the Board to organize the project, and produced frank and free discussion of techniques and experience which proved to be most fruitful and stimulating.

Two other conferences of these pathologists has been held with practically 100 percent representation and with steadily growing evidence that the principles underlying such a cooperative venture are sound and creative.

This pathologic-anatomic study of human lungs was begun in February 1955 and renewed for a second year in February 1956.

The twelve participants are widely scattered geographically for the purpose of randomizing the effects of environment upon the subjects of study.

Special attention is also being given to the nature and extent of lesions indicative of previous infection of the lung tissue.

Statistical study of the possible history of previous infection in relation to lung cancer may be an important matter and may give interesting leads for more extensive pathological investigations.

To make the data derived from the different laboratories comparable and standardized, a uniform system of recording was devised by Miss Mary Burke, the Scientific Advisory Board's statistical assistant, after consultation with Dr. E. B. Wilson, a member of the Board, and with the twelve cooperating investigators. This will greatly facilitate statistical analysis of the pooled data obtained and will thus augment the value of the effort.

IV. Direct application of tobacco derivatives (Drs. Reimann, Little, Jacobson)

Animals of different genetic background not only produce different characteristic incidence of lung and other types of cancer, but also react in different characteristic ways and degrees to challenging substances applied directly on the skin or elsewhere.

The Board, therefore, agreed on the policy of advocating the use of experimental animals of known genetic composition wherever possible. With this in mind, assistance is being given through a grant to the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine, to help stabilize and increase production of the desired strains and hybrids of mice, which are the most widely used and best analyzed experimental animals.

Recognizing also the great difference in response to chemical challenge as shown by different species of animals, the Board is interested in increasing knowledge of species susceptibility as well as strain susceptibilities.

In the same way, various sites at which the challenge is applied require careful study. The high degree of reactability of the skin of mice, rats and rabbits contrasts interestingly with the relative inertia of the skin of guinea pigs or of primates. Relationships of this sort indicate the need for extreme conservatism in extrapolating results obtained and conclusions drawn from one species to another, and even from the reactions of one tissue or organ system to another in the field of carcinogenesis.

Only an extensive series of experiments with animals in which species, strain, sex, age, dosage, and other factors are controlled and evaluated can give the sort of exact information on which reasonably safe conclusions can be based.

Another type of exposure is through inhalation. Dr. Kotin has had long experience in this field and is acting as a subcommittee of one to review applications relating to inhalation experiments.

Close contact is maintained by the Board within the whole field of experimental exposure. Efforts are continually being made to evaluate both the positive and negative results which various investigators have reported and are obtaining at present.

At best, work in animal exposure can produce only indirect evidence on the etiology of human lung cancer. However, there is real value in animal work; it can and often does sugegst problems and pathways in clinical research.

V. *Tissue culture Research* (*Drs. Jacobson, Lynch, Comroe, Reimann, Little*)

Since it is difficult to observe or to experiment on the animal or human lung or on the cardiovascular system *in situ*, efforts are being made to discover and develop techniques by which experimenters can obtain access to living lung tissue under conditions which alow direct observation and manipulation.

As was pointed out by Dr. Haagen-Smit, the real problem involved in the functional study of the derivatives of tobacco is in finding better and more accurate ways of determining the effects of these chemical substances on living tissue.

The Board, therefore, believes that among its major responsibilities is the development of new methods of bioassay that will allow the direct observation of reactions of human and animal tissue of various ages and origin.

To survey the possibility of initiating and supporting research into the maintenance and growth of lung tissue and of cardiovascular tissue in vitro, eleven of the leading American investigators in the field of tissue culture were invited by the Board to New York for an informal conference, exchange of views and discussion. Those in attendance were:

Dr. Lawrence Berman, Professor of Medicine, Wayne University College of Medicine, Detroit, Mich.

Dr. John J. Biesele, Head, Cell Growth Section, Sloan-Kettering Institute for Cancer Research, New York, N. Y.

Dr. Harry Eagle, Chief, Experimental Therapeutics, National Microbiological Institute, National Institutes of Health, Bethesda, Md.

Dr. Wilton R. Earle, Head, Tissue Culture Section, Laboratory of Biology, National Cancer Institute, National Institutes of Health, Bethesda, Md.

Mrs. Ruth Geiger, Neuropsychiatric Institute, University of Illinois, Chicago, Ill.

Dr. George O. Gey, Assistant Professor of Surgery, Johns Hopkins University School of Medicine, Baltimore, Md.

Dr. Margaret R. Murray, Associate Professor of Anatomy, Columbia University College of Physicians and Surgeons, New York, N. Y.

Dr. C. M. Pomerat, Professor of Cytology, the University of Texas, Medical Branch, Galveston, Tex.

Dr. Joseph J. Quilligan, Jr., Chief, Virus and Tissue Culture Laboratories, College of Medical Evangelists, Loma Linda, Los Angeles, Calif.

Dr. Charity Waymouth, Research Fellow, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.

Dr. Philip R. White, Research Associate, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.

The conference was most rewarding in its delineation of the present status of the art and science of cell culture, and encouraging with respect to its potential applications to the problems at hand. Following the conference, the Scientific Advisory Board recommended an appropriation be made to assist the summer training program of the Tissue Culture Association and to provide several fellowships for study of human lung tissues in vitro. The appropriation was subsequently approved by the Tobacco Industry Research Committee.

VI. Cardiovascular and coronary research (Drs. Cattell, Comroe) and chest physiology (Dr. Rienhoff)

Various relationships between nicotine, and perhaps other tobacco derivatives, and vascular and cardiac function have long been hypothesized and rumored.

Examination of the literature reveals a distinct need for further research to take full advantage of newer experimental techniques and to fill very real gaps in our knowledge in this field.

So evident is the need for careful and critical planning based on frank discussion of our present level of information that the Board held a conference in New York of research workers who are grantees of the Tobacco Industry Research Committee or who are associated with its program, together with a few guests. The following persons attended:

Grantees and Associates

Dr. Ernest O. Attinger, Boston City Hospital, Boston, Mass.
Dr. Richard J. Bing, Medical College of Alabama, Birmingham, Ala.
Dr. John W. Eckstein, Massachusetts Memorial Hospitals, Boston, Mass.
Dr. Frank C. Ferguson, Jr., Albany Medical College, Albany, N. Y.
Dr. Vincent J. Fontana, New York University-Bellevue Medical Center, New York, N. Y.
Dr. Jack Freund, Medical College of Virginia, Richmond, Va.
Dr. Carl C. Gruhzit, University of Pennsylvania, Graduate School of Medicine, Philadelphia, Pa.
Dr. Joseph H. Hafkenschiel, the Lankenau Hospital, Philadelphia, Pa.
Dr. Kelly T. McKee, Medical College of South Carolina, Charleston, S. C.
Dr. Hugh Montgomery, University of Pennsylvania, Philadelphia, Pa.
Dr. Hurley Lee Motley, University of Southern California, Los Angeles, Calif.
Dr. Walter Redisch, Goldwater Memorial Hospital, New York, N. Y.
Dr. Seymour H. Rinzler, Cornell University Medical College, New York, N. Y.
Dr. Hans Rottenstein, University of Pennsylvania, Philadelphia, Pa.
Dr. Maurice S. Segal, Boston City Hospital, Boston, Mass.
Dr. Janet Travell, Cornell University Medical College, New York, N. Y.
Dr. Richard Wechsler, Montefiore Hospital Institute of Research, Pittsburgh, Pa.
Dr. J. Edwin Wood, Massachusetts Memorial Hospitals, Boston, Mass.

Guests

Dr. Frank Davis, Johns Hopkins Hospital, Baltimore, Md.
Dr. Charles D. Marple, American Heart Association, Inc., New York, N. Y.
Mr. Felix E. Moore, National Heart Institute, Bethesda, Md.

Three half-day sessions were held. One was devoted to the heart and coronary circulation, one to the peripheral circulation, and one to lung function and pulmonary circulation. Preliminary findings were compared and newer methodologies were discussed at some length.

VII. Buccal and gastrointestinal research

The Board wants further research into tobacco in relation to these body sites, and is reviewing the field with the object of developing an orderly and coordinated program, rather than to support scattered and disconnected investigations that may be lacking in pertinency. Although priority has had to be given to other areas of research, and the program of research on the buccal and gastrointestinal sites is in its early stages, a few basic projects have been supported and progress reported. More attention to these phases of research is contemplated.

VIII. Smoking habits—Physical and psychological types (Drs. Wilson, Little)

Another fascinating but little-known field for investigation is into the types of persons who react to the strain, stress and frustration of intense modern living by the formation of habits of excess.

Common sense and everyday experience have proved that individuals differ from one another in their ease and degree of self-control and adjustment and

in their tempo of living. The same individual may vary in type and extent of reaction at different ages and under different kinds and intensity of stress.

These differences may reflect types and grades of physiopsychological interrelations and degrees of balance. Such factors would naturally affect the incidence of basic psychosomatic disturbances and diseases. They would thus be an important element in increasing the health risk and death rate of the individuals who present more critical phases of reaction to strain and stress.

It is, of course, difficult to reduce problems of this sort to exact scientific proportions and to devise sufficiently definite techniques of continued observation and measurement.

There are, however, several population groups with which studies have been progressing for some time under careful and well-trained investigators who have a keen appreciation of the difficulties involved. The Board has made arrangements to assist and implement certain of these studies so that smoking habits can and will be recorded and can later be correlated with various physiological and psychological types which are identified and defined.

Since we already know that hormonal secretion may be influenced by emotional or psychological stress and that hormonal secretion also can influence the origin and development of certain types of cancer, investigations in this field of "personality" may reveal important evidence of correlations between "personality type" and "health risk" in which habits of excess are "reflections" rather than being "causes" of instability, disease or death.

IX. Fellowship program

Recognizing the widely admitted serious scarcity of young, well-trained research workers in the biological sciences, the Board recommended research fellowships of $500 each for students in 52 medical schools in 1955. The Tobacco Industry Research Committee approved use of funds for this purpose.

This program was received with enthusiasm and for 1956 the TIRC, on recommendation of the Board, is making possible the award of 20 additional fellowships above the 1955 total.

Students who receive these fellowships are chosen directly by the dean or other administrative officers of the schools and assigned to research projects during summer or other off-terms. They may work on any problem selected by them and approved by the deans. The Committee keeps entirely out of contact with both students and schools until the selection has been made and the problems chosen. The Committee's only request is that the school notify it of the name of the student, the subject of the research and that, at the end of the fellowship period, an informal report be made on the purpose and progress of the research.

CONCLUSIONS

This report is designed to describe the current status and extent of the research program developed by the Scientific Advisory Board to the Tobacco Industry Research Committee. Clearly, any attempt to evaluate at this time the results of existing research projects would be extremely premature.

However, these general observations, based on existing scientific literature, may be made:

1. The very extent of the practice of smoking among our population is proof that it provides some kind of pleasure and satisfaction to those who engage in the habit. How and why it does so are among the uncertainties that need exploring. However, like any other human activity, the use of tobacco affects some individuals in different ways. The same may be said, of course, of almost anything else we come into contact with, including the food we eat.

2. Any possible role of smoking in the etiology of lung cancer remains an unresolved question. It cannot be said that smoking has been absolved from suspicion; neither have the charges that smoking has a role in lung cancer causation been proven. So many unknowns still obscure the whole field of cancer causation that it is not possible at this stage to say either "this is it" or "this is not it" about any single factor.

3. In the cardiovascular and coronary disease fields, the uncertainties regarding cause and even treatment remain innumerable. The heart attack experienced last fall by President Eisenhower dramatically brought to public attention how little we really know about the cause or prevention of heart ailments. Whether smoking has any significant part, and indeed, whether such a part may be positive or negative, is only one of the many areas in cardiovascular investigation that should be explored.

4. Preliminary study indicates that the physical, emotional and mental characteristics of people who become smokers may be of more significance than has

been so far recognized. It is a fact that many smoking studies involving humans have ignored or neglected personal information other than their smoking habits and one or two of the more obvious kinds of physical ailments.

The Tobacco Industry Research Committee's complete willingness to permit the Scientific Advisory Board to initiate and sponsor basic research into our health problems as well as to finance applied research into specific problems should prove a genuine and unselfish contribution to the gaining of new knowledge with a maximum of speed and efficiency.

ATTACHMENT F

TOBACCO INDUSTRY RESEARCH COMMITTEE

Original grants

No.	Grantee	Total value	Date activated
	1954		
8	Mann, David, Temple University School of Pharmacy	$5,500.00	Oct. 1, 1954.
6	Saltman, Paul D., University of Southern California	7,776.00	Do.
31	Clarke, Hans T., Columbia University, College of Physicians and Surgeons.	19,958.00	Nov. 1, 1954.
24	Haag, H. B., Medical College of Virginia	38,901.60	Nov. 1, 1954 (27 months).
7	Griffin, A. Clark, M. D., Anderson Hospital, University of Texas.	5,960.00	Dec. 1, 1954.
28	Moore, George E., Roswell Park Memorial Institute	30,542.40	Dec. 6, 1954.
	1955		
32	Cerecedo, Leopold, Fordham University	8,360.00	Jan. 1, 1955.
12	Freedlander, B. L., Mount Zion Hospital	8,900.00	Do.
42	Heath, Clark W., Harvard University	15,880.00	Do.
2	Segal, Maurice S., Tufts University School of Medicine	22,000.00	Do.
20	Stare, Frederick J., Harvard University School of Public Health.	13,613.00	Do.
54	Barnes, F. W., Jr., Johns Hopkins University School of Medicine.	11,000.00	Feb. 1, 1955.
25	Hawthorne, H. R., University of Pennsylvania Graduate School of Medicine.	30,000.00	Do.
27	McKee, Kelly T., Medical College of South Carolina	7,900.00	Do.
50	Pathologic-Anatomic Study A. R. W. Weller, $9,170.70, Feb. 1, 1955. B. W. O. Russell, $7,884, Feb. 1, 1955. C. K. P. Knudtson, $5,400, Feb. 1, 1955. D. R. S. Fisher, $8,855, Aug. 1, 1955. E. D. M. Angevine, $6,600, Feb. 1, 1955. F. E. D. Warner, $6,156, Feb. 1, 1955. G. E. M. Butt, $6,480, Feb. 1, 1955. H. R. L. Holman, $10,075, Feb. 1, 1955. I. T. C. Laipply, $5,724, Feb. 1, 1955. J. H. R. Pratt-Thomas, $6,875, Feb. 1, 1955. K. W. H. Carnes, $7,245, Mar. 1, 1955. L. Marvin Kuschner, $8,230, Feb. 1, 1955.	88,694.70	1955.
33	Sulzberger, Marion B., New York University Post-graduate Medical School.	15,000.00	Feb. 1, 1955.
44	Falk, Hans L., University of Southern California School of Medicine.	7,560.00	Mar. 1, 1955.
53	Montgomery, Hugh, University of Pennsylvania Medical School.	10,667.50	Do.
52	Murray, W. S., Roscoe B. Jackson Memorial Laboratory.	43,112.00	Do.
62	Hafkenschiel, J. H., the Lankenau Hospital	14,773.00	Apr. 1, 1955.
63	Simon, David L., University of Cincinnati, Cincinnati General Hospital.	2,401.15	Apr. 15, 1955.
80	Fellowship program	25,300.00	1955.
74	Leuchtenberger, Cecilie, Western Reserve University	23,199.24	June 1, 1955.
14	Motley, Hurley Lee, University of Southern California	21,000.00	Do.
59	Cooper, Philip, Boston University School of Medicine	15,000.00	June 15, 1955.
91	Bing, Richard J., the Medical College of Alabama	11,110.00	July 1, 1955.
66	Gruhzit, Carl C., University of Pennsylvania Graduate School of Medicine.	13,915.00	Do.
51	Pratt-Thomas, H. R., Medical College of South Carolina.	8,634.50	Do.
72	Rigdon, R. H., University of Texas Medical Branch	5,390.00	Do.
68	Sherwood, Charles E., the University of Rochester School of Medicine and Dentistry.	5,750.00	Do.
92	Travell, Janet, Cornell University Medical College	8,910.00	Do.
97	Wood, J. Edwin, Massachusetts Memorial Hospitals	4,000.00	Do.
93	Wechsler, Richard L., Montefiore Hospital Institute of Research.	10,000.00	July 20, 1955.
	Gertler, Menard M	1,000.00	Aug. 23, 1955
79	Kirsner, Joseph B., the University of Chicago	18,375.50	Sept. 1, 1955.
84	Schour, Isaac, University of Illinois College of Dentistry.	17,687.69	Do.

Original grants—Continued

No.	Grantee	Total value	Date activated
	1955		
104	Brozek, Josef,, University of Minnesota	15,000.00	Oct. 1, 1955
69	Ferguson, Frank C., Jr., Albany Medical College of Union University.	12,995.00	Oct. 1, 1955 (19 months).
29B	Homburger, Freddie, Tufts University School of Medicine.	25,760.00	Oct. 1, 1955.
85	Sorof, Sam, the Institute for Cancer Research and the Lankenau Hospital Research Institute.	16,583.00	Do.
89	Thomas, Caroline B., the Johns Hopkins University School of Medicine.	4,000.00	Do.
70	Freund, Jack, Medical College of Virginia	17,435.00	Oct. 15, 1955.
	1956		
95	Butt, E. M., Los Angeles County General Hospital	13,687.44	Jan. 1, 1956.
4A	Wender, Simon H., University of Oklahoma Research Institute.	8,970.00	Do.
116	Keys, Ancel, University of Minnesota	8,050.00	Feb. 1, 1956.
117	Richards, Victor, Stanford University School of Medicine	34,600.00	Mar. 1, 1956.
134	Lewis, Robert C., University of Colorado School of Medicine (tissue culture).	5,000.00	June 28, 1956.
126	Bonner, James F., California Institute of Technology	9,680.00	July 1, 1956.
128	Larson, Paul S., Medical College of Virginia	29,080.00	Do.
134A	Gey, George O., the John Hopkins Hospital (tissue culture).	8,000.00	July 3, 1956.
123	Jacobson, J. H., New York Eye and Ear Infirmary	4,200.00	Sept. 1, 1956.
86A	Rittenberg, S. C., University of Southern California	4,104.00	Oct. 1, 1956.
137	Bellet, Samuel, Philadelphia General Hospital	10,400.00	Nov. 15, 1956.
134B	Pomerat, C. M., the University of Texas Medical Branch (tissue culture).	7,000.00	Nov. 21, 1956.
	1957		
138	Grossman, Morton I., University of California Medical Center.	8,400.00	Jan. 1, 1957.
142	Saltman, Paul, University of Southern California School of Medicine.	7,560.00	Do.
146	Montgomery, Hugh, University of Pennsylvania School of Medicine.	10,500.00	Mar. 1, 1957.
154	Bock, Fred G., Roswell Park Memorial Institute	14,525.00	Apr. 1, 1957.
141	Kosak, Alvin I., New York University	6,038.00	June 1, 1957.
158	Bowery, Tom G., North Carolina State College	9,000.00	July 1, 1957.
150	Eckstein, John W., State University of of Iowa College of Medicine.	3,795.00	Do.
152	McCants, Charles B., North Carolina State College	3,000.00	Do.
157	Rigdon, R. H., University of Texas Medical Branch	5,278.50	Do.
148	Simon, David L., Cincinnati General Hospital	2,940.00	July 1, 1957 (?).
159	Leuchtenberger, Cecilie, Western Reserve University	22,928.12	Sept. 1, 1957.

Grant renewals

No.	Grantee	Total value	Date activated
	1955		
8R1	Mann, David, Temple University'School of Pharmacy	$1,988.00	Oct. 1, 1955 (7 months).
6R1	Saltman, Paul D., University of Southern California	7,776.00	Oct. 1, 1955.
31R1	Clarke, Hans T., Columbia University College of Physicians and Surgeons.	19,958.00	Nov. 1, 1955.
7R1	Griffin, A. Clark, M. D., Anderson Hospital, University of Texas.	5,850.00	Dec. 1, 1955.
28R1	Moore, George E., Roswell Park Memorial Institute	30,857.63	Dec. 1, 1955 (15½ months).
	1956		
42R1	Farnsworth, Dana L. (Heath), Harvard University	15,880.00	Jan. 1, 1956.
12R1	Freedlander, B. L., Mount Zion Hospital	11,800.00	Do.
2R1	Segal, Maurice S., Tufts University School of Medicine	16,500.00	Jan. 1, 1956 (18 months).
20R1	Stare, Frederick J., Harvard University School of Public Health.	14,863.00	Jan. 1, 1956.
54R1	Barnes, F. W., Jr., Johns Hopkins University School of Medicine.	9,000.00	Feb. 1, 1956.
32R1	Cerecedo, Leopold, Fordham University	8,360.00	Do.
25R1	Hawthorne, H. R., University of Pennsylvania Graduate School of Medicine.	30,000.00	Do.

Grant renewals—Continued

No.	Grantee	Total value	Date activated
	1956		
50R1	Pathologic-anatomic study AR1. R. W. Weller, $9,947, Feb. 1, 1956. CR1. K. P. Knudtson, $6,000, Feb. 1, 1956. DR1. R. S. Fisher, $4,445, Feb. 1, 1956. ER1. D. M. Angevine, $7,245, Feb. 1, 1956. FR1. E. D. Warner, $3,404, Feb. 1, 1956. GR1. E. M. Butt, $7,306, Feb. 1, 1956. HR1. R. L. Holman, $10,200, Feb. 1, 1956. IR1. T. C. Laipply, $5,724, Feb. 1, 1956. JR1. H. R. Pratt-Thomas, $6,875, Feb. 1, 1956. KR1. W. H. Carnes, $6,670, Feb. 1, 1956. LR1. Marvin Kuschner, $8,340, Feb. 1, 1956.	76, 156. 00	1956.
33R1	Sulzberger, Marion B., New York University, Postgraduate Medical School.	15, 000. 00	Feb. 1, 1956.
44R1	Falk, Hans L., University of Southern California School of Medicine.	12, 880. 00	Mar. 1, 1956.
53R1	Montgomery, Hugh, University of Pennsylvania Medical School.	10, 043. 75	Do.
52R1	Murray, W. S., Roscoe B. Jackson Memorial Laboratory.	32, 359. 00	Do.
80R1	Fellowship Program	35, 000. 00	1956.
62R1	Hafkenschiel, J. H., the Lankenau Hospital	7, 938. 00	Apr. 1, 1956.
74R1	Leuchtenberger, Cecilie, Western Reserve University	17, 282. 20	June 1, 1956 (9 months).
14R1	Motley, Hurley Lee, University of Southern California	19, 000. 00	June 1, 1956 (13 months).
91R1	Bing, Richard J., Washington University	9, 790. 00	July 1, 1956.
66R1	Gruhzit, Carl C., University of Pennsylvania Graduate School of Medicine.	13, 915. 00	Do.
51R1	Pratt-Thomas, H. R., Medical College of South Carolina.	8, 634. 50	Do.
72R1	Rigdon, R. H., University of Texas Medical Branch.	5, 290. 00	Do.
68R1	Sherwood, Charles E., the University of Rochester School of Medicine and Dentistry.	5, 750. 00	Do.
92R1	Travell, Janet, Cornell University Medical College	11, 000. 00	Do.
97R1	Wood, J. Edwin, Massachusetts Memorial Hospitals	4, 000. 00	Do.
93R1	Wechsler, Richard L., Montefiore Hospital Institute of Research.	10, 000. 00	July 20, 1956.
79R1	Kirsner, Joseph B., the University of Chicago	9, 188. 00	Sept. 1, 1956 (17 months).
84R1	Schour, Isaac, University of Illinois, College of Dentistry.	17, 687. 69	Sept. 1, 1956.
89R1	Thomas, Caroline B., the Johns Hopkins University School of Medicine.	10, 510. 00	Do.
85R1	Sorof, Sam, the Institute for Cancer Research	18, 855. 00	Oct. 1, 1956.
70R1	Freund, Jack, Medical College of Virginia	13, 077. 00	Oct. 15, 1956 (9 months).
31R2	Gottschall, Gertrude (Clarke), Columbia University, College of Physicians and Surgeons.	16, 458. 00	Nov. 1, 1956.
	1957		
12R2	Freedlander, B. L., Mount Zion Hospital	11, 800. 00	Jan. 1, 1957.
29BR1	Homburger, Freddie, Tufts University School of Medicine.	25, 760. 00	Do.
42R2	McArthur, Charles (Heath-Farnsworth), Harvard University.	7, 500. 00	Jan. 1, 1957 (6 months).
4AR1	Wender, Simon H., University of Oklahoma Research Institute.	8, 970. 00	Jan. 1, 1957.
54R2	Barnes, F. W., Jr., Johns Hopkins University School of Medicine.	9, 500. 00	Feb. 1, 1957.
32R2	Cerecedo, Leopold, Fordham University	10, 925. 00	Do.
25R2	Hawthorne, H. R., University of Pennsylvania Graduate School of Medicine.	30, 000. 00	Do.
50R2	Pathologic-anatomic study (5 months) AR2. R. W. Weller, $4,148, Feb. 1, 1957. BR1. W. O. Russell, $3,288, June 1, 1957. CR2. K. P. Knudtson, $2,502, Feb. 1, 1957. DR2. R. S. Fisher, $3,542, Feb. 1, 1957. ER2. D. M. Angevine, $3,021, Feb. 1, 1957. GR2. E. M. Butt, $3,047, Feb. 1, 1957. HR2. R. L. Holman, $4,253, Feb. 1, 1957. IR2. T. C. Laipply, $2,387, Feb. 1, 1957. JR2. H. R. Pratt-Thomas, $2,867, Feb. 1, 1957. KR2. W. H. Carnes, $2,781, Feb. 1, 1957. LR2. Marvin Kuschner, $3,478, Feb. 1, 1957.	35, 314. 00	1957.
44R2	Falk, Hans L., University of Southern California School of Medicine.	11, 385. 00	Mar. 1, 1957.
24R1	Haag, H. B., Medical College of Virginia	15, 630. 00	Mar. 1, 1957 (17 months).
52R2	Murray, W. S., Roscoe B. Jackson Memorial Laboratory.	32, 359. 00	Mar. 1, 1957.
117R1	Richards, Victor, Stanford University School of Medicine.	19, 000. 00	Do.
104R1	Brozek, Josef, University of Minnesota	15, 000. 00	Apr. 1, 1957.
80R2	Fellowship program	35, 300. 00	1957.
62R2	Hafkenschiel, J. H., the Lankenau Hospital	3, 969. 00	Apr. 1, 1957 (6 months).
116R1	Keys, Ancel, University of Minnesota	8, 800. 00	May 1, 1957.
91R2	Bing, Richard J., Washington University	9, 904. 00	July 1, 1957.
128R1	Larson, Paul S., Medical College of Virginia	23, 142. 90	Do.
51R2	Pratt-Thomas, H. R., Medical College of South Carolina.	8, 310. 50	Do.
92R2	Travell, Janet, Cornell University Medical College	16, 170. 00	Do.

ATTACHMENT G

FOR 1957 REPORT OF THE SCIENTIFIC DIRECTOR OF THE TOBACCO INDUSTRY RESEARCH COMMITTEE

EFFECT OF CIGARETTE SMOKING ON CORONARY BLOOD FLOW AND MYOCARDIAL METABOLISM

By L. M. Bargeron, Jr., M. D., D. Enmke, M. D., F. Gonlubol, M. D., A. Castellanos, M. D., A. Siegel, M. D., and R. J. Bing, M. D., Department of Experimental Medicine, the Medical College of Alabama, Birmingham. Circulation, Vol. 15, No. 2, pages 251–257, February 1957

Abstract

Purpose of studies: Despite the generally accepted actions of nicotine on specific tissue, the effect of smoking on the heart has been a controversial subject, and the literature has been contradictory. The purpose of this investigation was to measure by catheterization the changes caused by cigarette smoking on coronary blood flow, coronary vascular resistace, cardiac output, heart rate, and myocardial extraction of lactate, glucose, pyruvate, and ketones.

Experiments: A statistically significant rise in blood flow occurred after smoking a cigarette in 11 patients, from a mean of 69.8 ml. per min. per 100 gm. of heart muscle to 82.8 ml. Coronary vascular resistance fell significantly from 1.30 mm. to 1.05 mm. Hg per 100 gm. per min. Myocardial oxygen extraction declined from a mean of 12.7 volumes percent to 11.2 volumes percent: since it was proportional to the rise in blood flow, the oxygen usage was not significantly changed. The heart rate increased from an average of 87 to 93. Extraction of glucose fell significantly; of pyruvate fell, but not significantly; and extraction of ketones rose, but not significantly.

Findings: These results indicate that cigarette smoking in normal subjects does not result in constriction of the coronary blood vessels.

Other grantors: United States Public Health Service, Life Insurance Medical Research Fund, and American Heart Association.

CONTRACTILE PROPERTIES OF ACTOMYOSIN THREADS AND BANDS PREPARED FROM DOGS' HEARTS

By L. Dettli and R. J. Bing, Department of Experimental Medicine, the Medical College of Alabama, Birmingham. The American Journal of Physiology, Vol. 187, No. 1, pages 145–150, October 1956.

Abstract

Purpose of study: Previous researchers have been unable to establish a relationship between the shortening of actomyosin threads from animal hearts and the weight lifting capacity which results from application of ATP (adenosine triphosphate), the variable data being attributed to technical difficulties in handling these very breakable and sticky threads. The purpose of the present work is to investigate the dependence of the ATP-induced contraction upon both the load and the protein content of such threads. Four series of experiments were performed with improved apparatus.

Experiment: Series I (38 tests): These duplicated the work of previous workers and were attended by similar scattering of results, hence are not reported. Series II (25 tests): The length of the threads was held constant at 21 mm. Their thickness was varied by depositing different amounts of actomyosin in the solution, and nitrogen content of the threads was determined following contraction. Scattering of results persisted. Series III (30 tests): Exactly 1.5 milliliters of a .5% actomyosin solution was deposited, and the films were compressed to bands exactly 15 millimeters wide. The length of threads was constant at 21 millimeters, and the width varied to obtain different protein contents per unit length. There was a definite inverse linear relationship between shortening and load up to about 4.5 milligrams, above which the bands did not shorten at all and broke within a few minutes. Series IV: Isometric contractions in resting length were studied in a large number of bands. No relationship was found between tension developed and specific nitrogen content. The bands developed tensions up to 60 milligrams but then tore, as did the overloaded bands in Series III. The results therefore are not presented in detail.

Findings: The performance of the actomyosin bands described is limited by the speed of ATS transport into the protein. Therefore, this preparation is not suited for quantitative kinetic studies. However, since the error introduced by

diffusion is constant, the bands are a useful tool in comparative experimental studies.

Other grantors: National Heart Institute (H–1129), the American Heart Association, the Alabama Heart Association, the Life Insurance Medical Research Fund, and the Swiss-American Foundation for Scientific Exchange.

COMPARATIVE VASOCONSTRICTOR EFFECTS OF INHALING TOBACCO SMOKE IN WARM AND COOL ENVIRONMENTS AND BEFORE AND AFTER ABSTINENCE FROM TOBACCO

By John W. Eckstein, M. D., J. Edwin Wood, M. D., and Robert W. Wilkins, M. D., Cardiovascular Division, Evans Memorial, Massachusetts General Hospitals, and the Department of Medicine, Boston University School of Medicine. The American Heart Journal, Vol. 53, No. 3, Pages 455–462, March 1957

Abstract

Purpose of study: It is generally agreed that inhaling tobacco smoke causes vasoconstriction in the foot, but comparative data on the relative intensity of the vasoconstriction are not available. These experiments were designed to compare foot blood flow responses to smoking under different control conditions of vasomotor activity, to compare the vasoconstrictor response to smoking with that produced by a cool environment, and to determine whether or not a period of abstinence from tobacco produces a more profound vasoconstrictor response to smoking.

Experiments: A decrease in average foot blood flow occurred on smoking under all conditions of the study except in 3 of 31 experiments; in the 3 exceptions where no change in blood flow occurred, smoking was associated (as in all tests) with increases in blood pressure and pulse rate and reduction in the variations of blood flow. The range of responses in the warm and cool rooms expressed as percent reductions from control values were remarkably similar, and the distribution of variations of cool and warm room smoking responses was almost symmetrical. The ranges of smoking responses before and after 24 or 48 hours of smoking abstinence were very similar, as were the group averages.

Findings: The similarity of response under the conditions of this study suggests that the different control levels of vasomotor activity do not alter significantly the vasoconstrictor effects of smoking cigarettes. Under the conditions of this study smoking was a less intense vasoconstrictor stimulus than cooling the environment from 83° F. to 68° F.

EVALUATION OF SUBSTANCES CAUSING LOSS OF SEBACEOUS GLANDS FROM MOUSE SKIN

By Fred G. Bock, M. S., and Rhoda Mund, B. S., Roswell Park Memorial Institute, Buffalo, N. Y. The Journal of Investigative Dermatology, Vol. 26, No. 6, pages 479–487, June 1956.

Abstract

Purpose of study: Disappearance of sebaceous glands from mouse skin during the first days after exposure to certain carcinogens has been known since 1940. In 1954, W. E. Smith et al. (Proceedings, American Association for Cancer Research) reported that this effect could be used to predict the carcinogenic properties of certain petroleum fractions. With this in mind, the authors undertook to determine what factors might affect the destruction of sebaceous glands by various materials.

Experiment: The hair was shaved from large sections of the backs of Swiss mice, aged 55 to 65 days, and test solutions applied to the areas twice daily for three days. The mice were sacrificed one week after clipping and the treated skins stained to permit microscopic observation. Sebaceous gland suppression resulted from application of seven test solutions.

The suppression index was 10,000 for methylcholanthrene and dimethylbenzanthracene, 3,000 for dibenzanthracene and benzpyrene, and smaller for the other three compounds. Forty-two other compounds were found to have no demonstrable sebaceous gland suppression effect. The methylcholanthrene effects were not affected by time of day of painting or by the oxygen concentration of the air to which the animals were exposed.

Findings: Among the compounds studied, the sebaceous gland suppression effect is limited to polycyclic hydrocarbons and is parallel to the reported carcinogenic activity of compounds of this group. There is nearly additive

summation of the effects of several suppressor compounds when combined in one solution.

VENTILATION IN CHRONIC PULMONARY EMPHYSEMA. I. PRESSURE-VOLUME AND PRESSURE-FLOW RELATIONSHIPS. II. CORRELATION OF COMPLIANCE AND MECHANICAL RESISTANCE WITH ROUTINE PULMONARY FUNCTION TESTS

By Ernst O. Attinger, Merrill M. Goldstein, and Maurice S. Segal, Department of Medicine, Tufts University School of Medicine, and Lung Station (Tufts) and Department of Inhalation Therapy, Boston City Hospital, Boston, Mass. The American Review of Tuberculosis and Pulmonary Diseases, Vol. 74, No. 2, pages 210–219 and 220–228, August 1956

Abstract

Purposes of studies: I. There are still large gaps in our knowledge of the physical properties of the human lung which remain to be solved. The study is intended to demonstrate the importance of pressure-volume and pressure-flow relationships in the evaluation of patients with chronic pulmonary emphysema. II. The purpose is to report the results in 12 patients with chronic pulmonary emphysema in whom the mechanics of breathing were correlated with the conventional pulmonary function tests.

Experiments: I. The studies clearly demonstrate why patients with chronic bronchial asthma or chronic pulmonary emphysema can be fully incapacitated, despite very little X-ray or pathologic evidence of disability: a large part of their energy and oxygen uptake is spent on the process of breathing alone. II. Patients with chronic pulmonary emphysema in general show a decrease in the elastic properties of the lungs (compliance) and an increase in mechanical resistance (which expresses the pressure necessary for a given airflow).

Findings: I. Patients with chronic pulmonary emphysema produce lower flow rates, require greater pressure gradients to move air in and out of the lungs, and need proportionately more time for expiration than normal subjects. II. The work of breathing has a significant influence on the incapacity of these patients and is an important factor in determining the degree of disability.

Other grantor: None.

CHEMICAL CHANGES IN THE LUNGS OF TUMOR-BEARING RATS

By Leopold R. Cerecedo, Edward Bresnick, Harry Hochstrasser, Helen L. Rodriguez, Edward T. Schubert, and Edward J. Singer, Department of Biochemistry, Fordham University, New York, with the cooperation of Vincent S. Palladino, Department of Pathology, Meadowbrook Hospital, Hempstead, Long Island, N. Y. Biochimica et Biophysica Acta, Vol. 24, No. 1, Pages 58–61, April 1957

Abstract

Purpose of study: It was previously shown in this laboratory that significant changes occur in the nucleic acid concentration in certain tissues of tumor-bearing rats, and that the presence of the Walker 256 tumor caused an increase in the concentration of desoxyribonucleic acid (DNA) in the host lung. It was of interest to study this effect in some detail, as well as the ribonuclei acid (RNA) protein nitrogen (PN), cholesterol, and potassium in the lung, and similar relationships for the Murphy-Sturm lymphosarcoma.

Experiments: Male rats were implanted with either of the tumors at 4 to 6 weeks of age. The host animals were killed in groups concurrently with comparable groups of control animals. It was shown that during the growth of either tumor the dry weight concentration of host lung DNA increases markedly; that the RNA concentration remained fairly constant for the Murphy-Sturm lymphosarcoma hosts; and the RNA, PN, cholesterol, and potassium concentrations remained constant for the Walker 256 hosts.

Findings: If a constant amount of DNA per normal somatic diploid nucleus is assumed, the decreasing ratios to DNA of each of the other constituents occasioned by the rise in DNA concentration could be interpreted to mean that the host lung cell is being depleted of the four other substances during the progressive growth of the tumors. A possibility to be considered is that nutritional demands of the tumor may be responsible, but this is considered unlikely, since no overall losses occur when the data are expressed as total amounts per organ, normalized for body weight. Work is in progress to determine whether the effects observed are due to an increase in cell number. Whatever is the nature of the lung re-

sponse to the tumors, it may be assumed that it is mediated by an agent produced by the tumor and transmitted to the lung via the circulation. Experiments to investigate this possibility are also now in progress.

NUCLEIC ACID PATTERNS DURING GROWTH AND REGRESSION OF THE MURPHY-STURM LYMPHOSARCOMA IN THE RAT

By Leopold R. Cerecedo and Edward Bresnick, Department of Biochemistry, Fordham University, New York. Biochimica et Biophysica Acta, Vol. 23, No. 1, page 226, January 1957

Abstract

Purpose of study: It was observed in this laboratory that the growth of tumors in rats is accompanied by changes in the nucleic acid concentration of various tissues of the host, and that cortisone treatment of lymphosarcoma-bearing rats results in a retardation of tumor growth. It was of interest to determine whether regression of an implanted tumor would be accompanied by a complete reversal of the nucleic acid pattern to normal.

Experiments: Growth of the lymphosarcoma results in an increase in the desoxyribonucleic acid (DNA) content. Regression of the tumor is accompanied by a practically complete reversal of the increased DNA content to normal. On the other hand, the ribonucleic acid (RNA) concentration is not affected by either stimulus.

Findings: It has also been found that progressive growth of the lymphosarcoma results in an increase in the DNA concentration of the lungs, and that regression of the tumor results in a return of the lung DNA to normal values.

Other grantor: National Cancer Institute.

MITOTIC RATE OF GINGIVAL EPITHELIUM IN TWO AGE GROUPS

By Julia Meyer, B. S., Amarjit S. Marwah, B. D. S., and Joseph P. Weinmann, M. D., Division of Oral Pathology, University of Illinois, College of Dentistry. The Journal of Investigative Dermatology, Vol. 27, No. 4, pages 237–247, October 1956

Abstract

Purpose of the study: The findings by Bullough (J. Exp. Biol. 26: 261, 1949) that cell division (mitotic) frequently in mouse epidermis is considerably higher in middle-aged than in young animals contradicts the common concept of aging according to which the bodily processes slow down. Accordingly, it was decided to investigate whether human tissue in which maintenance depends on the perpetual proliferation of cells also shows higher regenerative rates in older individuals.

Experiments: A study of biopsy specimens of epithelium of the gums (attached gingiva) of 30 men aged 25 to 34 and of 30 aged 50 to 78 was made and cell size and density computed. The average mitotic index for the young men was 0.98 cells per 1000 and for the older 1.56 per 1000, the 50% increase in average frequency being statistically significant.

Findings: Epithelium of human gums behaves like epidermal epithelium in mice and men, suggesting that current concepts of age as slowing down the tissue maintenance processes may require revision in the case of epithelial tissue.

Other grantor: National Institutes of Health (C–2705), United States Public Health Service.

EFFECT OF CIGARETTE SMOKING ON EXCRETION OF UROPEPSIN AND CONCENTRATION OF PLASMA PEPSINOGEN

By Philip Cooper, M. D.,[1] Associate Professor of Clinical Surgery, Boston University School of Medicine, and Chief, Surgical Service, Veterans' Administration Hospital, Providence, R. I., with Harold L. Stein, M. D., Goldwyn F. Moore, B. S., and Harold W. Harrower, M. D. The Rhode Island Medical Journal, Vol. 40, No. 4, pages 215–216 and 251, April 1957.

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[1] Dr. Cooper is now Chief of Surgical Service at the Bronx Veterans' Administration Hospital and has been appointed as a Clinical Professor of Surgery at the Einstein Medical School.

Abstract

Purpose of study: Having found in previous research that no significant changes were produced in gastric secretions by smoking, the authors undertook to measure the excretion of uropepsin and the concentration of pepsinogen in plasma in a group of habitual smokers during and after a period of abstinence. The concentration of plasma pepsinogen in individual subjects is fairly constant from day to day, is not altered significantly by usual variations in diet and activity, and its laboratory determination is reliable.

Experiments: Twenty male cigarette smokers aged 22 to 65 (average, 34.1) in hospital for a variety of general conditions and free of any symptoms or signs of gastrointestinal, cardiac or urological conditions, and with normal urinalyses and blood urea nitrogen values, were observed for 13 days. The patients stopped smoking on the first day and resumed on the sixth day. No effort was made to regulate diet or fluid intake during the tests, and none of the patients received drugs which could affect peptic activity or renal excretion. The average daily number of cigarettes smoked in the second period was 22.3. The mean difference in the excretion of uropepsin between the two periods was not statistically significant. A small but statistically significant decrease occurred in the pepsinogen level during the smoking period compared with the nonsmoking period.

Findings: Uropepsin excretion determinations in this study on the same group of patients varied so widely that no conclusions relative to the effect or lack of effect of cigarette smoking could be drawn. Indeed, in some patients, day-to-day variations in the excretion of uropepsin during either test period exceeded the mean difference for the entire group between the two periods. Cigarette smoking after a period of abstinence produced a statistical significant decrease in the concentration of plasma pepsinogen.

IMMEDIATE EFFECT OF CHEWING TOBACCO ON CIRCULATION OF HABITUAL CHEWERS

By David L. Simon, M. D., Arnold Iglauer, M. D., John R. Braunstein, M. D., and Robert E. Rakel, Cardiac Laboratory, Department of Internal Medicine, Cincinnati General Hospital and Kettering Laboratory, University of Cincinnati, Ohio. The Journal of the American Medical Association, February 2, 1957, pages 354–356

Abstract

Purpose of study: Although 81,000,000 pounds of chewing tobacco are consumed annually in the United States, virtually no information is available about its physiological effect or specifically about its effect on the heart and circulation.

Experiments: Pulse rate, blood pressure, skin temperature and electrocardiograph and ballistocardiograph readings were taken of 24 male volunteers aged 34 to 71 who were habitual chewers of tobacco. Each subject chewed, on alternate days, a low-nicotine chewing tobacco (.31% to .47% nicotine), or a chewing gum placebo, and a regular commercial brand of tobacco (1.53% nicotine). Twenty-three of 24 subjects showed deterioration of the ballistic wave pattern on average 15 minutes after chewing commercial tobacco, and the patterns gradually returned to normal. No changes were observed 15 minutes after chewing the placebo gum. Pulse rate of one remained constant and of 14 increased markedly after chewing commercial tobacco; with low nicotine tobacco, the rate was constant in three, decreased in one, and rose moderately in nine subjects: with gum, one subject showed a slight rise and one a slight fall. Blood pressure measurements in 16 subjects after chewing commercial tobacco showed significant rise of all systolic pressures and 15 out of 16 showed significant rise of diastolic pressures. In one subject whose blood pressure rose significantly after chewing commercial tobacco, no rise was recorded after chewing gum. The skin temperature changes were those usually associated and previously reported with smoking.

Findings: The chewing of tobacco in a group of older men who habitually chewed tobacco produced an increase in pulse rate and blood pressure, a decrease in skin temperature, and a deterioration of the ballistocardiogram. It is believed that these changes are attributable to the nicotine absorbed during the chewing of tobacco.

TRAUMA AND CANCER. AN EXPERIMENTAL STUDY IN THE WHITE PEKIN DUCK

By R. H. Rigdon, M. D., Laboratory of Experimental Pathology, University of Texas Medical Branch, Galveston. AMA Archives of Pathology, vol. 61, No. 6, pages 443–449, June 1956

TUMORS INDUCED IN SKIN WITHOUT FOLLICLES. AN EXPERIMENTAL STUDY IN THE DUCK

By R. H. Rigdon, Cancer Research, vol. 16, No. 8, pages 804–807, September 1956

LYMPHOID HYPERPLASIA PRODUCED IN THE SKIN OF CHICKENS BY METHYLCHOLANTHRENE

By R. H. Rigdon, RES Bulletin (published by Society for Research on the Reticulo-Endothelial System), vol. II, No. 2, pages 40–46, fall 1956

HEMANGIOMAS. AN EXPERIMENTAL STUDY IN THE DUCK

By R. H. Ridgon, Jack Walker, and A. H. Teddle, Cancer, vol. 9, No. 6, pages 1107–1115, November–December 1956

CARCINOGENESIS IN THE WHITE PEKIN DUCK

By R. H. Rigdon, Texas, Reports on Biology and Medicine, vol. 14, No. 4, pages 508B527, winter 1956

Abstract

Purpose of studies: Various aspects of carcinogenesis need clarification and proof, such as the role of the lymphatic system, the hair follicles, the blood supply of cancer cells, and the influence of trauma. The absence of lymph glands in the duck and the availability of skin areas without feathers make it a useful laboratory animal to study these factors under varying conditions.

Experiments: The strong carcinogen methylcholantherene was applied in various ways, at varying times and in varying quantities to the web, bill, skin or implanted subcutaneously in white Pekin ducks and young chickens. Various types of tumors developed, mostly benign, including among others papillomas, ganglioneuromas, hemangiomas, Pacinian corpuscles, and a tumor with the morphological characteristics of a squamous cell carcinoma that regresses spontaneously, as did numbers of the other tumors.

Findings: 1. While tumors may occur without trauma, the plucking of feathers in the duck does have a cocarcinogenic effect when a carcinogen is applied to the plucked area; also, lymphocytes occurred in some chickens following incision by scalpel. 2. No metastatic tumors were found in the duck, indicating that a lymphatic system may be significant in the spread of tumors in mammals. 3. Tumors may result from application of a carcinogen in epithelial areas not associated with feather follicles, hence it would be unwise to consider that all skin cancers in man arise from hair follicles and to support this opinion by experimental observation in the mouse. 4. Development of blood vessels in tumors which unite with the vascular system of the host animal suggest that a histiocyte may be the cell stimulated by the carcinogen, leading to a proliferation of angioblasts associated with some enzymatic action. 5. Diminished blood supply to hemangiomas and the absence of a lymphatic system are suggested as related to spontaneous regression of tumors in fowls. A significant finding to be treated more fully in a later paper is the variety of mechanisms involved in spontaneous regression of tumors in ducks and chickens.

Other grantor: National Cancer Institute, United States Public Health Service.

EFFECT OF CIGARETTE SMOKING ON GASTRIC SECRETIONS OF PATIENTS WITH DUODENAL ULCER

By Philip Cooper, M. D.,[1] Associate Professor of Clinical Surgery, Boston University School of Medicine; Chief, Surgical Service, and Director, Surgical Research Laboratory, Veterans' Administration Hospital, Providence, R. I.; and James B. Knight, Jr., M. D., Resident in Surgery, Veterans' Administration Hospital, Providence, and Massachusetts Memorial Hospitals, Boston, Mass. The New England Journal of Medicine, vol. No. 255, pages 17–21, July 5, 1956

[1] Dr. Cooper is now Chief of Surgical Service at the Bronx Veterans' Administration Hospital and has been appointed as a Clinical Professor of Surgery at the Einstein Medical School.

Abstract

Purpose of study: Review of the literature fails to reveal any studies sufficiently detailed or adequately controlled to substantiate scientifically the many clinical opinions on the effect of smoking on gastric secretions.

Experiment: Two studies were conducted in an attempt to determine the facts. The first concerned 120 patients with duodenal ulcers, half of whom smoked and half of whom did not smoke during test periods in which gastric measurements were taken. The second was limited to measuring pH changes of gastric juice after smoking and broth stimulation and insulin stimulation, in 16 patients before and in 11 patients after partial gastrectomy for duodenal ulcer. First study: Within the "smoker" group and within the "control" group there were significant alterations in volume, petic concentration or peptic output in one or more periods, but comparisons between the "smokers" and "controls" revealed no significant differences between the volume, pH, "free acid," peptic concentration, or peptic output. Second study: It was found in both groups that the pH was elevated during the smoking period.

Findings: Under the conditions of the investigation, the smoking of cigarettes by patients with duodenal ulcer did not produce significant changes in gastric secretions.

CONTRACTILITY AND EXTRACTABILITY OF HEART ACTOMYOSIN AFTER DEATH

By L. Dettli, M. D., and R. J. Bing, M. D., Department of Experimental Medicine, the Medical College of Alabama, Birmingham. Circulation Research, vol. IV, No. 5, pages 519–522, September 1956

Abstract

Purpose of study: Benson (Circ. Res. 3: 221, 1955), by producing experimental chronic heart failure in dogs, was able to show that actomyosin from failing hearts differed from that of normal heart. These studies were based on the premise that characteristic properties of actomyosin do not change for a brief period after death. Accordingly, an investigation was begun which deals with a comparison of the properties of actomyosin from dogs' hearts immediately after death and one hour later.

Findings: Little difference was seen between fresh hearts and hearts extracted one hour after death in the yield of total and soluble protein and actomyosin per 100 gm. heart weight. This finding is not surprising, since post mortem stability of the contractile elements in the isolated muscle in vitro has been the basis for widely used pharmacological tests. The insult to the protein during its extraction is probably more severe than the denaturation that occurs during the first hour after death. Despite the vigorous treatments in the experiments, the contractile properties of the protein are preserved to a great extent. These studies may open the way for an investigation of the properties of actomyosin obtained from failing human hearts.

Other grantors: United States Public Health Service, the American Heart Association, the Alabama Heart Association, and the Life Insurance Medical Research Fund.

EXHIBIT 4A

A STUDY OF TOBACCO CARCINOGENESIS

II. DOSE RESPONSE STUDIES[1]

E. L. Wynder, P. Kopf, and H. Ziegler

Since epidemiological studies have repeatedly shown that the risk of lung cancer rises in proportion to the amount of tobacco consumed, it is important to know the amount of tobacco smoke which would not significantly influence the development of cancer. Such level has been suggested by statistical studies for man (2, 5). It was thought pertinent to determine it for the laboratory animal. If reduction to such a level is possible and practical, effective preventive measures may be at hand.

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[1] From the section of epidemiology, division of preventive medicine, Sloan-Kettering Institute, New York, N. Y.

METHODOLOGY

The methods of cigarette tar collection and application were the same as previously described (6). With a 1:1 tar-acetone solution the mice receive approximately 65 milligrams per painting.

The study, based upon 530 Swiss (Millerton) mice, was divided into the following groups:

Variation in frequency.—All mice were painted with a 1:1 tar-acetone solution. Group I was painted 5 times a week, group II, 3 times a week, group III, 2 times, and group IV, once a week. Group V was painted 3 times a week for 2 weeks, then painting was stopped for 2 weeks and then resumed for 2 weeks and so on. Group VI was painted alternately every 4 weeks, 3 times a week.

Variation in duration.—The animals were painted with a 1:1 solution 3 times a week, but painting was stopped after periods of 3, 6, 9, and 12 months respectively (groups VII–X). One group (II) was painted for the entire life span.

Variation in concentration.—The mice were painted with 1:1 and 1:2 solutions respectively (groups XI–XII). Two experimental groups painted with 1:3 and 1:4 solutions are still in progress.

The present experiment thus studied groups of mice receiving varying amounts of tobacco. By determining the total number of tumors obtained and the latent periods, the relative activities of the various tar concentrations and painting schedules were evaluated.

In the present studies it is realized that the term dose does not have the same meaning as it has in an experiment in which one injects a known amount of carcinogen into an animal or applies an exact amount of carcinogen to the skin. In the skin-painting technique as used here, one can only obtain an approximate idea about the amount of a substance given to each animal. However, this type of application of carcinogen is akin to that existing in human exposure and, more important than the evaluation of a given single experimental group, is the comparison from group to group.

RESULTS

Decreasing the frequency of tar application is followed by a significant decline in the number of tumors formed and an increase of latent periods (table 1). In fact, only 1 cancer (2 percent) was produced in the twice-a-week group, occurring at the 14th month in contrast to 8 cancers (16 percent) in the 3 times-a-week group, where the first cancer appeared in the 8th month. The group of animals receiving tar only once a week developed only two papillomas, the first of which occurred in the 15th month. In this group, no cancers were produced. The high mortality in the group painted 5 times a week (group I) makes this frequency of painting impractical. At the end of 1 year of tar application only 18 percent of the mice in this group were still alive. There is no indication that tumor formation was speeded up in the 5 times-a-week group.

The animals receiving tar 3 times a week for 2 and 4 weeks respectively and which were then not painted for a similar period of time developed a few papillomas after a long latent period, 13 and 11 months respectively, and no cancers (table 2).

Mice receiving tar 3 times a week for only 3 months developed no tumors (table 3). In the group painted for only 6 months, 4 percent of the animals developed papillomas. In the 9-month group, 34 percent developed papillomas and 12 percent cancers. The percent was greater in the 12-month group which yielded 58 percent papillomas and 30 percent cancers. In the group of animals painted for the entire life span, 38 percent developed papillomas and 16 percent cancers. It is noted, however, that the 12-month group at 12 months had more papillomas than the life span group (24 against 13), a finding in line with biologic variation. In the 9- and 12-month groups, cancers occurred long after the cessation of tar application (table 3, figure 1).

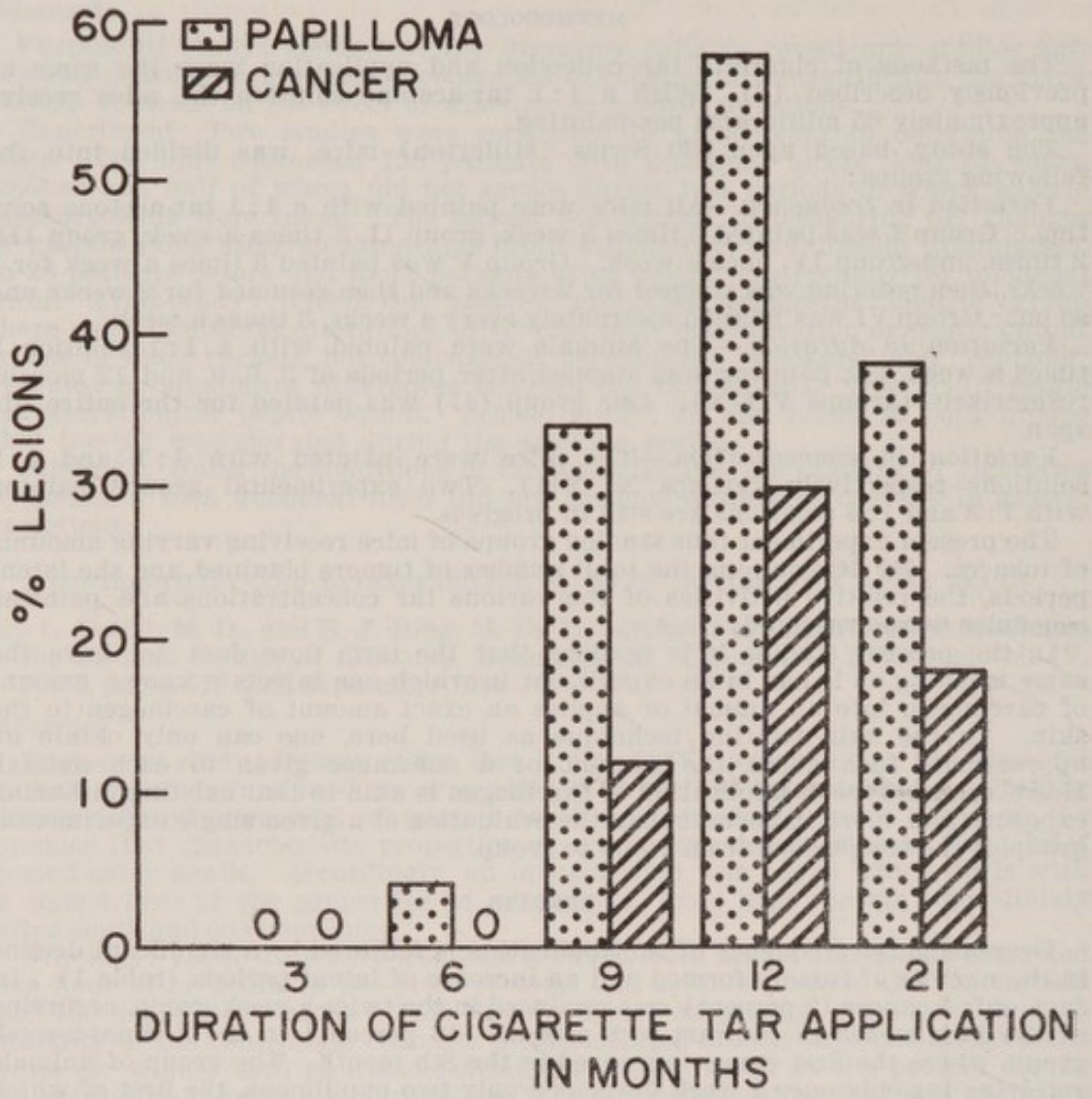


Figure 1

In the 9-month group (IX), no cancers were observed at the time painting was stopped. The first gross evidence of cancer was noted 4 months afterward and the last one 10 months after cessation of tar application. Similar observations are at hand for the 12-month group (X), where only 6 percent of the animals had gross evidence of cancer at the end of tar application.

The last new cancer was observed 8 months afterward. These experiments clearly indicate that carcinoma of the skin may develop in the experimental animal long after cessation of tar application, provided, of course, that a sufficient amount of the carcinogen has been applied to the animal.

Table 4 presents data on mice painted with 50- and 33-percent concentrations of tar. The papilloma yield in groups XI and XII is similar. There are differences, however, both in latent period and number of cancers produced. The fact that the survival time is shorter in the 1:1 group than in the 1:2 group tends further to increase the chance of cancer formation in the latter group.

Survival Rates.—The total number of cancers one can expect in any given group depend, of course, on the number of animals that survive long enough to enter the time period in which the first cancers can be expected. As far as tobacco tar is concerned, the survival rate is primarily dependent upon the amount of nicotine in the tar. Mice with cancer were sacrificed just prior to time when death was considered to be imminent. The survival rates that show significant differences among groups are the following:

1. The group of mice painted with a 50 percent tar-acetone solution 5 times a week have the highest mortality rate, in fact, it is twice as great as that of the group of animals painted only 3 times a week. By the 10th month, when the first cancer was noted, only 25 percent of the animals in group I were still alive com-

pared to 72 percent in group II. These data clearly indicate that the mice used in this experiment cannot tolerate a 5 times-a-week tar painting with 50 percent tar-acetone solution.

2. The group of animals painted for 3 months only, group VII, show a significantly longer survival as compared to the group painted for 9 months or longer. This is shown to a lesser, and not significant extent, by the 6-month group. At 15 months, for instance, 60 percent of the 3-month group were still alive in contrast to 22 percent in the group of animals painted for their entire life span. The difference between the groups painted for 9 months and 12 months, ad infinitum, were not significant.

3. The survival rate was higher in the group of mice painted with a 33-percent solution, group XII, up to the 10th month. At this time only 2 out of 30 mice in group XII had died in contrast to 12 out of 40 mice in group XI. After this time the survival rates of these two groups are similar.

Summary on data.—The results of decreased tar application and varying painting schedules on tumor formation are summarized in table V and figures 2 and 3. The statistical analysis of this data has been carried out with the assistance of Dr. I. Bross and R. Feldman. The figures graphically present the development of papillomas and cancers up to 18 months. They also indicate the confidence limits for each given group. When the confidence limits do not overlap for 2 given groups, the difference is statistically significant at the 5 percent level. More important than the contrast of two individual groups, however, is the general trend showing a decrease in tumor formation with decreasing doses.

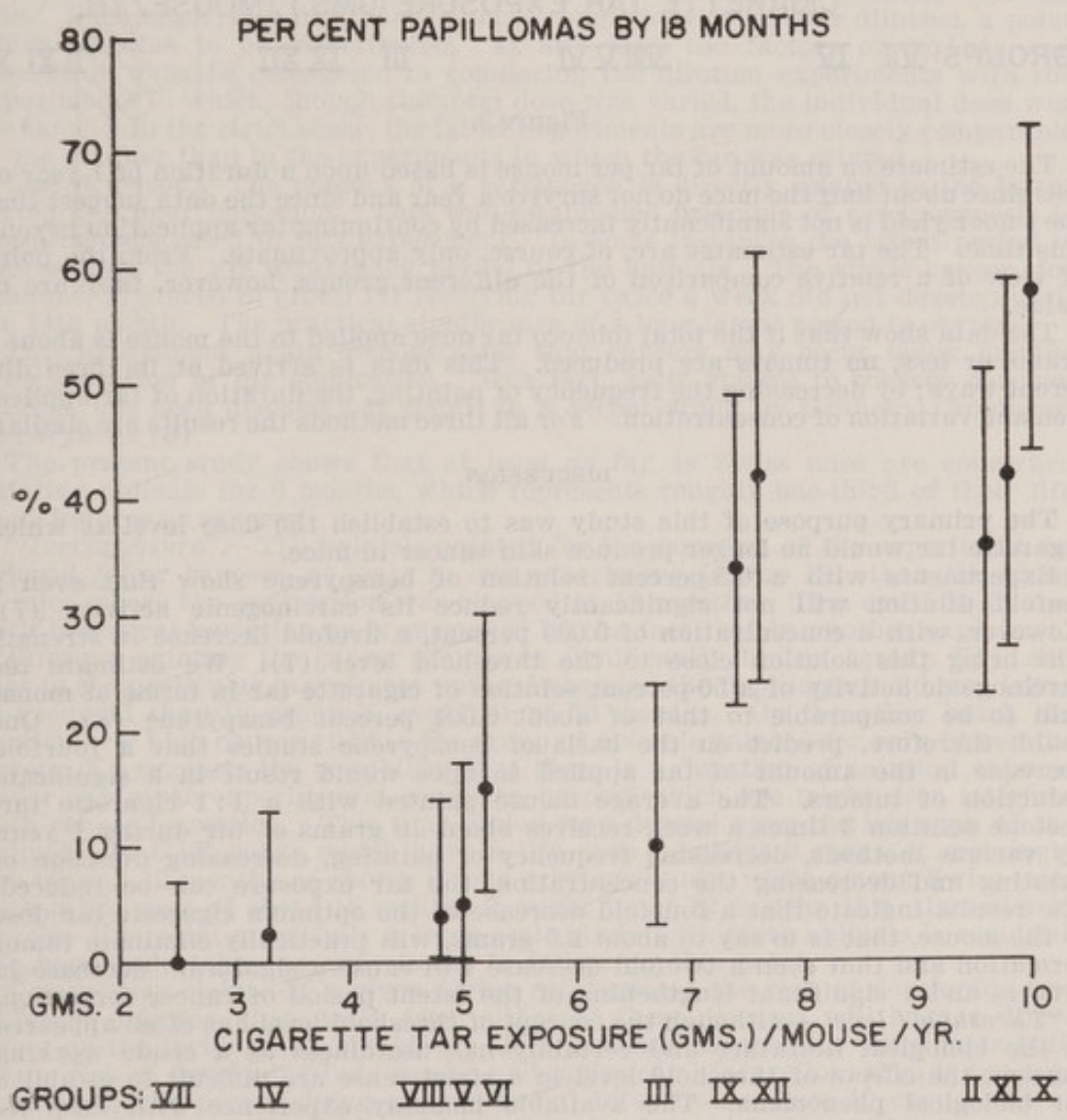


Figure 2.

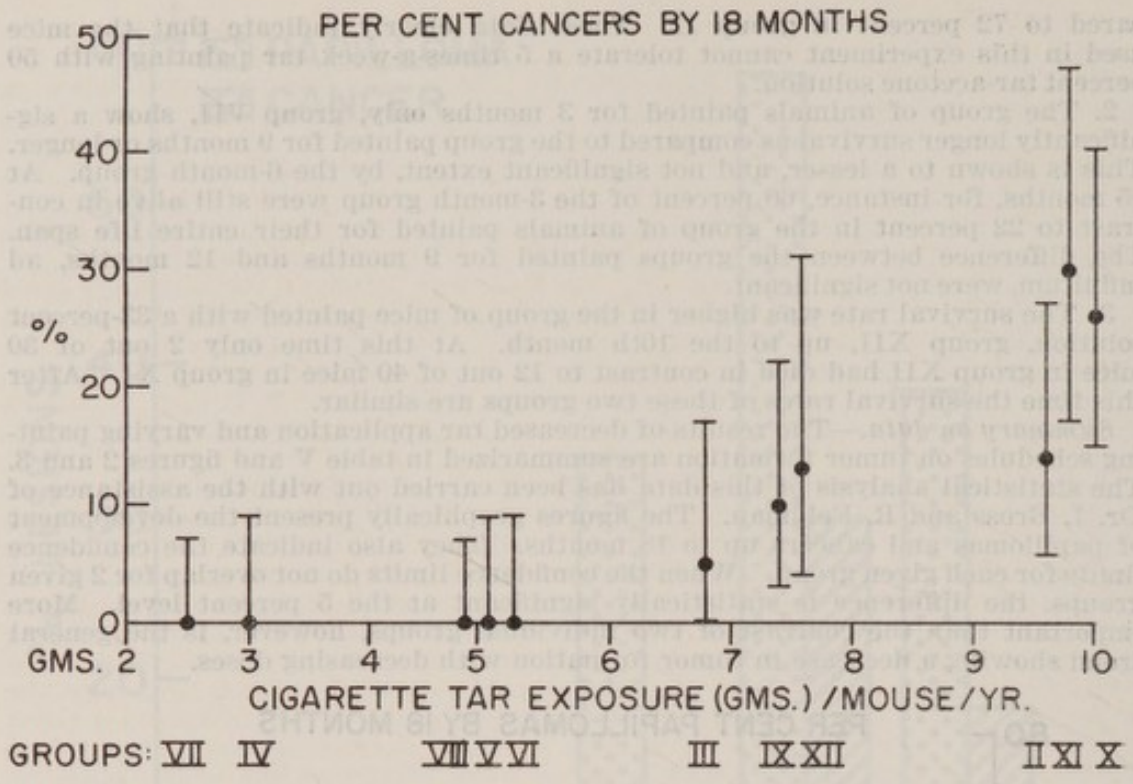


Figure 3.

The estimate on amount of tar per mouse is based upon a duration of 1 year or less since about half the mice do not survive a year and since the data suggest that the tumor yield is not significantly increased by continuing tar application beyond this time. The tar estimates are, of course, only approximate. From the point of view of a relative comparison of the different groups, however, they are of value.

The data show that if the total tobacco tar dose applied to the mouse is about 3 grams or less, no tumors are produced. This data is arrived at in three different ways; by decreasing the frequency of painting, the duration of tar application and variation of concentration. For all three methods the results are similar.

DISCUSSION

The primary purpose of this study was to establish the dose level at which cigarette tar would no longer produce skin cancer in mice.

Experiments with a 0.5-percent solution of benzpyrene show that even a tenfold dilution will not significantly reduce its carcinogenic activity (7). However, with a concentration of 0.005 percent, a fivefold decrease in strength will bring this solution close to the threshold level (7). We estimate the carcinogenic activity of a 50-percent solution of cigarette tar in terms of mouse skin to be comparable to that of about 0.004 percent benzpyrene (8). One could, therefore, predict on the basis of benzpyrene studies that a fourfold decrease in the amount of tar applied to mice would result in a significant reduction of tumors. The average mouse painted with a 1:1 cigarette tar-acetone solution 3 times a week receives about 10 grams of tar during 1 year. By various methods, decreasing frequency of painting, decreasing duration of painting and decreasing the concentration, the tar exposure can be reduced. The results indicate that a fourfold decrease of the optimum cigarette tar dose to the mouse, that is to say to about 2.5 grams, will practically eliminate tumor formation and that even a twofold decrease will cause a significant decrease in cancers and a significant lengthening of the latent period of cancer formation.

"Threshold" level.—Although the concept of threshold level has often appeared in the biological literature and certainly has usefulness as a crude working concept, the effects of threshold level in a strict sense are difficult to establish for biological phenomena. The available bioassay experience with dose response curves indicates that rarely is there an abrupt cutoff corresponding to a true threshold. The appearance of the response becomes increasingly rare as the dose is decreased so that in experiments of the size as used in this particular

study a response is not shown. This does not rule out, however, the possibility that if the number of animals used were increased to several thousand the response might actually appear. The present study, however, does show a dasage level at which the response is rare, if not necessarily nonexistent, and does establish levels at which the risk of developing tumors is so small and the latent period for tumor development so long that for all practical purposes this represents a "threshold" level, or at least a level which is desirable to attain.

The findings of this study offer an explanation for the apparent inability of some investigators to produce cancer in the experimental animal with tobacco tar upon applying solutions apparently under or near the threshold level (4). The present results also suggest practical measures through which the tobacco cancer problem can be approached. Such measures would have been difficult if the carcinogenic activity of tobacco tar was such that a fiftyfold or tenfold dilution were necessary. But if only a two or threefold dilution can be effective, practical preventive measures may be at hand.

Dilution effects.—Dilution experiments introduce factors not present when one applies the same amount of tar over different time periods. These include differences in survival time and in the rate of absorption. As one dilutes cigarette tar it is evident that its toxic effects become less and that more animals live to the time period at which cancer formation may be expected. In other words, the risk of the toxic effect has been lessened. Dilution also affects absorption as well demonstrated by the lesser carcinogenic response of whole tar as compared to a tar-acetone solution (9). It becomes clear even on gross observation that the tar contained in acetone is absorbed much more rapidly into the skin. Such absorption may be even more complete with greater dilution, a point which remains to be investigated. At any rate, the factors of toxicity and absorption must be considered in comparing the dilution experiments with the experiments in which, though the total dose was varied, the individual dose was the same. In the strict sense, the latter experiments are more closely comparable to one another than to the experiments in which the tar was diluted.

Latent period.—The potency of a carcinogen is directly related to the latent period of tumor induction. This well-established principle of carcinogenesis is again demonstrated in this experiment. In some instances the latent period is approaching the life expectancy period of the animals. The single cancer, for example, produced in group III receiving tar twice a week did not develop until the 14th month. The practical significance of a long latent period is obvious.

This experiment again demonstrates that cancers may occur many months after cessation of tar application. This fact, as it applies to tobacco carcinogenesis, has been discussed in detail in a recent study by Graham, Croninger, and Wynder (3).

The present study shows that at least as far as Swiss mice are concerned painting animals for 6 months, which represents roughly one-third of their life span, induces no cancers.

"Overexposure".—The animals painted for the duration of the experiment developed fewer cancers, at least in one experiment (group II), than the group of mice where tar application was stopped at 12 months. This could be in line with biological variation as is suggested by the fact that in another experiment of lifetime painting the cancer yield was significantly higher (group XI), and that in the particular experiment under discussion the papilloma yield was lower in group II than in group X even before 12 months. On the other hand, the particularly high cancer yield in the group of animals painted for only 12 months (group X) suggests the possibility that there is a duration of painting animals under any given experimental condition beyond which one might even interfere with cancer formation. This time period may depend upon the life span of the animal. In CAF_1 mice, which have a life span of about 2 years, it seemingly lies beyond 12 months as shown in our previous study, while among Swiss mice with a life span of about 18 months it might lie at 12 months (3). In order to develop this point more fully a large-scale study with more animals is required. We have undertaken an experiment to follow up this point.

Competitive risks.—If a given factor can increase the risk of developing two different diseases, then death from either of these diseases will have a direct effect on the incidence of the other. For instance, if smoking contributes both to the risk of developing lung cancer and myocardial infarction, then it becomes obvious that a person dying of myocardial infarction at age 40 cannot die of lung cancer at age 54. This evident, though often neglected principle, is well demonstrated in the present study. When the tar exposure of a given number of mice, as seen particularly in the group of animals painted five times a week, is

too great, the mortality rate is so high that relatively few of the animals reach the age at which the first cancers can be expected. Cigarette tar has, of course, a significant influence on the mortality rate among mice as is also seen from the fact that the group of mice painted for only 3 months had much better survival rates than mice painted for longer periods of time. These data clearly indicate that in evaluating the number of cancers one can obtain with the total cigarette tar or its fractions, it is important to consider the total survival rate in each one of these groups.

Relation to human cancer problem.—We have repeatedly stated that the primary proof for smoking as a cause of cancer in man is not based upon animal experimentation, but rests on human epidemiological, pathological, and clinical studies (6). If animal data confirm the carcinogenic activity of a substance previously suspected to be carcinogenic for man, the human evidence is strengthened.

The basic purpose of the animal experiment is to define the mechanism of carcinogenesis and to identify the specific carcinogens responsible for this activity. A function of the present animal experiments is also to explore the effects of various modifications of tobacco and its products, and to eliminate those possibilities which do not effectively reduce the risk of skin cancer in mice. Once established, one can only assume that these principles and agents also apply to the human problem. In view of the similarities established both in the field of chemotherapy and experimental carcinogenesis, much importance is based upon comparing human and animal work. Such comparisons gain importance if the human and animal data are consistent with one another. For instance, the positive animal experimental data on tobacco tar gain significance because of the established human data.

Studies determining the relative risk of lung cancer show the risk to have been increased in proportion to the amount of tobacco consumed (1). These data on risk have been established also in terms of absolute risks by Hammond and Horn and by Doll and Hill (2.5). Hammond and Horn have shown that the incidence of lung cancer for a person smoking 2 packs of cigarettes a day is 278; for a person smoking 1 to 2 packs of cigarettes a day, 119; for those smoking between one-half and 1 pack a day, 61; and for those who never smoke, 4.5. It is to be realized that in adidtion to difficulty in determining the rates in certain groups due to sample sizes, there is the lack of control on the dosage variable.

Doll and Hill's data show the death rate among British physicians smoking 25 grams of tobacco or more to be 205, for those smoking between 15 and 24 grams a day to be 106, those smoking between 1 and 14 grams a day 58, and for nonsmokers 9. Because of the number of cases involved, an exact comparison is again less significant than the general trend.

Both retrospective and prospective studies thus have shown the risk of lung cancer to rise in proportion with the amount of tobacco smoked.

There are several reasons why a dose response curve for mice can be expected to show both similarities and differences compared to the corresponding curves for humans. This includes differences in tissue susceptibility in response to toxic effects of the tar and probably other factors.

For mice, present data indicate that an exposure to 2.5 grams per year induce no or but rare tumors. Though it is difficult to estimate a comparable figure for man, the human data in line with the animal results indicate that a reduction in total tar exposure will be followed by a decrease in tumor formation. For this reason, measures directed toward this end are of utmost importance.

Measures which can succeed in reducing the tar exposure of man include the following:

1. More effective filtration. It seems feasible to produce a filter which will remove 40 percent of the tar from a given cigarette and which will still have a satisfactory pressure drop and flavor.
2. Modification in the types of tobacco so as to use a blend which is as low as possible in tar and nicotine content.
3. Regulation of the size of the cigarette so as to yield as little tar as possible per cigarette.

These measures taken together, in addition to a moderation of smoking habits, can be effective in reducing the risk of smokers to develop cancer. It may be predicted that if the average smoker is exposed to only one-half the amount of tobacco tar to which the smoker of regular-size cigarettes is exposed at present, he will significantly reduce his cancer risk. Any measure designed to thus reduce man's exposure to tobacco tar, be it through modification of the tobacco or the cigarette, or through more effective filtration, can significantly contribute toward this end.

SUMMARY AND CONCLUSIONS

1. The purpose of the present study was to determine the dose response level of tobacco carcinogenesis in mice by varying the duration, frequency and concentration of tobacco-tar application.

2. There is an optimum as well as a minimum concentration of tobacco tar that will produce papillomas and cancers in mice. The optimum concentration is in part dependent upon the toxicity of the tobacco tar.

3. The minimum amount of tar capable of producing papillomas in mice is about one-third, and of producing cancer one-half that of the optimum dose.

4. The practical implications of this data and their relationship to the human cancer problem have been emphasized.

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TABLE 1.—*Tumor formation by frequency of cigarette tar application in percent*

Number of months	Group I			Group II			Group III			Group IV		
	Number surveyed	Percent papilloma	Percent cancer	Number surveyed	Percent papilloma	Percent cancer	Number surveyed	Percent papilloma	Percent cancer	Number surveyed	Percent papilloma	Percent cancer
1	50	0	0	50	0	0	40	0	0	40	0	------
2	42	0	0	50	0	0	40	0	0	40	0	------
3	34	0	0	49	0	0	37	0	0	40	0	------
4	32	2	0	48	0	0	37	0	0	39	0	------
5	28	2	0	47	0	0	35	0	0	36	0	------
6	27	4	0	45	4	0	33	0	0	36	0	------
7	22	6	0	39	6	0	32	0	0	36	0	------
8	19	6	0	38	12	2	22	3	0	31	0	------
9	18	6	0	36	12	2	20	5	0	29	0	------
10	13	8	2	36	20	2	19	5	0	25	0	------
11	11	8	2	31	20	6	18	8	0	24	0	------
12	9	10	2	30	26	6	15	8	0	24	0	------
13	8	10	4	26	28	12	13	8	0	21	0	------
14	------	------	------	21	30	12	10	10	3	18	0	------
15	------	------	------	17	36	12	7	10	3	15	3	------
16	------	------	------	11	36	12	4	10	3	12	3	------
17	------	------	------	9	36	12	0	10	3	11	3	------
18	------	------	------	7	36	14	------	------	------	7	3	------
19	------	------	------	5	36	14	------	------	------	3	3	------
20	------	------	------	3	38	16	------	------	------	3	3	------
21	------	------	------	2	38	16	------	------	------	2	3	------
22	------	------	------	0	38	16	------	------	------	2	6	------

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TABLE 2.—*Tumor formation by frequency of cigarette tar application in percent*

Number of months	Group V			Group VI		
	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer
1	40	0		40	0	
2	40	0		39	0	
3	37	0		38	0	
4	37	0		38	0	
5	35	0		35	0	
6	33	0		35	0	
7	27	0		27	0	
8	23	0		25	0	
9	23	0		19	0	
10	23	0		19	0	
11	22	0		18	3	
12	20	0		17	3	
13	17	3		15	8	
14	15	5		13	15	
15	11	5		7	15	
16	8	5		7	15	
17	1	5		2	15	
18	1	5		1	15	
19	0	5		0	15	

TABLE 3.—*Tumor formation by duration of cigarette tar application in percent—Duration of application of 50 percent cigarette tar/acetone solutions*

Number of months	Group VII			Group VIII			Group XI			Group X			Life span II		
	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer
1	50			50	0		50	0	0	50	0	0	50	0	0
2	45			50	0		45	0	0	48	0	0	50	0	0
3	43			48	0		43	0	0	48	0	0	49	0	0
4	43			45	0		43	0	0	48	0	0	48	0	0
5	43			43	0		40	0	0	47	0	0	47	0	0
6	42			40	2		38	0	0	43	4	0	45	4	0
7	42			36	2		38	2	0	39	6	0	39	6	0
8	42			35	2		37	6	0	39	10	0	38	12	2
9	42			35	2		34	12	0	36	24	0	36	18	2
10	42			35	2		31	22	0	35	26	2	36	20	2
11	40			32	4		27	26	0	31	36	2	31	20	6
12	37			29	4		25	28	0	26	48	6	30	26	6
13	34			28	4		21	28	4	19	50	10	26	28	12
14	34			25	4		19	28	4	16	54	12	21	30	12
15	30			20	4		15	32	8	14	56	24	11	36	12
16	26			17	4		13	32	8	7	56	24	11	36	12
17	23			16	4		8	34	10	6	58	26	9	36	14
18	19			15	4		8	34	10	2	58	26	7	36	14
19	14			10	4		4	34	12	2	58	28	5	36	14
20	8			8	4		3	34	12	1	58	30	3	38	16
21	4			7	4		2	34	12	0	58	30	2	38	16
22							1	34	12				1	38	16

TABLE 4.—*Tumor formation by degree of dilution of cigarette tar/acetone solution application in percent*

Number months	Group XI			Group XII		
	Number surgery	Percent papilloma	Percent cancer	Number surgery	Percent papilloma	Percent cancer
1	40	0	0	30	0	0
2	36	0	0	30	0	0
3	35	0	0	30	0	0
4	35	5	0	29	0	0
5	34	8	0	29	0	0
6	32	13	0	28	3	0
7	30	28	0	28	9	0
8	30	30	3	28	9	0
9	28	30	3	28	12	0
10	28	33	8	28	15	0
11	21	33	8	15	27	0
12	19	35	8	14	30	0
13	17	35	18	13	39	0
14	14	35	18	10	42	0
15	12	38	23	6	42	6
16	6	40	25	5	42	6
17	4	43	28	4	42	6
18	3	43	30	0	42	12
19	2	45	35			
20	0	45	35			

TABLE 5.—*Dose response to cigarette tar—Summary table*

Group	Number of mice	Concentration in percent	Frequency per week	Duration in months	Estimated amount tar per year	Percent papilloma	Percent cancer	1st papilloma month	1st cancer month
I	(50)	50	5	Life span	16.0	10	4	4	10
II	(50)	50	3	do	10.0	38	16	6	8
X	(50)	50	3	12	10.0	58	30	6	10
XI	(40)	50	3	Life span	10.0	45	35	4	8
XII	(30)	33	3	do	7.5	42	12	6	15
IX	(50)	50	3	9	7.5	34	12	7	13
III	(40)	50	2	Life span	6.7	10	3	8	14
V	(40)	50	(1)	do	5.0	5	0	13	
VIII	(50)	50	3	6	5.0	4	0	6	0
VI	(40)	50	(2)	Life span	5.0	15	0	11	0
IV	(40)	50	1	do	3.3	6	0		
VII	(50)	50	3	3	2.5	0	0		

[1] 3 every other 2 weeks.
[2] 3 every other 4 weeks.

III. FILTERED CIGARETTES [1]

Ernest L. Wynder and Jona Mann

With the introduction of filtered-tip cigarettes and their use on a wide scale during recent years, it became of interest to determine whether the tar obtained from the smoke of these cigarettes differs in respect to carcinogenic activity on experimental animals. For this reason we undertook studies in which the tar obtained from smoking four different filtered cigarettes was applied in the same concentration with which we have applied tar obtained from unfiltered cigarettes. The purpose of this study was to determine possible differences in biological behavior of filtered and unfiltered cigarette tar.

One brand of popular regular size (brand UR I) and 2 brands of popular king size (brands UK II and UK III) cigarettes, all unfiltered, were compared against 2 regular-size (brands FR I and FR II) and 2 king size filtered cigarettes (brands FK III and FK IV). The tar-removal efficiency of the filters of these brands varied. At the time when these cigarettes were smoked their approximate efficiency in terms of tar removal rated as follows: Brand FR I, 30%;

[1] From the section of epidemiology, division of preventive medicine, Sloan-Kettering Institute, New York, N. Y.

brand FR II, 25 percent; brand FK III, 20 percent; and brand FK IV, 10 percent.

The methods of obtaining the cigarette tar and applying it to mice were the same as previously described (6).

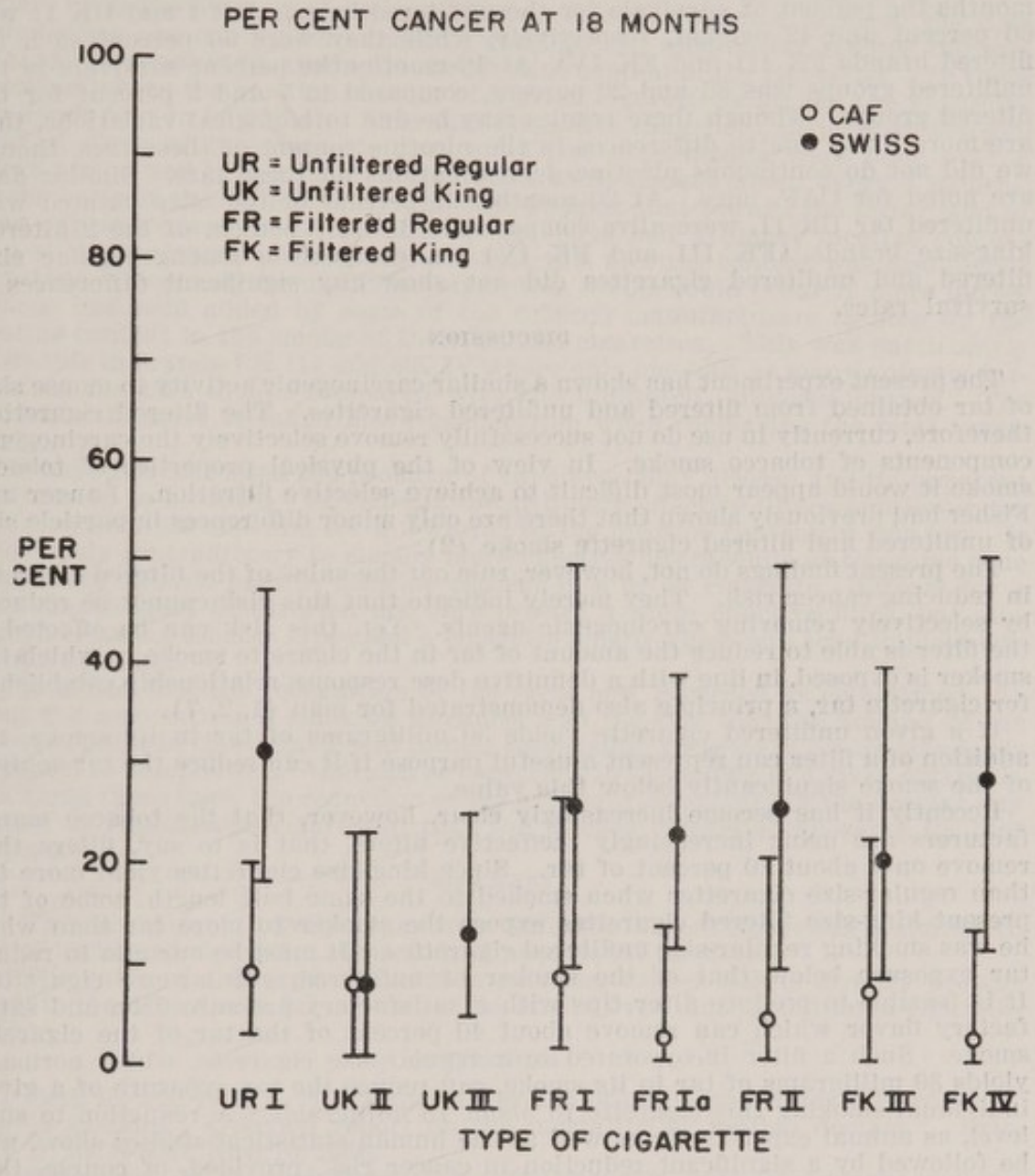


Figure 1

RESULTS

In tables 1 and 2 we summarize the survival rates and the percent of papillomas and cancers formed upon applying tar from unfiltered and filtered cigarettes to CAF_1 (Jackson) and Swiss (Millerton) mice. The results demonstrate that on a gram to gram basis the carcinogenic activity of a variety of filtered cigarettes as judged from the latent period and percent of papilloma and cancer formation is similar to that of unfiltered cigarettes. There are no statistically significant differences between any of the groups, as graphically demonstrated in figure 1, which shows all confidence limits to overlap. The statistical analysis of this data has been carried out by Dr. I. Bross and R. Feldman. Variations that are noted are in line with biologic variation as observed upon applying even the same tar to a group of animals of the size used in this study.

The results of this study indicate that the Swiss mice are more susceptible to cancer formation as a result of tobacco application than CAF_1 mice, while at the same time the CAF_1 mice have a longer survival period. These data confirm those previously reported on tobacco tar, as well as tobacco fractions (9).

Survival rates.—There is some variation in survival rates between the filtered and unfiltered groups within the CAF_1 and Swiss study groups. These differences apply particularly to the king-size filtered cigarettes. The major differences in the Swiss group occurred toward the latter part of the experiment. At 12 months the percent of survivals for the unfiltered brands UR I and UK II was 60 percent and 42 percent, respectively, while they were 30 percent each for filtered brands FK III and FK IV. At 16 months the percent survivals in the unfiltered groups was 33 and 20 percent, compared to 7 and 5 percent for the filtered groups. Though these results may be due to biological variations, they are more likely due to differences in the nicotine content of these tars, though we did not do continuous nicotine determinations on these tars. Similar data are noted for CAF_1 mice. At 20 months, 27 percent of the mice painted with unfiltered tar UK II_1 were alive compared to 3 and 8 percent of the 2 filtered, king-size brands (FK III and FK IV). A comparison among regular size, filtered and unfiltered cigarettes did not show any significant differences in survival rates.

DISCUSSION

The present experiment has shown a similar carcinogenic activity to mouse skin of tar obtained from filtered and unfiltered cigarettes. The filtered cigarettes, therefore, currently in use do not successfully remove selectively the carcinogenic components of tobacco smoke. In view of the physical properties of tobacco smoke it would appear most difficult to achieve selective filtration. Langer and Fisher had previously shown that there are only minor differences in particle size of unfiltered and filtered cigarette smoke (2).

The present findings do not, however, rule out the value of the filtered cigarette in reducing cancer risk. They merely indicate that this risk cannot be reduced by selectively removing carcinogenic agents. Yet, this risk can be effected if the filter is able to reduce the amount of tar in the cigarette smoke to which the smoker is exposed, in line with a definitive dose response relationship established for cigarette tar, a principle also demonstrated for man (1, 2, 7).

If a given unfiltered cigarette yields 30 milligrams of tar in its smoke, the addition of a filter can represent a useful purpose if it can reduce the tar content of the smoke significantly below this value.

Recently it has become increasingly clear, however, that the tobacco manufacturers are using increasingly ineffective filters, that is to say, filters that remove only about 10 percent of tar. Since king-size cigarettes yield more tar than regular-size cigarettes when smoked to the same butt length, some of the present king-size filtered cigarettes expose the smoker to more tar than when he was smoking regular-size unfiltered cigarettes. It must be our aim to reduce tar exposure below that of the smoker of unfiltered, regular-size cigarettes. It is feasible to produce filter tips with a satisfactory pressure drop and satisfactory flavor which can remove about 40 percent of the tar of the cigarette smoke. Such a filter incorporated in a regular-size cigarette, which normally yields 30 milligrams of tar in its smoke, can reduce the tar exposure of a given individual smoking this cigarette to about 18 milligrams. A reduction to such level, as animal experiments as well as the human statistical studies show, will be followed by a significant reduction in cancer risk, provided, of course, that the number of cigarettes smoked is kept constant.

Effect of butt length on tar-nicotine content of cigarette smoke.—The observation that king-size cigarettes yield relatively more tar than regular-size cigarettes when smoked to the same butt length has been confirmed in a recent study by Monahan and Miller. King-size cigarettes were smoked to the following butt lengths: 75, 65, 55, 45, 35, 25, and 10 milligrams.

The data are summarized in table 3. For tar content the data show a marked increase per each 10 millimeters of cigarette smoked. For instance, at a butt length of 75 millimeters, the average tar content of the smoke was 4.5 milligrams. This increased to 9.5 milligrams at 65 millimeters, an increase of 5 milligrams. In contrast, the differential between 35 and 25 millimeters was 9.1 milligrams, an increase per 10 millimeters which is nearly double that found between 75 and 65 millimeters.

The increase in nicotine content is more linear than that of tar content. Between 65 and 55 millimeters it rises from 0.3 to 0.6 milligrams, and then rises steadily to 2.4 milligrams at 25 millimeters and 2.8 milligrams at 10 millimeters.

The data thus indicate that the tar content of the smoke rises with the length to which a cigarette is smoked. This observation is, of course, due to the fact that the tobacco itself acts as a filter and as we smoke the cigarette toward the

end we are reburning the tar which has accumulated. The length to which a cigarette is smoked, therefore, is an important determinant in the amount of tar to which the smoker is exposed.

Nicotine effects.—The cancer yield in a given animal group depends, of course, in part upon the number of animals that survive long enough to reach the time period when one can expect cancer to develop. In tobacco tar studies the survival rate depends in particular on the nicotine content of the cigarettes. In those instances where the nicotine content of a given cigarette is higher, the mortality rate tends also to be higher. This fact may have influenced the cancer development of some of the cigarette groups. These experiments were done over a 2-year period and new groups of cigarettes were smoked about every 3 months. On the basis of animal mortality, the data suggest that toward the latter part of these experiments some of the filtered cigarettes had a higher nicotine content than at the beginning of the study and that this nicotine content was also higher than that of some of the unfiltered cigarettes. This could occur if more burley tobacco has been added by some of the tobacco manufacturers to step up the nicotine content in the smoke of their filtered cigarettes. This was particularly noticeable in brands FK III and FK IV where only one-half as many animals survived to the 16th month compared with the unfiltered, king-size brands. The survival rate must be taken into consideration when comparing the total tumor yield.

We have not discussed the problem of cardiovascular disease and its apparent relationship to nicotine. In striving toward more effective filters and lower tar exposure we must also aim for a lowering of the nicotine content in this tar. It is obviously contradictory to employ more effective filters while at the same time using more tobacco high in nicotine content in a given cigarette. Efforts for a safer smoke must be directed not only toward a more effective filter, but also toward choosing tobacco types and a cigarette length which will tend to yield less tar and nicotine.

It is hoped that the recognition of the facts now regarded as established (5) about the association of smoking to cancer and the role that the amount of tar exposure plays in this regard, will induce responsible agencies to set up standards in respect to amount of tar that can be contained in the smoke of a given cigarette. It is along these lines, the reduction of tar exposure below that of the present day smoker of regular-size cigarettes, that the filtered cigarette can serve its best purpose.

SUMMARY

1. The carcinogenic acivity of tar from different filtered cigarettes is similar to that of unfiltered cigarettes when judged upon equal tar doses.

2. The value of a filtered cigarette in reducing cancer risk is directly proportional to the decrease in tar content of the smoke over that in unfiltered cigarette smoke.

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TABLE I.—*Filtered and unfiltered cigarette tar—Survival rates and percent papilloma and cancer formation in Swiss mice*

Number of months	UR I[1]			UK II			UK III			FR I			FR I (a)			FR II			FK III			FK IV		
	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer
1	40	0	0	30	0	0	50	0	0	20	0	0	40	0	0	20	0	0	30	0	0	40	0	0
2	36	0	0	30	0	0	48	0	0	20	0	0	37	0	0	20	0	0	28	0	0	39	0	0
3	35	0	0	29	0	0	43	0	0	20	0	0	36	0	0	20	0	0	27	0	0	37	0	0
4	35	5	0	28	0	0	41	0	0	20	0	0	33	0	0	20	0	0	26	0	0	33	0	0
5	34	8	0	28	0	0	41	4	0	20	5	0	32	0	0	18	0	0	24	0	0	30	0	0
6	32	13	0	28	0	0	41	8	0	20	5	0	24	5	0	18	0	0	23	0	0	24	8	0
7	30	28	0	27	10	0	40	8	0	20	10	0	28	5	0	17	0	0	22	3	0	27	8	0
8	30	30	3	27	10	0	37	12	0	20	10	0	26	5	0	16	0	0	22	13	0	24	15	0
9	28	30	3	24	18	0	37	14	0	20	15	0	26	13	0	15	20	0	21	17	0	19	23	3
10	23	33	8	22	18	0	33	18	0	19	20	0	25	13	0	15	20	0	17	20	3	17	33	5
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18	3	43	30	5	40	8	5	28	14	2	30	25	8	40	23	3	60	25	1	33	20	0	43	28
19	2	45	35	3	40	8	------	------	------	1	30	25	4	40	30	3	60	25	0	33	20	------	------	------
20	1	45	35	1	40	8	------	------	------	0	30	25	3	40	30	3	60	25	------	------	------	------	------	------
21	0	45	35	0	40	8	------	------	------	------	------	------	0	40	30	0	60	25	------	------	------	------	------	------

UR=unfiltered regular
UK=unfiltered king.
FR=filtered regular.
FK=filtered king.

[1] Same group as group XI in paper on A Study of Tobacco Carcinogensis II Dose Response Studies.

TABLE 2.—*Filtered and unfiltered cigarette tar —Survival rates and percent papilloma and cancer formation in CAF$_1$ mice—Cigarette tar applied in 50 percent concentration*

Number of months	UR I			UK II			FR I			FR I (a)			FR II			FK III			FK IV		
	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer	Number of survivors	Percent papilloma	Percent cancer
1	50	0	0	30	0	0	25	0	0	40	0	0	25	0	0	30	0	0	40	0	0
2	42	0	0	30	0	0	25	0	0	40	0	0	25	0	0	29	0	0	40	0	0
3	42	0	0	30	0	0	22	0	0	40	0	0	25	0	0	29	0	0	40	0	0
4	41	0	0	30	0	0	22	0	0	40	0	0	25	0	0	29	0	0	40	0	0
5	41	0	0	29	0	0	22	0	0	40	0	0	25	0	0	28	0	0	38	0	0
6	41	0	0	29	0	0	22	0	0	40	0	0	25	0	0	28	0	0	35	0	0
7	41	0	0	28	0	0	22	0	0	37	0	0	25	0	0	27	3	0	29	0	0
8	41	2	0	23	0	0	22	0	0	25	0	0	24	0	0	23	10	0	28	0	0
9	40	10	0	22	7	0	21	0	0	25	3	0	24	0	0	20	13	0	23	3	0
10	40	18	0	22	13	0	21	0	0	25	3	0	24	0	0	17	27	0	22	15	0
11	40	20	0	20	17	0	21	12	0	24	5	0	24	4	0	14	37	0	22	20	0
12	40	28	0	18	17	0	21	12	0	23	10	0	24	8	0	14	37	0	20	25	0
13	40	28	0	18	27	0	21	16	0	20	17	0	24	12	0	14	40	0	19	28	3
14	37	32	2	17	33	3	20	20	0	20	17	0	24	16	0	13	47	3	19	35	3
15	36	34	4	14	33	7	18	28	0	20	25	0	23	28	0	9	50	7	16	45	3
16	32	28	4	14	43	7	17	32	4	17	25	0	21	40	0	3	53	7	8	48	3
17	28	42	6	11	50	7	16	40	8	16	25	3	11	44	0	3	53	7	7	48	3
18	23	42	8	9	53	7	11	48	8	14	25	3	5	48	4	3	53	7	7	48	3
19	17	46	10	9	53	13	5	52	12	13	33	5	3	48	4	3	53	7	4	50	3
20	12	46	10	8	53	13	5	52	12	12	35	5	3	48	4	1	53	13	3	50	3
21	7	46	12	6	53	17	5	60	12	8	37	5	1	48	4	1	53	13	3	50	3
22	4	46	12	4	53	20	0	60	20	4	37	5	1	48	4	0	53	13	3	50	3
23	3	46	12	3	53	27	------	------	------	3	37	8	1	48	4	------	------	------	3	50	5
24	1	46	12	0	53	27	------	------	------	------	------	------	1	48	4	------	------	------	2	50	5

UR=unfiltered regular
UK=unfiltered king.

FR=filtered regular.
FK=filtered king.

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TABLE 3.—*Cigarette tar and nicotine in milligrams*

Length of butt (millimeters)	Tar, in milligrams	Nicotine, in milligrams
75	4.5	0.03
65	9.5	.3
55	15.2	.6
45	22.7	1.3
35	28.4	1.9
25	37.5	2.4
10	48.8	2.8

EXHIBIT 4B

[Reprinted from the British Medical Journal, January 5, 1957, vol. i, p. 1]

TOWARDS A SOLUTION OF THE TOBACCO-CANCER PROBLEM

By E. L. Wynder, M. D., From the Section of Epidemiology, Division of Preventive Medicine, Sloan-Kettering Institute, New York

HUMAN EVIDENCE

Statistical and epidemiological evidence linking smoking to cancer of the lung and also to cancer of other sites of the upper respiratory tract is so extensive that it hardly needs to be recited for the physician and the scientist who have followed these data. Sixteen separate investigations from seven different countries, studying smoking habits of over 6,000 patients with lung cancer, are all in agreement that lung-cancer patients smoke significantly more than any group of controls and that the relative risk of developing lung cancer rises in proportion with the amount of tobacco and particularly of cigarettes consumed. Two prospective studies, carried out in England and in the United States, that followed population groups whose smoking habits were known, also showed that the heavy cigarette smoker has a far greater chance of developing lung cancer than the nonsmoker. In fact, the study by Hammond and Horn (1954 and personal communication) indicated that for a person smoking 40 cigarettes a day this chance was 70 times greater than for a nonsmoker. To account for such magnitude of difference on the basis of statistical errors would require errors of equal magnitude. Such errors have so far not been demonstrated.

The belief that smoking is a major cause of lung cancer is not only based upon statistical evidence but it also is supported by the general epidemiological pattern of lung cancer. In countries, such as Great Britain and the United States, with a high cigarette consumption already 30 years ago the present lung-cancer rates are high, while in countries, such as Iceland, where the cigarette consumption was low, the current rate of lung cancer is low. Among males in every country lung cancer is more common than among women. This fact has always presented a particular challenge. Any factor found to be significant statistically has to account, at least in part, for this sex difference. In a recent study we have shown that the present sex ratio of lung cancer is quite consistent with the long-term smoking habits of the two sexes. For instance, while some 20 percent of American males over the age of 50 smoke in excess of one packet a day, only 2 percent of the women in this age group smoke this amount. This study also showed that, among women who had smoked, the risk of developing epidermoid cancer of the lung or its undifferentiated types is about the same as that for men (Wynder et al., 1956).

To consider smoking as a major cause of lung cancer does not mean that it is the only cause of this disease. Obviously there is the factor of internal predisposition which must exist, since not everyone who smokes develops lung cancer. Obviously, too, there are other exogenous factors that may produce cancer of the lung. Among these are a number of occupational exposures. There is also evidence that general air pollution and/or motor exhaust fumes may be a contributing factor (Stocks and Campbell, 1955; C. Mills, personal communication). However, with the possible exception of a few isolated cities, such as Liverpool, air pollution is at best only a secondary factor. This belief is based on the fact that among nonsmokers lung cancer occurs but rarely, that the incidence of lung cancer among women, who are also exposed to city air, is low, and that the age distribution of epidermoid lung cancer with its peak in the fifth to sixth decade and its subsequent decline is more compatible with an exogenous factor

to which only a certain population group was exposed some 30 to 40 years ago rather than with air pollution, which would expose an entire population group at the same time (Levin, 1953).

While most students of the problem of the aetiology of lung cancer admit to an association between smoking and lung cancer, some question whether this association also represents causation. Until proved otherwise, an association means at least a contribution toward if not a causation of a disease. For any given patient it matters little whether a factor has contributed to or caused a disease. In either case the elimination of this factor will lead to reduction of the disease. In this sense, smoking, and especially cigarette smoking, is to be regarded as an important factor in the development of lung cancer. In fact, among the exogenous factors that influence the development of this disease it represents the most important one. Present incidence data indicate that some 80 percent of all lung-cancer cases occurring in males today would not have occurred were it not for tobacco smoking, and that about 10 percent of males over the age of 25 who smoke in excess of 20 cigarettes daily will develop lung cancer by the age of 75.

In view of these data, which gain significance because lung cancer represents the most common cause of cancer death among males in many countries, it becomes paramount to ask what we can do about solving this problem. Ideally this problem could be largely overcome by abolishing the use of tobacco. It is evident, however, that because of economic factors and because of firmly entrenched habits such advice is not likely to be heeded, regardless of how strong the evidence. For this reason we became interested in experiments that might lead to a reduction of carcinogens in tobacco and its products.

ANIMAL EVIDENCE

Experimental work is not carried out to prove that smoking causes cancer in man. Such proof can come only from human statistical and epidemiological studies. The purpose of an animal experiment is to define the mechanism by which a given agent produces cancer and to determine the specific substances responsible for this activity. In a series of experiments with different strains of mice we have demonstrated that condensed cigarette tar obtained in a manner simulating human smoking habits will produce skin cancers in a significant number of animals, varying from 10 to 44 percent, depending upon the strain of mice used (Wynder, Graham, and Croninger, 1953, 1955; Wynder, Lupberger, and Grener, 1956). Preliminary data show that some 20 percent of rabbits whose ears have been painted with tobacco tar for more than 5 years also develop cancer (Graham, Croninger, and Wynder, unpublished data). Thus carcinogenic activity of cigarette tar has been demonstrated in two species of animal and in several strains of mice. By itself, this observation might be only of academic significance, but taken together with the human evidence already at hand it gains practical significance.

Among the well-established principles of carcinogenesis is the relationship of dose to the latent period and number of tumors that may be obtained. The fulfillment of this principle is one of the reasons smoking is regarded as a cause of lung cancer in man. The risk of developing lung cancer rises in proportion to the amount of tobacco smoked. It has been of interest to determine whether with tobacco this principle could also be established for the experimental animal. Preliminary work leaves no doubt that this is the case (Wynder, Kopf, and Ziegler, unpublished data). When tar was applied to the back of Swiss mice twice a week, instead of three times weekly as is standardly followed in our laboratory, only 10 percent of 40 mice developed papillomas and 3 percent cancers at the end of 17 months. Of the animals painted three times a week for the same interval, 58 percent had developed papillomas and 26 percent cancers. The first papilloma in the group painted twice a week occurred 2 months later than in the group of animals painted 3 times a week. 40 mice painted once a week for 17 months developed only 1 papilloma.

A comparable reduction in tumor formation and lengthening of latent periods is also noted when one decreases the amount of tar applied. A 33 percent solution produced about 20 percent fewer cancers and increased the latent period by 3 months over that obtained with a 50 percent solution.

In still another way we can demonstrate the dose-response principle by stopping the painting after a given period of months. Painting animals 3 times a week for only 3 months and then stopping produced no tumors after 16 months of observation; painting 50 mice for 6 months produced 2 papillomas which subsequently regressed; 9 months' painting produced a significant number of papillomas and cancers, but this was exceeded if animals were painted for 1 year.

Thus, animal experimentation has demonstrated a definite relationship between the amount and duration of tar applied to an animal and the total number of tumors and the duration of the latent period observed. The importance of this demonstration serves not so much as an example for those investigators who report negative results after painting animals with insufficient amounts of tar or over a too short time period, but serves chiefly to reemphasize the existence of a threshold value for tobacco tar below which tumors will not or but infrequently occur after a long latent period. Here again human and animal data confirm one another. The realization of this principle has practical aspects that are discussed below.

TOBACCO CARCINOGENS

Having established the carcinogenic activity of cigarette tar, one needs to determine the nature of the carcinogens responsible for this activity. The benzypyrene content of cigarette tar is not more than 2 parts per million, which, according to our experiments, is not sufficient to produce the type of activity noted in our animals painted with tobacco tar (Wright and Wynder, 1955, 1956). The arsenic content of our tar sample is about 35 parts per million. Again this is not sufficient to induce the number of skin cancers produced. Cooper and Lindsey (1955) have found traces of 1 : 2-benzpyrene, and G. L. Wright (unpublished data) observed spectrographic evidence of traces of 2 methyl,3 : 4-benzphenanthrene. At present there is no evidence of a known carcinogen in tobacco tar in quantities large enough to account for the observed animal activity. A summation effect of the substances just mentioned or perhaps a cocarcinogenic effect of other substances remains to be ruled out. It seems more likely, however, that there are carcinogenic substances in tobacco tar which are as yet unestablished.

Most, though not all, of the carcinogenic activity of the cigarette tar lies in the neutral fraction (Wynder and Wright, 1956). Within the neutral fraction the carcinogenic components are those that are eluted by hexane and particularly by carbon tetrachloride or solvents of similar polarity. Both of these fractions proved highly active for the rabbit, and the latter particularly potent for the mouse (Wynder and Wright, 1956). The benzpyrene content of both fractions is low. In the concentration as applied to the animals it was not more than 2 parts per million. In these fractions there is spectrographic evidence of other higher aromatic hydrocarbons which are present in 50 times the concentration of benzpyrene. We are currently engaged in various separation schemes trying to isolate these substances.

FORMATION OF THE TOBACCO CARCINOGENS

Present evidence suggests that the majority, though not all, of the carcinogens in tobacco are formed during pyrolysis. Human statistics comparing the risk of cancer among tobacco chewers with that of tobacco smokers, and our own experimental work comparing the activity of cigarette tar obtained by extracting cigarettes with methanol and hexanes with that of condensed cigarette smoke, indicate far greater activity for the latter in each case (Wynder and Wright, 1956). Thus there is good reason to believe that the majority of the carcinogens are formed during combustion. In fact, ever since the classical work of Kennaway (1925) and his associates showing that under pyrolytic conditions by which they are formed the pyrolysis of isoprene and that of acetylene in turn form potent carcinogens, it has been apparent that higher aromatic hydrocarbons can be formed by the pyrolysis of saturated or terpinoid aliphatic hydrocarbons. Most recently this was again demonstrated by Lam 1956a, 1956b), who obtained such substances upon pyrolysis of dicetyl as well as of aliphatic tobacco hydrocarbons. In this respect one recalls Kennaway's recent remark (personal communication) that in view of the high temperatures associated with smoking he was surprised that tobacco smoke was not more carcinogenic than present evidence indicates.

It is our working hypothesis that a major proportion of the tobacco-tar carcinogens are formed through pyrolysis of substances naturally occurring in tobacco, which thus serve as precursors to tobacco-tar carcinogens. The practical aspects of research in this field must be directed towards reducing the exposure to and the formation of carcinogens in tobacco tar.

PRACTICAL ASPECTS

Practical aspects of solving the tobacco-cancer problem center in the following suggestions and research programs.

1. *Moderation of smoking habits.*—All a physician can do is to present the facts to the public. In general, of course, the public is unable to evaluate the present facts fully, in part because of the confusion that special interests in some countries have added to this situation. In the final instance it must become the function of the public health services of different countries to evaluate the data at hand, and, if convinced by the evidence that tobacco plays a part in the development of lung cancer, decide whether lives are more important than economic considerations. In view of the apparent difficulty that may be foreseen in arriving at a decision on this issue, research must be carried forward that may give a practical solution to the problem at hand.

2. *Effective filtration.*—During recent years filtered cigarettes have become increasingly popular, initially because of "supposed health protection" and today because of a combination of factors, which include large advertising campaigns, the perhaps more pleasant feeling of a filtered tip, and a somewhat "milder smoke." According to present knowledge filtration to remove specific components from the particulate phase of the smoke seems not possible. Thus we must concentrate on mechanical filtration permitting a minimum of tobacco smoke to pass, a minimum compatible with a satisfactory pressure drop and tobacco taste. In the United States some tobacco companies have taken advantage of the public's desire for filtered cigarettes and its equal wish for good tobacco flavor by marketing increasingly ineffective filters. Since both human and animal evidcence indicates that the risk of developing cancer is directly related to the amount of tar exposure, it stands to reason that a given filter which will permit x milligrams of tar to pass is more effective than one which will permit the passing of $3x$ milligrams of tar. Sooner or later, regulations must be passed that establish criteria for the amount of tar which may pass through a given filter and that will make it a requirement for the manufacturer to state the effectiveness of a given filter. Present work indicates that it is entirely possible to develop a filtered cigarette with a good pressure drop and satisfactory tobacco taste which will remove about 40 percent of nicotine and tar from a given cigarette. Uniform acceptanace of a filter in this range will be a partial answer to the present problem, provided, of course, that the smoker does not decide to smoke twice as many cigarettes, and provided, too, that the tobacco selection, cut, or packing is not altered in such a way as to yield increasingly more tar.

3. *Removal of precursors.*—If the hydrocarbons naturally occurring in tobacco represent one of the major precursors of tobacco-tar carcinogens, a reduction of such substances may have a direct effect in reducing the carcinogens. Most of the aliphatic hydrocarbons are in the waxes that cover the tobacco leaf. Terpenes are also present. It may be practicable to remove all or part of these substances from tobacco by washing it with eluents such as hexane. Experiments testing this hypothesis are now in progress.

4. *Modification of pyrolysis.*—Since pyrolysis plays an important part in the formation of carcinogens, modifications of pyrolysis may affect the formation of carcinogens. This problem can be studied from the point of view of all those factors that affect the process of pyrolysis of tobacco. This includes the amount of oxygen supply, the temperature levels reached and maintained, the catalysis available, the cut of the tobacco leaf as such, the length to which a cigarette is smoked, and even the cigarette paper used. Experiments are now in progress for studying the interrelation of these factors so as to determine how a modification of the factors might lead to a reduction of the carcinogens formed during pyrolysis.

In this respect recent work of Lam (1956a, 1956b) is of particular interest. Pyrolysing aliphatic tobacco hydrocarbons, he found smaller amounts of aromatic compounds formed at 700° C. than at 800° C. At the latter temperature Lam found 3,4-benzpyrene to account for 1.3 percent of the total aromatic compounds and at 700° C. for 0.5 percent. The respective figures in parts per million were 340 and 30. At 600° C. Lam was unable to find evidence of aromatic substances, but studies by Wright indicate that some polynuclear aromatics are found even at lower temperatures (G. Wright, unpublished data).

CONCLUSION

The practical aspects outlined above may not individually lead to a tobacco tar that will have less tumorigenic activity, but a combination of these factors may be effective.

This discussion has dealt only with the problem of carcinogens and not with the equally important problems of nicotine and its apparent effect on the cardio-

vascular system. Jointly with work carried out toward reducing the carcinogens in tobacco tar to a subthreshold level, work should also be carried out on nicotine effects to determine whether it can also be brought to a subthreshold level so far as its effects on man are concerned.

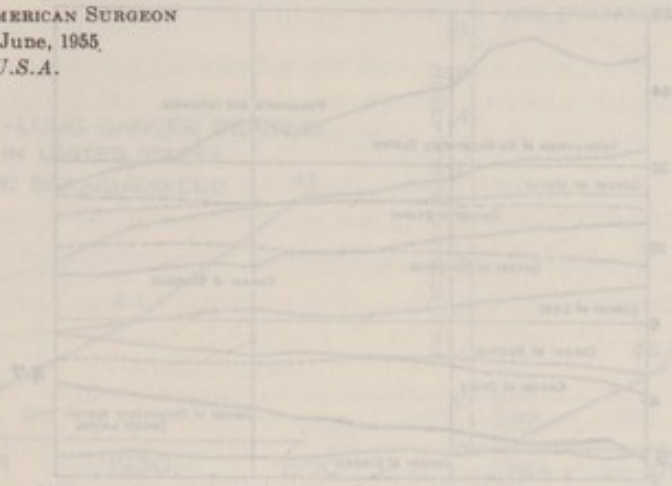
A final point concerns a comparison of human and animal data referred to in the beginning of this discussion. It was there stated that the proof that smoking is the cause of cancer in man rests entirely upon human statistical and epidemiological data. Animal data chiefly serve to indicate the nature of the carcinogens in tobacco tar. Obviously they can only indicate the nature of a carcinogen to a specific animal strain, species, and tissue. This truism applies not only to this specific instance but to all phases of cancer research. In cancer chemotherapy, as in carcinogenesis, many similarities and also many differences are known to exist between human and animal data. On having identified the specific carcinogens in tobacco tar as they apply to the experimental animal, one can only assume that these carcinogens are also responsible for the human cancer problem. If an agent has been found to be carcinogenic to a variety of animals, and human epidemiological data are not inconsistent with this finding, then the burden of proof lies upon those who claim that these substances are not carcinogenic to man. For that reason we should try to establish measures, if at all practicable, that can reduce established carcinogens from tobacco tar and apply these to the manufacturing of smoking products and the smoking habits of the population. This problem, as any other problem in life, cannot be solved by ignoring its existence or depreciating its importance. It can only be solved by facing it directly. In view of the work already completed and the investigations in progress now, a satisfactory solution should be attainable. The lives of people everywhere who will in years to come die from cancer of the lung and cancer of the upper respiratory tract demand that we give this problem our fullest attention.

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EXHIBIT 5A

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THE RELATIONSHIP OF SMOKING AND CANCER OF THE LUNG

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Undoubtedly many lay persons as well as physicians have been concerned by the increased incidence of cancer generally. The increase in most cancers is due to the fact that cancer primarily is a disease of older persons and because at present life expectancy is considerably greater than 25 years ago; there are more persons living to a cancer age than ever before. In fact, of all persons 90 years of age a greater percentage will develop cancer than those 80; of those 80 years of age a greater percentage will develop cancer than those 70, and so on. This is true of every type of cancer except one—cancer of the lung—which has increased and is increasing more than any other cancer in the body.

Whereas cancer of the lung occurs primarily in men (at the present time about 90 per cent of cancers of the lung occur in males), it is increasing in both sexes. According to the Public Health statistics cancer of the lung increased in women from 2.3 per 100,000 population in 1933 to 4.7 per 100,000 population in 1948 (fig. 1). The respective increases in men in these two periods was from 4.6 to 17.8 (fig. 2). If one considers the increase after age standardization, cancer of the lung increased in women from 0.6 per 100,000 population in 1914 to 4.3 per 100,000 population in 1950 (fig. 3); whereas, in men, the increase was from 0.7 per 100,000 population in 1914 to 19.6 per 100,000 population in 1950 (fig. 4). There was an increase of 411 per cent in the incidence of cancer of the lung in men from 1930 to 1948 (fig. 5). The deaths from lung cancer in both sexes increased from 3,900 in 1930 to 27,000 in 1953. Clemmesen,[2] in a recent communication, believes that bronchogenic cancer is increasing so rapidly that it can be expected that in men it will equal in frequency all other cancers combined. He refers to the increase in the incidence of bronchogenic cancer as being *pandemic*.

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Presented during the Atlanta assembly of The Southeastern Surgical Congress, Feb. 21–24, 1955.

DEATH RATES FOR SELECTED RESPIRATORY DISEASES AND SITES OF CANCER
AMONG WHITE FEMALES, United States, 1933-1948

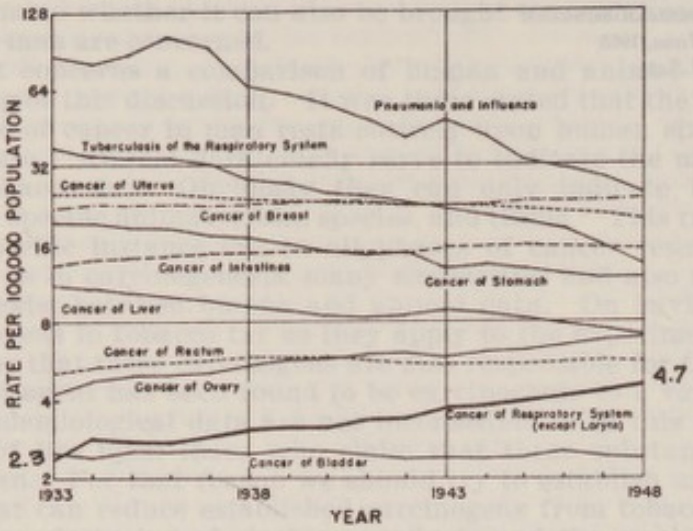


FIG. 1

DEATH RATES FOR SELECTED RESPIRATORY DISEASES AND SITES OF CANCER
AMONG WHITE MALES, United States, 1933-1948

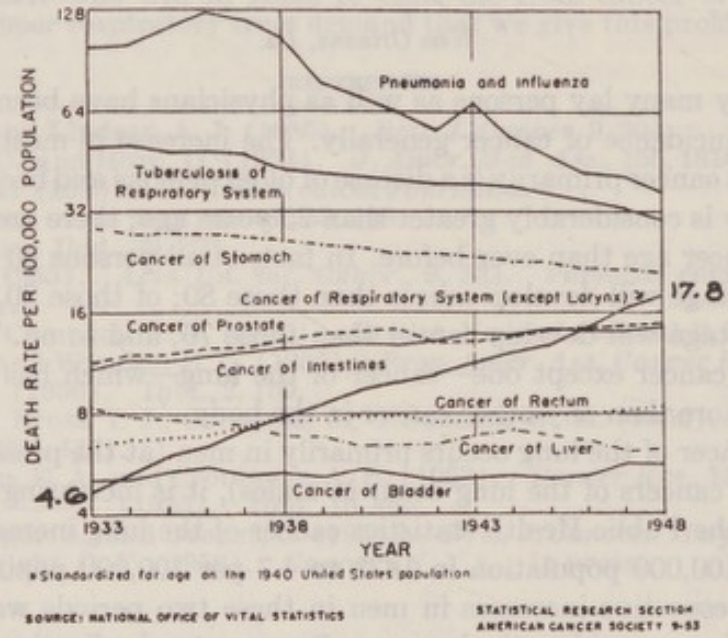


FIG. 2

Although, as stated previously, cancer is increasing generally, cancer in females has actually decreased. This is a real tribute to the educational program of the American Cancer Society and its efficacy in that women have learned that it pays to consult their physicians regularly with the result that precancerous lesions are being detected and corrected, producing a decrease in the incidence of cancer in women. On the other hand, cancer in men has increased very remarkably in the past decade and this has been due almost entirely to the tremen-

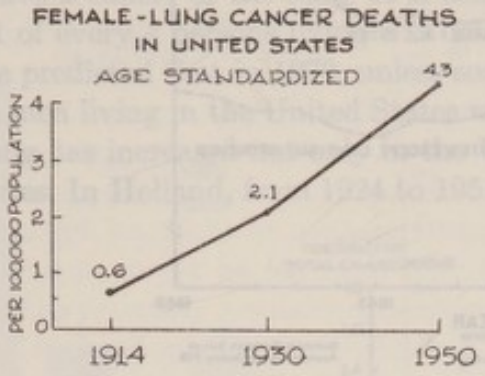


FIG. 3

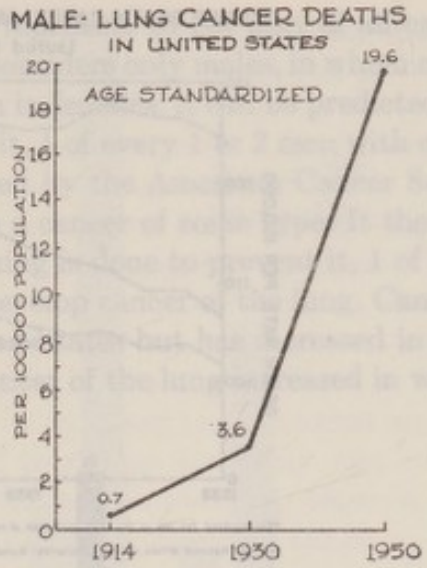


FIG. 4

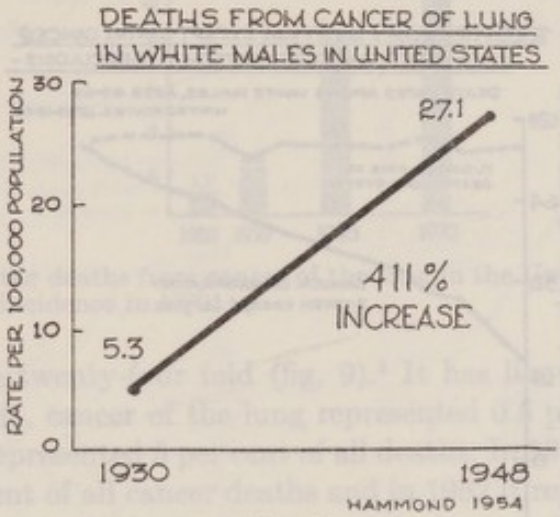


FIG. 5

dous increase in the incidence of bronchogenic cancer. If one excludes bronchogenic cancer, there has been very little increase in the incidence of cancer in men in the past quarter of a century (fig. 6).

There are many who maintain that cancer of the lung is not increasing but that it is simply being diagnosed at the present time, whereas previously it was not recognized and the condition was diagnosed as some other lesion. If this were the case, the one lesion with which cancer of the lung might be confused would be tuberculosis. Whereas there has been a general decrease in the incidence of tuberculosis in recent years, there has been no decrease in the incidence of tuberculosis in the older age group in which cancer of the lung occurs. In fact, if the increase in the incidence of cancer of the lung were due to better diagnosis at the present time, at the same time there should be a decrease in the incidence of tuberculosis in the older age group commensurate with the increase in the

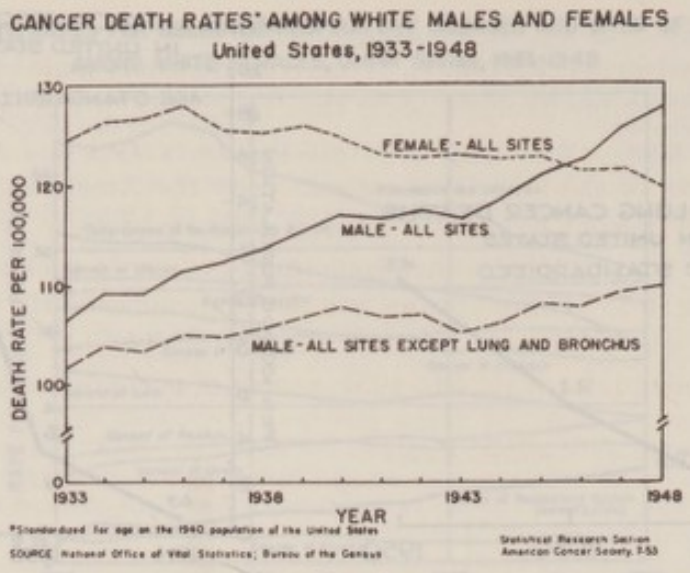


FIG. 6

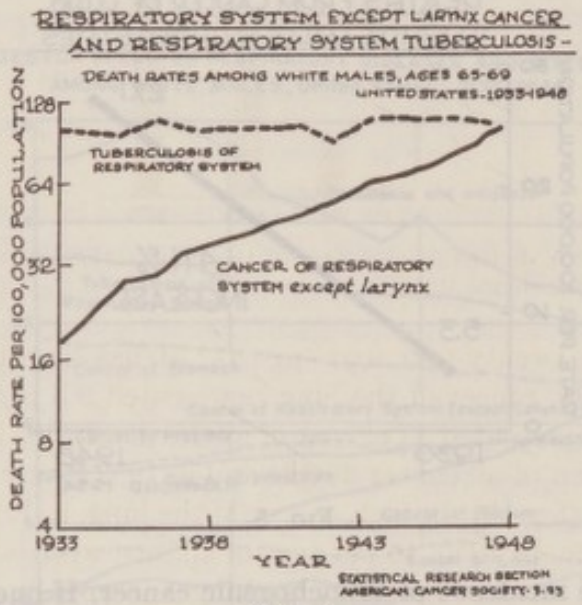


FIG. 7

incidence of cancer of the lung, but such is not the case (fig. 7). Another reason why I am convinced that there has been an actual increase in the incidence of bronchogenic cancer and not a relative one due to more correct diagnoses, is that in those countries where autopsies have been done routinely for many years, such as the Germanic countries, bronchogenic cancer has increased as it has in this country, and it is inconceivable that the well-trained, meticulous German pathologist would miss bronchogenic cancer at autopsy.

In the United States, cancer of the lung in 1920 represented 1.1 per cent of all cancers; in 1930, 2.2 per cent of all cancers; in 1948, 8.3 per cent of all cancers. Upon the basis of this increase from 1920 to 1948 and because of the presence of certain carcinogenic factors which will be discussed later, we have ventured to

predict that in 1970 cancer of the lung will represent 18 per cent of all cancers or approximately 1 in every 5 (fig. 8). If one considers only males, in whom cancer of the lung is much more likely to occur than in females, it can be predicted that in 1970, unless something is done to prevent it, 1 of every 1 or 2 men with cancer will have a cancer of the lung. It is estimated by the American Cancer Society that 1 of every 4 persons living will develop a cancer of some type. It therefore can be predicted that in 1970, unless something is done to prevent it, 1 of every 4 or 8 men living in the United States will develop cancer of the lung. Cancer of the lung has increased not only in the United States but has increased in other countries. In Holland, from 1924 to 1951, cancer of the lung increased in women

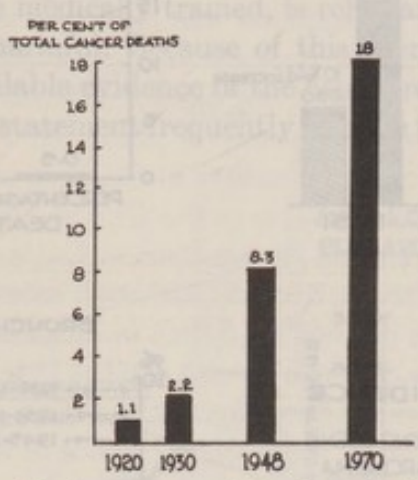


FIG. 8. Increase in the deaths from cancer of the lung in the United States from 1920 to 1948 with a predicted incidence in 1970.

tenfold and in men twenty-four fold (fig. 9).[5] It has likewise increased in the British Isles. In 1931, cancer of the lung represented 0.5 per cent of all deaths, whereas in 1952 it represented 5 per cent of all deaths. In 1931, cancer of the lung represented 5 per cent of all cancer deaths and in 1952 it represented 26 per cent of all cancer deaths (fig. 10). In fact, cancer of the lung, in England, is by far the most common cancer. In 1950, 10 per cent of all men who died between the ages of 45 and 55 died of primary cancer of the lung—an appalling fact![3]

As mentioned previously, cancer of the lung is the one cancer in which the incidence does not correspond to the pattern followed by other cancers. Whereas all other cancers increase with advancing age past 50, cancer of the lung increases precipitously to reach a peak age at the present time of 55 years following which there is a rapid decline in incidence (fig. 11). This exceptional behavior of cancer of the lung is not new; in fact, 10 years ago, the age at which the peak incidence occurred was 65 but the peak was not as high as it is at the present time. Five years ago, the peak age was 60 and the height of the peak was somewhere between that which was present 5 years ago and that at the present time (fig. 12). The lack of conformity of the lung cancer increase to the pattern followed by all other cancers is due to a definite reason. For many years I have been convinced that the unprecedented increase in the incidence of cancer of the lung is due to the carcinogenic effect of cigarette smoke, and I am sure that this accounts for the

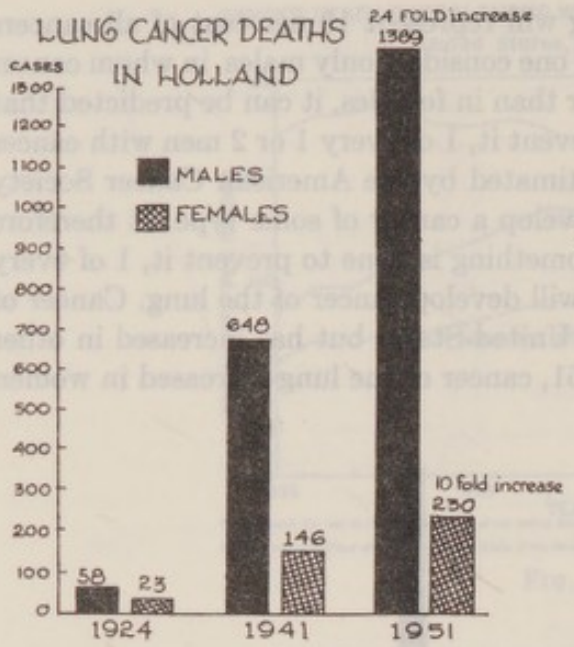


FIG. 9

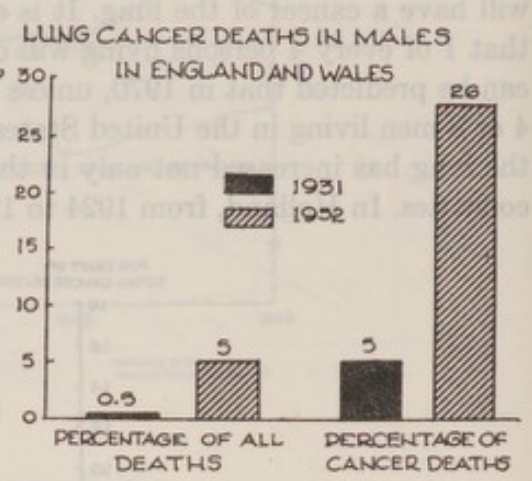


FIG. 10

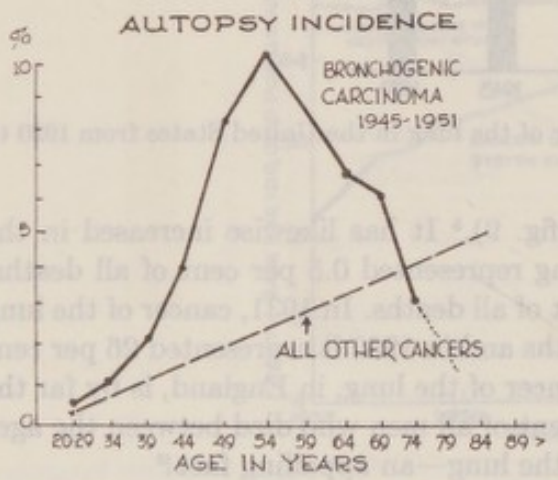


FIG. 11

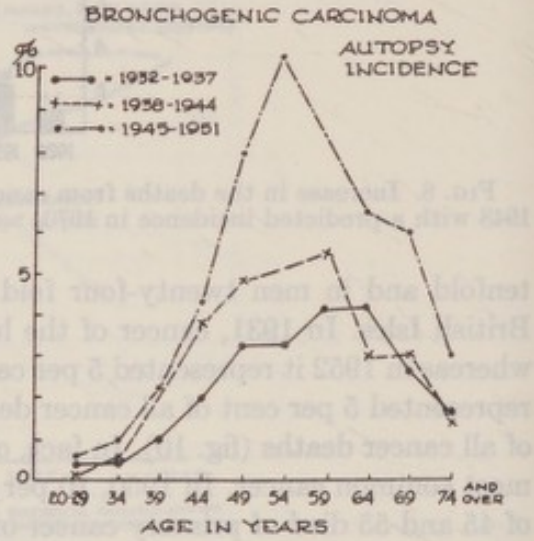


FIG. 12

peculiar behavior in the pattern followed by cancer of the lung. The tremendous increase in the incidence of cancer of the lung is due to the fact that cigarette smoking has increased enormously. The peak age is becoming higher and at an earlier age, because more people are smoking heavily and beginning to smoke at an earlier age and, therefore, the carcinogenic effect is being produced at an earlier age today than it was 10 years ago and an even earlier age than it was 5 years ago. The fall-off in the incidence of cancer of the lung after the peak age is obtained is due to the detrimental effect of the tobacco on the heart and blood vessels of individuals who have smoked heavily, producing coronary heart disease resulting in the death of the individual before the carcinogenic effect has had a chance to exert itself. It is a well known fact that cigarette smoking produces cardiovascular damage.

The annual consumption of cigarettes per capita in the United States has inincreased tremendously (fig. 13); in fact, in persons 15 years of age and older the number increased from 630 in 1920 to 3,500 in 1953 (fig. 14). There also is a distinct parallelism between the consumption of cigarettes in the United States and the increase in the incidence of bronchogenic cancer (fig. 15).

It is thoroughly understandable that there are many individuals who are unwilling to accept a causal relationship between smoking and bronchogenic cancer. Obviously those concerned with the raising of tobacco, its marketing, the processing and the sale of the final product are reluctant to accept any such causal relationship. In addition, the cigarette user who has become addicted to its use, even though he might be medically trained, is reluctant to admit that anything that he does might be harmful. Because of this there are many who will not accept the presently available evidence of the causal relationship between smoking and lung cancer. The statement frequently is made that there can be no causal

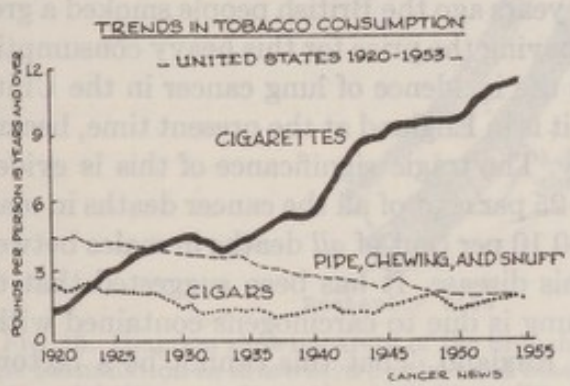


FIG. 13

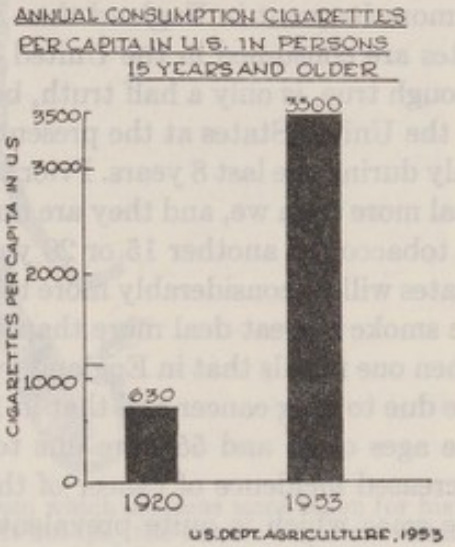


FIG. 14

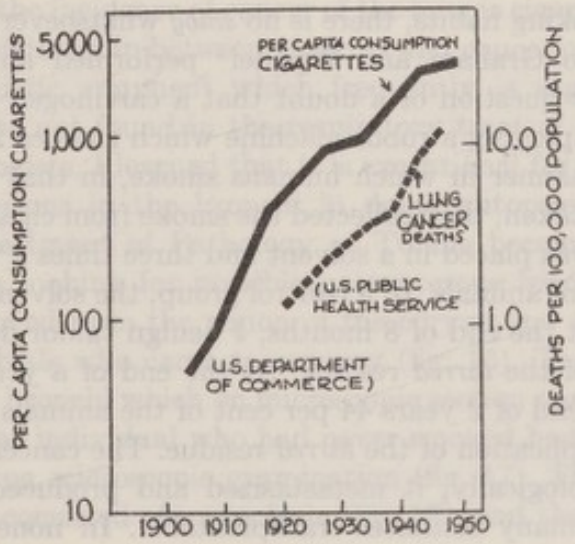


FIG. 15

relationship between smoking and lung cancer because, if there were, there should be a commensurate increase in the fatality incidence from cancer of the larynx which is not the case. Whereas it is true that the death rate from cancer of the larynx has not been proportionate to the death rate from cancer of the lung, it is equally as true that cancer of the larynx has been and is increasing with the same frequency as cancer of the lung. The difference between cancer of the larynx and cancer of the lung is that cancer of the larynx is readily diagnosed, usually when the lesion is early, and can be very satisfactorily treated with cure in a large percentage of instances, whereas cancer of the lung is difficult to diagnose early and because of this the death rate from cancer of the lung has been high. At one time, the death rate from appendicitis was high, but no fair minded individual would hazard a statement that appendicitis has decreased or is not a common disease at the present time because few cases are found at autopsy.

Another half truth which frequently is presented against a possible causal relationship between smoking and cancer of the lung is that cancer of the lung is more frequent in England than it is in the United States although more cigarettes are consumed in the United States than in England. This statement, even though true, is only a half truth, because, although more cigarettes are consumed in the United States at the present time than in England, such has been the case only during the last 8 years. Prior to 8 years ago the British people smoked a great deal more than we, and they are now paying the price for this heavy consumption of tobacco. In another 15 or 20 years the incidence of lung cancer in the United States will be considerably more than it is in England at the present time, because we smoke a great deal more than they. The tragic significance of this is evident when one recalls that in England over 25 per cent of all the cancer deaths in males are due to lung cancer and that in 1950 10 per cent of *all* deaths in males between the ages of 45 and 55 were due to this disease. It has been suggested that the increased incidence of cancer of the lung is due to carcinogens contained within the *smog* which is quite prevalent in England. That this cannot be a factor is illustrated by the fact that across the English Channel in Denmark, where the incidence of cancer of the lung has increased as it has in England and is commensurate with smoking habits, there is no *smog* whatsoever.

Several years ago Graham and Wynder[8] performed an experiment which proved without any question of a doubt that a carcinogen exists in the smoke from cigarettes. Employing a robot machine which smokes 24 cigarettes almost identically to the manner in which humans smoke, in that every 60 seconds a *drag* of 2 seconds is taken, they collected the smoke from cigarettes and cooled it. The *tarred* residue was placed in a solvent and three times a week it was applied to the skin surfaces of animals. In a control group, the solvent without the *tarred* residue was used. At the end of 8 months, 1 benign tumor developed at the site of the application of the *tarred* residue. At the end of a year 1 real cancer developed and at the end of 2 years 44 per cent of the animals developed a cancer at the site of the application of the *tarred* residue. The cancer was identical with human cancer histologically, it metastasized and produced the death of the animal and was in many instances transplantable. In none of the controls in

which the solvent alone was applied to skin of animals did either a benign or malignant tumor develop. The statement frequently has been made that one cannot compare animal and human cancer. In this investigation no attempt was made to make this comparison. The investigation simply proves that contained within the smoke from cigarettes is a carcinogen. Since it is an established fact that cancer of the lung is increasing more than any other cancer in the body and also that there is a parallelism between the consumption of cigarettes in the United States and the incidence of cancer of the lung and because the investigation of Graham and Wynder has shown that there is a carcinogen in the smoke from cigarettes, it is a perfectly logical conclusion that the cause for the unprec-

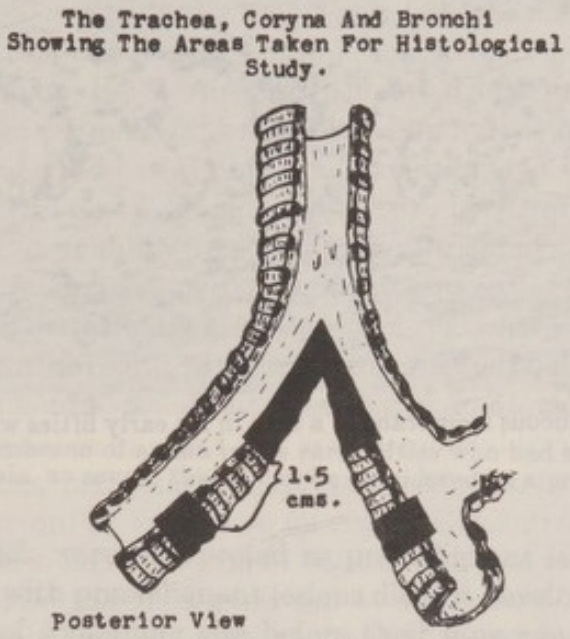


FIG. 16. Diagram showing areas of the bronchi from which sections were taken for histologic examination as illustrated by the solid black. At autopsy the coryna was removed and portions of the mucous membrane, 1.5 cm. distally in each main stem bronchus.

edented increase in the incidence of cancer of the lung is cigarette smoking, establishing the causal relationship between smoking and cancer of the lung.

Another antagonistic argument which frequently is suggested is that precancerous lesions are not found in the respiratory tract of smokers. After consulting with pathologists, I learned that it is exceptional for pathologists to look for precancerous lesions in the bronchi in doing autopsies. Approximately 2 years ago the Department of Pathology at Tulane became interested in this problem and began looking for possible precancerous lesions in the blocks of bronchial wall removed from the region of the coryna and also from each main bronchus of individuals who came to autopsy (fig. 16). They found that there were changes in the bronchi which on microscopic section varied according to the amount smoked. The individual who had never smoked had a normal bronchial mucous membrane on microscopic examination (fig. 17). The moderate smoker had changes which consisted of metaplasia (fig. 18), and the heavy smoker had



FIG. 17. Bronchial mucous membrane of a man in his early fifties who had never smoked.

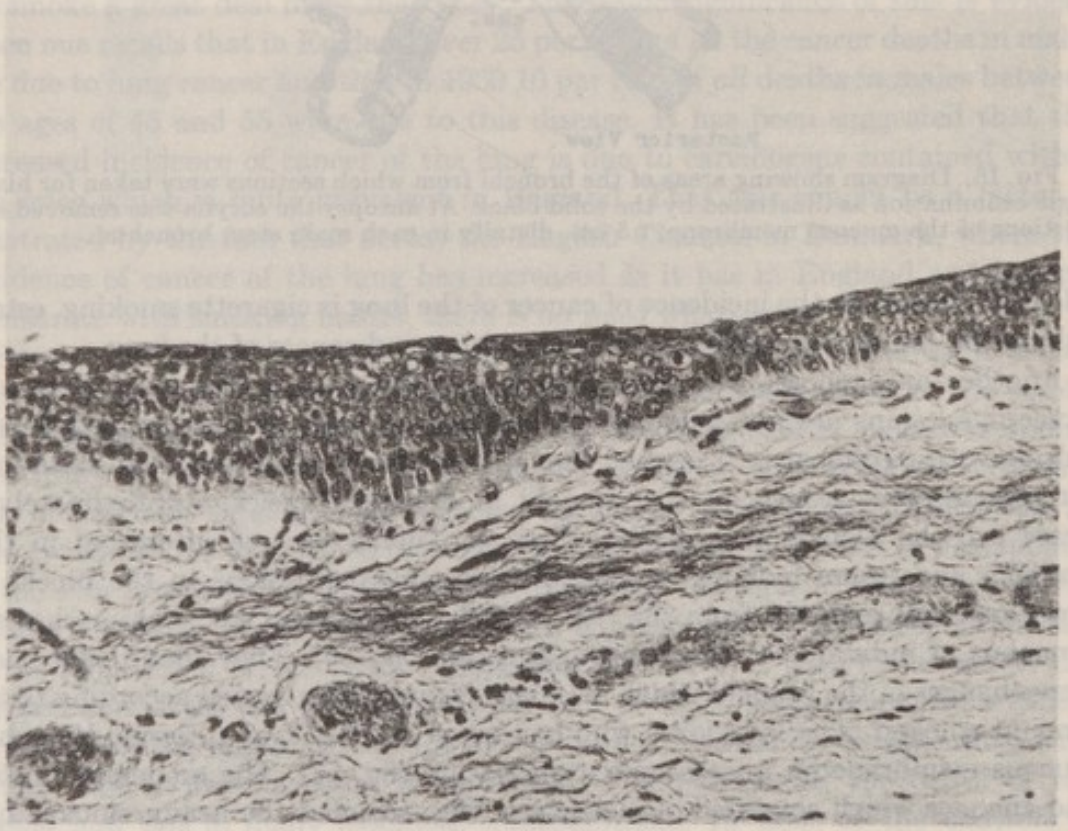


FIG. 18. Bronchial mucous membrane of a man in his early fifties who had smoked moderately. In contrast to the mucous membrane in figure 17, there is definite metaplasia in the mucosa.



FIG. 19. Mucous membrane of a man in his early fifties who had smoked heavily. There is tremendous metaplasia, so severe that it can be considered as a precancerous lesion.

marked changes which were interpreted as premalignant lesions (fig. 19). The only reason patients with premalignant lesions did not develop cancer of the lung was that they died of something else before their lung cancer developed. This study demonstrates that cigarette smoking is associated with precancerous lesions in the bronchial mucosa and that the degree of metaplasia increases with the amount smoked.

For many years those who have been concerned with the treatment of patients with bronchogenic cancer have been impressed with the high frequency of heavy smokers among men who have this disease. In fact I have been so impressed by this that for the past 5 years I have categorically made the statement that if any man who has a pulmonary condition which might be bronchogenic cancer, if he is not a smoker, the lesion is not an epidermoid cancer but is either an adenocarcinoma, which is not produced by a carcinogen, or is an inflammatory lesion. In 5 years time I have been wrong only once. The positive history of smoking at present is our best method of making a diagnosis of a malignant lesion of the lung. Well over 95 per cent of men with cancer of the lung are cigarette smokers as contrasted with a control group of from 65 to 75 per cent without cancer of the lung. Recently, Breslow and associates[1] showed, on the basis of careful examination of 518 patients with bronchogenic cancer as compared with 518 patients of the same age and sex without lung cancer that in men over 50 years of age,

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lung cancer was 4 to 11 times as frequent among smokers as nonsmokers and 7 to 27 times as frequent among heavy smokers.

The criticism frequently is made that a high incidence of smokers in patients with cancer of the lung is of no significance, but until it is known what the incidence of lung cancer is among smokers as compared to nonsmokers, a possible relationship cannot be considered. The answer to this question was not available until the preliminary report of the investigation by the American Cancer Society, which was made before the American Medical Association in San Francisco in June, 1954.[4] The survey conducted by the American Cancer Society briefly was as follows: Twenty-two thousand volunteer workers in urban areas, in highly industrialized areas, and in rural areas interviewed approximately 200,000 men between the ages of 50 and 70, because this is the age group in which cancer of the lung occurs most frequently. A very elaborate questionnaire was filled out concerning the man's smoking history. One year later the interviewer reinterviewed each of the men whom she had interviewed the year before. In the meantime some of the men interviewed had died. A photostat of the death certificate was secured and, if the individual had died of cancer and had had a biopsy or autopsy, an attempt was made to obtain the microscopic slide of the tissue removed. Two years after the original interview, a third interview was secured. Approximately 5,000 men had died since the beginning of the survey, representing 2.6 per cent of the entire group interviewed. The results of this investigation are extremely illuminating. The investigation showed that there has been a tremendous increase in the incidence of smoking in recent years. Of the men in the age group 65 to 70, 21.6 per cent had never smoked and 20.3 per cent were heavy smokers; whereas in the age group 50 to 55 only 15 per cent had never smoked and 43 per cent were heavy smokers (fig. 20). The study showed that there was a definite increase in the death rate from all causes among the individuals who smoked cigarettes as compared with those

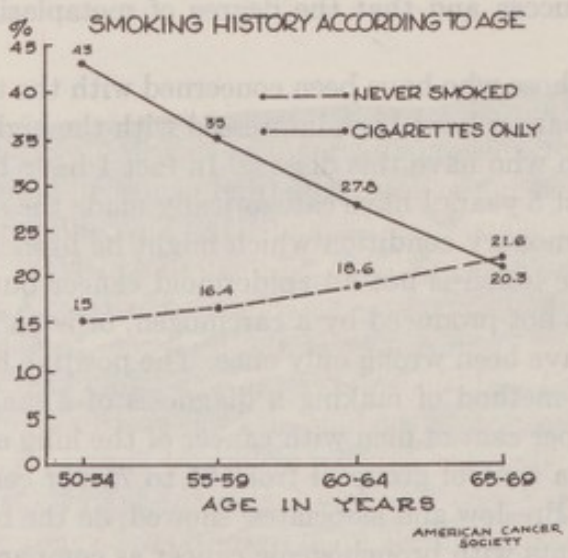


FIG. 20. Diagram showing that men in the fifty to fifty-five age group smoke much more than men in the sixty-five to seventy year age group.

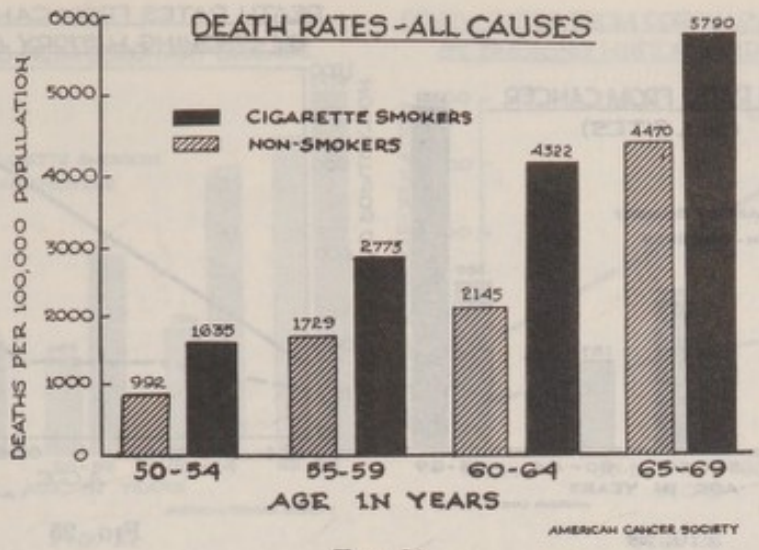


FIG. 21

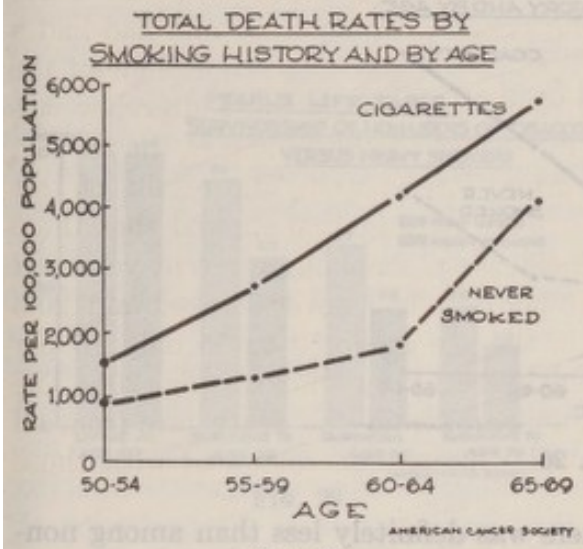


FIG. 22

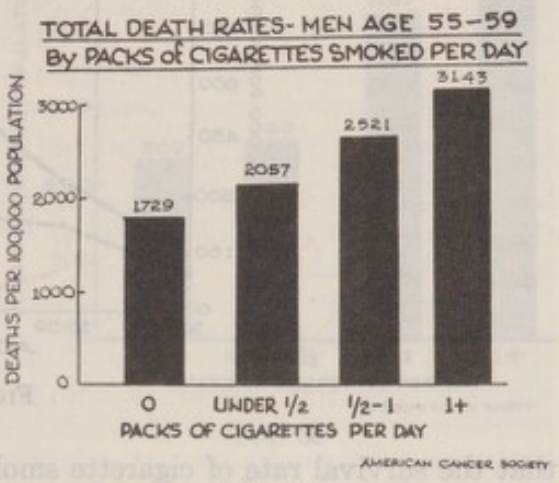


FIG. 23

who did not smoke; and this was true in all the age groups and the death rate varied according to the amount smoked (figs. 21, 22, and 23). There was considerable difference in the death rate from cancer in all sites among cigarette smokers and nonsmokers (figs. 24 and 25). This difference was particularly great in the age groups 60 to 65 and 65 to 70 in which the death rate was over twice as great among the smokers as compared with nonsmokers (fig. 25). Although the death rate from cancer of the lung was particularly higher among smokers as compared with nonsmokers, it was found that the death rate from other cancer besides cancer of the lung also was greater among cigarette smokers than among nonsmokers (fig. 26). An incidental finding was that the death rate from coronary disease was definitely higher in cigarette smokers than in nonsmokers and this was particularly great in the 60 to 65 age group (figs. 27 and 28). This was not unanticipated, because many years ago Raymond Pearl[7] showed

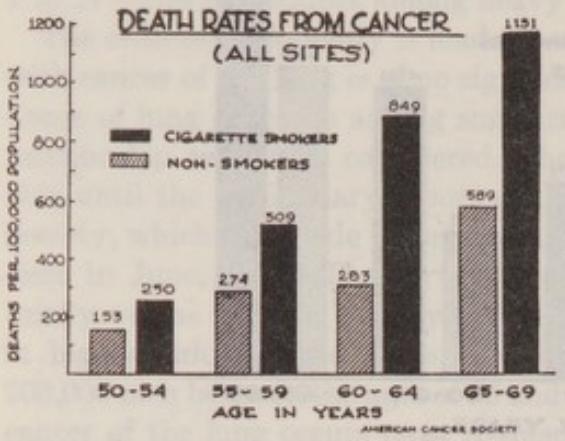


FIG. 24

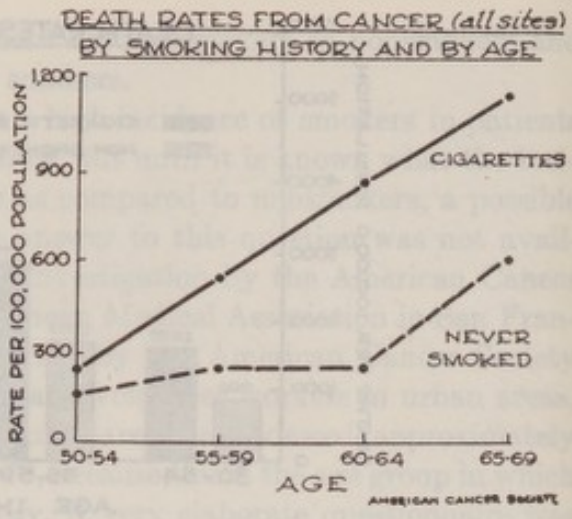


FIG. 25

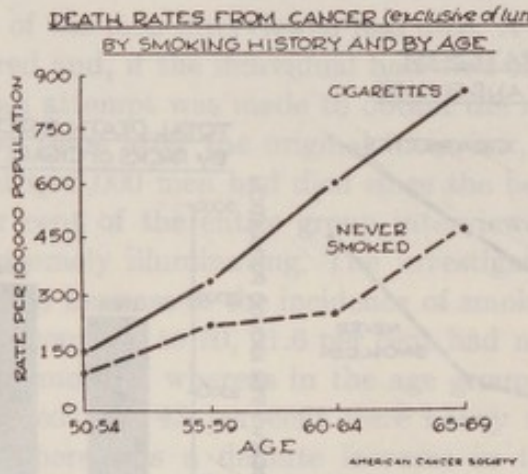


FIG. 26

that the survival rate of cigarette smokers was definitely less than among nonsmokers (fig. 29). Also, the American Cancer Society study[4] showed that the death rate from coronary disease was proportionate to the amount smoked. Of the group that smoked none at all there were 689 per 100,000 population deaths; of those who smoked less than a half pack there were 769; of those who smoked from a half a pack to a pack there were 1,287, and of those who smoked more than a pack there were 1,477 (fig. 30). To recapitulate, the study showed that among heavy cigarette smokers the over-all death rate was 75 per cent higher than among nonsmokers. The death rate from heart disease was 95 per cent higher than among nonsmokers; the death rate from all cancers was 156 per cent higher than among nonsmokers, and the death rate from lung cancer was 400 per cent higher than among nonsmokers (fig. 31)!

The study by the American Cancer Society[4] which was made in a completely impartial manner and which includes a large enough group of cases to be statistically significant proves without any reasonable question of doubt that there is a causal relationship between smoking and cancer—particularly lung cancer—as well as a causal relationship between cigarette smoking and heart disease. In

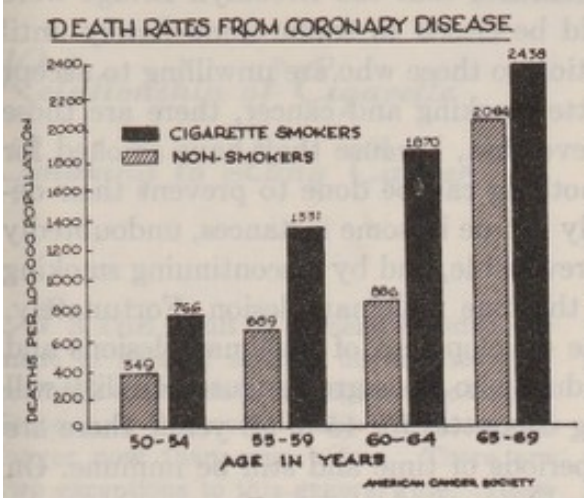


FIG. 27

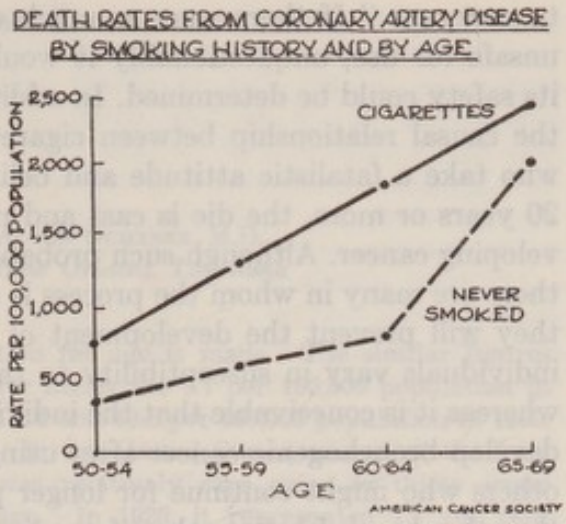


FIG. 28

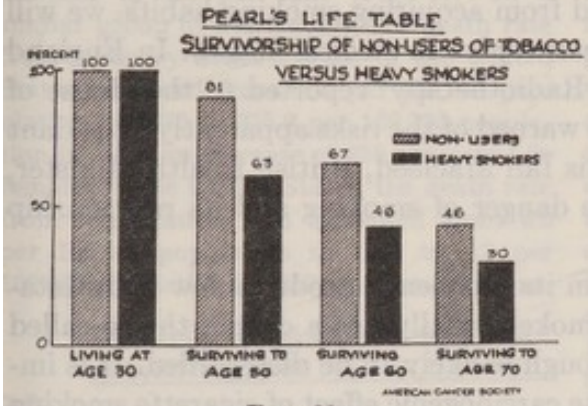


FIG. 29

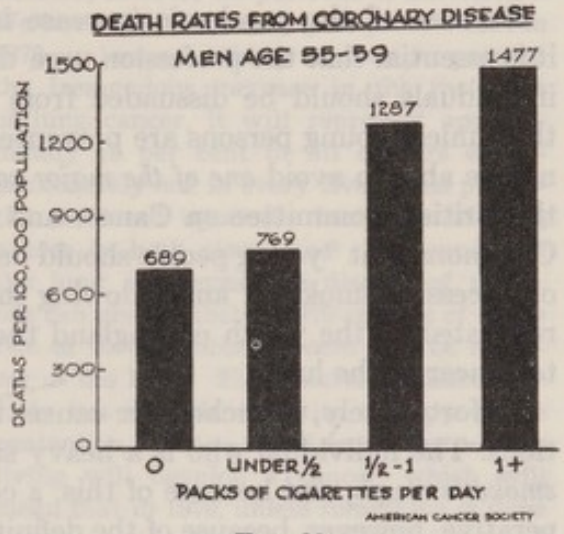


FIG. 30

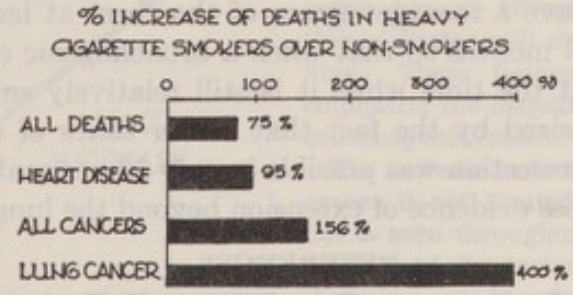


FIG. 31

spite of this there are many who are reluctant to accept this relationship. Clemmesen,[2] an eminent Danish pathologist, who has been very much interested in epidemiology of cancer for years, states: "It is true that we have no warrant of the effect, but where were the warrants in the combat against epidemics in the past? Let no one believe that the attitude of the public will remain indifferent when in one or two decades the extent of the catastrophe will become apparent

to everyone." If there were as much evidence that the Brooklyn Bridge were unsafe for use, unquestionably it would be closed to traffic immediately until its safety could be determined. In addition to those who are unwilling to accept the causal relationship between cigarette smoking and cancer, there are those who take a fatalistic attitude and believe that, because they have smoked for 20 years or more, the die is cast and nothing can be done to prevent their developing cancer. Although such probably is true in some instances, undoubtedly there are many in whom the process is reversible, and by discontinuing smoking they will prevent the development of the true malignant lesion. Fortunately, individuals vary in susceptibility to the development of malignant lesions and whereas it is conceivable that the individual who has a greater susceptibility will develop bronchogenic cancer after using cigarettes for 15 or 20 years, there are others who might continue for longer periods of time and still be immune. On the other hand, I believe that if one lives long enough and continues smoking he will ultimately develop bronchogenic cancer.

Because of the pandemic increase in the incidence of bronchogenic cancer, it is essential that the profession urge discontinuance of smoking and that young individuals should be dissuaded from acquiring the habit. Clemmesen[2] states that unless young persons are prevented from acquiring smoking habits, we will not be able to avoid *one of the major catastrophes in medical history*. In England the British Committee on Cancer and Radiotherapy[6] reported to the House of Commons that "young people should be warned of the risks apparently attendant on excessive smoking" and following this Ian Macleod, British Health Minister, reiterated to the youth of England the danger of smoking and its relationship to cancer of the lung.

Unfortunately, bronchogenic cancer in its incipiency produces few manifestations. The individual who is a heavy smoker usually has a cough, the so-called *smoker's cough*, and because of this, a cough is likely to be disregarded. It is imperative, however, because of the definite carcinogenic effect of cigarette smoking that men who smoke heavily, whether they are members of the profession or lay people, should have a roentgenogram of the chest at least every 6 months or preferably every 3 months so that when a bronchogenic cancer does develop it can be detected at the time while it is still relatively small. Need for such precaution is emphasized by the fact that in our series of over 1,500 cases of bronchogenic cancer resection was possible in only 32 per cent and in only 10 per cent was there no gross evidence of extension beyond the lung.

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EXHIBIT 5B

Relationship of Cigarette Smoking to Lung Cancer*

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New Orleans, Louisiana

AS THE result of tremendous advances made in medical science, death rates generally have decreased throughout the United States and the world. Life expectancy is longer now than ever before. There are two exceptions to this general rule. These two are cancer of the lung and coronary heart disease. In white males in the United States, the death rate from cancer of the lung increased from 5.3 per 100,000 population in 1930 to 27.1 per 100,000 in 1948, an increase of 411 per cent (Fig. 1). In a similar group of individuals, the death rate from coronary heart disease in the United States increased from 61.1 per 100,000 population in 1930 to 235.6 per 100,000 population in 1948, an increase of 286 per cent.[1] In females in the United States, the death rate from lung cancer has increased from 0.6 per 100,000 population in 1914 to 4.3 per 100,000 population in 1950 when standardization for age is made. The similar figures for males are 0.7 per 100,000 population in 1914 and 19.6 per 100,000 population in 1950.

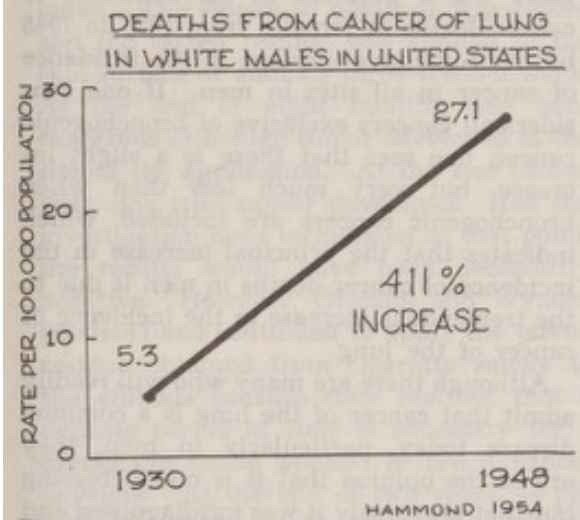


Fig. 1. An increase of 411 per cent in 18 years in deaths from lung cancer.

In the United States, cancer of the lung was relatively rare prior to thirty years ago. In 1920, it represented 1.1 per cent of all cancers; in 1930, 2.2 per cent of all cancers; in 1948, 8.3 per cent of all cancers. We have had the temerity to predict that in 1970, unless something is done to prevent the tremendous increase in the incidence of lung cancer, it will represent approximately 18 per cent of all cancers or approximately one in every five. This prediction is based upon the incidence of lung cancer in both sexes, and since cancer of the lung is primarily a disease of males, one can predict that in 1970 one out of every two or three cancers in men will be a cancer of the lung. The American Cancer Society has reliable statistics to support the contention that one out of every five persons living will develop a cancer, which will mean that in 1970, unless something is done to prevent the increase in the incidence of lung cancer, approximately one out of every ten or fifteen men living in the United States will develop a cancer of the lung. These figures are staggering and emphasize the magnitude of the problem of bronchogenic cancer.

The increase in incidence of bronchogenic cancer is not limited to the United States but is seen throughout the civilized world. In Holland, for instance, from 1924 to 1951, there was a twenty-fourfold increase in the incidence of lung cancer deaths in men and a tenfold increase in women during this same period of time. In 1931, cancer of the lung represented 0.5 per cent of all the deaths in England and Wales in males, whereas in 1952, this percentage had in-

*Presented before the annual session of the Colorado State Medical Society at Colorado Springs, September 21-24, 1954. From the Department of Surgery, Tulane University School of Medicine, and the Ochsner Clinic.

creased to 5 per cent. In 1931, cancer of the lung represented 5 per cent of all cancer deaths and in 1952, this percentage had increased to 26. In England during the year 1950, 4 per cent of all the male deaths were due to cancer of the lung and between the ages of 45 and 54, 10 per cent of the deaths in males were due to this disease.[2]

As stated previously, deaths from all causes except cancer of the lung and coronary heart diseases have been decreasing. It is a well known fact, however, that with each advancing year past 40 years of age there is an increasing incidence of cancer with increasing age. Cancer is primarily a disease of older persons. This is true of all cancers except cancer of the lung, which does not correspond to the pattern of a progressive increase in the incidence of the disease with each advancing year of life. At the present time, cancer of the lung increases very rapidly, much more rapidly than other cancers, to reach a peak incidence at approximately 55 years, following which there is a decrease in its incidence. This deviation from the pattern followed by all other cancers is seen not only at the present time but has been present for at least the past ten years, the only difference being that ten years ago the peak incidence was not as high as it is at the present time and occurred at a later date. Whereas, ten years ago, the peak incidence occurred at approximately 65 years, and five years ago at approximately 60 years, at the present time the peak incidence is 55 years.

In a survey by the American Cancer Society[3] it was found that among white males between the ages of 50 and 55 who were interviewed, 15 per cent had never smoked whereas 43 per cent had been heavy cigarette smokers, in contrast to a group of men between the ages of 65 and 70, in which 20.3 per cent smoked cigarettes heavily and 21.6 per cent had never smoked (Fig. 2). It is thus seen that heavy smoking is much more common in younger men today and the incidence of non-smoking much less than it is in older men. This is because youngsters who previously began smoking after 21 now begin smoking in the first decade of life.

The lack of conformity of lung cancer to the pattern followed by all other cancers we believe is due to the fact that there is a causal relationship between smoking and lung cancer and also between smoking and coronary heart disease. The increasing incidence occurring at an earlier age in the more recent period of time is due to the fact that men who are now 55 began smoking approximately ten years earlier than men who were 55 ten years ago. They have subjected their bronchi and cardiovascular systems to the deleterious effects of tobacco, and if they develop cancer they develop it at an earlier age than that at which is previously occurred. The reason for the decrease in the incidence of bronchogenic cancer after the peak is obtained is that individuals who are heavy smokers have subjected their hearts and blood vessels to the deleterious effects of tobacco and many develop coronary heart disease, succumb to it, and do not live long enough to develop bronchogenic cancer. One might facetiously state that a dubious advantage of cigarette smoking is that one might be spared a lung cancer death because of the possibility of developing coronary thrombosis and dying from it before lung cancer has a chance to develop.

According to the Public Health statistics, there was a decrease in the incidence of cancer deaths in females from 1933 to 1948 but a progressive increase in the incidence of cancer in all sites in men. If one considers all cancers exclusive of bronchogenic cancer, one sees that there is a slight increase, but very much less than when bronchogenic cancers are included, which indicates that the principal increase in the incidence of cancer deaths in men is due to the tremendous increase in the incidence of cancer of the lung.

Although there are many who will readily admit that cancer of the lung is a common disease today, particularly in men, they are of the opinion that it is not increasing but that previously it was misdiagnosed and that the increase in the incidence is not real but only apparent. There are two reasons why we are of the opinion that this contention is not true. One is that in those countries, such as the Germanic countries,

where autopsies are done routinely and have been done for the past hundred years, the incidence of bronchogenic cancer has increased throughout the years. Even by the greatest stretch of imagination, one cannot envision a well-trained German pathologist overlooking a bronchogenic cancer at autopsy twenty or thirty years ago. Another reason is that if incorrect diagnoses were made previously, there should be a decrease in the conditions which were erroneously diagnosed as bronchogenic cancer. If one considers the older age group in which bronchogenic cancer is frequent, it is evident that there was no decrease in the incidence of pulmonary tuberculosis between the years 1933 to 1948, during which time there was a tremendous increase in the incidence of bronchogenic cancer.

As mentioned previously, it is our conviction that there is a definite causal relationship between cigarette smoking and lung cancer and that the cancer is due to a carcinogen in cigarette smoke. That carcinogens are present in the smoke from cigarettes has been shown by Graham and Wynder.[4] These investigators obtained cigarette smoke by smoking cigarettes in a robot machine in a comparable manner to the way humans smoke cigarettes. Every sixty seconds a drag of two seconds was taken. The smoke so obtained was cooled and the tarred residue was applied to the skin surface of animals three times a week. At the end of eight months, one noncancerous or benign tumor developed at the site of the application. At the end of one year, one true cancer developed. Had the investigation been terminated at that point, the results would have been necessarily negative. However, Graham and Wynder persisted and continued to apply the tarred residue obtained from cigarette smoke to the animals because they realized that a definite period of time is necessary for a carcinogen to be applied before a cancer develops. At the end of two years, 44 per cent of the animals developed a true cancer which was indistinguishable from human cancer, histologically and biologically. It metastasized, killed the animal, and in many instances was transplantable. The criticism has been raised that one cannot compare human and animal cancer. In citing the investigative work of Graham and Wynder, no attempt is made to compare human and animal cancer. This simply demonstrates that contained within the smoke from cigarettes is a factor which contains carcinogens which will produce a cancer.

The criticism is frequently raised that there can be no causal relationship between the incidence of smoking and cancer of the lung because precancerous conditions are not found in the bronchi, which should be the case were the lung cancer the result of the carcinogenic effect of tobacco. Unfortunately, in performing autopsies, very little attention is paid to the microscopic changes in the bronchial mucosa, and it is because of this that the early changes are not known. Relatively recently a senior medical student[5] at Tulane University examined the bronchi of patients coming to autopsy. He found that there was considerable difference in the histologic picture of individuals who had never smoked, those who had smoked moderately and those who had smoked heavily. As a matter of fact, the individual who had smoked cigarettes heavily had definite metaplastic changes in the bronchial mucosa which could easily be termed precancerous lesions.

The objection that is also frequently raised to a causal relationship between cigarette smoking and cancer of the lung is that if such a relationship did exist, there should be a proportionate increase in deaths from laryngeal cancer, which is not the case. Although the death rate from laryngeal cancer is not increasing proportionately, as is the death rate from lung cancer, the incidence of laryngeal cancer *is* increasing in proportion to the incidence of lung cancer. The difference between laryngeal cancer and lung cancer is that laryngeal cancer is readily diagnosed because the patient, fortunately, consults a physician early; it is a relatively slow growing lesion; and it is as amenable to curative therapy as any cancer in the body. The reason why the death rate from laryngeal cancer is not increasing is that patients with laryngeal cancer are cured and a relatively small proportion die. One might similarly state that the incidence of appendicitis is

extremely low if one based it upon death statistics. Although fifty years ago the incidence of appendicitis based upon autopsy statistics was extremely high, at the present time the death rate is extremely low, for the simple reason that patients with appendicitis are operated upon and do not die.

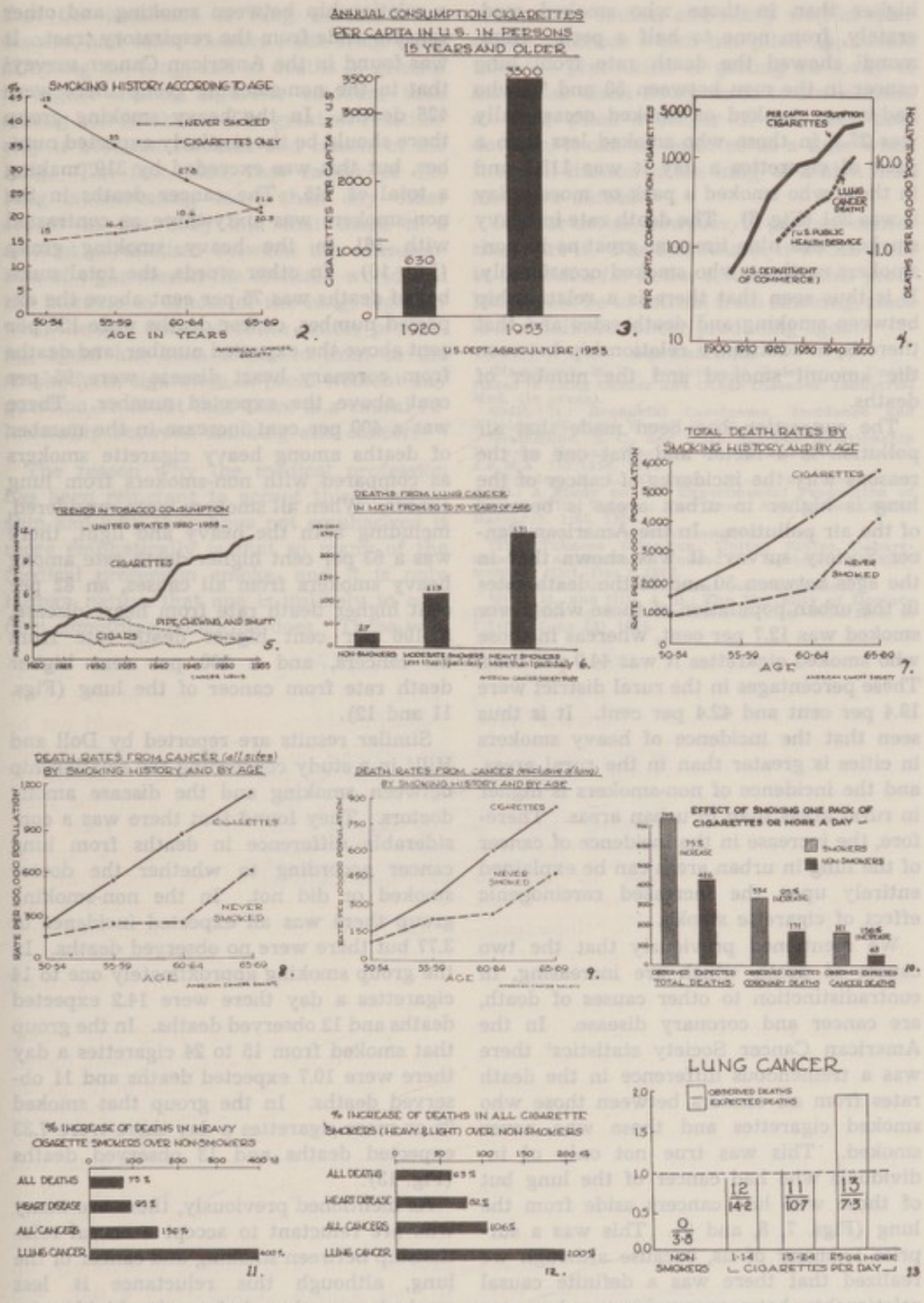
In addition to the fact that smoke from cigarettes contains a carcinogen which can produce cancer in animals, there is a distinct parallelism between the sale of cigarettes and the incidence of cancer of the lung. The annual consumption of cigarettes per capita in the United States in persons 15 years of age and older increased from 630 in 1920 to 3,500 in 1953, an increase of 456 per cent in thirty-three years. During the same time the amount of tobacco in cigars decreased about a half of its value in 1920[6] (Figs. 3-5).

There is tremendous impetus at the present time to the use of measures to cut down the deleterious effects of tobacco. There are few companies which do not advertise that its particular brand is less harmful, less poisonous, and less injurious than that of its competitors. In essence, the advertising, which is the most negativistic type that could be used, is simply, "Our product will kill you, but it won't kill you quite as fast as will our competitor's." The measures which are used to cut down the deleterious effects of tobacco are king-sized cigarettes, presumably the butt end of the cigarette acting as a filter, and the use of various filters. It has been shown that these measures, whether it is a long cigarette or the filters which are now used, exert little effect on the absorption of tars and nicotine. Tar is the factor which contains the carcinogen, nicotine the factor which is deleterious to the heart and blood vessels. Therefore, one can get little comfort in the use of filters or king-sized cigarettes except a possible psychic effect.

For many years those of us who have been convinced that there was a causal relationship between cigarette smoking and cancer of the lung have had considerable difficulty in convincing others, many of whom were physicians, because the statement has been frequently made, "The fact that over 97 per cent of patients with cancer of the lung are heavy smokers, as contrasted with approximately 85 per cent of the general population as a whole, means nothing. Until one knows what the incidence of cancer of the lung is among smokers as contrasted with non-smokers, one cannot say with any certainty that there is a causal relationship." It was difficult to understand why individuals, particularly physicians, were reluctant to accept the probable causal relationship, because the profession and industry have been perfectly willing to accept a causal relationship between the incidence of cancer of the lung and certain industrial hazards such as chromium and uranium.

It remained, however, for the American Cancer Society to prove finally, without any question or doubt, that there is a causal relationship between smoking and lung cancer. Approximately three years ago, a survey was undertaken by the American Cancer Society.[3] Twenty-two thousand volunteer workers in the states of New Jersey, Pennsylvania, New York, southeastern Michigan, Illinois, Wisconsin, Minnesota, Iowa, and California interviewed 204,547 white men between the ages of 50 and 70. Deducting those cases which could not be used, there were 190,134 which were usable. Of these, 98.8 per cent were successfully followed. At the end of a year, the same volunteer workers interviewed each man again. At the original interview and at subsequent interviews his smoking history was obtained, whether he smoked at all, what he smoked, if he smoked, and how much he smoked. At the end of two years, a total of 4,854, or 2.6 per cent of the entire group, had died.

It was found that the death rate among cigarette smokers was 65 per cent higher in the age group 50 to 54 than in the group in which there was no smoking. The death rate from all causes among those who smoked a pack or more cigarettes a day was 102 per cent higher than that in the group in which none had smoked from the ages 50 to 54. It was also found that the death rate in those who smoked moderately, up to half a pack of cigarettes a day, was higher than in those who never smoked, and that the rate in those who smoked from half a pack to a pack of cigarettes was



Figs. 2 to 13. Factual data upon relationship of lung cancer to cigarette smoking. Figs. 2, 6, 7, 8, 9, 10, 11, 12, after Hammond, E. C., and Horn, Daniel: The Relationship Between Human Smoking Habits and Death Rates: A Follow-Up Study of 187,776 Men, J.A.M.A., Aug. 7, 1954. Letters from Dr. Hammond and the Journal of the American Medical Association give permission to reproduce these figures.

higher than in those who smoked moderately, from none to half a pack. Hammond[7] showed the death rate from lung cancer in the men between 50 and 70 who had never smoked or smoked occasionally was 27.2, in those who smoked less than a pack of cigarettes a day it was 113.1 and in those who smoked a pack or more a day it was 251 (Fig. 6). The death rate in heavy smokers was nine times as great as in non-smokers or those who smoked occasionally. It is thus seen that there is a relationship between smoking and death rates and that there is, in addition, a relationship between the amount smoked and the number of deaths.

The suggestion has been made that air pollution is a factor and that one of the reasons why the incidence of cancer of the lung is higher in urban areas is because of the air pollution. In the American Cancer Society survey[3] it was shown that in the ages between 50 and 54 the death rates in the urban population of those who never smoked was 12.7 per cent, whereas in those who smoked cigarettes it was 44.9 per cent. These percentages in the rural district were 19.4 per cent and 42.4 per cent. It is thus seen that the incidence of heavy smokers in cities is greater than in the rural areas, and the incidence of non-smokers is higher in rural areas than in urban areas. Therefore, the increase in the incidence of cancer of the lung in urban areas can be explained entirely upon the increased carcinogenic effect of cigarette smoke.

We mentioned previously that the two causes of death which are increasing, in contradistinction to other causes of death, are cancer and coronary disease. In the American Cancer Society statistics[3] there was a tremendous difference in the death rates from all cancers between those who smoked cigarettes and those who never smoked. This was true not only of individuals who had cancer of the lung but of those who had cancers aside from the lung (Figs. 7, 8, and 9). This was a surprise to many of us, because although we realized that there was a definite causal relationship between smoking and cancer of the respiratory tract, including the mouth, we were not aware of the fact that there is a relationship between smoking and other cancers aside from the respiratory tract. It was found in the American Cancer survey[3] that in the non-smoking group there were 426 deaths. In the heavy smoking group there should be the similarly expected number, but this was exceeded by 319, making a total of 745. The cancer deaths in the non-smokers was sixty-three as contrasted with 161 in the heavy smoking group (Fig. 10). In other words, the total number of deaths was 75 per cent above the expected number, cancer deaths were 156 per cent above the expected number, and deaths from coronary heart disease were 95 per cent above the expected number. There was a 400 per cent increase in the number of deaths among heavy cigarette smokers as compared with non-smokers from lung cancer. When all smokers were considered, including both the heavy and light, there was a 63 per cent higher death rate among heavy smokers from all causes, an 82 per cent higher death rate from heart disease, a 106 per cent higher death rate from all cancers, and a 200 per cent higher death rate from cancer of the lung (Figs. 11 and 12).

Similar results are reported by Doll and Hill[8] in a study concerning the relationship between smoking and the disease among doctors. They found that there was a considerable difference in deaths from lung cancer according to whether the doctor smoked or did not. In the non-smoking group there was an expected incidence of 3.77 but there were no observed deaths. In the group smoking approximately one to 14 cigarettes a day there were 14.2 expected deaths and 12 observed deaths. In the group that smoked from 15 to 24 cigarettes a day there were 10.7 expected deaths and 11 observed deaths. In the group that smoked 25 or more cigarettes a day there were 7.33 expected deaths and 13 observed deaths (Fig. 13).

As mentioned previously, there are many who are reluctant to accept a causal relationship between smoking and cancer of the lung, although this reluctance is less marked now than before the highly significant investigation by the American Cancer Society.[3] The evidence now is irrefutable

that the incidence of lung cancer, heart disease, and other cancers aside from those affecting the lung, and of deaths in general is higher among cigarette smokers than among non-smokers. The greatest relationship is between smokers and cancer of the lung. The fact, also, that cancer of the lung is increasing more than any other cancer in the body and that there is a distinct parallelism between the consumption of cigarettes in the civilized world and the increase in cancer of the lung, together with the fact that a definite cancer producing agent has been found to exist in the smoke from cigarettes, is proof without any question or doubt that there is a causal relationship between smoking and cancer.

The reason why the medical profession has been reluctant to accept this relationship and why they have not emphasized it more emphatically is that so many of the medical profession smoke, and it is only human nature that one is unlikely to condem something which he does. Those who continue to smoke, and many will in spite of the evidence which has piled up, should take the precaution of getting an x-ray of his or her chest every three to six months, so that, when a cancer of the lung does develop, it can be detected at a time when it is still limited to the lung, at which time most are curable.

It is far better, however, to prevent cancer than cure it. For this reason, it would seem to me that a far better solution of this problem would be to refrain from smoking, since it is a definite cancer producing agent.

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The Influence of Smoking on the Respiratory Tract

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THE INFLUENCE OF SMOKING ON THE RESPIRATORY TRACT

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Since smoking is intimately associated with respiration and because the smoke from tobacco comes in intimate contact with the mucous membrane of the lips, tongue, mouth, larynx, trachea, and bronchi, there is a possibility that smoking may exert an effect on the respiratory tract. Although all physicians may not be in accord with the concept that cancer of the lung is caused by smoking, there is probably no physician who would deny the fact that smoking does exert a causative effect in the production of cancer of the mouth and lip. For a long time it has been known that cancers of the lips, tongue and mouth are seen much more frequently in individuals who use tobacco and apparently it does not make much difference whether the tobacco is smoked or whether it is chewed. In fact, not only is tobacco known to be the cause of cancer but also individuals who use snuff are likely to develop cancer in the areas of the mucous membrane with which the snuff comes in contact. Bissinger and Proehl[1] of the University of Minnesota Medical School in Minneapolis found that individuals who chewed tobacco for twenty years or more were more likely to develop cancer of the mouth than those who did not use tobacco. Twenty-six of forty men with mouth cancers were long-term tobacco or snuff chewers, and eighteen of twenty-three patients with leukoplakia were tobacco chewers. Fewer patients in the same age group with non-cancerous lesions chewed tobacco. Levin, Goldstein, and Gerhardt[2] found that cancer of the lips and tongue was more frequently associated with pipe and cigar-smoking than with cigarette-smoking. We have frequently observed that individuals who always held a cigar or cigarette in one portion of the mouth developed cancer at that particular site. We had one patient who developed a cancer on one side of the mouth where he held his cigarette. The cancer was successfully treated and although he was told to refrain from smoking, he declined to do so, but did change his habits in that he held his cigarette in the opposite corner of the mouth, following which he developed a cancer at that site. A frequent precursor of cancer of the mouth is leukoplakia and although in the past leukoplakia has been thought to be associated with syphilitic infection, it is now generally accepted that leukoplakia with very few exceptions is caused almost entirely by the irritating effect of tobacco. Leukoplakia is definitely a precancerous lesion and should be energetically treated if cancer is to be prevented. The adequate treatment of leukoplakia will prevent cancer which is, of course, the desideratum. Because of definite causative relationship between the use of tobacco and leukoplakia, it is imperative that the patient with leukoplakia refrain from the use of tobacco completely and for the rest of his life. In addition to this, careful mouth hygiene is essential. We have also found that the administration of stilbestrol, one to two milligrams three times a day, the use of brewer's yeast, and, as suggested by Nathanson,[3] the administration of liver extract approximately every ten days, usually cause a regression of the leukoplakia. If however, the leukoplakia resists this form of therapy, extirpation of the hyperkeratotic area is essential. X-ray is of no value and actually can do harm. Generally, excision can be accomplished under local analgesia and without a great deal of difficulty.

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A frequently raised objection to a causal relationship between cancer of the lung and cigarette-smoking is that if such a relationship existed why should not the mortality rate from cancer of the larynx increase proportionately as has that from cancer of the lung. Sadowsky, et al,[4] although convinced as a result of their study that there is an association between cigarette-smoking and cancer of the lung, raised the possible objection to this association and noted that an explanation is needed for no apparent associated increase in incidence of cancer of the larynx. Contrary to their presumption, cancer of the larynx *is* increasing as is cancer of the lung, but cancer of the larynx differs from cancer of the lung in that it is a slowly growing lesion, is readily diagnosed, and is extremely amenable to treatment. Because of this, although cancer of the larynx *is* increasing, relatively few people die from the disease and studies based upon autopsy and the vital statistics records would show a relatively low incidence of cancer of the larynx. Similarly, one might state that appendicitis is common but fortunately is diagnosed and treated and the patient does not come to autopsy. There is a definite causal relationship between smoking, particularly cigarette-smoking, and cancer of the lung. As in the mouth, precancerous lesions such as leukoplakia are caused by smoking but are correctable if the patient will refrain from smoking and conservative therapy is instituted.

Cancer of the lung, until approximately a quarter of a century ago, was an extremely rare disease. In 1912, Adler,[5] who was able to collect only three hundred and forty-seven cases from the entire world literature, stated: "There is complete consensus of opinion that primary malignant neoplasms of the lung are among the rarest form of disease". At the present time cancer of the lung is increasing more than any other cancer of the body and has become the most frequent form of cancer in men. In 1920, cancer of the lung represented 1.1 per cent of all cancers, in 1930, 2.2 per cent of all cancers, and in 1948, 8.3 per cent of all cancers. Based upon the number of people who are smoking today, we have predicted that in 1970, cancer of the lung will represent 18 per cent or approximately one out of every five persons with cancer. If one considers only men, in whom cancer of the lung is more prevalent, it is our belief that cancer of the lung in another quarter of a century will involve one out of every two or three men with cancer. Since it has been estimated by the American Cancer Society that one out of every five persons living today will develop cancer, it can be predicted that, unless measures are instituted to prevent the unprecedented increase, one out of every ten or fifteen men living in 1970 will have cancer of the lung.

It is frequently suggested that cancer of the lung is not increasing but that the increase is only apparent in that better diagnosis is responsible for more cases being detected. It is inconceivable that cancer of the lung can be missed by the pathologist in performing an autopsy. It must be concluded therefore that cancer of the lung actually is increasing and that the increase is not just relative and only apparent but is actual. Korteweg[6] stated: "Before long in the Netherlands and in many other countries lung cancer mortality will amount to 25 to 40 per cent of the total cancer mortality in males". He further added, "Unless all indications are misleading, tobacco-smoking is one of the most important causes of lung cancer. Through the preventive measure of giving up heavy smoking it must be possible to greatly reduce the significance of the disease." He believes that, "In view of the hundreds of thousands who by overconsumption of tobacco are preparing the ground for future disease, the omission to warn the public against their danger is no longer justified". He showed that in the Netherlands cancer of the lung between 1924 and 1951 in-

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creased 24-fold in men and 10-fold in women.

The increase in cancer of the lung has been suggested by some as due to the causes which are responsible for the increase in cancer generally. It is true that there are more people developing cancer today than twenty years ago. The increase in cancer generally is due to the increase in the age of the population and in all cancers there is a progressive increase in the incidence with advancing age, i.e., of all the persons ninety years of age a greater proportion will have cancer than those eighty years of age. This is because cancer is a disease primarily of advancing age. This is true of all cancers except cancer of the lung. Cancer of the lung increases markedly to reach a peak incidence at approximately fifty-five years of age at the present time, following which there is a definite decrease in the incidence. The age of the peak incidence in cancer of the lung is becoming less. Whereas, fifteen years ago, the highest incidence was between sixty-five and seventy years of age, it is now between fifty and fifty-five years of age. The decrease in the incidence of lung cancer after the peak incidence of fifty-five years is probably due to the fact that individuals who have been heavy smokers for many years, have subjected their heart and blood vessels to the deleterious effects of smoking and many develop heart disease, die of coronary thrombosis, and do not live long enough to develop lung cancer. A dubious advantage of cigarette-smoking, therefore, is that it might protect one from developing cancer by being responsible for an early death from heart disease.

As stated previously cancer of the lung is increasing more than any other cancer of the body in an unprecedented manner. There is, however, a distinct parallelism between the incidence of cancer of the lung and the sale of cigarettes in the Unted States and in other countries of the world where cancer of the lung is increasing. It has been suggested that this parallelism is only coincidental and well it might be if it were not for the fact that it has been shown by experimental methods that the smoke obtained from cigarettes is cancer-producing. One of the most significant investigations concerning this phase has been performed by Drs. Wynder and Graham[7] who showed that the smoke obtained from cigarettes which are smoked comparable to the manner in which individuals smoke them produces cancer in 44 per cent of the animals at the site of the application of the tar obtained from smoke. This investigation and other similar confirmatory ones demonstrate that in the smoke from cigarettes is an agent which will produce cancer which is identical with cancer seen in human beings in that it metastasizes (spreads) to other parts of the body and produces death of the animal. Since it has been proved that there is in smoke from cigarettes a cancer-producing agent and since there is a distinct parallelism between the sale of cigarettes and the unprecedented increase in the incidence of cancer of the lung, it is logical to assume that the increase in the incidence of cancer of the lung is due to the cancer-producing agent in cigarette smoke.

The most frequent cancer of the lung is epidermoid cancer, and it is this type of cancer which is increasing more than any other type. Epidermoid cancers are the ones which frequently occur as the result of cancer-producing agents. A relatively rare type of cancer of the lung is adenocarcinoma or glandular cancer which develops on the basis of an embryologic rest and is not caused by a cancer-producing agent. Epidermoid cancer of the lung is extremely rare in individuals who have not been heavy smokers. Koulumies[8] found that of 812 men with lung cancer over 99 per cent were smokers as contrasted to 82 per cent without lung cancer. The average age at which smoking began in the lung cancer group was 15.5 years as contrasted with 18.8 years for the control group. There was a large number of boys who started smoking at the age of

ten or younger in the lung cancer group. There was an average of eight to ten years longer smoking history in the lung cancer group than in the control series, that is, over forty years in the former and thirty-two years in the latter. Wynder and Graham[9] found that of 605 male patients with bronchogenic cancer, only 2.6 per cent did not smoke or smoked only minimally, whereas over 96 per cent had smoked heavily for over twenty years. In the control male hospital population without cancer these figtures were 14.6 per cent and 7.3 per cent respectively. Similarly, Levin, Goldstein, and Gerhardt[2] found that there were more smokers among lung cancer patients than non-cancer patients and that the patients with cancer usually smoked cigarettes. Doll and Hill[10] state, "About the age of forty-five, the risk of developing the disease (lung cancer) increases in simple proportion with the amount smoked and it is approximately fifty times as great among those who smoke twenty-five or more cigarettes a day as among non-smokers." In a more recent study Doll and Hill[11] state, "There is a real association between carcinoma of the lung and smoking. We believe that smoking is a factor and an important factor in the production of carcinoma of the lung. We also found that as the population becomes more highly urbanized the proportion of non-smokers and pipe-smokers decreases and the proportion of heavy smokers, especially cigarette-smokers, increases." In our own statistics only 1.5 per cent of men with epidermoid carcinoma were non-smokers, whereas 60 per cent of the individuals without cancer of the lung did not use tobacco. I have been so convinced of the causal relationship of smoking and epidermoid cancer of the lung that for the past five years in a patient in whom all clinical evidence suggests lung cancer but who is not a heavy cigarette-smoker, I have categorically diagnosed his condition as being either an adenocarcinoma or an inflammatory lesion. As yet, I have not been wrong. Of course, there will be the exception to prove the rule but the "average" up to the present time is 100 per cent.

Cancer of the lung is increasing more than any other cancer and since there is a parallelism between cancer of the lung and the sale of cigarettes and because it has been demonstrated that cigarette smoke contains a cancer-producing agent, it behooves all men who have been heavy smokers who are possible candidates for cancer of the lung to have chest x-rays taken at least every six months, but preferably every three months, so that when a cancer develops it can be detected at a time when it is still limited to the lung and can be cured by removal of the lung. Unfortunately, the symptoms of cancer of the lung occur insidiously and are likely to be disregarded. Cough is without a doubt the most prominent manifestation but because most, if not all, heavy cigarette-smokers have a cough often, it is considered of no significance. Spitting of blood always demands investigation as does the development of a wheeze in a man past forty who previously had not wheezed and who has been a heavy smoker.

The diagnosis of cancer of the lung is not difficult generally. X-ray is of real value because of the ease with which it can be performed but the diagnosis is only presumptive. A positive diagnosis is made either by finding tumor cells in the sputum or by visualizing the tumor on bronchoscopic examination and obtaining a piece of tissue for microscopic examination. In a series of over 1400 cases it was possible, however, to make a positive diagnosis preoperatively in approximately 80 per cent. In other words, in approximately 20 per cent of patients with cancer of the lung a positive diagnosis cannot be made until operation at which time either a portion of the tumor or a portion of the lung can be removed and an immediate examination made to determine the presence or absence of cancer.

The only curative treatment of cancer of the lung is removal of involved

lung and the lymph nodes which drain the lung. This is the standard type of treatment which is used in the surgical treatment of all malignant processes. Unfortunately, the five-year salvage rate of removal of the lung for cancer today is not as high as it should be but this is due to the fact that there has been procrastination on the part of the patient and failure to take cognizance of the symptoms which are suggestive of lung cancer. Far too frequently the patient disregards the cough or discomfort in the chest or the slight bleeding from the lung or wheezing or possibly is treated for a number of weeks or even months for virus pneumonia. Most cases of cancer of the lung are entirely curable if the condition is suspected and the diagnosis made relatively early. Unfortunately, however, there are occasional cases in which the tumor is so rapidly growing that it becomes widespread by the time the first symptom is manifest. It is for this reason that routine x-rays of the chest should be made in all men past forty who have been heavy smokers in order that when cancer of the lung develops a diagnosis can be made while the condition is still limited to the lung.

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EXHIBIT 6

REPORT OF STUDY GROUP ON SMOKING AND HEALTH [1]

The study group on smoking and health was organized in June 1956, at the suggestion of the American Cancer Society, the American Heart Association, the National Cancer Institute, and the National Heart Institute, to review the problem of the effects of tobacco smoking on health, and to recommend further needed research to the sponsoring organizations.

The study group has held six 2-day conferences, has examined the pertinent literature and more recent unpublished reports, and has consulted with scientists representing specialized areas of research concerned with the subject.

The study group, cognizant of the implications of its conclusions and recommendations, now submits the following joint report.

I. LUNG CANCER [2]

At least 16 independent studies carried on in 5 countries during the past 18 years have shown that there is a statistical association between smoking and the occurrence of lung cancer.[3] These retrospective studies have been reinforced by two investigations in which large male populations have been followed prospectively. Lung cancer occurs much more frequently (5 to 15 times) among cigarette smokers than among nonsmokers, and there is a direct relationship between the incidence of lung cancer and the amount smoked.

It is estimated that on a life-time basis, 1 of every 10 men who smoke over 2 packs a day will die of lung cancer. The comparable risk among nonsmokers is estimated at 1 out of 275.[4] Self-selection and sampling bias have been mentioned as possible sources of error. Examination of the evidence shows that any distortion resulting from these sources does not invalidate the conclusions.[5]

Epidemiologic studies also indicate that cigarette smoking cannot account for all cases of epidermoid cancer of the lung. There are other causative environmental factors, the most important of which are probably various atmospheric pollutants. As in other diseases, various other influences such as sex, nutrition, and heredity may modify its occurrence.[6]

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[1] Sponsored by American Cancer Society, American Heart Association, National Cancer Institute, and National Heart Institute.

[2] As used in this report, the term "lung cancer" refers to epidermoid and undifferentiated types of carcinoma of the lung, but not adenocarcinoma.

[3] Fourteen (10, 20, 38, 41, 48, 50, 53, 60, 61, 62, 67, 68, 73, 74) of the retrospective studies have been reviewed by Cutler (18). Stocks and Campbell (63) have studied the association between smoking and lung cancer in terms of place of residence. An additional recent retrospective study by Wynder (71) is concerned with the occurrence of lung cancer in women.

[4] Doll and Hill (21) in England and Hammond and Horn (31) in this country reported their first results in 1954. Since then Doll and Hill have published a followup report (22) and Hammond has reported additional data (30).

Although the statistical evidence has often been quoted as indicating an association between heavy smoking and lung cancer, there is no evidence of a threshold level below which the risk disappears. The best available estimates for both the United States and England indicate that the lung-cancer disk is statistically significant for half pack a day smokers and that there is a correlation of risk with amount smoked (22, 30, 63).

[5] By numerical illustration, Berkson (7) has indicated that, in epidemiological studies similar to the one conducted by Hammond and Horn, it is possible to obtain a spurious statistical association between smoking and a disease as a result of sampling bias, even though no real association exists. It has been pointed out by Cornfield (17), Korteweg (36), Levin (42), and Lilienfeld (44), that for the degree of association between cigarette smoking and lung cancer observed by Hammond and Horn to be a result of sampling bias, it would be necessary to assume an unreasonably large degree of such bias. Furthermore, a study of smoking habits of a probability sample of the United States population reported by Haenszel and associates (28) indicates that necessary degree of sampling bias was not present in the Hammond-Horn study. Berkson also indicated that the influence of sampling bias would disappear with the passage of time. Doll and Hill (22) have recently reported that over a 4-year period of observation, the gradient of lung cancer mortality in relation to the amount smoked has remained remarkably constant during each of the years the study group was followed.

The issue of self-selection is raised as an argument against interpreting the statistical association of cigarette smoking and lung cancer as a causal relationship. Self-selection assumes that there is a factor that both causes a person to smoke and also causes lung cancer; thus, individuals are selected for both smoking and lung cancer by a third mutually related factor. Such a hypothesis does not appear likely. Doll and Hill (22) and Levin (42) have indicated that such a hypothethesis would be inconsistent with the marked increase of lung cancer mortality in recent years. The likelihood of this hypothesis is diminished further by the biological and pathogenetic evidence discussed in this statement. However, information concerning the characteristics of smokers and nonsmokers would be valuable for further evaluation of this theoretical possibility.

[6] Epidemiological studies indicate that cigarette smoking cannot account for all cases of lung cancer; nonsmokers do develop lung cancer and variations in frequency of lung cancer in different population groups are not completely related to variations in frequency of

The two prospective studies further suggest that cessation of smoking by chronic smokers decreases the probability of such individuals developing lung cancer.

The epidemiologic evidence is supported by laboratory studies on animals. At least five independent investigators have produced malignant neoplasms by tobacco smoke condensates. Although the active material has not been identified chemically, some progress has been made in localizing the activity in purified fractions.[7] Although the demonstration of carcinogenic activity in animals does not constitute proof of carcinogenicity in the lungs of human beings, this is important contributory evidence that strengthens the concept of causal relationship.

Studies on pathogenesis of human lung cancer are also compatible with the causal relationship. Physiologic observations indicate that foreign material is trapped in the tracheobronchial tree, particularly where ciliary action is inhibited or the ciliated epithelium is destroyed.[8]

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cigarette smoking. The most significant observation on this point is the higher death rate from lung cancer in urban as compared with rural areas (27, 29). This excess urban death rate has been attributed by most investigators (34, 37, 63) to the probable influence of air pollution. As has been indicated by Hammond (29) and Haenszel and Shimkin (27), part of this urban excess may be attributed to the higher proportion of cigarette smokers among urban populations, but they have also pointed out that even after adjusting for differences in smoking habits, the urban rate is still higher than the rural rate. Estimates have been made of the relative contribution that cigarette smoking and the "urban factor" (probably air pollution) makes to the lung-cancer deaths. From studies in Liverpool, Stocks and Campbell (63) have estimated that 50 percent of the lung-cancer deaths result from smoking and about 35 percent from air pollution. Hammond (29) estimated that about 31 percent of lung-cancer deaths are due to air pollution. It is recognized that these estimates may not be very precise, but they do afford some idea of the relative importance of these two factors. Recently, Hueper (34) and Kotin (37) have defended their views that air pollution is the major etiological factor in lung cancer, with cigarette smoking playing a minor role, if any. Cigarette smokers have higher lung-cancer death rates than nonsmokers in urban areas, where presumably both of these groups have had similar exposure to general air pollution. A definite need exists for determining the chemical nature of these pollutants, and estimating more precisely the role of air pollution, particularly in relation to the effect of cigarette smoking.

Several studies indicate that occupational exposure to chromate ore (4), radioactive dust, and other agents (34), increase the risk of lung cancer. However, specific occupational exposures have been demonstrated to be responsible for only a small percent of lung-cancer deaths.

It is also possible that additional factors increase host susceptibility to these environmental factors. There is suggestive evidence that hormonal factors may have some influence (43). The possible existence of endogenous factors influencing susceptibility to exogenous agents should be further investigated since such information may have some bearing on control efforts.

[7] In addition to the 5 recent demonstrations of carcinogenic activity in tobacco smoke (33, 52, 54, 64, 75), Roffo (59) and Woglom (69) had previously reported the production of cancers from tobacco tar. Evidence for a coarcinogenic effect of tobacco smoke condensate has been obtained by Gellhorn (25).

Several workers have now reported the presence of 3, 4-benzpyrene in tobacco smoke (9, 11, 16, 40, 76). It has been demonstrated to be present in the neutral fraction of tobacco smoke condensate at concentrations around 2 micrograms per hundred cigarettes. According to Wynder (70) the concentation is so low that the biological activity observed in his most purified fractions cannot be due solely to 3, 4-benzpyrene. Other carcinogenic polycyclic hydrocarbons are being sought. In addition to 3, 4-benzpyrene, Bonnet and Neukomm found other polycyclic hydrocarbons, including 3, 4; 9, 10-dibenzpyrene at an estimated concentration of 1 microgram per hundred cigarettes.

Recently Lacassagne, Buu-Hoi, and others (39) produced sarcomas in mice by 3 subcutaneous injections totaling 1.8 milligrams of this compound per animal. The quantitative aspects of this problem obviously need investigation.

[8] In the normal human lung particulate matter upon inhalation may penetrate diffusely throughout and into the alveoli. This is evident from many observations, both clinical and experimental. O. H. Robertson (58) in an exhaustive investigation and review of this subject presents evidence to indicate that such factors as particle size and concentration, the depth of ventilation, as well as time of exposure are important determinants as to the amount of particulate matter that will enter the lung. It is estimated that the range of particulate size in cigarette tobacco smoke is between 0.3 to 1.0 microns. Particles of this size upon inhalation may readily be distributed diffusely throughout the lung.

Increased depth of ventilation, such as seen in inhalation while smoking, favors the entrance of greater amounts of particulate matter into the lungs. Particles that penetrate the upper air passages come in contact with the mucus-covered surface of the bronchi where they tend to adhere, and depending on the size and nature of the particles may remain in contact with bronchial mucosa for varying periods of time.

An irritant such as smoke may bring about alterations in the ciliary activity of the bronchial mucosa. It is this ciliary activity that is the main mechanism available for the removal of particulate matter from the lung (47). Slight temporary alteration in this ciliary activity will allow for the retention of particulate matter. Furthermore, any alteration in the quantity or quality of mucus in the tracheobronchial tree will also interfere with the removal of particulate matter. Particles may become concentrated in outdrifting mucus and remain in contact with bronchial mucosa for significant periods of time. Hilding (32) demonstrated the possible role of paralyzed cilia or deciliated areas in accumulating cigarette tar by exposing recently removed calf lungs to cigarette smoke.

Fluorescent substances present in cigarette smoke have been shown to enter the cells of the buccal mucosa.[9] Detailed histological studies of the tracheobronchial tree indicate that basal cell hyperplasia, atypical hyperplasia, squamous metaplasia, and areas of morphologic alteration with all characteristics of carcinoma in situ are encountered more frequently among cigarette smokers than among nonsmokers.[10] Thus, every morphologic stage of carcinogenesis, as it is understood at present, has been observed and related to the smoking habit.

The sum total of scientific evidence establishes beyond reasonable doubt that cigarette smoking is a causative factor in the rapidly increasing incidence of human epidermoid carcinoma of the lung.

The evidence of a cause-effect relationship is adequate for considering the initiation of public-health measures. Nevertheless, additional research is needed to clarify many details and to aid in the most effective development of a program of lung cancer control. The need for information in the following areas appears to be most important:

(*a*) The isolation, identification and possible elimination from tobacco smoke of chemicals that produce cancer in animals.[11]

(*b*) The role of atmospheric pollutants and additional environmental factors other than cigarette smoking in the causation of lung cancer in man.

(*c*) The effect of cessation of smoking upon the occurrence of lung cancer in man.[12]

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[9] After smoking, according to the degree of keratinization, some chronic cigarette smokers tested have been found by Mellors (49) to have fat-soluble fluorescent substances in scrapings of the buccal mucosa. Such substances are not observed, to any appreciable degree, 12 hours after smoking.

[10] It is well established that changes may be observed in epithelial surfaces that precede established forms of invasive carcinoma. These alterations consist of atypical hyperplasia, metaplasia, and carcinoma in situ. Although there may be disagreement in the terminology of these early morphologic changes, their presence and significance is generally accepted. It has been demonstrated by Black and Ackerman (8) that epidermoid carcinoma in situ is an important stage in the histogenesis of lung cancer. If smoking is related causally to the development of epidermoid carcinoma of the lung, then one should expect to find a greater incidence of these early morphologic changes in the bronchial tree in smokers as compared to nonsmokers. A study investigating this hypothesis was carried out by Auerbach and associates (3). A total of 28,638 slides was made from the tracheobronchial tree of 150 autopsied individuals in whom a cigarette-smoking history had been obtained. Such changes as basal cell hyperplasia, stratification, squamous metaplasia and carcinoma-in-situ were found both quantitatively and qualitatively to significantly greater degree in smokers than in nonsmokers. The findings were accentuated in autopsied cases of bronchogenic carcinoma, all of which patients had been smokers. Carcinoma in situ was found in 1 percent of the slides of those who never smoked regularly, whereas it was found in 6 percent of the slides in those individuals who smoked over one pack of cigarettes a day. The incidence of the various stages of these lesions as related to sex, age, and exact anatomic distribution awaits the investigation of larger numbers of cases. The alterations recorded by Auerbach were similar in almost all details to those found preceding and developing into epidermoid carcinoma of the uterine cervix. Chang (13) and Chang and Cowdry (14) studied whole mounts of bronchial epithelium obtained at autopsy from smokers and nonsmokers. They found metaplasia and thickening of the epithelium more common among the smokers than the nonsmokers. These results are in good agreement with those of Auerbach. In addition, they observed that the cilia were shorter and that Goblet cells were more numerous in smokers' lungs than in nonsmokers.

[11] It is possible that an innocuous cigarette could be developed without prior identification of the active agents, and efforts along this line should parallel the work on characterization of the active materials. However, if empiric reduction is achieved, it would still be urgent to define the chemical nature of the agents and their mode of action.

Reduction in tumor activity might be sought by the removal or inactivation of carcinogenic, cocarcinogenic (promoting) factors, or the precursors of either. Possibilities suggesting themselves include selection of tobacco strain, extraction of tobacco leaves, alteration of the burning temperature of the cigarette, and, possibly, selective removal of the active material from tobacco smoke. In this connection, it should be noted that filters presently in use do not appear to be selective in action; rather, they reduce tumor activity only to the extent that they remove whole smoke (70). In the cass of most commercial cigarette filters, efficiency is not high and accordingly the amount of smoke reaching the lungs is not reduced to any major degree.

An incidental but urgent requirement for the identification studies are improved assay procedures since the work is presently hampered by the necessity for relying on slow and relatively insensitive methods.

[12] The most desirable method of obtaining information concerning the effect of cessation of smoking would be by a human experiment. A description of one of several designs follows. A group of smokers, who would volunteer to be included in the study, would be randomly allocated to an experimental and a control group. The control group would continue to smoke, whereas the experimental group would be asked to give up smoking. These groups would be followed for a number of years to determine the risk of dying in general and the risk for specific causes of death. Even though all individuals in the experimental group had not ceased smoking, comparisons of mortality rates in the two groups would be made. Obviously, success of the experiment would depend upon the proportion of the experimental group who had stopped smoking; if it is 50 percent or more, meaningful results could be expected from the experimental design examined by the study group.

(*d*) Measurement of possible physiological, sociological, and psychological differences between smokers and nonsmokers and the relation of host differences to the occurrence of lung cancer.[13]

II. LONGEVITY AND CARDIOVASCULAR DISEASES

At least three satistical investigations shown an association of tobacco smoking with a decrease in longevity, probably referable to a higher risk for male smokers of dying from cardiovascular disease. The mortality among smokers in certain age groups is reported to be approximately double that of the nonsmokers.[14]

The study group is of the opinion that the significance of this observation requires further research before its meaning can be determined. Cardiovascular diseases account for well over half of all adult male deaths. Even a relatively small proportionate excess in the cardiovascular death rate could, therefore, contribute a larger number of deaths than a much larger excess in the lung cancer death rate.

In the few reports available, there is no convincing biological or clinical evidence to indicate that smoking per se has a causative role in cardiovascular disease. Many physicians have the clinical impression that patients with coronary artery disease should not smoke, on the basis that smoking may be detrimental to previously damaged coronary arteries. A few studies have provided some validating evidence for this hypothesis. However, further systematic investigation is essential.[15]

Consideration of sample sizes required for this experiment indicated that very large samples would be necessary to obtain results with regard to lung cancer because of the relatively low death rate from this cause. However, the estimated sample sizes for studying the effect of cessation of smoking on total mortality, particularly on cardiovascular disease, appeared to be within practical limits; at least, they were such that further exploration of the feasibility of such an experiment appears warranted. Prior to conducting a full-scale experiment, a pilot study should be undertaken. Such a study is important not only from a biological but also from a public health administrative viewpoint, in that it would be a means of evaluating the feasibility of certain control efforts.

It should be recognized, of course, that such an experiment would fail to show a difference between experimental and control groups if the damage to the organ system had been irreversibly established at the time smoking was stopped.

Further information on the effect of cessation can be obtained from the prospective studies of Hammond, Doll, and Hill, and the veterans study being conducted by the National Cancer Institute. It is recommended that the necessary data continue to be collected with regard to this aspect of these studies.

[13] A large prospective study on the effect of smoking on lung cancer and other causes of death among approximately 220,000 veterans of World War I is now in progress by the National Institutes of Health, under the direction of Dr. Harold F. Dorn. This study offers an excellent opportunity to explore, by additional questionnaires and interviews, some of the possible differences that may exist between smokers and nonsmokers. In addition, this study could also be expanded to include cardiovascular evaluation, perhaps even to the extent of physical examinations and laboratory determinations on subsamples of the smoker and nonsmoker groups.

The study group has been in contact with Dr. Dorn and has suggested that additional questionnaires aand clinical and laboratory determinations might be valuable. It is hoped that these suggestions will be developed into research actions with the help of the National Heart Institute and the Veterans' Administration.

There is no evidence at present to indicate that the occurrence of cancer of the lung among cigarette smokers is limited to an undefined subgroup of susceptibles. Nevertheless, certain characteristics may well be correlated with greater or lesser susceptibility to lung cancer or heart disease. Research on host factors that may be associated with or modify certain disease states is of great importance. This is particularly true of chronic diseases in which the etiologic situations are often multiple and in which the host response may play a paramount role.

[14] Pearl (57), Hammond and Horn (31), and Doll and Hill (22) have all shown an increase in mortality associated with smoking. Pearl found the mortality rate for all causes among cigarette smokers to be just twice that of nonsmokers among men 60 to 65 years of age. In the same age group, Hammond and Horn reported a ratio of 1.9. Doll and Hill found the mortality rate to increase progressively with the amount smoked, so that heavy smokers for all age groups over 35 had a mortality rate 42 percent higher than nonsmokers.

For coronary artery diseases, Gertler and White (26) studied smoking habits, results of which showed a significantly higher number of smokers among the heart patients than among controls. English, Willius, and Berkson (23) in 1940 found a greater incidence of coronary disease in male smokers than among nonsmokers particularly at ages under 50. Hammond and Horn (31) found the death rates for men smokers of a pack or more of cigarettes a day dying of coronary artery diseases to be more than twice as high as the death rate of men who had never smoked; this was true for age groups from 50 to 65. The results of Doll and Hill on death from coronary thrombosis agree broadly with those of Hammond and Horn, but the increase is less marked.

[15] Evidence pointing to the effect of nicotine on the coronary circulation was obtained by Bargeron and associates (5), who found that cigarette smoking increases the coronary blood flow in individuals without heart disease. That nicotine may cause a decrease in coronary blood flow if the coronary vessels are atherosclerotic has been suggested by ballistocardiographic studies. Davis and associates (19) have confirmed the reports of others showing definite ballistocardiographic abnormalities in patients with coronary heart disease; these alterations were not present in individuals with normal hearts.

The study group strongly urges a research program of wide scope that would clarify the relationship and association between smoking and cardiovascular diseases. The following areas are considered of particular importance:

(*a*) Epidemiologic studies with appropriate consideration of the roles of other factors such as diet, physical activity, and blood lipides.[16]

(*b*) The chronic effects of tobacco and nicotine in animals, with and without experimental atherosclerosis.

(*c*) Further physiologic studies on man, particularly on the effect of smoking upon coronary artery blood flow in normal individuals and in individuals with coronary insufficiency.

III. OTHER DISEASES

The study group considered reports of association of smoking with a number of other disease entities, including carcinoma of the larynx, oral cavity, esophagus, bladder, and stomach; peptic ulcer; bronchitis and tuberculosis; and thromboangitis obliterans.[17]

The evidence relating smoking directly to thromboangitis obliterans is based upon repeated, well-confirmed demonstrations that the disease is materially ameliorated by cessation of smoking, and strikingly aggravated by continued smoking.[18]

For the other conditions mentioned, the relationships have been studied only to a degree to indicate statistical association. Further research on the individual conditions is necessary before valid conclusions can be drawn.

IV. CONCLUSIONS

The study group concludes that the smoking of tobacco, particularly in the form of cigarettes, is an important health hazard. The implications of this statement are clear in terms of the need for thorough consideration of appropriate control measures on the part of the official and voluntary agencies concerned with the health of the people. The lack of specific recommendations in this regard reflects no lack of interest. Rather, it reflects the desire of the study group to limit its recommendations to the area of research needs according to the instructions it received.

The study group recommends that further research on smoking and health be vigorously pursued. The recommendations made in section I are for research into means of coping with the lung cancer hazard which has been established for cigarette smoking. The recommendations of sections II and III are directed

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Experimentally, Travell (65) at Cornell University has demonstrated that nicotine causes a diminution in coronary blood flow in perfused hearts of rabbits previously made atherosclerotic through dietary measures.

It is felt that one of the most pressing problems consists in direct investigation of the coronary circulation of patients with arteriosclerotic heart disease during smoking. The techniques for this investigation are being developed, but the present methods are not yet applicable.

[16] In contrast to the situation in lung cancer, the number of studies concerned with the relationship of smoking to cardiovascular diseases have been relatively few. Most epidemiological investigations of heart disease have been concerned with evaluating the etiological role of diet, physical activity, and blood lipides. A definite need exists for both retrospective and prospective studies on smoking and heart disease. Several independent studies should be conducted in various parts of the country. Because of the possible influence of diet, physical exercise and blood lipides, they should be taken into account to determine possible interrelationships of these factors to smoking. In addition, it is possible that studies of heart disease, already in progress, could be expanded to obtain further information concerning the effects of smoking. The study group considered the veterans' study, being conducted by the National Institutes of Health, as offering an excellent opportunity for expansion to include the suggestions made above.

[17] A few references to studies relating smoking to some of these diseases should be noted. Lilienfeld, Levin, and Moore (45) demonstrated a statistical association between smoking and cancer of the urinary bladder and felt it was highly suggestive of a cause-and-effect relationship. Friedell and Rosenthal (24) and Moore and associates (51) have discussed the evidence for carcinogenic activity of unburned tobacco in producing oral cancers. Wynder (72) has reported some evidence that alcohol consumption increases a smoker's susceptibility to laryngeal cancer. The evidence regarding the etiological role of smoking in gastrointestinal disease is highly controversial (6, 66).

Case and Lea (12) found an association between chronic bronchitis and lung cancer in World War I pensioners. Unfortunately, smoking histories were not available. However, Joules (35), Palmer (56), Oswald and associates (55), and Abbott and associates (1) found chronic bronchitis to be closely associated with heavy smoking. (See (15).) Lowe (46) found heavy smokers to be significantly more common among tuberculosis patients than among control patients.

[18] For references to the voluminous literature on this condition, see Allen, Barker and Hines (2).

primarily at establishing whether smoking is one of the contributory factors in other diseases, particularly coronary artery disease. Many of these answers can best be obtained by a cooperative research effort.

The study group on Smoking and Health approves dissemination of this report as desired by the sponsoring agencies and hereby terminates its activities.

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In the United States
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EXHIBIT 7

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Tobacco Smoking Patterns In the United States

William Haenszel

Michael B. Shimkin, M.D.

Herman P. Miller, Ph.D.

Including an addendum

Tobacco Consumption In the United States, 1880 to 1955

Benno K. Milmore, M.D., M.P.H., and Arthur G. Conover, M.A.

PUBLIC HEALTH MONOGRAPH NO. 45

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Introduction

Tobacco, the comforting weed of the American Indian that botanical nosologists renamed *Nicotiana tabacum*, was introduced into Western European culture during the midportion of the 16th century. Fernandes took it to Spain in 1558, Nicot to France a year later, and Lane and Drake to England in 1585.

The use of tobacco spread rapidly and became of economic and social importance throughout Europe and the Orient. By 1615, tobacco was a staple crop of Jamestown and the principal currency of the colony. It was first recommended for its supposed medicinal virtues but soon came to be recognized as a luxury. As such, it was soon regarded as a promising source of tax revenue. Corti (*1*) has described the diffusion of the use of tobacco in our society down to the end of the 19th century.

The Statistical Abstract of the United States (*2*) recounts that farm income from raising tobacco reached $1,094,000,000 in 1953. The net value added to national income by industrial processing of tobacco amounted to another $580,000,000. More than 100,000 persons were employed in tobacco manufacturing in addition to those engaged in tobacco farming. Almost 2 percent of the average current family expenditures was devoted to the purchase of tobacco. In 1953, the excise taxes on tobacco collected by the Internal Revenue Service amounted to $1,655,000,000. References to some aspect of tobacco are found on 62 pages of the Statistical Abstract, covering such topics as acreage, advertising, income, manufacturing, prices, taxes, and trade; in fact, almost everything except information on people who use these products. Recently an official of the United States Department of Agriculture stated, "We have no reliable figures on the number of actual cigarette smokers in the United States" (*3*). He did go on to say that "most trade sources place the number in the neighborhood of 60,000,000."

The relative lack of information on consumers may be somewhat surprising when one recalls the questions frequently raised concerning the effect of tobacco smoking on health. Adverse opinions on the effect of tobacco appeared as early as the 17th century when Simonis Paulli published De Abusu Tabaci. This book must have achieved some currency in its time since the Latin text was translated into English (with additions) by Dr. George James and reissued in 1746 (*4*). The systematic collection of observations bearing on the possible ill effects of tobacco, which meet some of the accepted standards for evidence, did not appear until the 20th century. In 1928 Lombard and Doering reported heavy smoking to be more common among cancer patients than among persons in a control group (*5*). In 1938, Pearl reported that heavy smokers had a shorter life expectancy than did nonsmokers (*6*).

More recently, several retrospective studies of lung cancer patients and matched controls have reported an association of cigarette smoking with lung cancer (*7*). The preliminary results of forward studies on British physicians (*8*) and on persons interviewed by American Cancer Society workers (*9*) also agree in finding excess lung cancer risks among cigarette smokers. One other study, conducted by the National Cancer Institute among World War I veterans holding Government life insurance policies, is under way (*10*). In addition, the findings of the American Cancer Society study indicate an excess mortality from all causes, particularly coronary disease, among smokers. A recent book has summarized some of the known biological effects of tobacco, with emphasis on the clinical and experimental aspects (*11*).

A survey of smoking habits for a cross section of the population of the United States would provide data which might help answer some of the questions which have been raised concerning

the effects of tobacco use and give further insight into the meaning of the reported association between smoking and lung cancer.

Lung cancer is widely distributed in the population of the Western World. It is not an explosive nor geographically limited disease, and further tests are desirable to protect against possible erroneous inferences from the observed association. Possible criticisms which might be advanced against the results obtained to date from retrospective and forward studies include the not completely representative selection of groups studied prospectively and retrospectively and the further suggestion that the relationship might turn out to be an indirect one, reflecting merely a further association of smoking with other unidentified causative factors.

These points deserve careful attention in the absence of convincing experimental reproduction of lung cancer in suitable laboratory animals exposed to tobacco smoke (*12*), or of confirmation of an etiological relationship through the successful application of control measures. It might also be remembered that the forward studies have not yet been conducted long enough (from an actuarial standpoint) to nullify the effect on the result of any selection biases that may have been present originally in the cohorts followed. There is some difference of opinion as to the importance and magnitude of the biases which may be present in the early results of the forward studies (*13, 14*).

Comprehensive information on the distribution of smoking habits with respect to age, sex, residence, race, and other characteristics would serve at least two purposes. First, it would provide a check as to whether the number of lung cancer deaths is consistent with estimates of the excess risk among smokers and among the population of smokers affected. To illustrate in another context what is meant, the number of persons exposed to excessive lung cancer risk in the chromate industry could account for only a small fraction of the observed lung cancer deaths, and it is clear that other etiological candidates must be sought. If this test of consistency is met, a reconciliation of the excess risk among smokers with the observed pattern of lung cancer morbidity and mortality in the general population by age, sex, residence, and other variables may be attempted to see how much of the variation would be accounted for, when smoking habits were controlled statistically in the comparison. This approach would supplement with broader coverage the findings derived from the smaller special groups studied.

The latter goal might be achieved through collecting smoking histories for a sample of all lung cancer deaths registered in a study area, or, more simply, by using a well-known indirect technique of adjustment, which takes account of the information on excess risks among various smoking classes reported by previous studies to yield an "expected" distribution of lung cancer deaths to compare with the distribution actually observed in the general population.

None of the surveys on smoking referred to in the literature are completely suitable for the purposes of systematic epidemiological inquiry. Many surveys have been undertaken for commercial organizations, but the results usually have been guarded as trade secrets and have not found their way into technical or scientific journals. Berkson alludes in a recent article to an estimate on the proportion of smokers secured from trade sources (*13*). Details on the definitions employed and the phrasing of questions put to respondents are lacking, so there are difficulties in interpreting such unpublished results.

In this country, data on smoking collected primarily for market research have been published by *Fortune* (*15*) and by the Gallup poll. The *Milwaukee Journal* has for many years included questions on smoking in an annual survey of the Milwaukee area (*16*). The *Hulton Press* provided a similar service in Great Britain (*17*). Clemmesen has reported a Danish market survey of smoking habits made available to him by a tobacco company (*18*). Doll and Hill referred to some estimates of smokers and nonsmokers from a British social survey which do not appear in print elsewhere (*19*). Mills and Porter (*20*), and Kreyberg (*21*), whose primary interests were lung cancer, collected data on smoking from the population outside hospitals, in order to protect against the possible inadequacies, for control purposes, of data limited to hospital patients.

Mills and Porter restricted themselves to one city, Columbus, Ohio, and had no information on regional or urban and rural differences.

In Norway, Kreyberg surveyed several distinct occupational and geographic clusters, which he thought would be useful "to provide background information on factors which must be taken into consideration when designing a retrospective control study on smoking habits and the incidence of lung cancer" but which cannot supply systematic information on gradients among population groups. Kirchoff and Rigdon recently published a report on smoking histories collected in Texas, the principal source of information being hospital admissions (*22*). The most recent article, "Tobacco Consumption in Denmark," in the *Danish Medical Bulletin* for December 1955 (*23*), reported a survey undertaken as a part of the investigations into the health status of the Danish population carried out in the years 1951–54.

For use in concert with morbidity and mortality data for the general population, the smoking histories must be collected from a representative cross section of the general population, a condition best met by a survey using probability sampling methods. This requirement is more stringent than that usually placed on prospective studies, in which representativeness of coverage may be sacrificed to insure adequate followup without destroying the value of these studies. Similar study situations have been recognized in epidemiological work on other cancer sites. Stocks remarked, in the course of examining mortality data on stomach cancer, "Statistics of the prevalence of different cooking habits in different parts of England and Wales are lacking, and it would be worth while trying to ascertain by a sampling survey what kinds of fats and oils are used for cooking. . . . If the statistical averages so arrived at for any kind of fat or oil corresponded at all closely in distribution with that for stomach cancer among women, there would be good reason to pursue the question further by intensive research" (*24*). These views are easy to express. To implement them is a more formidable task.

Fortunately, the National Cancer Institute was able to contract with the Bureau of the Census for the collection of smoking histories as a supplement to the Current Population Survey (CPS) for February 1955. The arrangements made available all the other demographic details collected as regular features of the survey. This is the largest sample survey regularly undertaken in the United States population. It yields both regional and urban and rural estimates, and the technical standards for sampling procedures are not surpassed by any other statistical organization. This survey provided estimates of smoking characteristics with measurable sampling errors. In addition to sampling variability, however, the data may reflect varying degrees of response errors which arise from the degree of understanding, interest, motivation, knowledge, and skill of the interviewer and respondent. One desirable aspect of this study was that the questions on smoking habits could be phrased in a manner almost identical with those employed in the American Cancer Society and the Veterans Administration-National Cancer Institute forward studies. Besides getting information on current smoking status and consumption rates, inquiries were also made on age at which smoking started and maximum consumption rate when this was in excess of the current rate, in an effort to explore some of the dynamics of tobacco smoking. Results requiring memory of age at which smoking started, past purchases, or usage, may be affected by failure to recall or lack of knowledge of the respondent. A more complete discussion of response errors in this survey is given on page 9.

Because of the comprehensive geographic and subject matter scope of this survey on tobacco use, hitherto unduplicated, every effort has been made to report the findings in detail. These data may prove useful to many other persons interested in the social, economic, and marketing aspects of tobacco use, as well as to investigators interested in lung cancer. This report is devoted solely to presentation of the survey findings. The application of the data to testing proposed models for lung cancer etiology in the manner suggested earlier in this introduction has been reserved for another paper (*25*).

Definitions and Explanations

Urban and Rural Residence

The definition of urban and rural areas used in this survey was the same as that used in the 1950 census. According to this definition, the urban population comprises all persons living in (*a*) places of 2,500 inhabitants or more incorporated as cities, boroughs, and villages; (*b*) incorporated towns of 2,500 inhabitants or more except in New England, New York, and Wisconsin, where "towns" are simply minor civil divisions of counties; (*c*) the densely settled urban fringe, including both incorporated and unincorporated areas, around cities of 50,000 or more; and (*d*) unincorporated places of 2,500 inhabitants or more outside of any urban fringe. The remaining population is classified as rural.

Region

The States included in each region of the United States are shown in Figure 1.

Size of Place

The urban population is classified as living in urbanized areas or in urban places outside urbanized areas. According to the definition used in this survey and in the 1950 census, the population in urbanized areas comprises all persons living in (*a*) cities of 50,000 inhabitants or more in 1940 or according to a special census taken between 1940 and 1950, and (*b*) the densely settled urban fringe, including both incorporated and unincorporated areas, surrounding these cities. Residents of urbanized areas were classified according to the size of the entire area rather than by the size of the place in which they lived. The remaining urban population is classified as living in the smaller urban places not in the urbanized areas.

Farm and Nonfarm Residence

The rural population is subdivided into the rural farm population, which comprises all rural residents currently living on farms, and the rural nonfarm population, which comprises the remaining rural population. The method of determining farm and nonfarm residence in this survey is the same as that used in the 1950 census. Persons on "farms" who were paying cash rent for their house and yard only were classified as nonfarm; furthermore, persons in institutions, summer camps, "motels," and tourist camps were classified as nonfarm.

Race

The term "race" refers to the division of the population into two groups, white and nonwhite. The nonwhite group includes Negroes, Indians, Japanese, Chinese, and other nonwhite races.

Age

The age classification is based on the age of the person at his last birthday.

Employment

Employed persons comprise those who, during the survey week, were either (*a*) "at work"—those who did any civilian work for pay or profit or who worked without pay for 15 hours or more on a family farm or business, or (*b*) "with a job but not at work"—those who did not work and were not looking for work but had a civilian job or business from which they were temporarily absent because of vacation, illness, industrial dispute, bad weather, or layoff with definite instructions to return to work within 30 days of layoff. Also included are persons who had new jobs to which they were scheduled to report within 30 days.

Unemployed persons include those who did not work at all during the survey week and who were looking for work. Also included as unemployed are persons who would have been looking for work except that (*a*) they were temporarily ill, (*b*) they expected to return to a job from which they had been laid off for an indefinite period, or (*c*) they believed no work was available in their line of work or in the community.

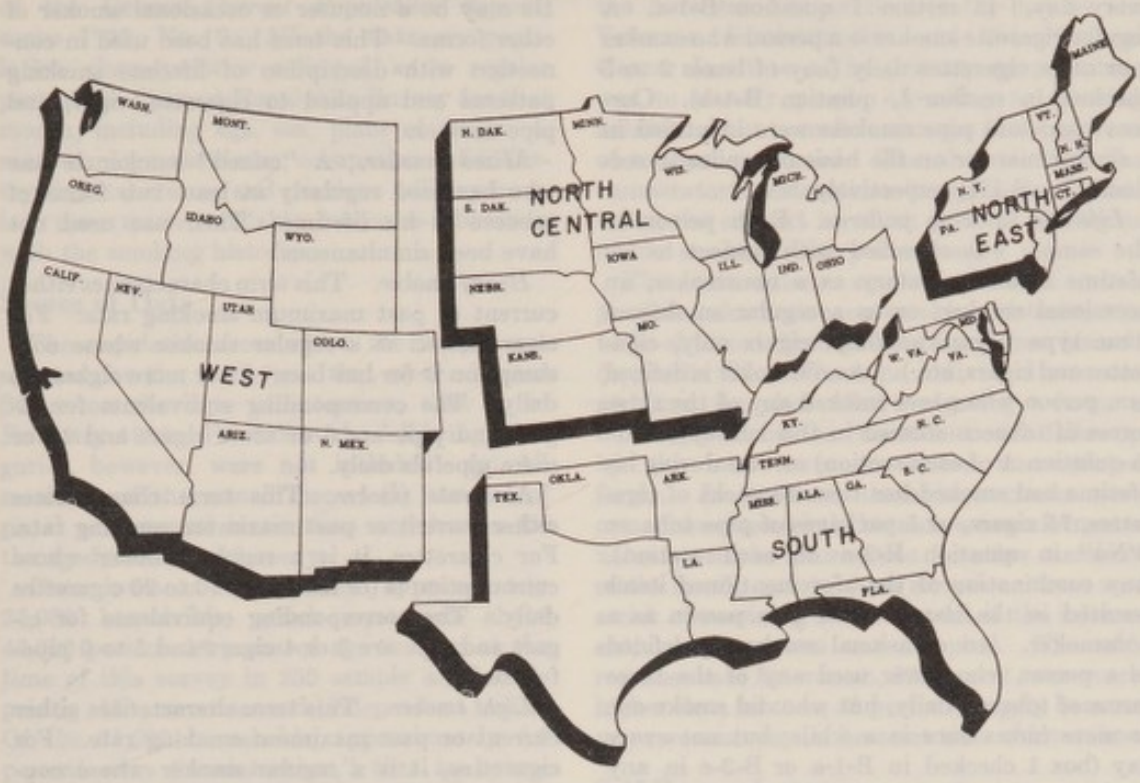
Labor Force

Persons are classified as in the labor force if they were employed as civilians or were unemployed during the survey week.

Not in Labor Force

All civilians 14 years of age and over who are not classified as employed or unemployed are

Figure 1. Regions of the United States.



defined as "not in the labor force." These persons are further classified as "keeping house," "in school," "unable to work" because of disability, and "other," the last group including for the most part retired persons, those too old to work, seasonal workers for whom the survey week fell in an "off" season, and the voluntarily idle. Persons doing only incidental unpaid family work (less than 15 hours) are also classified as not in the labor force.

Occupation, Industry, and Class of Worker

The data on occupation, industry, and class of worker of employed persons refer to the job held during the survey week. Persons employed at two or more jobs were reported in the job at which they worked the greatest number of hours during the week. Persons who were unemployed during the survey week are classified according to their last civilian job. The occupation and industry groupings used here are largely the same as the major groups in the classification systems used in the 1950 Census of Population.

Military service. Males are classified as having previous military service if they had ever been in any of the Armed Forces of the United States during peacetime or wartime.

Tobacco Smoking Concepts

A tobacco smoking questionnaire (appendix A) was completed for each person in the sample 18 years old and over. The information was then coded and tabulated according to current smoking patterns and lifetime smoking patterns. These concepts and others based on the tobacco smoking questionnaire are defined below:

Current smoking pattern. Each person in the sample was classified as a nonsmoker, an occasional smoker, or a regular smoker of each form of tobacco (cigarettes, cigars, or pipes) at the present time. For example, a nonsmoker of cigarettes is a person who never smoked cigarettes ("No" in section I, question A) or who had smoked less than 10 packs of cigarettes in his lifetime ("Yes" in section I, question A, and "No" in question B-2-a). An occasional cigarette smoker is a person who

checked box 1, "smoke once in a while, not every day," in section I question B-1-a. A regular cigarette smoker is a person who smokes 1 or more cigarettes daily (any of boxes 2 to 5 checked in section I, question B-1-a). Current cigar and pipe smokers were identified in a similar manner on the basis of replies to sections II and III, respectively.

Lifetime smoking patterns. Each person in the sample was classified with respect to his lifetime smoking history as a nonsmoker, an occasional smoker, or as a regular smoker of some type (cigarettes only, cigars only, cigarettes and cigars, etc.). A nonsmoker is defined as a person who never smoked any of the three forms of tobacco studied in this survey ("No" in question A of each section) or who during his lifetime had smoked less than 10 packs of cigarettes, 75 cigars, or 5 packages of pipe tobacco ("No" in question B-2-a of each section). Any combination of the aforementioned items resulted in the classification of a person as a nonsmoker. An occasional smoker is defined as a person who never used any of the three forms of tobacco daily, but who did smoke one or more forms once in a while, but not every day (box 1 checked in B-1-a or B-2-e in any section combined with "No" in question A or "No" in question B-2-a in the other sections). A regular smoker is defined as a person who smoked one or more forms of tobacco every day (any of boxes 2 to 5 checked in question B-1-a or B-2-e in any section).

Discontinued smoker. A discontinued smoker is defined as a person who once was a regular smoker as defined above, but who was a nonsmoker or an occasional smoker at the time of the survey.

Pure smoker. A "pure" smoker is one who has used regularly only one form of tobacco. He may be a nonuser or occasional smoker of other forms. This term has been used in connection with description of lifetime smoking patterns and applied to cigarette, cigar, and pipe smokers.

Mixed smoker. A "mixed" smoker is one who has used regularly at least two forms of tobacco in his lifetime. Their use need not have been simultaneous.

Heavy smoker. This term characterizes either current or past maximum smoking rate. For cigarettes, it is a regular smoker whose consumption is (or has been) 21 or more cigarettes daily. The corresponding equivalents for cigars and pipe are 5 or more cigars and 10 or more pipefuls daily.

Moderate smoker. This term characterizes either current or past maximum smoking rate. For cigarettes, it is a regular smoker whose consumption is (or has been) 10 to 20 cigarettes daily. The corresponding equivalents for cigars and pipe are 3 or 4 cigars and 5 to 9 pipefuls daily.

Light smoker. This term characterizes either current or past maximum smoking rate. For cigarettes, it is a regular smoker whose consumption is (or has been) less than 10 cigarettes daily. The corresponding equivalents for cigars and pipe are 1 or 2 cigars and less than 5 pipefuls daily.

Years stopped smoking. Discontinued smokers are classified by the number of years they have stopped smoking. When this variable relates to a particular type of tobacco, it is based directly on the reply to B-2-d, all sections. When it relates to lifetime history, however, it represents the number of years since the person last smoked any form of tobacco.

Study Procedures and Forms and Reliability of Estimates

This survey of tobacco smoking was sponsored by the National Cancer Institute of the United States Public Health Service and was conducted by the trained field staff of the Bureau of the Census as a supplement to its February 1955 Current Population Survey. This was the first nationwide survey of tobacco smoking ever conducted in the United States by the use of probability sampling procedures. A rather complete description of the Current

Population Survey sample appears in Bureau of the Census *Current Population Reports*, series P-23, No. 2. All the data on population characteristics collected as a regular part of the Current Population Survey for that month, including age, sex, place of residence, race, employment status, occupation and industry, marital status, and previous military service, were made available for cross-tabulation with the smoking histories.

Source of Data

The population from which the sample was selected for the study was that of the United States on the date of interview, that is, in February 1955. Persons in the following categories, however, were not included: (*a*) All members of the armed forces; (*b*) inmates of penal and mental institutions and homes for the aged, infirm, and needy.

Data were collected from approximately 21,000 households, which contained about 45,000 persons 18 years of age or over at the time of this survey in 230 sample areas comprising 453 counties and independent cities. Of the 21,000 households, approximately 4.4 percent were "noninterview," that is, households for which no information was recorded because no interview could be obtained. In order to account for these schedules, the weights assigned to other schedules for households of similar characteristics residing in the sample areas were increased. In addition, tobacco smoking information was not reported for about 4.5 percent of the persons. Substitutions were not made for these schedules.

The estimating procedure used in the survey involved, as a final step, the inflation of the weighted sample results to agree with independent estimates of the civilian noninstitutional population of the United States by age, sex, and race. These independent estimates were based on statistics from the 1950 census of population and on statistics of births, deaths, and net immigration.

Questionnaire and Interview

A separate form was used by the interviewer for recording smoking histories for all household members 18 years and over (appendix A). The phrasing of the questions was very similar to that employed in recent studies conducted by the American Cancer Society (*9*) and by the Veterans Administration and the National Cancer Institute. The primary purpose here was to classify people according to smoking habits and not to make estimates of the amount of tobacco consumed. The abstract from the enumerator's instructions in appendix B illustrates the context in which some of the terms on the smoking history were used.

The instructions emphasized that when possible information on smoking histories was to be obtained directly from the person himself (appendix B). When the individual was not at home, the plan called for leaving a form to be completed by the respondent with followup by the interviewer to secure uncompleted forms. In practice, these instructions were not adhered to strictly because some informants volunteered the information for other members of the family and in these situations there was a risk of receiving no data at all if the informant's response was not accepted. The self-response rate for women, 83 percent, was much higher than for men, 58 percent, largely because more women were at home at time of interview.

Self-Respondents

Under these conditions, scrutiny of the data supplied by persons responding for themselves and for others seems advisable. Table 1 shows that the proportion of self-respondents increased for men in the older age groups, but was rather stable among all women over 25 years.

Appendix table 12 compares the distribution of current cigarette smoking patterns obtained

Table 1. Percentage of smoking histories obtained from persons responding for themselves, by age and sex

Age (years)	Males	Females
All ages	57.9	83.0
18–24	43.3	71.4
25–34	53.5	86.8
35–44	56.4	85.8
45–54	58.0	85.4
55–64	64.0	84.8
65 and over	73.6	79.2

Table 2. Percentage of persons 18 years of age and over in selected cigarette smoking history categories for total sample and for self-respondents, by sex

Sex	Never smoked cigarettes	Now non-smoker, previously smoked cigarettes regularly	Current, regular cigarette smokers	
			Total	21 or more cigarettes daily
Male:				
Total sample	32.0	9.8	52.6	13.6
Self-respondents	28.7	11.1	54.5	14.9
Female:				
Total sample	67.6	3.1	24.5	2.4
Self-respondents	65.9	3.5	25.5	2.4

NOTE: Percentages for self-respondents adjusted to age distribution of population surveyed. Smoking histories not reported in total sample distributed proportionately to other smoking categories.

from self-respondents with that reported on behalf of others. In the latter group, the proportion of persons reported as having never smoked cigarettes at any time was higher and the proportion of previous regular smokers was lower than among self-respondents. The differences are partially compensatory and obviously suggest that persons replying for others have more complete knowledge concerning current smoking habits than of past practices. The reported proportion of regular smokers, including those using more than one pack of cigarettes daily, was somewhat greater among self-respondents. These relationships held true generally within individual age groups, particularly for males.

Although the results according to availability for personal interview may reflect some selection effect, it would not be unreasonable to believe that the true distribution of smoking histories for persons not replying for themselves should approximate that for self-respondents in each sex and age group. For this situation, an estimate of the downward effect resulting from reliance on the total sample rather than on self-respondents alone may be gauged from table 2. The shift in numerical values of the percentages due to an adjustment of this nature would not be great and would not affect inferences drawn from comparisons based on the total sample as reported in tables of this monograph, particularly age comparisons. The results as stated in the tables probably minimize the true sex differentials in smoking histories. Data on age smoking started and maximum rates of tobacco use are subject to greater bias than those on current tobacco use.

Males in the group 18–24 years of age had the lowest proportion of self-respondents, and errors attributable to this response bias should be greatest here. This fact may be responsible for much of the lag noted among males now 18–24 years of age in the cumulative percentage curve for starting age of regular cigarette smoking when contrasted with the experience of older cohorts (fig. 4).

Reliability of Estimates

Sampling Errors

Since the estimates are based on a sample survey, they are subject to sampling variability. The reliability of an estimated percentage depends upon both the size of the percentage and the size of the total on which it is based. Table 3 shows the approximate standard errors of estimates of selected sizes. Table 4 presents the approximate standard errors of estimated percentages that may be computed by using data from the report for both numerator and denominator.

The standard error is a measure of sampling variability. The chances are about 2 out of 3 that the difference due to sampling variability between an estimate and the figure that would have been obtained from a complete count of the population is less than the standard

error. The amount by which the standard error must be multiplied to obtain other odds deemed more appropriate can be found in most statistical textbooks. For example, the chances are about 19 out of 20 that the difference is less than twice the standard error and 99 out of 100 that it is less than 2½ times the standard error.

In addition to sampling variation, the figures are subject to errors of response and nonreporting, the possible effects of which are not included in the above measures of reliability.

Table 3. Standard error of estimated numbers published in this report (range of 67 chances out of 100)

Males		Females	
Estimated number	Standard error	Estimated number	Standard error
50, 000	15, 000	10, 000	5, 000
100, 000	21, 000	25, 000	8, 000
200, 000	30, 000	50, 000	12, 000
500, 000	47, 000	100, 000	16, 000
1, 000, 000	70, 000	200, 000	25, 000
2, 000, 000	95, 000	500, 000	35, 000
3, 000, 000	115, 000	750, 000	45, 000
5, 000, 000	145, 000	1, 000, 000	50, 000
10, 000, 000	190, 000	2, 000, 000	70, 000
15, 000, 000	220, 000	3, 000, 000	90, 000
20, 000, 000	235, 000	5, 000, 000	110, 000
25, 000, 000	240, 000	10, 000, 000	150, 000
		25, 000, 000	190, 000

Response Error

No scheme for validation of responses was built into the survey plan and no direct measures of the extent of response errors can be attempted. The extent of the possible bias associated with smoking history responses given on behalf of other persons has been discussed elsewhere in this chapter. The internal stability of the relationships in the smoking data when cross-classified by other population variables and between sample clusters of families is indirect evidence of the consistency of responses elicited by the questions.

It should be stressed that the questions were designed to classify people and not to estimate the amount of tobacco consumed. For the latter purpose the proper approach would have been to include questions on recent purchases or on the number of units consumed in a given day. Couching smoking questions in terms of "on the average" probably exerts a downward bias. The respondent may be encouraged to think of some smoking pattern which he rarely falls below and to neglect amounts in excess of this figure in his response. Since the primary objective of this survey was to classify groups of people for purposes of comparison, such biases would not be important so long as they were not large nor selective in character.

One of the questions on the smoking history form called for the number of units smoked.

Table 4. Standard error of estimated percentages published in this report (range of 67 chances out of 100)

Estimated percentage	Base of percentage								
	500, 000	1, 000, 000	2, 000, 000	3, 000, 000	5, 000, 000	10, 000, 000	15, 000, 000	25, 000, 000	50, 000, 000
	Males								
2 or 98	1. 3	0. 9	0. 7	0. 5	0. 4	0. 3	0. 2	0. 2	0. 1
5 or 95	2. 1	1. 5	1. 0	. 9	. 7	. 5	. 4	. 3	. 2
10 or 90	2. 9	2. 0	1. 4	1. 2	. 9	. 6	. 5	. 4	. 3
25 or 75	4. 1	2. 9	2. 1	1. 7	1. 3	. 9	. 8	. 6	. 4
50	4. 8	3. 4	2. 4	2. 0	1. 5	1. 1	. 9	. 7	. 5
	Females								
2 or 98	1. 0	0. 7	0. 5	0. 4	0. 3	0. 2	0. 2	0. 1	0. 1
5 or 95	1. 6	1. 1	. 8	. 7	. 5	. 4	. 3	. 2	. 2
10 or 90	2. 2	1. 6	1. 1	. 9	. 7	. 5	. 4	. 3	. 2
25 or 75	3. 2	2. 3	1. 6	1. 3	1. 0	. 7	. 6	. 5	. 3
50	3. 7	2. 6	1. 8	1. 5	1. 2	. 8	. 7	. 7	. 4

96946 O—57——30

From the responses to this question it was possible to estimate the class midpoint value of average daily consumption for categories of regular smokers. For cigarettes they were as follows:

Less than 10	4.8
10–20	18.3
21–40	35.0
More than 40	55.0

The class midpoint values obviously reflect very heavy concentration in responses on 20, 30, 40, 50, and 60 cigarettes.

The tax data indicate an average daily consumption in February 1955 of approximately 1 billion cigarettes. Utilizing the above information on average daily consumption rates in each smoking class, an estimate of present consumption of cigarettes may be derived from the survey data. The authors are indebted to A. G. Conover of the Department of Agriculture for the following calculation: With allowances for consumption by the military population in continental United States, inmates of institutions, persons under 18 years of age, persons in Alaska and Hawaii, and the occasional smokers, the survey data would estimate the national daily consumption of cigarettes at 813 million, about 19 percent under the true value. Substituting the experience of self-respondents for that of the total sample would raise the survey estimate by a factor of 1.07, or to 87 percent of the figure determined from tax sources. This underestimate of a national aggregate was noted also in a Danish survey of tobacco consumption (*23*). Considering the different intent for which the smoking questionnaire was designed, this correspondence between an estimate from the survey and the tax figure seems reasonable.

Analysis of Data

Form of Presentation

A primary objective in collecting smoking histories is to use the data in examining the epidemiological characteristics of lung cancer morbidity and mortality and to see whether the latter are consistent with the distribution of the smoking habit in various population groups. For this purpose, it is convenient to gather the data on smoking practices pertaining to each population variable in separate sections. This section discusses the total national data with emphasis on age and sex patterns. Subsequent sections deal with variation by place of residence; geographic region; race; employment status, occupation, and industry; previous military service; and marital status. In the appendix tables, the results from the sample studied have been appropriately weighted in order to estimate the national population totals classified by age, sex, and other variables, with appropriate age and sex detail. Variation in smoking patterns by age and sex is quite pronounced, and, for many comparisons, these two factors need to be controlled.

Analysis of the survey on smoking has been classified and discussed from the following aspects: (*a*) Lifetime history of the use of tobacco; (*b*) age at which smoking was started; (*c*) current consumption rate; (*d*) maximum consumption rate reached at any time; and (*e*) discontinuance rate. The last four items may be considered separately for the three forms of tobacco smoking—cigarette, cigar, pipe. Since cigarettes now and in the immediate past have accounted for the bulk of tobacco smoking, major emphasis in presentation has been directed to that form. Discontinuance rates might be treated from many viewpoints, for example, with age at which smoking was started, since by striking a balance between entries and withdrawals from the smoking population, its net size at any period could be estimated. Discontinuance will be discussed after presentation of current and maximum consumption rates because information on discontinuance seems most useful for interpreting and reconciling these two indexes of classifying the smoking habits of the population.

Lifetime History

Male respondents are classified as to whether they had ever smoked regularly cigarettes, cigars, pipe, or a combination of these forms of tobacco. Since women smoke cigarettes almost exclusively, no subdivision of lifetime history by form of tobacco smoked is attempted for women. Occasional smokers, persons who never smoked regularly at any time, are grouped into one category in appendix tables 2–6 without regard to the form in which tobacco was used. The other classification comprises persons who had "never smoked."

Of the 49,581,000 males and 55,096,000 females 18 years of age and over in the civilian, noninstitutional population of the United States as of February 1955, an estimated 11,360,000 males and 37,202,000 females, or 22.9 and 67.5 percent, respectively, have never smoked. These estimates include a prorating of persons in the sample for whom smoking histories were not obtained. Totals for histories "not reported" are carried systematically in the appendix tables; 5.8 percent of the men and 3.8 percent of the women fall into this category. In view of the relatively small proportion with unreported histories and the stability of this figure in most population subgroups, the "not reported" groups have not been routinely distributed among other smoking classes in the text tables and charts, except as noted. Prorating would influence but slightly the comparisons drawn, and the reader interested in such estimates of the magnitude of certain smoking categories can make these adjustments readily.

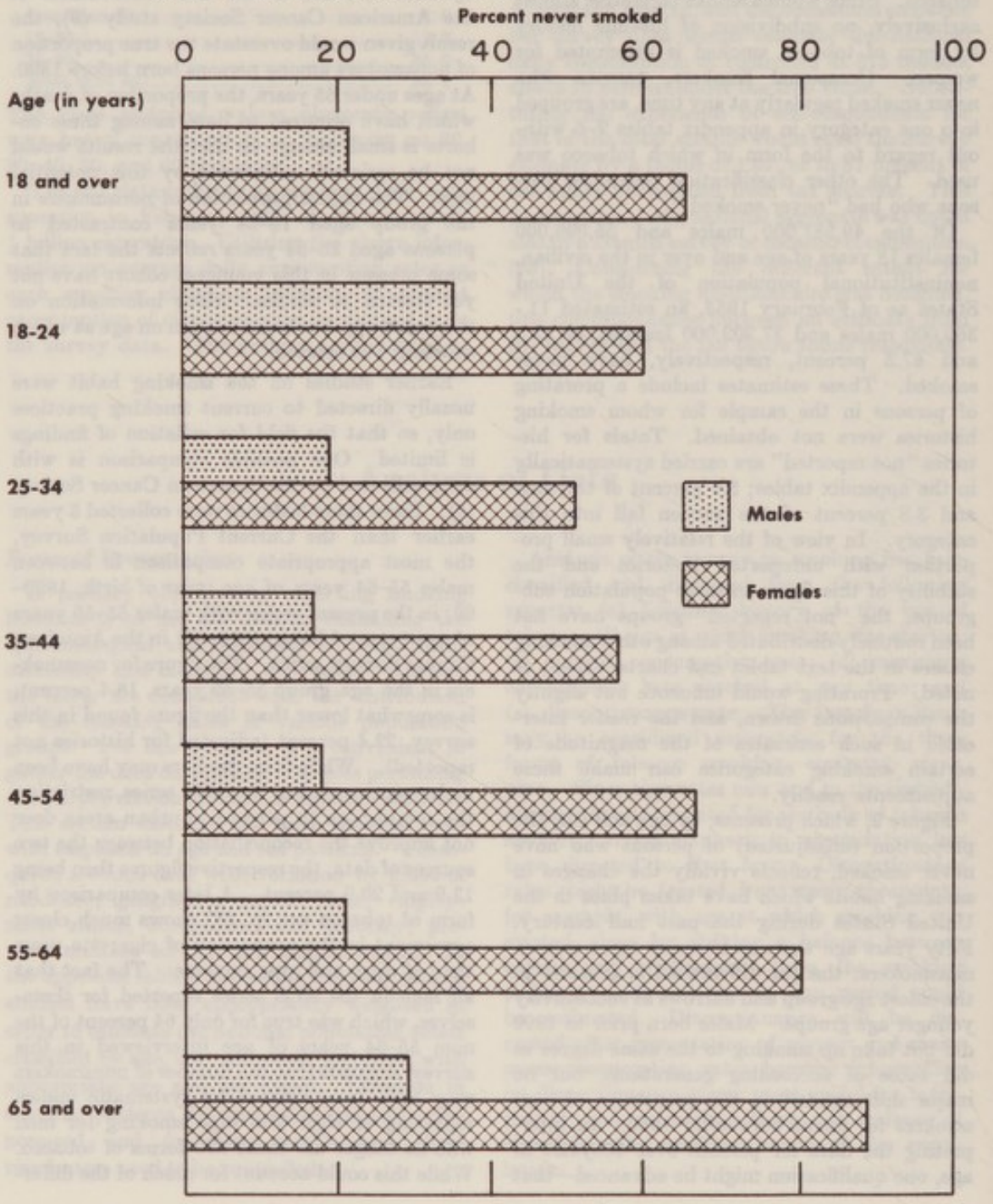
Figure 2, which presents, by age and sex, the proportion (unadjusted) of persons who have never smoked, reflects vividly the changes in smoking habits which have taken place in the United States during the past half century. Fifty years ago there were many more women nonsmokers; the sex differential is greatest in the oldest age group and narrows at successively younger age groups. Males born prior to 1890 did not take up smoking to the same degree as did those of succeeding generations, but no major shift appears in the proportion of nonsmokers for males born after 1900. In interpreting the data for persons over 65 years of age, one qualification might be advanced—that the information describes the cohort survivors rather than the original total cohort.

If smokers have been subject to an excess mortality risk from all cause on the order noted by Pearl (*6*) and in the first observations of the American Cancer Society study (*9*), the result given would overstate the true proportion of nonsmokers among persons born before 1890. At ages under 65 years, the proportion of deaths which have occurred to date among these cohorts is small enough so that the results would not be seriously influenced by this potential bias. The higher proportion of nonsmokers in the group aged 18–24 years contrasted to persons aged 25–34 years reflects the fact that some persons in this youngest cohort have not yet started to smoke. More information on this point appears in the section on age at which smoking was started.

Earlier studies on the smoking habit were usually directed to current smoking practices only, so that the field for collation of findings is limited. One possible comparison is with data collected by the American Cancer Society (*9*). Since these histories were collected 3 years earlier than the Current Population Survey, the most appropriate comparison is between males 55–64 years of age (year of birth, 1890–99) in the present series with males 55–59 years of age (year of birth, 1893–97) in the American Cancer Society series. The figure for nonsmokers in the age group 55–59 years, 16.4 percent, is somewhat lower than the figure found in this survey, 22.4 percent (adjusted for histories not reported). While farm dwellers may have been under-represented in the ACS series, restricting the comparison to persons in urban areas does not improve the reconciliation between the two sources of data, the respective figures then being 13.9 and 20.0 percent. A later comparison by form of tobacco use (p. 18) shows much closer agreement in the proportion of cigarette users than of cigar and pipe smokers. The fact that all men in the ACS series reported for themselves, which was true for only 64 percent of the men 55–64 years of age interviewed in this survey, suggests that the number of nonsmokers may have been inflated by systematic underreporting of cigar and pipe smoking for men who no longer use these two forms of tobacco. While this could account for much of the differ-

ence it should be remembered that the primary objective of the ACS study was to recruit persons for further observation and was not intended as a representative sample from which to estimate the proportion of smokers in the in the general population. Persons interviewed

Figure 2. Percentage of persons who have never smoked, by sex and age.

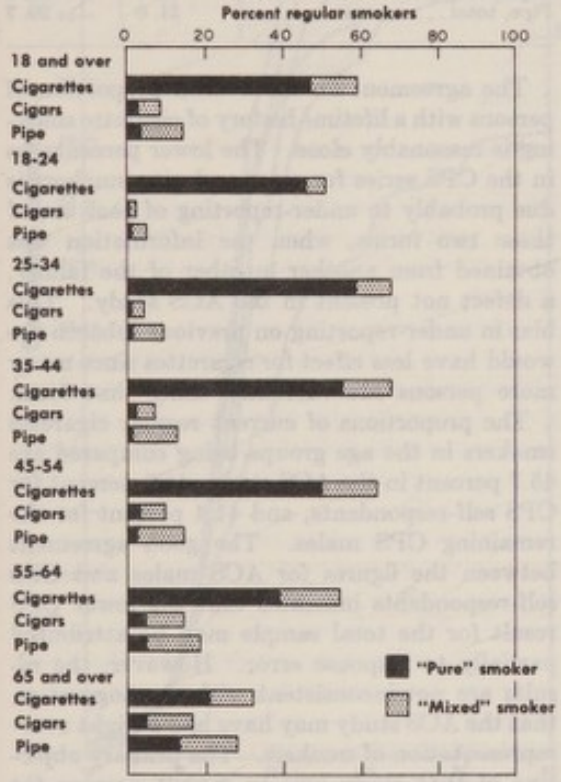


in the ACS study were generally known to the interviewers and it seems entirely possible that a slight tendency existed to overselect smokers for interview and followup.

Occasional smokers, numerically, are not an important category. The proportion does not vary much by age and sex and in only a few instances amounts to more than 5 percent. Regular smokers comprise the vast majority of persons who have ever indulged in smoking. Bearing in mind also the relative constancy of smoking histories not reported, one gains, from visualization of the complements to the bars in figure 2, a fairly accurate picture of the variation in the proportion of regular smokers by age and sex. Complete data on this point are presented in appendix table 2.

Figure 3 distributes regular male smokers with respect to the lifetime history of use of specific forms of tobacco. Data on females are omitted since cigarettes accounted for nearly all tobacco used by women. Persons using two or more forms of tobacco have been counted in each category and the portion of the bars for "mixed" smokers represents duplicate counts of individuals. Information on the exact patterm of use by individuals is also shown in appendix table 2.

Figure 3. Percentage of males who have regularly smoked at some time cigarettes, cigars, or pipe, by age.



NOTE: A "pure" smoker has used regularly only one form of tobacco.

Cigarettes are the preponderant form in which tobacco is used; 59.1 percent of all males over 18 years of age reported that they had used cigarettes regularly at some time. In contrast, cigar and pipe smokers are represented by 8.7 and 14.4 percent, respectively, of the total male population. Among men over 65 years of age, pipe smoking rivals cigarette smoking in popularity. The differential in favor of cigarettes widens at the younger ages. Moreover, in the younger cohorts there seems to be a shift to "pure" cigarette smoking, accompanied by a lessened tendency to smoke cigars or a pipe exclusively. The great majority of cigar and pipe smokers, especially those under 55 years of age, have smoked in combination with another form of tobacco, usually cigarettes.

Answers as to the number of smokers in the population depend, of course, on the definitions and screening questions employed. The data in appendix table 2 on lifetime history of cigarette use cannot be reconciled completely with estimates from trade sources, which place the number of cigarette smokers in the neighborhood of 60 million (*3*). If the 60 million is intended to refer to present consumers only (and a determination on this point is difficult since the phrasing of the question in market surveys is usually some variant of "Do you ever smoke . . .") the gap between the data in appendix table 13 on current cigarette smokers and trade estimates would be greater. Adjusting for persons whose smoking history was unreported, the present survey estimates that approximately 47 million persons over 18 years of age in the civilian population are now or have been regular cigarette smokers. Inclusion of occasional smokers would raise the total to 52 million. An allowance for persons in military service and in institutions, not covered in the survey of the civilian population, and for smokers presently under 18 years of age, would not add more than 3 million smokers at most.

Thus, the maximum estimate of persons using cigarettes to be derived from this survey is about 55 million. Some of the discrepancy between this estimate and those of trade sources may be traced to differences concerning the number of women smokers.

For men, the survey estimate for cigarette smokers, including occasional users, comes very close to 65 percent. Wrather states that "several authorities have concluded that about 65 to 70 percent of the adult males and around 35 percent of the women use cigarettes" (*3*). The *Milwaukee Journal's* market survey for the Milwaukee metropolitan area (*16*) has included a question on cigarette smoking from its inception in 1923, and since 1937 its index of the proportion of men smoking cigarettes has remained almost unchanged—at about 66–68 percent.

For women, the maximum estimate of cigarette smokers, including occasional users, which might be derived from this survey is 32 percent. The results of the present survey clearly point to a marked diminution in the proportion of smokers in passing from the age group 45–54 years to ages 55–64 and 65 and over. In this respect, the survey results check well with those of the Columbus study of Mills and Porter (*20*), which reported percentages of women smokers in line with the present study and also showed a sharp drop in the proportion of women over 50 years of age who smoke. They also seem consistent with a figure of 20.5 percent of smokers among women over 40 years old in a general hospital population (not lung cancer patients) found by Graham (*26*). Market survey information which has come to our attention agrees substantially with the present results for younger women smokers; the serious discrepancies appear in estimates for older women smokers, particularly those over 50 years of age. It may be that trade and market survey sources have not polled representative samples of older women.

Despite some differences in estimates on the number and proportion of smokers in the population there are many features on which this survey and trade information agree. In general, the variation in smoking patterns by urban or rural residence, major regions of the country, age—especially for male smokers—and form of tobacco use, as reported here, is consistent both with trade impressions and with a recent survey (unpublished) made for the Cigar Institute of America.

The present data from the Current Population Survey (CPS) on proportion of regular smokers among males 55–64 years of age (adjusted for histories not reported) can also be compared with an almost corresponding cohort in the American Cancer Society (ACS) study. In both surveys, the questions on smoking were couched in almost identical language:

Form of tobacco use	Percentage of men with lifetime history of regular smoking	
	CPS, 1955 (55–64 years)	ACS, 1952 (55–59 years)
Cigarette, total	58.0	60.5
Cigarette only	41.3	35.5
Cigarette with cigar and/or pipe	16.7	25.0
Cigar, total	15.3	23.8
Pipe, total	21.0	29.7

The agreement on the overall proportion of persons with a lifetime history of cigarette smoking is reasonably close. The lower percentages in the CPS series for cigar and pipe smokers is due probably to under-reporting of past use of these two forms, when the information was obtained from another member of the family, a defect not present in the ACS study. This bias in under-reporting on previous tobacco use would have less effect for cigarettes since many more persons are currently using that form.

The proportions of current regular cigarette smokers in the age groups being compared are 45.7 percent in the ACS study, 44.7 percent for CPS self-respondents, and 41.4 percent for the remaining CPS males. The good agreement between the figures for ACS males and CPS self-respondents indicates that the lower CPS result for the total sample may be attributed partially to response error. However, the results are not inconsistent with the suggestion that the ACS study may have had a slight overrepresentation of smokers. The primary objective of that study was to recruit persons for further observation, and it was not intended to

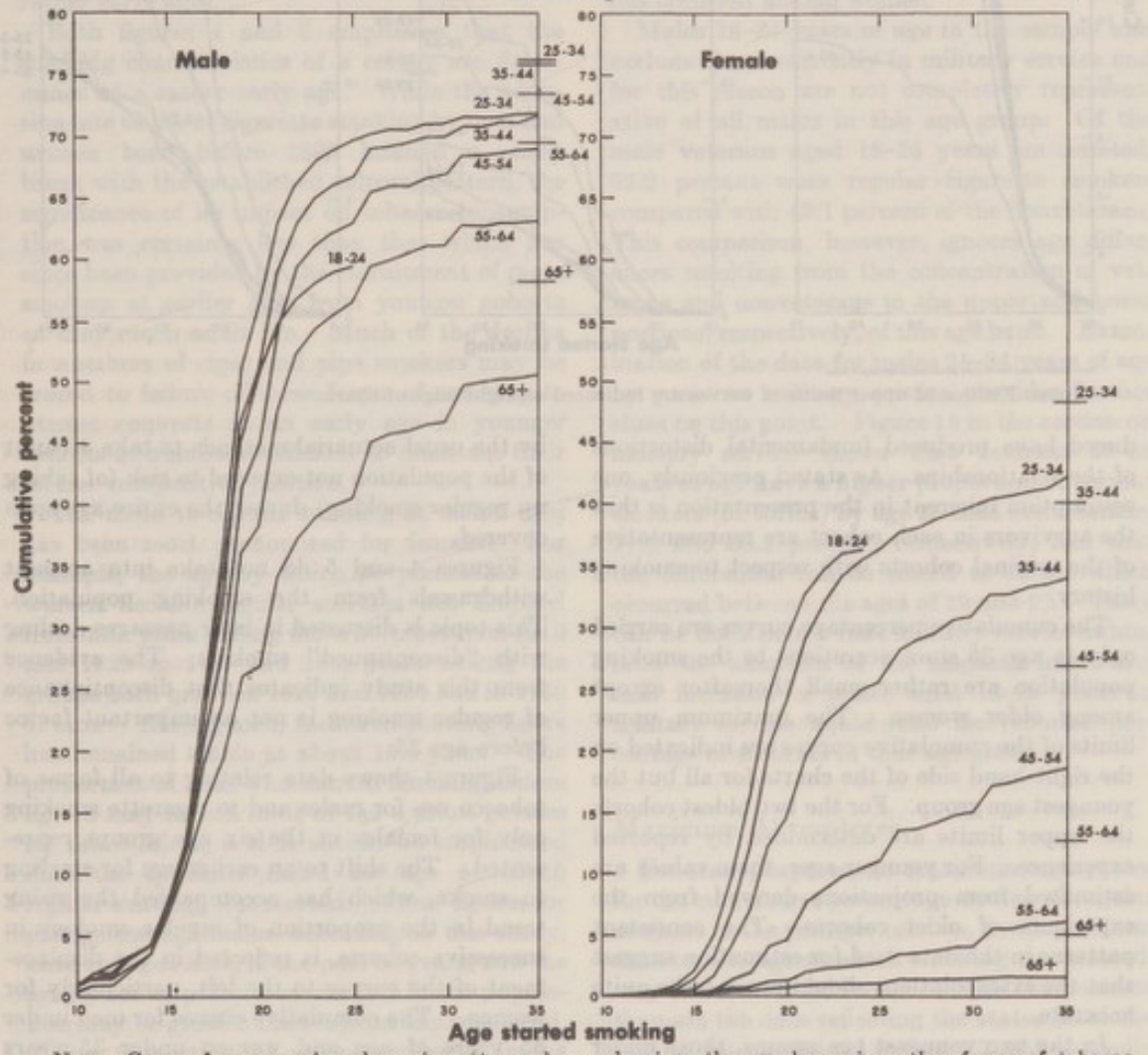
estimate the proportion of smokers in the general population. Since persons interviewed in the ACS study were known to the interviewers, it is possible that a very slight tendency existed to overselect smokers for interview and followup.

The gradients in the proportion of smokers by successive age groups in the CPS data are duplicated within the limited age range covered in the ACS study.

Age Started Smoking

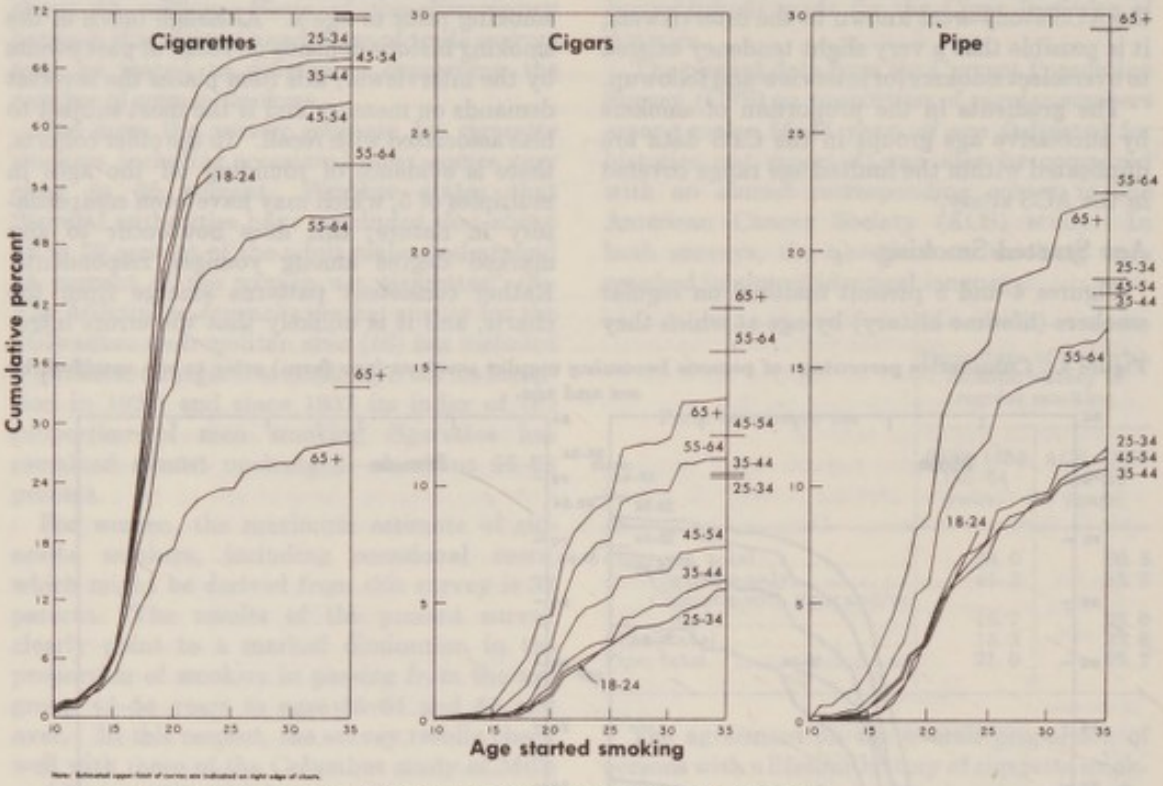
Figures 4 and 5 present material on regular smokers (lifetime history) by age at which they started smoking, with the results expressed in terms of the cumulative percentage who started smoking prior to age x. Although much of the smoking history depends on recall of past events by the interviewer, this item places the severest demands on memory and is the most subject to bias associated with recall. In the older cohorts, there is evidence of rounding off the ages in multiples of 5, which may have been compensatory in nature; this does not occur to any marked degree among younger respondents. Rather consistent patterns emerge from the charts, and it is unlikely that the errors intro-

Figure 4. Cumulative percentage of persons becoming regular smokers (any form) prior to age specified, by sex and age.



NOTE: Curves for women based on cigarette smokers only since the number using other forms of tobacco is very small. Estimated upper limits of curves are indicated on right edge of charts.

Figure 5. Cumulative percentage of males becoming regular cigarette, cigar, or pipe smokers prior to age specified, by age.



NOTE: Estimated upper limits of curves are indicated on right edge of charts.

duced have produced fundamental distortions of the relationships. As stated previously, one assumption inherent in the presentation is that the survivors in each cohort are representative of the original cohorts with respect to smoking history.

The cumulative percentage curves are carried only to age 35 since accretions to the smoking population are rather small thereafter except among older women. The maximum upper limits of the cumulative curves are indicated on the right-hand side of the charts for all but the youngest age group. For the two oldest cohorts the upper limits are determined by reported experience. For younger ages, these values are estimated from projections derived from the experience of older cohorts. The consistent patterns in the data used for estimation suggest that the extrapolations should prove to be quite accurate.

In the two youngest age groups, those under 35 years, the cumulative curves are adjusted by the usual actuarial methods to take account of the population not exposed to risk (of taking up regular smoking) during the entire age span covered.

Figures 4 and 5 do not take into account withdrawals from the smoking population. This topic is discussed in later passages dealing with "discontinued" smokers. The evidence from this study indicates that discontinuance of regular smoking is not an important factor before age 35.

Figure 4 shows data relating to all forms of tobacco use for males and to cigarette smoking only for females in the six age groups represented. The shift to an earlier age for starting to smoke, which has accompanied the rising trend in the proportion of regular smokers in successive cohorts, is reflected in the displacement of the curves to the left, particularly for women. The cumulative curves for men under 55 years of age and women under 35 years exhibit recognizable S-shaped patterns, imply-

ing the existence of a stable set of age-specific probabilities for taking up smoking. Women over 35 years of age showed progressive departure from the S-pattern; this reflects the large-scale adoption of the smoking habit by women in the 1920 and 1930 decades. A rather regular pattern still appears for males between 45 and 65 years old, but for men over 65 years the curve shows evidence of an adoption of the smoking habit later in life by that cohort. In the past, proportionately more women than men have taken up smoking after age 35. This situation may not persist among women born after 1920, because a sizable proportion of them has started to smoke at rather early ages.

Both figures 4 and 5 emphasize that the smoking characteristics of a cohort are determined at a rather early age. While the adoption late in life of cigarette smoking by men and women born before 1890 marked a sharp break with the established cultural pattern, the significance of its impact on tobacco consumption was certainly less than that which has since been provided by the recruitment of more smokers at earlier ages from younger cohorts as they reach adult life. Much of the decline in numbers of cigar and pipe smokers may be traced to failure of these forms of smoking to attract converts at an early age in younger cohorts in numbers sufficient to maintain their former competitive position.

The trend to regular smoking at earlier ages has been most pronounced for females. For example, the age by which 20 percent of the women become regular smokers has dropped from 21.3 years among those born between 1910 and 1920 to 19.0 and 18.5 years among the groups born between 1920 and 1930 and in 1930 or later. Among men, the corresponding figure has remained stable at about 15.6 years. The proportion of men who started smoking before age 15 may exceed most of the a priori guesses on this subject, and it should be emphasized that the question called for age at which regular smoking was started. These figures for young teen-age males, according to this study, have changed little in the past 50 years, and the proportion of precocious smokers in the population may be greater than is generally suspected.

The proportion of self-respondents among males 18–24 years of age is considerably smaller, 43 percent, than in any other sex-age group. Furthermore, this particular group shows the greatest difference between self-respondents and those for whom data were supplied by others with respect to the proportion of cigarette smokers (see appendix table 12). This suggests some children may have successfully concealed their smoking practices from the rest of the family. Therefore, the observation that the cumulative percentage of smokers among men now 18–24 years of age is falling behind the corresponding experience of the next older cohort (25–34 years) must be accepted with reservation, particularly since no similar shift has occurred among women.

Males 18–24 years of age in the sample also exclude those currently in military service and for this reason are not completely representative of all males in this age group. Of the male veterans aged 18–24 years enumerated, 62.2 percent were regular cigarette smokers compared with 43.1 percent of the nonveterans. This comparison, however, ignores age differences resulting from the concentration of veterans and nonveterans in the upper and lower portions, respectively, of this age band. Examination of the data for males 25–34 years of age by previous military service provides further clues on this point. Figure 19 in the section on military service shows that veterans 25–34 years of age have a higher proportion of regular smokers (all forms) by age 25 than nonveterans, 71.0 and 66.1 percent, respectively, and that the differential can be traced to events which occurred between the ages of 19 and 22. There can be little doubt that military service stimulates the adoption of the smoking habit and that inclusion of males aged 18–24 years in military service would raise the reported percentage of smokers in that age group.

Maximum Asymptotes

For some purposes, the maximum asymptotes for the cumulative percentage curves of regular smokers in the younger cohorts, developed from data on the age at which smoking was started, may be more useful for age and sex comparisons than are the data reflecting the status as of the survey date contained in figure 2 and appendix

table 2. Contrasts of these respective measures have been set forth for cigarette smokers in table 5. Because of the later age at which women take up smoking, the differences between current status and projected maximum percentage are greater for that sex.

Table 5. Comparison of percentage of regular cigarette smokers (lifetime history) as of the survey date with estimated future maximum percentage for the corresponding cohort, by age and sex

Age (years)[1]	Males		Females	
	At time of 1955 survey	Estimated future maximum	At time of 1955 survey	Estimated future maximum
25–34	68.7	71.3	39.8	47.8
35–44	68.9	70.7	35.7	40.5
45–54	65.3	66.3	25.1	26.7
55–64	55.8	56.5	12.6	12.6
65 and over	33.7	33.7	4.6	4.6

[1] As of date of survey.

Table 6. Median age started smoking, regular cigarette, cigar, and pipe smokers, by age and sex

Age (years)[1]	Age started smoking			
	Male			Female (cigarette)
	Cigarette	Cigar	Pipe	
25–34	17.9	33.2	29.5	20.0
35–44	18.2	30.6	27.4	21.3
45–54	18.4	25.9	26.7	26.0
55–64	18.6	25.0	23.7	35.3
65 and over	19.3	23.2	22.5	39.9

[1] As of survey date.

Cigarette, Cigar, and Pipe Smoking

The changing patterns in the use of tobacco by males are accentuated when individual forms are considered (fig. 5, but note the different scale for cigar and pipe smokers). Since cigarettes are the predominant form, the comments on male smokers in general apply to cigarette smokers as well. For cigarettes, the cumulative curve for males over 65 years of age departed even more markedly from the predominant S-shaped representation for the younger male cohorts than was the case for all smoking forms combined. Comparing the successive cohorts, it is evident that the rise in the number of male cigarette smokers is not completely at the expense of cigar and pipe. More widespread use of cigarettes in the younger cohorts has increased the proportion of regular smokers and seems to have promoted the practice of smoking at earlier ages.

Figure 5 shows that the smaller number of males taking up pipe smoking adopt it later in adult life, with cigars generally being the form of tobacco taken up latest in life. Table 6 records the estimated median starting age for smokers of the three forms of tobacco smoking. The median ages were estimated from the charts, the readings being taken at half the estimated upper limit of the cumulative percentage curves. With the decreasing popularity of pipe and cigars, the median starting age for these two forms of smoking has risen in the younger age groups.

Figure 6. Current status of men with respect to smoking cigarettes, cigars, and pipe, by age.

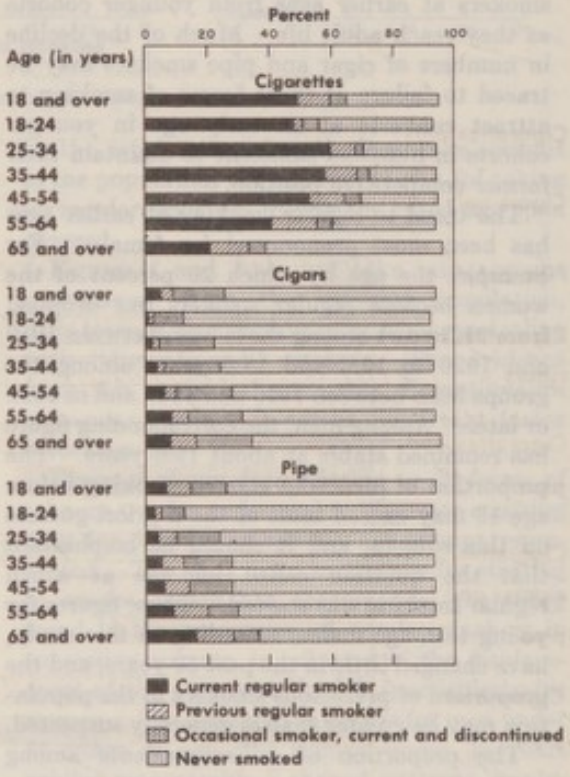
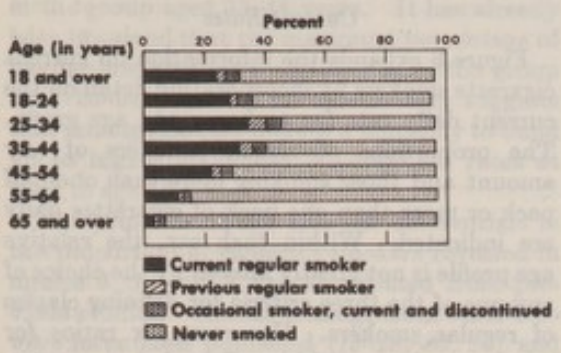


Figure 7. Current cigarette smoking status of women, by age.

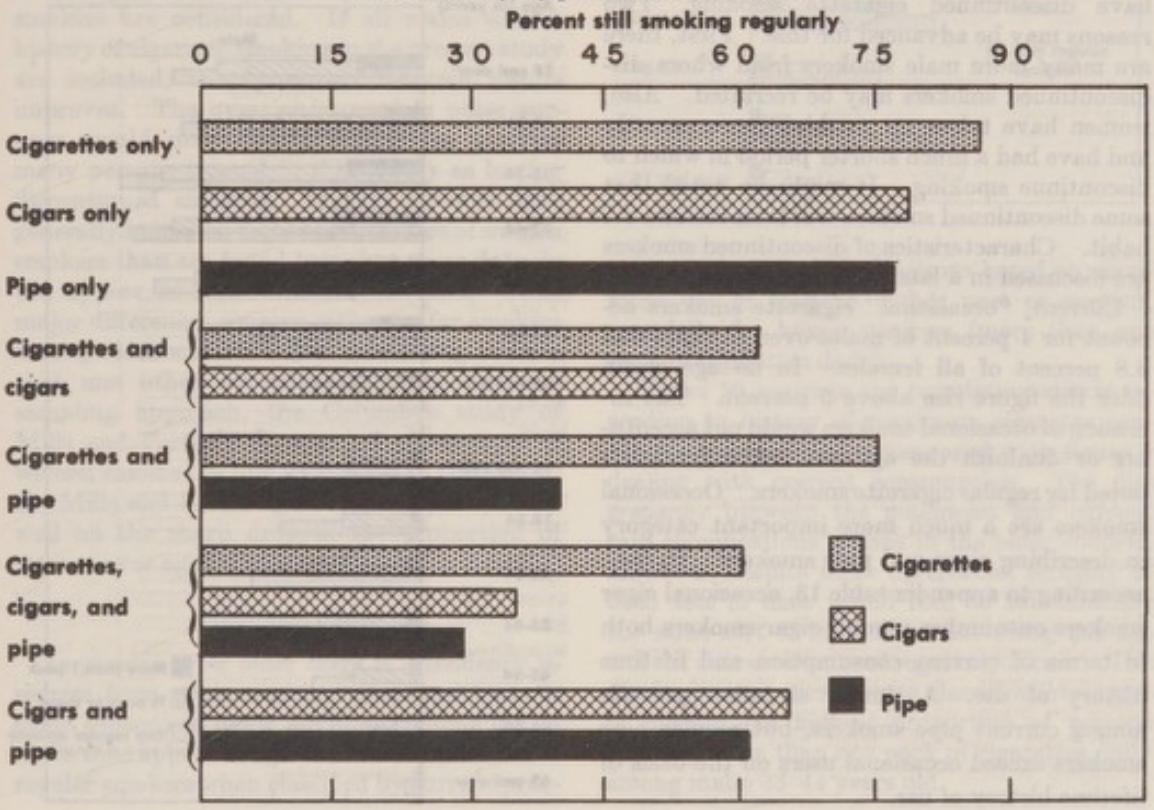


Smoking Status

Figure 6 presents information on the current smoking status among men; data for cigarettes, cigars, and pipe smoking are given separately. The corresponding data for females for cigarettes only are in figure 7. Along with the proportion of current, regular smokers the remainder have been identified as to whether they were once regular smokers, occasional smokers only, or had never smoked. The components do not total 100 percent because of the exclusion of persons for whom smoking histories were not reported. The data in figure 6 on males who have never smoked cigars, cigarettes, or pipe supplement those in figure 2 concerning complete abstention from tobacco in any form. Inspection of figures 2 and 7 and the corresponding source tables (appendix tables 2 and 13) reveals the negligible number of women cigar and pipe smokers estimated by the survey (20,000).

Current, regular cigarette smokers exhibit a systematic age pattern for both males and females, the highest percentage appearing at ages 25–34 years and tapering off in successively older groups. The differences between the groups aged 18–24 and 25–34 years should diminish in time as more persons in the younger cohort take up smoking. The number of current smokers depends on the interplay of rates

Figure 8. Percentage of all regular smokers 18 years of age and over still smoking regularly at time of survey, by pattern of tobacco use.



for entering and leaving the smoking population. Figures 6 and 7 also summarize the nature of differences obtained from classification by current status as opposed to considering lifetime history. Comparisons by current status of cigarette smoking tend to minimize the sex differentials and, among males, to maximize the age differentials. In drawing comparisons among males, current status also stresses the relative predominance of cigarette smoking. The latter point is emphasized in figure 8, which shows, by the use of various forms of tobacco smoking, the proportion of regular smokers (lifetime history) who have continued to smoke regularly up to the time of the survey. Not only is this figure higher among "pure" cigarette smokers as opposed to "pure" cigar and pipe smokers, but, among persons who have smoked cigars and pipes in conjunction with cigarettes, there is an unmistakable tendency to continue to smoke cigarettes and to discard regular use of cigars and pipes.

Men who have discontinued cigarette smoking proportionately outnumber the women who have discontinued cigarette smoking. Two reasons may be advanced for this. First, there are many more male smokers from whom disdiscontinued smokers may be recruited. Also, women have taken up smoking more recently and have had a much shorter period in which to discontinue smoking. It might be noted that some discontinued smokers will later resume the habit. Characteristics of discontinued smokers are discussed in a later separate section.

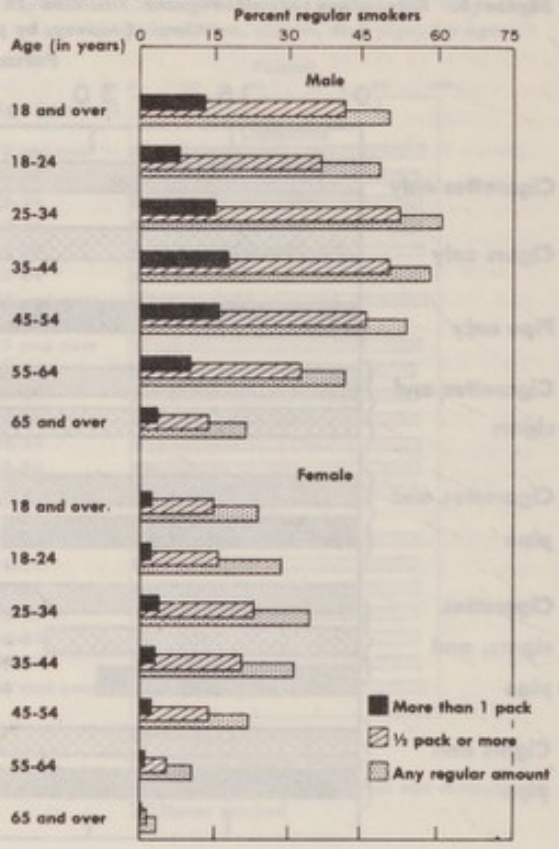
Current, "occasional" cigarette smokers account for 4 percent of males over 18 years and 3.8 percent of all females. In no age group does the figure rise above 5 percent. The inclusion of occasional smokers would not accentuate or diminish the age and sex differentials noted for regular cigarette smokers. Occasional smokers are a much more important category in describing cigar and pipe smokers. In fact, according to appendix table 13, occasional cigar smokers outnumber regular cigar smokers both in terms of current consumption and lifetime history of use. A similar situation prevails among current pipe smokers, but regular pipe smokers exceed occasional users on the basis of lifetime history of use.

Cigarette Consumption Rates

Current Rates

Figure 9 expands the information on current cigarette smokers by incorporating detail on the current daily rate for each sex and age group. The proportions of regular smokers of any amount and those smoking more than one-half pack or more than one pack of cigarettes daily are indicated. Within each sex, the relative age profile is not greatly altered by the choice of any one of the three criteria for defining classes of regular smokers. However, sex ratios for regular smokers would be affected since proportionately more women than men fall into the category of those who smoke under one-half pack of cigarettes daily. The largest sex differentials are obtained when comparison is restricted to heavy smokers consuming more than one pack of cigarettes daily.

Figure 9. Percentage of persons currently smoking cigarettes regularly, by sex and age, with additional detail on current daily rate.



Among men, the highest proportion smoking more than one pack of cigarettes daily is found in the group aged 35–44 years. It has already been observed that the maximum percentage of regular smokers (any amount) is in the group aged 25–34 years. This relationship suggests that among smokers there is a tendency to build up to higher maximum consumption rates at least to age 35.

The presence of age and sex differentials in the proportion of cigarette smokers revealed in figures 6, 7, and 9 was anticipated from previous studies. Similar findings from other surveys have been published (*15–18, 20, 22*) and have also appeared in unpublished market surveys. No precise comparison of the age and sex pattern with other results can be attempted because of differences in concepts for classifying smoking habits, the areas and time periods covered, and the coarse age groupings used in some reports. The magnitude of the age differentials among men are reasonably consistent with other reports although the present data indicate lower smoking rates, regular and occasional, than reported elsewhere when only current smokers are considered. If all males with a history of cigarette smoking in the present study are included, the agreement between results improves. The question(s) used in other surveys would undoubtedly include as smokers many persons treated in this survey as having discontinued smoking. Market surveys have generally indicated higher proportions of women smokers than are found here, but these data do not appear as representative or valid. The major differences concern estimates for smokers among older women. It is interesting to note that one other investigation using an area sampling approach, the Columbus study of Mills and Porter (*20*) reported percentages of women smokers in line with those in this study; the Mills and Porter figures agreed particularly well on the sharp drop in the proportion of women over age 50 who smoke.

Maximum Rates

As persons grow older there is a tendency to regress from peak smoking rates and to consume tobacco in lesser amounts. Some differences thus appear in age and sex comparisons of regular smokers when classified by current practices as opposed to comparisons based on maximum use of tobacco, either past or present, especially for heavy smokers (more than one pack of cigarettes daily).

Figure 10. Percentage of persons smoking cigarettes regularly (lifetime history), by sex and age, with additional detail on maximum daily rate.

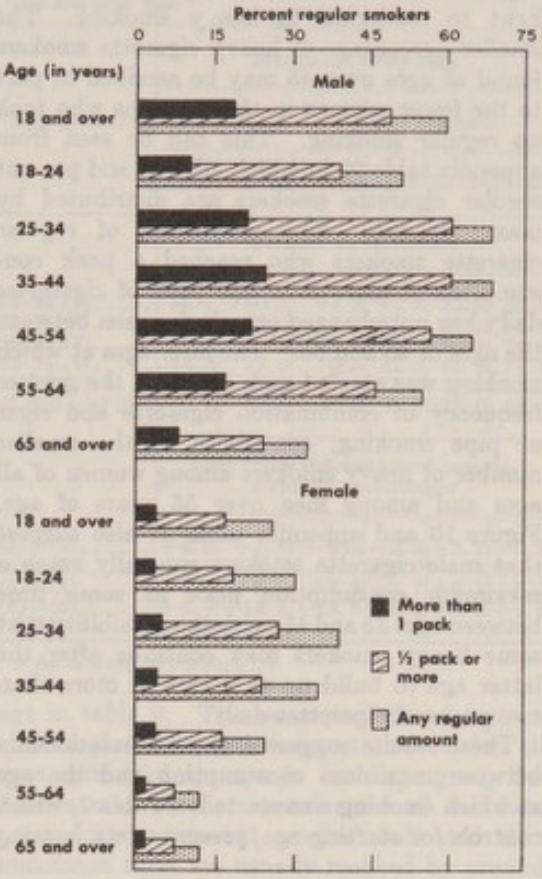


Figure 10 portrays the population status according to history of maximum cigarette consumption and may be compared with figure 9 dealing with current consumption. The discrepancy between the maximum and current rate classifications widens in the older cohorts. However, despite these differences, scrutiny of both sets of data would lead to substantially the same sort of inferences concerning the age and sex characteristics of regular smokers. The maximum and current rate classifications agree in placing the highest proportion of persons smoking more than one pack of cigarettes daily among males 35–44 years old.

The yield of heavy smokers in a cohort depends on the proportion that took up smoking, as well as on a rate of progression from light to moderate to heavy smoking. The smaller percentage of heavy cigarette smokers found at ages over 45 may be ascribed in part to the fewer persons in these groups who took up regular smoking. This can be seen from appendix table 22 in which all past and present regular cigarette smokers are distributed by maximum rate. The proportion of regular cigarette smokers who reached a peak consumption of more than one pack of cigarettes daily has not changed greatly for men between the ages of 25 and 55. The later ages at which smoking was started and, for men, the greater frequency of combination cigarette and cigar or pipe smoking, contribute to the smaller number of heavy smokers among women of all ages and among men over 55 years of age. Figure 10 and appendix table 22 also suggest that male cigarette smokers generally reach a maximum consumption peak at some time between ages 35 and 45, with the possibility that some heavy smokers may continue after the latter age to build up to a rate of more than two packs of cigarettes daily.

These results suggest a possible relationship between maximum consumption and the age at which smoking was started. Table 7, which controls for starting age, presents data bearing on this point. There is a definite rise in the proportion of regular cigarette smokers who have reached a daily consumption of more than one pack among those who started smoking at very young ages. The increase is particularly marked for both men and women who started before age 18. Two interpretations of the data are possible. The first would regard the higher proportion of heavy smokers as the result of a longer period of exposure and conditioning, whereas the other would introduce a selection element and view those smokers who started at an early age as having, to a more marked degree, some endogenous factor predisposing toward heavier smoking. If the selection hypothesis were true, one would expect that, as the total proportion of regular smokers increased in the younger age groups, light or moderate smokers would be represented to a greater degree since the additional smokers recruited should constitute a more marginal group and hence should be less likely to become heavy smokers. The results observed do not support the selection thesis. Rather, they suggest that the forces which lead smokers to maintain or increase consumption rates are associated with or are of the same character as those which determine the proportion of smokers in a cohort.

Table 7. Percentage of regular cigarette smokers (lifetime history) who have reached a maximum rate of more than 1 pack daily, according to age started smoking, by sex and age

Age (years)[1]	Age started smoking (years)			
	Under 18	18–19	20–21	22 and over
Male:				
25–34	35.3	29.1	22.1	22.8
35–44	40.8	33.9	30.6	26.5
45–54	38.6	31.4	32.6	25.2
55–64	38.2	27.0	24.9	23.3
65 and over	30.1	19.1	19.1	14.1
Female:				
25–34	20.9	12.2	10.2	6.9
35–44	19.7	11.5	9.4	8.8
45–54	26.5	24.9	15.2	13.1
55–64	23.3	29.3	10.6	11.4
65 and over	13.3	[2]	[2]	7.1

[1] As of date of survey.
[2] Insufficient data.

Continuance at Maximum Rate

Cross tabulations of current and maximum rates of tobacco consumption such as are contained in appendix table 22 can be cast into measures of persistence at the maximum rate. Obviously, the proportion of smokers who continue smoking at the maximum class rate attained is a crude measure and does not take into account small reductions within the broad class limits. The proportions, however, may be more sensitive indexes than appear at first glance. Within each class, definite concentrations on certain daily rates appear. In the class "10–20 cigarettes," most persons report 20 cigarettes; for "21–40 cigarettes" and "41+ cigarettes," the modal units are 30, 40, 50, and 60 cigarettes. More than mere rounding of numbers in reporting would seem to be involved. These peaks undoubtedly arise from the practice of vending cigarettes in packs of 20 and

from individual programing of daily consumption in units of half-packs.

Among men with a lifetime history of regular cigarette smoking, 73.6 percent have continued to smoke at their maximum class rate. The corresponding figure for women is 79.7 percent. Cigar and pipe smokers have retreated from their maximums in greater numbers than cigarette smokers, the proportions still remaining at the maximum class rates being 49.1 and 44.0 percent, respectively. As a group, cigar and pipe smokers are older than cigarette smokers, but adjusting the comparison for age does not eliminate the difference. Assuming the same age distribution for cigar and pipe smokers as for cigarette smokers, the respective proportions remaining at the maximum would be 51.7 and 42.5 percent.

Table 8 shows for each form of tobacco smoking the proportion of regular smokers continuing to smoke at their highest attained rate.

Table 8. Percentage of regular cigarette, cigar, and pipe smokers (lifetime history) 18 years of age and over continuing to smoke at their maximum, by maximum rate attained

Form of tobacco smoking	Maximum daily rate			
	Light	Moderate	Heavy	Very heavy
Cigarette:				
Male	76.3	78.7	68.3	47.7
Female	82.3	82.8	66.6	48.8
Cigar (male)	53.6	56.5	52.4	46.8
Pipe (male)	46.6	45.9	49.1	50.7

Light = Less than 10 cigarettes, 1–2 cigars, less than 5 pipefuls.
Moderate = 10–20 cigarettes, 3–4 cigars, 5–9 pipefuls.
Heavy = 21–40 cigarettes, 5–8 cigars, 10–19 pipefuls.
Very heavy = More than 40 cigarettes, more than 8 cigars, more than 19 pipefuls.

Among men and women cigarette smokers, the probabilities of regressing from the "over 40 cigarettes" and "21–40 cigarettes" categories are greater than for those who smoke lesser amounts. Little difference by maximum rate class was noted when the data for pipes and cigars were examined. This strengthens the belief that much of the cutback from maximum for the latter two forms has been due to a switch to cigarettes.

The cigarette data have been subdivided by

Table 9. Percentage of regular cigarette smokers (lifetime history) continuing to smoke at their maximum according to maximum rate attained, by sex and age

Age (years) [1]	Maximum daily rate					
	Male			Female		
	<10	10–20	21–40	<10	10–20	21–40
	Percent at maximum					
18–24 (1)	89.1	90.6	74.3	86.4	83.8	54.5
25–34 (2)	82.7	83.0	70.3	81.1	81.2	64.5
35–44 (3)	76.2	81.2	70.5	83.3	84.9	74.0
45–54 (4)	74.3	77.9	70.5	82.3	85.3	71.5
55–64 (5)	68.3	68.9	61.9	78.6	76.2	62.2
65 and over (6)	62.4	55.6	43.3	69.3	72.9	52.4
	First order differences of complementary percentages					
(3)−(2)	+6.5	+1.8	−0.2	−2.2	−3.7	−9.5
(4)−(3)	+1.9	+3.3	.....	+1.0	−.4	+2.5
(5)−(4)	+6.0	+9.0	+8.6	+3.7	+9.1	+9.3
(6)−(5)	+5.9	+13.3	+18.6	+9.3	+3.3	+9.8

[1] As of date of survey.

age in table 9. The same persistence pattern with regard to maximum rate noted for all ages combined appears in each age group for both sexes. Earlier examination of the data on maximum amount smoked indicated that maximum rates are usually reached by around age 35. An indirect approach to measuring changes in smoking patterns, typified by the index to be discussed here, was adopted because of limitations imposed by the questionnaire. Time would not permit and informants might not have been able to supply a detailed chronology on shifts in smoking habits. However, further advances may be made indirectly by examining the first-order differences of the proportions continuing at a maximum (or better, their complements—the proportions dropping back from a maximum) in the various age groups, which are presented in the second half of table 9. The percentages representing persistence at, or dropping back from, a maximum may be regarded as cumulative in character from age 35 on, that is, the experience for persons in the group aged 55–64 years reflects the events occurring at any previous age. A very rough estimate of the proportion of persons

55–64 years of age dropping back from a maximum within the preceding 10 years may then be obtained by subtracting from this cumulative figure the corresponding value for persons 45–54 years of age.

The first-order differences for a maximum rate "less than 10 cigarettes" were rather constant for men, suggesting a uniform amount of curtailment throughout most of the smoking-life span. When the maximum was "over 20 cigarettes," the differences became much greater after age 55, indicating that for heavier smokers the probability of curtailment increases sharply with advancing age.

Discontinuances

Once a smoker has reached his maximum rate he may maintain it, drop back to a lower rate, or discontinue regular smoking. This section discusses the last course of action. Regular smokers may discontinue by reverting to occasional use or by becoming abstainers; abstention is the more frequent course of action. Table 10 shows the proportion of discontinued smokers for combinations of tobacco use subdivided into abstainers and occasional smokers. For "pure" cigarette smokers, the proportions are almost the same for the two sexes. Cigar, pipe, and combination smokers exhibit higher discontinuance risks than smokers of cigarettes alone although the differential would be narrowed somewhat if adjusted for the older ages of the former groups. Reversion to occasional smoking is more frequent for cigars and pipe than for cigarettes.

Table 10. Percentage of all regular smokers 18 years of age and over who have discontinued regular smoking, by pattern of tobacco use and sex

Pattern of use and sex	Total	Abstainers	Now occasional smokers
Cigarette only:			
Male	13.2	11.4	1.8
Female	12.7	10.8	1.9
Cigar only (male)	21.2	16.3	4.9
Pipe only (male)	22.7	17.8	4.9
Cigarette and cigar (male)	18.1	16.5	1.6
Cigarette and pipe (male)	12.3	10.8	1.5
Cigarette, cigar, and pipe (male)	20.6	19.6	1.0
Cigar and pipe (male)	19.5	16.2	3.3

In the survey, the determination of his status as a current or discontinued smoker was made by the respondent and depended in part on his views as to the meaning of "present time" in reporting on smoking practices as well as on his current intentions regarding them. There is some overstatement of the percentage of persons who discontinued smoking since a number of presently discontinued smokers will later resume their regular habits. This tendency would have a smaller effect percentagewise on the estimates for current smokers. For example, if all males 18 years and over who stated that they had discontinued cigarette smoking during the previous 18 months were counted as current smokers, the figure for number of current, regular cigarette smokers in appendix table 13 would be raised 4.3 percent, from 24,667,000 to 25,722,000. Distributions of the total number of discontinued regular smokers without reference to the time of discontinuance are carried in appendix tables 13, 20, and 22.

There is another effect which compensates partially for the systematic overstatement of discontinuances mentioned above. From comparison of the results on cigarette smoking in appendix table 12 for self-respondents and for persons for whom the information was supplied by others, it appears that some discontinued smokers in the latter group must have been reported as nonsmokers. When the data for self-respondents are analyzed separately, the age pattern of discontinuance rates follows closely that reported for the total sample although the former are 6–8 percent higher. In view of the compensatory nature of the biases, the data on discontinuance rates based on the total sample are probably more accurate approximations than those relating to self-respondents alone. The former have been used exclusively in this report.

A separate tabulation for cigarette smokers only was prepared according to age at which regular smoking was stopped, by single years to age 60. From the corollary information on age at which smoking was started, the population at risk of discontinuance can be defined, thus permitting age-specific discontinuance rates for cigarette smokers to be computed for each cohort. The detail of rates for single

Table 11. Age-specific discontinuance[1] rates per 100 regular cigarette smokers for 5-year intervals ($5q_x$) from age 15 to 60, by sex

Period (years) between two exact ages stated	Age (years)[2]					
	18–24	25–34	35–44	45–54	55–64	65 and over
	Males					
15–20	2.2	0.8	0.7	0.6	0.8	0.5
20–25	10.5	2.2	.8	.8	.8	1.3
25–30	------	7.4	1.4	1.0	1.2	1.6
30–35	------	11.4	3.6	1.2	1.5	2.4
35–40	------	------	7.0	2.6	2.2	1.4
40–45	------	------	9.7	3.8	2.1	2.1
45–50	------	------	------	7.1	2.4	2.3
50–55	------	------	------	7.0	5.6	2.1
55–60	------	------	------	------	9.8	3.0
	Females					
15–20	5.5	0.9	1.2	------	[3]	[3]
20–25	11.6	5.0	1.3	[3]	------	------
25–30	------	6.2	1.0	1.8	------	------
30–35	------	7.5	2.5	1.2	------	------
35–40	------	------	3.5	1.5	------	------
40–45	------	------	5.9	4.3	------	------
45–50	------	------	------	4.3	------	------
50–55	------	------	------	6.2	------	------
55–60	------	------	------	------	------	------

[1] Includes reversion to occasional smoking as well as abstinence.
[2] As of date of survey.
[3] Experience too limited to warrant presentation.

years of age has been consolidated in table 11, covering 5-year age intervals. In actuarial notation the rates shown are substantially equivalent to $5q_x$—the risk that regular smokers attaining age x will discontinue before reaching age $x+5$.

Review of table 11 reveals that the rates for the older age intervals covering experience in the years immediately preceding 1955, which are to the left of the diagonal line, are higher and depart sharply from the more uniform progression toward higher rates observed in the other age intervals. This reflects the effect of overreporting of recent discontinuances, already mentioned, as well as a possible upturn in discontinuance rates in the years immediately preceding the survey. Because of the difficulties in interpreting the responses, the latter possibility should not be regarded as an established fact. The rates to the right of the diagonal line should be free of this distortion in the data, and it is interesting to note that within a specific age interval those discontinuance rates for the several cohorts have shown little tendency to change. This stability suggests that discontinuance of regular cigarette smoking may be governed more by biological factors than by shifts in cultural patterns.

The rates as expressed in table 11 take into account the effective number at risk of discontinuance and hold constant the length of exposure to risk of discontinuance as a factor influencing the number of discontinuances. Because of the sex differences for duration of the smoking habit, these rates are particularly valuable for sex comparisons of discontinuance. The sex differences in rates are very small for the age- and time-intervals reported in table 11. When these results are compared with the observed proportion of discontinuances as reported in tables 14 and 15, it is obvious that much of the sex differences in discontinuance of cigarettes for all ages combined in table 14

Table 12. Approximate annual discontinuance (abstention) rates per 100 regular, "pure" cigarette, cigar, and pipe smokers 18 years of age and over, for 4 time intervals

Time (in years) since discontinuance	Cigarettes		Cigars (males)	Pipe (males)
	Males	Females		
Within past 1½	2.5	2.7	2.4	1.8
1½ to 2½	1.5	1.2	1.2	1.6
2½ to 4½	.7	.8	.5	1.0
4½ to 9½	.4	.5	.7	.6

Table 13. Approximate annual discontinuance (abstention) rates per 100 regular, "pure," male cigarette smokers for 4 time intervals, by age

Time (in years) since discontinuance	Age (years)				
	25–34	35–44	45–54	55–64	65 and over
Within past 1½	2.7	2.3	2.1	2.6	3.4
1½ to 2½	1.5	1.4	1.3	2.3	3.3
2½ to 4½	.8	.6	.9	.9	1.1
4½ to 9½	.3	.5	.5	.7	.9

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and for the four oldest age groups in table 15 must be due to the shorter exposure of women to the smoking habit.

Increase in Reported Discontinuances

The extent of the overstatement of discontinuances may be gauged by taking into account the length of time discontinuance has been maintained, for which some information is supplied in appendix tables 9–11. Appendix table 7 also carries data on "interrupted" smokers, defined as current smokers whose reported duration of smoking was two or more years less than the interval between current age and age at which smoking was started. The figures on "interrupted" smokers are subject to considerable discount since there is some question concerning the accuracy and consistency with which the data on age started smoking and duration of smoking were reported. Even so, it appears that some persons must have resumed regular smoking even after a break of a year or longer.

Table 12 presents approximate annual discontinuance rates for "pure" cigarette, cigar, and pipe smokers (to eliminate the effect of switching to other forms) for intervals within the past 10 years, which emphasize the higher reported rates for the 1953–54 period. The data for male cigarette smokers are further subdivided by age in table 13. Inspection of these results reinforces the conclusions on overreporting of recent discontinuances drawn from table 11. Substantially the same pattern appears, however, for each form of tobacco and for each age group. Excluding discontinuances within the past 1½ years would lower the overall discontinuance rate by one-fourth and one-third for men and women cigarette smokers, respectively, the effect of such an exclusion being somewhat greater at the younger ages.

The results indicate that, although the number of permanent discontinuances may be overestimated, (*a*) the data on total discontinuances are still useful for comparative purposes, and that (*b*) inferences on the age and sex profile for discontinuance rates do not differ materially when based on total discontinuances or on the fraction excluding the more recent discontinuances. This is fortunate since data on length of discontinuance could not be incorporated in the great majority of the appendix tables.

Influence of Smoking Rate

The proportion of regular cigarette, cigar, and pipe smokers, classified by rate of use, who have discontinued smoking completely appears in table 14. For cigarette smokers of each sex, discontinuances for light and very heavy smokers are greater than for moderate smokers. The proportion of discontinuances for cigars appears to increase according to maximum rate of use, but no trend by rate of rise is evident for pipes. Table 15 shows that the higher dis-

Table 14. Percentage of all regular cigarette, cigar, and pipe smokers 18 years of age and over who now abstain from smoking, by maximum rate of use attained

Form of tobacco smoked	Maximum daily rate			
	Light	Moderate	Heavy	Very heavy
Cigarette:				
Male	22.5	14.2	12.1	23.0
Female	15.9	8.1	7.9	17.5
Cigar (male)	22.7	34.8	47.3	51.6
Pipe (male)	42.8	41.0	48.0	48.2

Light = Less than 10 cigarettes, 1–2 cigars, less than 5 pipefuls.
Moderate = 10–20 cigarettes, 3–4 cigars, 5–9 pipefuls.
Heavy = 21–40 cigarettes, 5–8 cigars, 10–19 pipefuls.
Very heavy = More than 40 cigarettes, more than 8 cigars, more than 19 pipefuls.

Table 15. Percentage of all regular cigarette smokers who now abstain from smoking, according to maximum rate of use attained, by age and sex

Age (years)[1]	Maximum daily rate (cigarettes)					
	Male			Female		
	<10	10–20	21–40	<10	10–20	21–40
	Percent abstainers					
18–24 (1)	9.3	4.0	3.0	12.5	5.1	5.3
25–34 (2)	15.1	11.4	8.3	16.7	9.3	8.3
35–44 (3)	22.1	12.3	11.6	14.6	7.9	7.7
45–54 (4)	24.6	14.6	12.6	16.4	7.5	7.8
55–64 (5)	30.6	23.2	18.3	19.5	10.0	12.6
65 and over (6)	36.9	29.7	29.3	28.2	13.9	9.5
	First order difference					
(3)−(2)	+7.0	+0.9	+3.3	−2.1	−1.4	−0.6
(4)−(3)	+2.5	+2.3	+1.0	+1.8	−.4	+.1
(5)−(4)	+6.0	+8.6	+5.7	+3.1	+2.5	+4.8
(6)−(5)	+6.3	+6.5	+11.0	+8.7	+3.9	−3.1

[1] As of date of survey.

continuance rates noted for light cigarette smokers of both sexes persist within each age group; data on those smoking more than two packs of cigarettes daily are not shown separately because of the small numbers involved.

The age-specific proportions of discontinued smokers which can be derived from appendix tables 13 and 22 convey no direct information on age at discontinuance. Since the percentages are cumulative to the survey date, a rough notion on trends by age intervals may be secured by examining the first-order differences. The reasoning would be analogous to that employed with persistence rates in table 9. This approach would open the way to study the influence of other variables—maximum amount smoked, place of residence and so on—on discontinuance rates. Inspection of the first-order differences in table 15 according to maximum amount smoked shows much the same pattern as the results in table 9 on cutbacks from a maximum smoking rate. Men with a daily maximum of less than 10 cigarettes tend to have a more uniform number of discontinuances in each age interval, which changes among smokers of more than one pack of cigarettes daily to an accelerating dropout at the older ages. No consistent pattern emerges for women.

Influence of Age Started Smoking

The percentage of discontinued smokers does not vary greatly by age at which smoking was started. Only in the numerically unimportant group of men who start to smoke cigarettes after age 22 does the risk of discontinuance rise markedly (see table 16 and appendix table 24).

Influence on Comparisons of Groups

For cigarettes in particular, the discontinuance rates fall into consistent age patterns, show some stability over a variety of population groups, and do not seem inordinately large. As a rule, it does not appear necessary to qualify group comparisons based either on past or current smoking histories, by taking into account the effect of discontinuance on duration of exposure to the smoking habit. Exposure time is more strongly influenced by age at which smoking was started and is automatically taken care of when the latter factor is controlled in comparisons. Control of date or age at which smoking started is most important in handling sex comparisons.

Table 16. Percentage of all regular "pure" cigarette smokers 18 years of age and over who now abstain, according to age started smoking, by sex

Age started smoking (years)	Male	Female
Under 16	10.9	10.1
16–17	9.8	11.8
18–19	10.8	11.4
20–21	12.1	9.2
22 and over	18.2	11.2

The regularity of the rates for discontinuing smoking may also imply that the smoking survey data will not be quickly outdated. Unless profound changes in public acceptance of smoking habits occur, one may be able to take the survey information on proportion of smokers, discontinuance rates, persistence at maximum rate, and so on, and, for cohorts born prior to 1930 project fairly accurate estimates on the distribution of smokers at future anniversaries. If smoking histories continue to be the subject of further surveys, the collation of future survey findings on some of the cohorts with projections from the present survey might be attempted to check this point.

Urban and Rural Residence

Aside from the differences associated with age and sex, urban and rural residence is the population characteristic which differentiates smoking habits most sharply. Data for urban, rural nonfarm, and farm populations are presented in appendix tables 2, 4, 13–15, and 23; an urban-rural dichotomy only is found in appendix tables 8, 9, 11, and 21. The tabulation programs had to be planned in advance and full recognition of the important distinction between the nonfarm and farm populations came too late to permit this detail to be carried in all tables. However, the essential features of this relationship are indicated.

Lifetime History

The percentage of nonsmokers diminishes with the impact of urban life. Rural nonfarm persons tend to resemble the urban dwellers in smoking habits, and the sharp demarcation, for women particularly, appears between the nonfarm and farm population. Figure 11 shows these gradients persisting at all ages for both sexes. Smoking is a custom well-entrenched among men, and changes in the urban-rural gradients over a succession of cohorts are less pronounced for men than for women. According to figure 11, most women over 65 years of age in the three population subdivisions were nonsmokers. The reduction in the proportion of nonsmokers with each younger cohort is greater for urban and nonfarm

Figure 11. Percentage of persons who have never smoked, urban, rural nonfarm, and rural farm population, by sex and age.

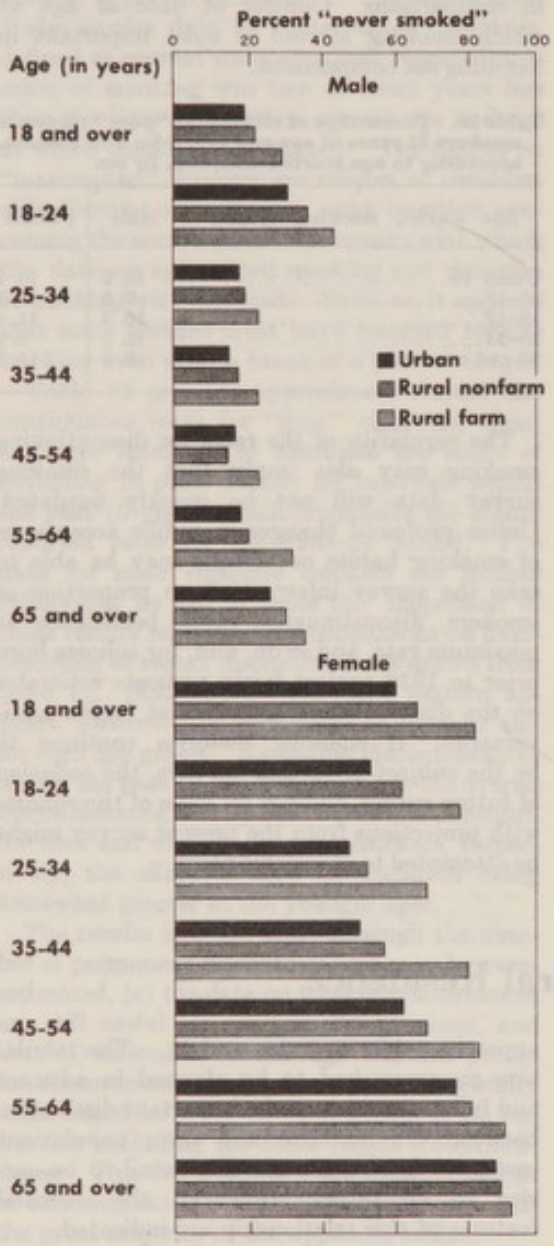
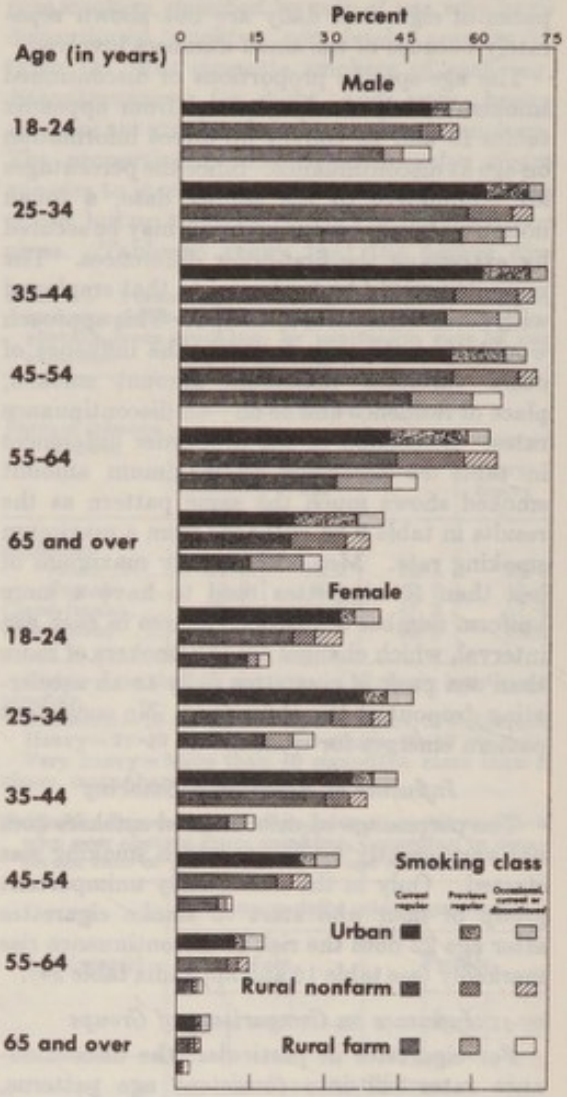


Figure 12. Current cigarette smoking status for urban, rural nonfarm, and rural farm population, by sex and age.



women, thus widening the gap between these groups and farm women. Not until the data on farm women as young as 25–34 years are studied, does a major decrease in the number of nonsmokers appear.

The relationships for cigarette smokers, set forth in figure 12, are corollaries of those observed for nonsmokers. The results for cigarettes do not differ materially whether one considers current regular smokers, all regular smokers (lifetime history), or regular and occasional smokers combined. Attention might be directed to the higher percentage of occasional smokers among urban women of all ages, a feature not duplicated among men. The data for women may be a more sensitive index of cultural diffusion. They imply that in an urban setting, cigarette smoking, regularly or occasionally, has been increasingly accepted by women born after 1890. The habit was acquired less rapidly on the farm and a sizable acceptance was delayed until women born after 1920 were reached, almost one generation later than smoking's acceptance in the city.

Urban-rural differences in smoking habits have been noted in other surveys. The *Fortune* survey in 1935 (*15*) reported a sizable excess of cigarette smokers in the urban population, particularly for women. Nor has this phenomenon been confined to the United States. A Danish survey in 1948 (*18*) also observed large urban-rural differentials in smoking patterns for both sexes. Some of the retrospective studies on the association of smoking and lung cancer have contained estimates of an excess of smokers in urban areas. Doll and Hill (*19*) provided some rather detailed information on this point for England and Wales.

Appendix tables 3 and 14 indicate that within the urban area there are practically no differentials by size of community, extending to urban fringe groups, in the proportion of smokers for both sexes, including cigarette smokers. Little trace of a gradient appears

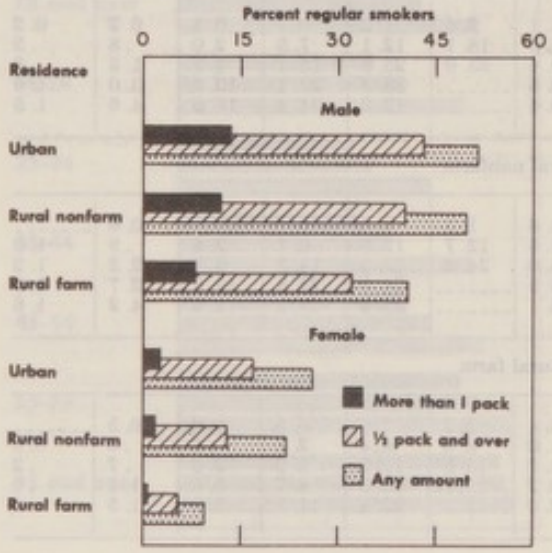
Table 17. Cumulative percentage of persons becoming regular cigarette smokers prior to age specified, by sex and age, for urban, rural nonfarm, and rural farm population

Started smoking prior to age—	Sex and age (years)											
	Males						Females					
	18–24	25–34	35–44	45–54	55–64	65+	18–24	25–34	35–44	45–54	55–64	65+
	Urban											
15	6.9	6.4	7.1	6.5	6.5	5.1	2.6	1.2	0.7	0.3	0.2	0.2
18	34.9	35.8	31.1	26.7	22.7	14.5	18.1	12.1	7.8	2.0	.8	.2
20	51.0	56.2	49.8	42.7	35.6	19.4	33.0	25.3	16.1	4.9	1.3	.6
25	-------	68.1	64.8	58.9	50.3	26.6	-------	38.9	29.1	12.0	3.0	1.0
30	-------	68.9	67.3	62.1	53.4	29.4	-------	42.2	34.4	16.6	4.6	1.5
	Rural nonfarm											
15	6.5	6.6	7.1	7.6	7.7	4.6	1.3	2.0	0.4	0.5	0.6	-------
18	33.1	36.8	32.4	32.2	25.9	14.6	12.7	12.6	6.7	2.6	.9	1.0
20	49.9	54.9	50.0	48.5	40.1	19.0	24.2	23.3	14.3	5.7	2.2	1.2
25	-------	65.7	63.6	62.6	50.7	25.9	-------	34.8	23.3	11.5	2.7	1.5
30	-------	66.4	66.2	65.2	52.9	28.1	-------	39.4	29.5	14.6	4.2	1.6
	Rural farm											
15	4.2	7.5	8.2	6.2	4.1	3.4	1.2	2.0	0.5	0.2	0.5	-------
18	29.5	30.9	35.0	26.4	15.9	11.0	8.8	8.4	2.7	.8	.5	0.2
20	39.6	51.7	52.1	38.8	26.3	14.6	13.8	15.2	5.0	2.0	.7	.2
25	-------	63.5	61.5	52.2	36.2	19.2	-------	20.3	8.7	3.5	1.5	.6
30	-------	64.4	62.5	54.6	39.1	22.0	-------	22.4	11.3	5.0	1.5	.6

even when unpublished data for persons over 45 years of age are examined separately. This finding is at variance with the earlier *Fortune* survey, which reported some systematic differences as of 1935 for men and women, by size of urban community. However, differences which existed 20 years ago could well have been diluted by the recent outward migration of population from central cities to the surburbs. This, along with the narrowing differences in smoking patterns of the younger urban and nonfarm cohorts, suggests that the events of the past 20 years have disposed of community size as an important determinant in shaping smoking habits.

Viewing figure 12 from another perspective, it may be observed that the proportion of discontinued smokers, as related to the total of all regular cigarette smokers, is about the same for each of the three residence categories (urban, rural nonfarm, and farm). The age patterns for discontinuance rates in the three population groups do not appear to depart greatly from the total national experience for men and women as discussed in connection with table 15, and the discussion is not repeated here.

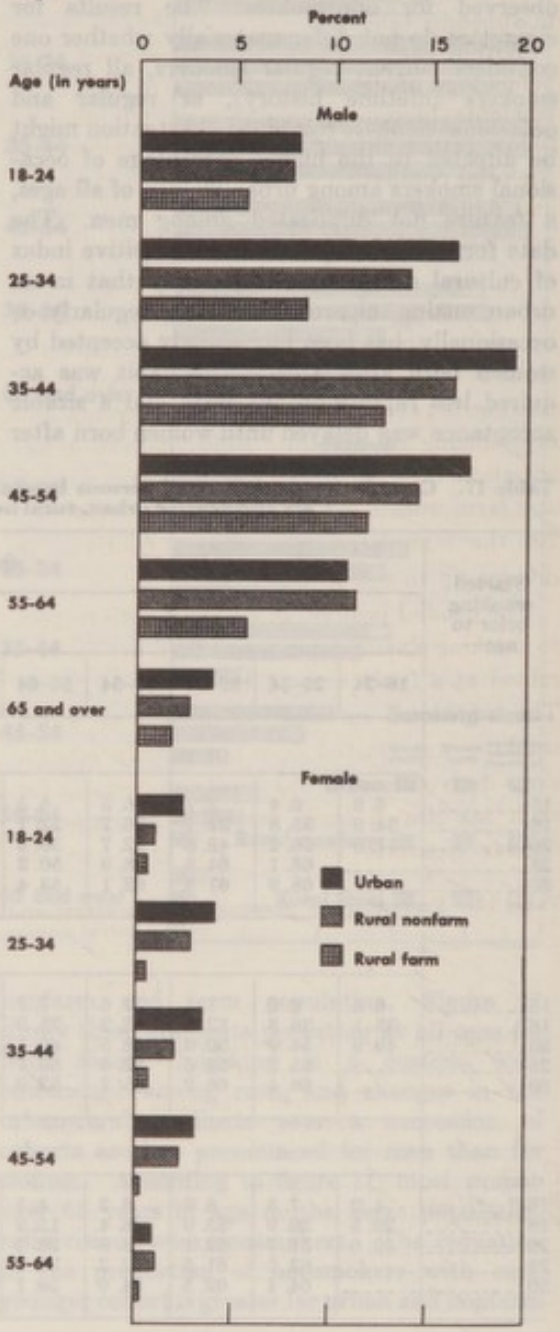
Figure 13. Percentage of persons 18 years of age and over currently smoking cigarettes regularly, by sex, with additional detail on current daily rate, for urban, rural nonfarm, and rural farm population.



Age Started Smoking

Table 17 presents the cumulative percentage of persons who had started to smoke cigarettes

Figure 14. Percentage of persons currently smoking more than one pack of cigarettes daily, for urban, rural nonfarm, and farm population, by sex and age.



regularly prior to the beginning of age x, at selected ages up to age 30, for urban, rural nonfarm, and farm populations. The technical comments, which accompanied figure 4 on this subject in the previous section, apply to table 17 as well. Additional data on age at which smoking of any form of tobacco was started by men residing in urban and rural areas are given in appendix table 8.

Within each sex-age group, the cumulative percentage of urban, rural nonfarm, and farm residents becoming regular cigarette smokers follow rather closely the curves descriptive of the total population shown in figure 4 (females) and figure 5 (males). The farm and nonfarm populations do not acquire the smoking habit at a later age than the urban population. Among males, the proportion of regular smokers by age 15 continues to be nearly equal for the three residence groups, the differentiation appearing between the 15th and 20th years and crystallizing by age 20. Although the differentiation between urban and nonfarm women and farm women is also well established by age 20, the cumulative percentages continue to diverge up to age 30.

The general position of a cohort with respect to smoking classification seems then to be determined at a fairly early date, by age 18 or 20. The additional information on age at which smoking was started does not modify conclusions derived from urban-rural comparisons for each sex drawn from overall percentages of regular cigarette smokers, and this variable would normally not have to be controlled for such comparisons.

Heavy Cigarette Smokers

Figure 13 incorporates detail on the present rate of consumption in comparisons of the proportion of current cigarette smokers in urban, rural nonfarm and farm populations. For males particularly, the differentiation among the three groups is sharpened when the proportion of current heavy and moderate smokers is the criterion employed. These data on current cigarette smokers are supplemented in figure 14 with age detail on the proportion currently smoking more than one pack of cigarettes daily. In all age groups of both sexes except persons 55–64 years of age, a systematic gradient from urban to rural farm appears. No clear trend toward narrowing urban-rural differentials for heavy smokers, especially between farm and nonfarm populations, has developed among the younger cohorts of either sex. Data for the three residence groups agree that the highest proportion of males currently smoking more than one pack of cigarettes daily is to be found in the 35–44 age bracket.

The urban and rural populations are also classified in appendix table 23 with respect to the maximum regular rate of cigarette smoking ever attained. On the whole, the urban, nonfarm and farm population contrasts with respect to amount smoked, by age and sex, obtained from the maximum rate data of appendix table 23, would lead to substantially the same conclusions to be gained from inspection

Table 18. Percentage of persons smoking cigars and pipes regularly (current status and lifetime history) for urban, rural nonfarm, and rural farm population

Population	Cigars				Pipes			
	18 years and over		65 years and over		18 years and over		65 years and over	
	Current status	Lifetime history	Current status	Lifetime history	Current status	Lifetime history	Current status	Lifetime history
Urban	6.1	10.3	10.6	21.2	6.7	14.1	15.9	27.4
Rural nonfarm	4.5	8.2	6.2	14.9	7.9	16.7	18.1	34.0
Rural farm	2.9	5.7	4.4	8.6	9.7	17.8	17.9	30.3

of figure 14. Only one modification is indicated. Classification by maximum rate yields smaller urban-nonfarm differences for heavy cigarette smokers than does classification by current consumption status for males between the ages of 35 and 65. This discrepancy would imply that urban males using more than one pack of cigarettes daily tend to persist at this maximum rate without cutback for longer periods than nonfarm males. When farm and nonfarm populations are combined for comparison with the urban population, both classification methods agree on the extent of urban-rural differences in the prevalence of heavy cigarette smoking.

Cigar and Pipe Smokers

The basic information on urban-rural differences for cigar and pipe smoking is entered in appendix table 13. The brief summary in text table 18 reveals opposite gradients for these two forms of smoking. Cigar smoking is and has been a custom more frequently pursued in urban areas. Although the differential for pipe smoking is smaller, this form of smoking is somewhat more common among nonfarm and farm men. Adjustment for age would not alter these statements. The data for persons over 65 years of age show that the relationship noted for all ages holds true for the older population as well.

Geographic Region

The size of the sample surveyed permits presentation of some results for four major regions of the country (appendix tables 3, 4, 14, and 15). The States comprising each region are shown in figure 1.

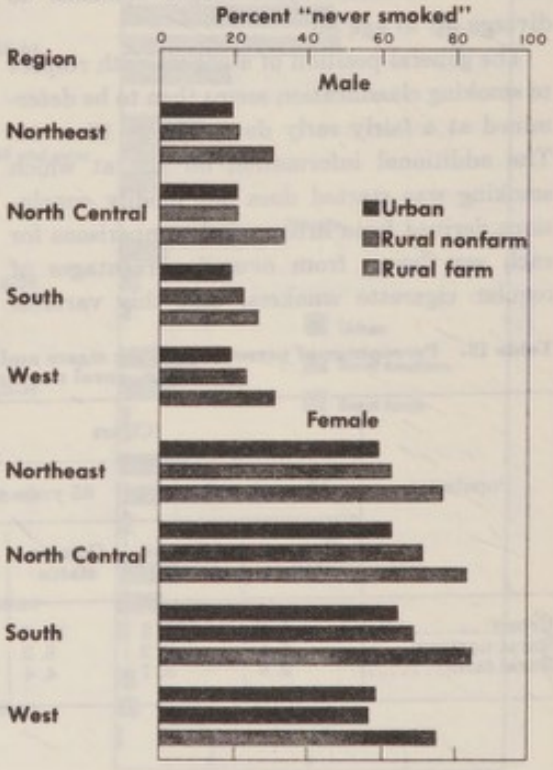
Lifetime History

The proportion of nonsmokers among persons 18 years of age and over by region for urban, rural nonfarm, and farm populations is presented separately in figure 15. The urban-rural gradients in nonsmokers of each sex observed nationally are repeated within each region. For men, the overall regional variation in nonsmokers is small, ranging from 19.9 percent in the Northeast to 22.8 percent in the North Central States. The lower figure for the Northeast is influenced partly by its large proportion of urban residents. Within each of the three residence components the regional agreement on nonsmokers is good for males and standardization for place of residence would further minimize the observed regional variation.

The proportion of female nonsmokers is lower in the Northeastern and Western regions than in the North Central and Southern regions.

The regional data pertaining to persons 45 years of age and over has been summarized in table 19, in the absence of age detail for regions in the appendix tables. Many of the comments for nonsmokers 18 years of age and over apply to the older group also. The irregularities noted in some of the latter results might easily be

Figure 15. Percentage of persons 18 years of age and over who have never smoked, for urban, rural nonfarm, and rural farm population, by sex and geographic region.



attributed to the smaller number of persons sampled in the older age group.

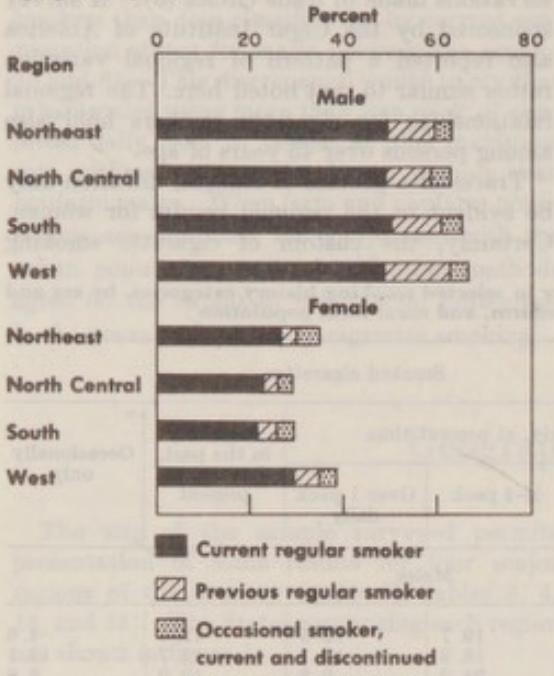
Similar regional relationships appear in the data on cigarette smokers in figure 16. The regional variation for male cigarette smokers appears minimal using any one of three criteria—current regular smokers, regular smokers (lifetime history), or all regular and occasional smokers combined. On the other hand, each of the measures suggests that cigarette smoking is more prevalent among women in Northeastern and Western States than elsewhere in the country. This latter finding agrees with observations made in trade circles (*3*). A survey sponsored by the Cigar Institute of America also reported a pattern of regional variation rather similar to that noted here. The regional relationships for cigarette smokers hold also among persons over 45 years of age.

Traces of a process of cultural diffusion may be evident in the regional results for women Certainly, the custom of cigarette smoking

Table 19. Percentage of persons 45 years of age and over in selected smoking history categories, by sex and geographic region, for urban, rural nonfarm, and rural farm population

Geographic region and residence	Never smoked in any form	Smoked cigarettes				
		Regularly, at present time			Regularly in the past, but not at present	Occasionally only
		Any amount	½–1 pack	Over 1 pack daily		
	Males					
Northeast	20.6	39.9	19.7	12.5	12.4	4.6
Urban	20.1	39.7	18.9	13.1	12.7	13.1
Rural nonfarm	22.0	42.0	24.2	9.8	10.9	9.8
Rural farm	26.7	36.3	17.9	11.6	10.0	11.6
North Central	23.4	38.8	21.8	8.9	11.3	4.9
Urban	20.7	42.8	23.9	10.1	11.5	4.7
Rural nonfarm	20.7	36.7	21.8	9.3	13.4	6.3
Rural farm	34.6	27.3	15.2	4.8	8.8	4.2
South	23.0	41.8	23.3	10.0	13.0	5.2
Urban	20.8	44.7	24.8	11.1	12.5	5.5
Rural nonfarm	22.3	44.6	25.5	10.9	12.7	4.7
Rural farm	28.2	33.1	18.2	6.8	14.2	5.4
West	18.6	45.2	25.3	10.5	15.6	4.7
Urban	16.8	45.6	25.8	11.0	16.2	4.5
Rural nonfarm	22.1	44.9	25.1	8.0	14.6	5.8
Rural farm	25.7	41.5	21.2	12.9	12.4	4.1
	Females					
Northeast	73.5	15.3	6.9	2.6	2.8	3.9
Urban	73.0	15.3	6.6	2.6	2.9	4.2
Rural nonfarm	74.1	16.5	9.0	3.0	2.2	2.9
Rural farm	83.0	7.9	4.4	1.3	1.3	1.3
North Central	81.0	11.4	5.9	1.0	1.4	2.5
Urban	78.5	13.8	7.0	1.1	1.3	2.8
Rural nonfarm	83.7	8.9	4.8	1.0	2.3	2.7
Rural farm	88.4	4.4	2.4	.2	.5	1.4
South	81.0	9.9	5.2	1.0	2.5	2.9
Urban	76.6	12.5	6.4	1.2	3.1	3.5
Rural nonfarm	82.1	9.4	5.6	1.1	2.1	3.2
Rural farm	91.7	3.5	1.5	.4	1.5	.9
West	74.5	17.3	9.8	.9	2.4	3.8
Urban	72.9	17.9	10.4	1.1	2.3	4.5
Rural nonfarm	77.0	17.7	8.7	.6	2.2	1.6
Rural farm	85.2	9.4	5.9	----	3.9	1.0

Figure 16. Current cigarette smoking status for persons 18 years of age and over, by sex and geographic region.



spread more rapidly among women in the industrialized Northeast than in the farm economy of the Midwest and South. Selection factors associated with migration may have played a role in the higher Western rates. Population growth through immigration has been greatest in that part of the country and there may be an association between a willingness to move and to adopt new customs. Unfortunately, separate data on the smoking habits of migrants are not available to test this point.

Age Started Smoking

Regional information on ages at which men and women started smoking any form of tobacco is reported in appendix table 25. When the data are subdivided by age as well, the sampling errors increase and too much importance should not be attached to any single result. The general impression gained from inspection of appendix table 25 is that for males no great regional variation exists in age at which smoking is started. Men might start smoking at a slightly earlier age in the Northeastern and Southern States, and over the years women in the Northeast may have started smoking at earlier ages than women elsewhere. However, the differences which may exist are small and do not warrant the adjustment of regional comparisons to take account of differences in duration of exposure to smoking.

Heavy Cigarette Smokers

Figure 17 carries regional information on current cigarette smokers for each sex, incorporating detail on present daily rate of consumption. The corresponding data based on maximum consumption rate ever attained are not available. As observed in the discussion of the influence of urban-rural residence, the proportion of heavy smokers, those smoking more than one pack of cigarettes daily, is the item which discriminates best among males. Although the proportion of all male cigarette smokers is almost constant from region to region, the Northeast appears to have an excess of heavy smokers. The latter finding probably reflects the true situation rather than an unusual result from the sample interviewed. The forces which influence the regional distribution of all women cigarette smokers may operate on

Figure 17. Percentage of persons 18 years of age and over currently smoking cigarettes regularly, by sex and geographic region, with additional detail on current daily rate.

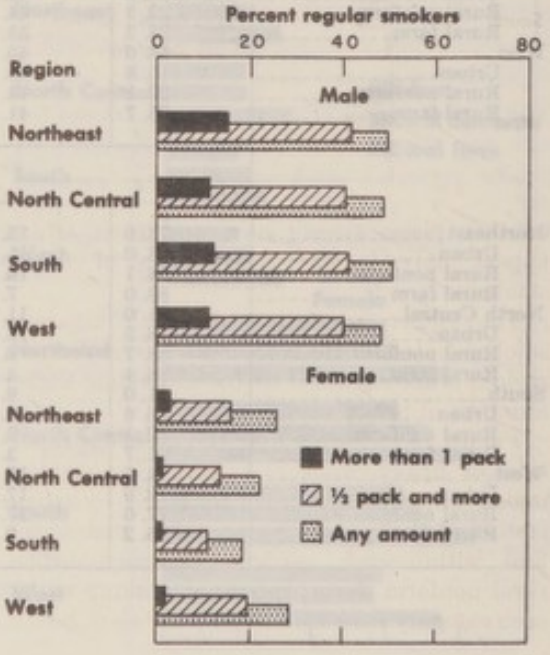
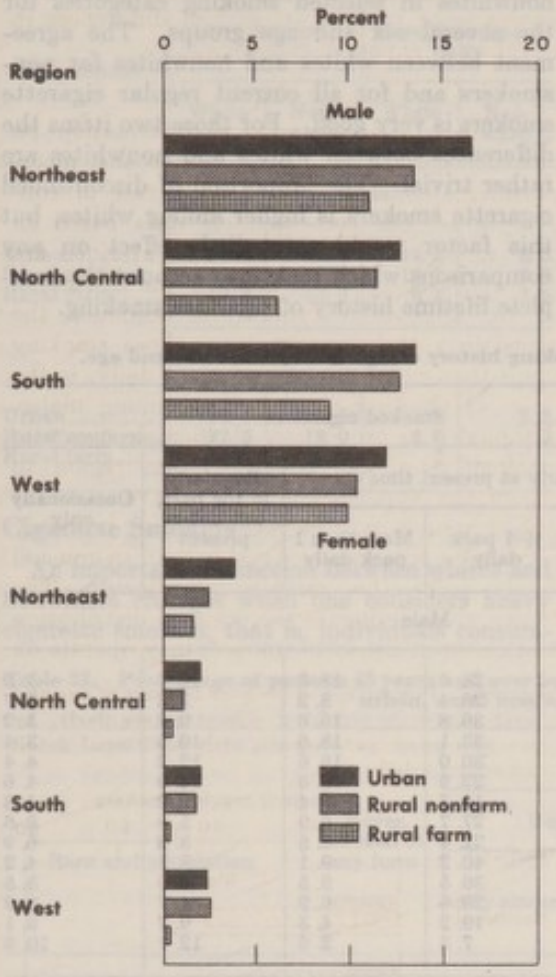


Figure 18. Percentage of persons 18 years of age and over currently smoking more than one pack of cigarettes daily, for urban, rural nonfarm, and rural farm population, by sex and geographic region.



another plane among men and their effects appear only in the distribution of heavy smokers.

The higher proportion of female cigarette smokers noted for the Northeastern and Western regions is accompanied by corresponding higher numbers of heavy smokers.

Figure 18 indicates that the greater proportion of heavy smokers among Northeastern males cannot be accounted for solely by urbanization of that area. Northeastern males rank first within each residence category—urban, rural nonfarm, farm; the usual ranking within each category runs as follows: Northeast, South, North Central, West. The low proportion of heavy smokers in the male farm population of the North Central States provides one exception. The higher farm figure for the West is another irregularity. Although this last result could be a sampling artifact, it may reflect a true difference in smoking pattern. The character of agriculture in the West, with its emphasis on specialized crops in California, requiring a large migrant labor force, and on livestock grazing elsewhere, differs from family farm operations in other parts of the country, and this difference may well influence smoking practices.

Among women, the proportion of heavy smokers in the Northeast is consistently higher for each residence component. The higher rate for women on Northeastern farms underscores the fact that cigarette smoking has gained its greatest acceptance in that portion of the country. The sex differences in regional pattern which appear for all regular cigarette smokers turn up for heavy smokers also. Although Southern males as a class rank second in heavy cigarette smoking to Northeastern males, the proportion of heavy smokers among Southern women is decidedly lower than in the Northeast or West and instead is on a par with experience reported from the North Central region.

Many of the comments concerning regional data for heavy cigarette smokers of both sexes apply as well to the population over 45 years of age (table 19).

Cigar and Pipe Smokers

Data on current and previous regular cigar and pipe smokers are given in table 20. Cigar smoking is more common in the Northeast. Pipe smoking appears to be most prevalent in the North Central States although in this respect there is little difference between the Northeastern and North Central States.

Table 20. Percentage of persons 18 years of age and over smoking cigars and pipe regularly (current status and lifetime history), by geographic region

Geographic region	Cigars		Pipe	
	Current status	Lifetime history	Current status	Lifetime history
Northeast	7.1	10.7	8.0	14.9
North Central	4.8	8.2	8.3	16.0
South	4.8	7.2	6.2	11.8
West	3.9	8.8	6.5	15.4

Race

Nonwhites, mostly Negroes, comprise approximately 10 percent of the population of the United States and are proportionately represented in the sample surveyed. Data for whites and nonwhites are incorporated in appendix tables 4 and 15. Because of the relatively small number of nonwhites, age detail had to be sacrificed in examining the nonwhite experience by region and place of residence.

Table 21 shows the proportion of whites and nonwhites in selected smoking categories for the several sex and age groups. The agreement between whites and nonwhites for nonsmokers and for all current regular cigarette smokers is very good. For those two items the differences between whites and nonwhites are rather trivial. The proportion of discontinued cigarette smokers is higher among whites, but this factor would exert little effect on any comparisons which took into account the complete lifetime history of cigarette smoking.

Table 21. Percentage of persons in selected smoking history categories, by sex, race, and age.

Race and age (years)	Never smoked in any form	Smoked cigarettes				
		Regularly at present time			Regularly in the past, but not at present	Occasionally only
		Any amount	½–1 pack daily	More than 1 pack daily		
	Male					
White	21.5	49.9	28.6	13.3	10.7	4.0
18–24	31.8	49.0	29.4	8.2	3.3	4.7
25–34	19.0	59.9	36.8	15.6	9.2	3.2
35–44	16.9	59.1	33.1	18.6	10.9	3.4
45–54	17.3	54.1	30.0	16.6	12.2	4.4
55–64	21.2	41.5	22.9	10.6	14.9	4.6
65 and over	29.3	21.6	10.7	3.4	12.2	4.8
Nonwhite	22.8	48.4	27.7	6.9	5.8	6.6
18–24	38.0	42.6	22.4	4.5	3.2	6.9
25–34	18.8	62.1	40.2	9.1	3.1	4.2
35–44	19.6	52.7	30.5	9.5	6.9	5.5
45–54	18.2	51.2	28.6	6.9	4.7	6.9
55–64	20.1	38.7	19.2	4.3	9.7	9.1
65 and over	27.8	19.9	7.3	2.6	12.1	10.6
	Female					
White	65.2	23.6	13.1	2.4	3.7	3.9
18–24	58.7	28.8	14.9	2.0	3.2	5.0
25–34	51.5	34.2	20.2	3.7	5.8	4.7
35–44	56.0	32.1	18.7	3.1	4.6	4.0
45–54	66.5	22.4	12.1	3.0	3.3	4.5
55–64	80.7	10.6	4.9	1.2	2.2	3.1
65 and over	90.2	3.5	1.4	.2	1.2	1.5
Nonwhite	62.8	22.9	9.4	1.7	2.5	5.7
18–24	59.9	25.7	10.4	1.4	3.4	5.0
25–34	49.5	35.6	14.9	2.4	2.3	7.5
35–44	58.0	24.4	9.8	2.6	3.2	7.4
45–54	71.4	18.0	7.1	1.2	1.8	4.1
55–64	79.8	10.0	4.4	.4	1.2	4.4
65 and over	82.5	2.1	1.5	----	1.9	2.9

Table 22. Percentage of persons 18 years of age and over currently smoking cigarettes regularly, any amount and more than 1 pack daily, by sex and race for urban, rural nonfarm, and rural farm population

Population	Any amount		More than 1 pack daily	
	White	Nonwhite	White	Nonwhite
	Male			
Urban	51.5	51.8	14.6	8.3
Rural nonfarm	50.5	37.2	12.7	3.8
Rural farm	40.8	43.2	8.5	3.5
	Female			
Urban	26.4	25.5	2.8	2.2
Rural nonfarm	22.5	18.0	2.0	.3
Rural farm	9.2	12.3	.7	----

Cigarette Smokers

An important distinction between whites and nonwhites emerges when one considers heavy cigarette smokers, that is, individuals consuming more than one pack of cigarettes daily. The proportion for white males, 13.3, is almost double that for nonwhites, 6.9. There is a similar excess of heavy smokers among white females, 2.4 percent compared with 1.7 percent for nonwhite females. In their survey of Columbus, Ohio, Mills and Porter (*20*), using a different smoking history classification, also discovered an excess of heavy cigarette smokers among whites, in the presence of similar overall proportions of cigarette smokers among both races. Kirchoff and Rigdon (*22*) recently found a lower proportion of heavy cigarette smokers among nonwhites in Texas, but refrained from attaching any special significance to the observation. The fact that the excess of heavy cigarette smokers among whites has now turned up in three independent studies greatly strengthens the credence to be placed in the white-nonwhite differential reported here.

Since there is good agreement on nonsmokers and all regular cigarette smokers, a reason must be sought as to why fewer nonwhites proceed to build up to a peak consumption of more than one pack of cigarettes daily. Some sort of

Table 23. Percentage of persons 45 years and over in selected smoking history categories, by sex and race for urban, rural nonfarm, and rural farm population

Race and population	Never smoked in any form	Smoked cigarettes				
		Regularly at present time			Regularly in the past, but not at present	Occasionally only
		Any amount	½–1 pack	More than 1 pack daily		
	Male					
White:						
Urban	19.8	42.3	22.6	12.0	13.3	4.7
Rural nonfarm	21.6	42.9	24.7	10.4	13.1	4.3
Rural farm	31.7	31.8	17.6	7.1	12.0	4.2
Nonwhite:						
Urban	21.5	44.7	23.1	6.4	8.1	6.7
Rural nonfarm	23.6	28.2	16.7	1.6	8.5	13.1
Rural farm	16.5	29.6	12.7	3.0	6.7	12.0
	Female					
White:						
Urban	75.4	14.8	7.3	1.8	2.4	3.7
Rural nonfarm	80.4	12.0	6.8	1.6	2.3	2.6
Rural farm	89.9	4.3	2.4	.4	1.4	1.1
Nonwhite:						
Urban	75.8	12.7	5.5	.9	2.2	4.0
Rural nonfarm	75.3	10.1	3.5	----	----	5.6
Rural farm	83.3	8.1	2.7	----	----	.9

economic barrier is the most plausible explanation, fewer nonwhites being able to afford the expenditures entailed. Unfortunately, income data are not available at the present time, so the hypothesis cannot be directly put to the test here. The hypothesis may be supported indirectly by noting that only 5.7 percent of nonwhite males in the South smoke more than one pack of cigarettes daily compared with 8.3 percent elsewhere. The corresponding percentages for females are 0.7 and 2.8. Northern Negroes have greater disposable incomes than those in the South.

Among nonwhite women the regional differences on heavy cigarette smokers carry over to current smokers of any amount, the figure for the South, 16.7 percent, being slightly more than half that reported elsewhere, 30.9 percent. This disparity for all regular cigarette smokers is not so pronounced for nonwhite males, 46.1 percent in the South compared to 51.1 percent elsewhere.

Another interesting distinction appears in table 22, which contrasts the urban-rural gradients for whites and nonwhites. Among white males the rural nonfarm experience on all current and heavy smokers closely resembles that of the urban population. For nonwhites the reverse is true, the rural nonfarm figures tending to approach those of the farm population. A similar, but less pronounced, relationship appears for females. These relationships hold for persons over 45 years of age (table 23) as well as for the entire population 18 years of age and over. The probable explanation lies in the different character of the rural nonfarm population of the two races. The nonfarm whites appear to be engaged primarily in urban occupational pursuits. The occupation and industry data for the nonwhites suggest, on the other hand, that a sizable proportion of the nonfarm group work in the country, and hence have been less subjected to urban environmental influences.

Table 24. Percentage of persons 18 years of age and over smoking cigars and pipe regularly (current status and lifetime history), by race

Race	Cigars		Pipe	
	Current status	Lifetime history	Current status	Lifetime history
White	5.2	9.3	7.5	15.6
Nonwhite	6.2	8.6	5.8	10.3

Cigar and Pipe Smokers

Table 24 indicates that nonwhite males may smoke more cigars than white males although the difference is not statistically significant. Pipe smoking appears to be practiced more frequently by whites.

Employment Status, Occupation, and Industry

Employment Status

The classification of the population with respect to employment status and labor force participation, for which some results are given in appendix tables 5, 6, 16, and 17, provides some insight into the relationship of these variables with smoking patterns.

Lifetime History

The proportion of persons who had never smoked was somewhat lower among the unemployed. Among persons unemployed at the time of the survey, 17 percent of the men and 58 percent of the women had never been regular or occasional smokers any time during their lives. The corresponding percentages for employed men and women are 21 percent and 63 percent, respectively (table 25). The differences between the employed and unemployed in this respect are not striking. This is to be expected since the employment status classification, which relates to activity during the survey week, could not be directly connected with lifetime smoking patterns, particularly during a period of relatively full employment when joblessness typically is of a short duration.

However, there are small, but persistent, differences in the smoking rates for the employed and the unemployed which are not readily explained. The proportion of nonsmokers remains smaller among the unemployed even when differences in the age composition of each group are allowed for in the age-adjusted rates in table 25. (An age breakdown of occupation and industry data has not been incorporated in the appendix tables because age standardization does not significantly affect the comparisons.)

Similarly, the industry composition of the unemployed does not account for their higher smoking rates. There is a possibility of reporting error. Appendix table 12 gives evidence of some overstatement in negative smoking histories when the data were supplied by a respondent other than the individual himself. Unemployed persons may have been in a position to report for themselves more frequently in this survey than employed persons although this possibility could scarcely account for the total differential. The use of tobacco may have a small adverse selection effect among applicants for employment. Or some nonsmokers may tend to start smoking during a period of stress, such as unemployment, although at the present time, most of the unemployed tend to get jobs within a very short period.

The proportion of nonsmokers was greater among men and women not in the labor force during February 1955 than among those in the labor force. For men, there is no significant difference in the crude proportion of nonsmokers among those unable to work and those not in the labor force because they were retired, in school, or, for other reasons, were not working. Among women, there is a noticeable difference in the percentages of nonsmokers among housewives and among those not in the labor force because of inability to work and for other reasons; 69 percent of the housewives had been nonsmokers during their entire lives as compared with 86 percent of women who were unable to work and 79 percent of women not in the labor force for other reasons. As a factor influencing smoking rates, age adjustment produces a more important effect for men not in the labor force than for men working or looking for work. The age-adjusted proportion of nonsmokers among men unable to work rises slightly, but the figure for women in this same category drops from 86 to 74 percent. For men and women not in the labor force due to retirement, in school, and for other reasons, the adjustment revises the observed proportion downward but it produces little effect on the result for housewives.

Table 25. Percentage [1] of persons 18 years of age and over who have never smoked, by sex and employment status

Employment status	Male		Female	
	Crude	Age-adjusted [2]	Crude	Age-adjusted [2]
Employed	21.4	22.4	62.7	65.2
Unemployed	16.8	16.6	58.1	63.1
Not in labor force	33.6	30.0	70.2	68.6
Unable to work	32.6	34.2	86.1	74.3
Keeping house	------	------	69.4	68.1
Other reasons	33.7	26.0	79.3	73.5

[1] Based on persons reporting smoking pattern.
[2] Adjusted to population of persons 18 years of age and over enumerated in Current Population Survey for February 1955.

Current Smokers

If the analysis is restricted to the relationship between current employment status and current smoking practices, the differences among the employed, the unemployed, and persons not in the labor force are somewhat greater than those previously described. Data on the current consumption of cigarettes, cigars, and pipe tobacco by these groups are found in appendix table 17.

Among men, about 61 percent of the unemployed currently smoke cigarettes regularly as compared with 53 percent of the employed and only 28 percent of those not in the labor force. Although unemployed men tend to have higher cigarette smoking rates than employed men, the proportion of heavy cigarette smokers is about the same for each group. About 14 percent of the employed men and 13 percent of the unemployed men smoke more than one pack of cigarettes daily. In the case of men who were not in the labor force at the time of the survey, only 4 percent were heavy smokers. A similar situation prevails among women. About one-third (32 percent) of the unemployed women were regular cigarette smokers as compared with 28 percent of the employed women, 22 per-

cent of those who were keeping house, and 10 percent of those not working for other reasons. Since heavy smoking rates are uniformly low for all women, there was no significant difference in the proportion of heavy smokers among the various employment status groups.

Occupation

Smoking may be related to occupation in several ways. First, income differences among occupations produce variations in the ability to purchase and use tobacco. In addition, there are differences in the opportunity to smoke or in the acceptance of smoking in various kinds of work. Finally, occupation itself may reflect differences in background and training which are in turn reflected in smoking rates. These factors often tend to operate in opposite directions in terms of their impact on smoking rates and, therefore, add complexity to the relationship between occupation and smoking.

Lifetime History

The observed proportions of nonsmokers in major occupation groups are shown in table 26 along with the companion adjusted rates, which eliminate differences attributable to variation in the age composition of the groups. Standardization of the percentages for age does not significantly affect any of the conclusions drawn on the basis of the unadjusted data.

Farmers and farm managers had the lowest smoking rates among men. Over 32 percent of the men employed in this pursuit had a history of no regular smoking. The high proportion of nonsmokers among farmers is consistent with the generally low smoking rates found for the total farm population. Of men employed at nonfarm jobs, professional and technical workers clearly had the most nonsmokers despite the fact that they also had the highest average incomes, almost three-tenths of the professional men falling into this category. The proportion of nonsmokers was somewhat lower for men in independent professional practice than for salaried professional workers. The proportions of nonsmokers for men employed at other nonfarm jobs fall into a relatively narrow range and, indeed, are often not significantly different from each other. These figures range from 17 to 23 percent. There is some evidence of ordering by social class in the data, the white-collar groups (professional workers, managers, clerks, sales workers) showing more nonsmokers than craftsmen and operatives. Still, the similarity of smoking rates for such disparate groups as nonfarm laborers and businessmen suggests strongly the role of other noneconomic factors, as well, in the determination of smoking rates.

There was somewhat greater diversity in smoking rates among employed women. The highest percentage of women nonsmokers was found among farm laborers, who are usually housewives employed as unpaid family workers on their own farms. Among nonfarm women, private household workers had the lowest smoking rates, with 72 percent reported as nonsmokers. The rate for this group may be related to the fact that a large number of the workers are nonwhites, whose smoking rates are

Table 26. Percentage [1] of persons 18 years of age and over who have never smoked, by sex and occupational group

Occupational group	Male		Female	
	Crude	Age-adjusted [2]	Crude	Age-adjusted [2]
Professional, technical, and kindred workers	29.0	29.9	66.3	69.3
Self-employed	26.1	23.3	[3]	[3]
Salaried	29.7	30.5	66.8	70.0
Farmers and farm managers	32.4	32.6	[3]	[3]
Managers, officials, and proprietors	20.2	22.6	61.8	60.0
Self-employed	20.2	24.7	64.5	60.2
Salaried	20.2	20.8	57.4	58.3
Clerical and kindred workers	22.5	22.9	56.4	59.9
Sales workers	19.9	21.0	62.6	62.7
Craftsmen, foremen, and kindred workers	17.0	18.4	[3]	[3]
Operatives and kindred workers	18.3	19.2	65.8	68.1
Private household workers	[3]	[3]	71.6	69.3
Service workers, except private household	21.6	21.8	59.1	60.0
Farm laborers and foremen	27.8	23.0	90.2	90.1
Laborers, except farm and mine	20.4	20.8	[3]	[3]

[1] Based on persons reporting smoking pattern.
[2] Adjusted to population of persons 18 years and over enumerated in Current Population Survey for February 1955.
[3] Percent not shown when there were fewer than 100 cases reporting in the sample.

generally below those reported for whites. The fewest nonsmokers among employed women were reported for service workers (other than domestics) and white-collar workers in the clerical and managerial fields. While any social class gradient among women must be small, smoking may be a little more prevalent in women with above-average incomes.

Current Smokers

An analysis of current smoking patterns by occupational groups does not modify the picture described on the basis of lifetime history of smoking. The data on current tobacco consumption by occupation are in appendix table 17, and the results of age adjustment contained in text table 26 indicate that this latter refinement is not required for their interpretation.

For women, the ranking of major occupational groups with respect to current cigarette smoking rates and lifetime history of smoking is identical. Moreover, there are no significant differences in the proportion of smokers using more than one pack of cigarettes daily among the various occupations open to women. The proportion of heavy cigarette smokers ranges from 2 percent for private household workers to about 5 percent for managerial workers.

Among men, the ranking of occupations with respect to current cigarette smoking rates closely resembles those obtained from classification according to lifetime experience. The multiplicity of factors affecting smoking rates is again evident from the fact that farmers have a lower proportion of regular cigarette smokers (40 percent) than farm laborers (51 percent), and that professional workers, who are among the highest paid workers, rank lower (43 percent) than nonfarm laborers (54 percent). The ordering by social class noticeable for nonsmokers and all regular cigarette smokers is blurred when data for those smoking more than one pack of cigarettes daily are examined. Economic factors may play their most important role in determining the group percentages of heavy smokers. For example, sales and managerial workers show a somewhat higher proportion of heavy cigarette smokers (18 and 16 percent) than the lower-paid clerks (14 percent) in the presence of about the same overall proportion of smokers. This tendency is also suggested by the excess of heavy smokers among skilled craftsmen (17 percent) compared with factory operatives (15 percent). Despite the greater frequency of smoking among farm laborers, the proportion of heavy cigarette smokers was the same for both farmers and farm laborers (7 percent).

While the findings in this section concern effects which are not large in magnitude and must be interpreted cautiously, they agree substantially with those of the Hulton survey for Great Britain (*17*). In the latter report, social class was defined by a combination of social and economic criteria. Men in the working class had a higher proportion of smokers (cigarette and pipe) than did the well-to-do and middle classes (82 vs. 79 percent). The relationship was also reversed for heavy cigarette smokers (23 cigarettes or more daily), this proportion being 13 percent for well-to-do and middle-class

Table 27. Percentage [1] of persons 18 years of age and over who have never smoked, by sex and industry group

Industry group	Male		Female	
	Crude	Age-adjusted [2]	Crude	Age-adjusted [2]
Agriculture, forestry, and fisheries	31.1	31.3	86.5	85.2
Construction	18.1	18.9	[3]	[3]
Manufacturing:				
Durable goods	17.7	18.3	53.4	56.2
Nondurable goods	20.8	21.4	65.5	67.5
Transportation, communications, and other public utilities:				
Railroads	21.3	23.4	[3]	[3]
Other	17.3	18.6	57.8	61.8
Trade:				
Wholesale	17.5	19.4	62.6	67.0
Retail	21.9	22.2	59.6	61.3
Finance, insurance, and real estate	20.2	19.9	57.1	57.9
Business and repair services	18.0	20.1	55.8	57.7
Private and personal services	20.0	21.1	68.1	67.5
Entertainment and recreation	19.0	18.1	[3]	[3]
Professional services	31.5	31.8	65.8	68.3
Public administration	23.0	23.5	59.1	62.6

[1] Based on persons reporting smoking pattern.
[2] Adjusted to population of persons 18 years and over enumerated in Current Population Survey for February 1955.
[3] Percent not shown when there were fewer than 100 cases reporting in the sample.

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men and 9 percent for working-class men. Among women in the Hulton survey, the social class differential for cigarette smokers was small (well-to-do and middle class, 41 percent; working class, 39 percent), and this relationship carried over to average and heavy smokers.

Industry

The analysis of smoking patterns by industry groups parallels that for occupation in many respects. Table 27 shows the proportion of nonsmokers by industry. As found in the discussion of occupation, the standardization of the industry data by age produces no significant changes.

The striking differences in percentage of male nonsmokers were found in agriculture and professional services, where they were 31 and 32 percent, respectively. For men employed in industries, the proportion ranged within relatively narrow limits, from 17 to 23 percent. The variation in smoking rates, by industry, was somewhat greater for women, with the lowest proportion of nonsmokers in manufacturing (durable goods), business services, and finance.

The proportion of men smoking more than one pack of cigarettes daily, reported in appendix table 16, is greater for construction, wholesale trade, mining, and finance and insurance than for business, professional, and personal services.

Previous Military Service

Corti, in his History of Smoking (*1*), points out that since tobacco was first introduced into our culture, every great war has been followed by an increase in the smoking habit. He cites its spread after the Thirty Years' War, the revival of smoking in the form of the cigar, during the Napoleonic campaigns, the popularity of the cigarette during and after the Crimean War and, of course, the increase in cigarette use which took place at the time of World War I. Since Corti had no systematic collection of facts to guide him, he no doubt sacrificed accuracy to hyperbole when he declared, in referring to World War I, "If there were any among all those millions of soldiers who were nonsmokers when the war began, there were none by the time it was over." Every observer would agree that the tedium and strain of military life promote and encourage the smoking habit, and none would question the accuracy of the general impression of an increase in tobacco use among members of the armed services.

One of the CPS questions for the survey month the smoking history supplement was used dealt with previous military service, without regard to whether it was performed in peacetime or in wartime. The cigarette-smoking histories for males have been tabulated according to this item, and the results appear in appendix table 19, which excludes the few persons for whom military service status was not reported. As survey coverage was restricted to the civilian population, persons still on active military duty are not included. Direct comparisons should not be attempted between veterans and nonveterans 18–24 years of age; the veterans as a group must be older since they have fulfilled their service require-

Table 28. Percentage of males currently smoking cigarettes regularly, any amount and more than 1 pack daily, by age and previous military service

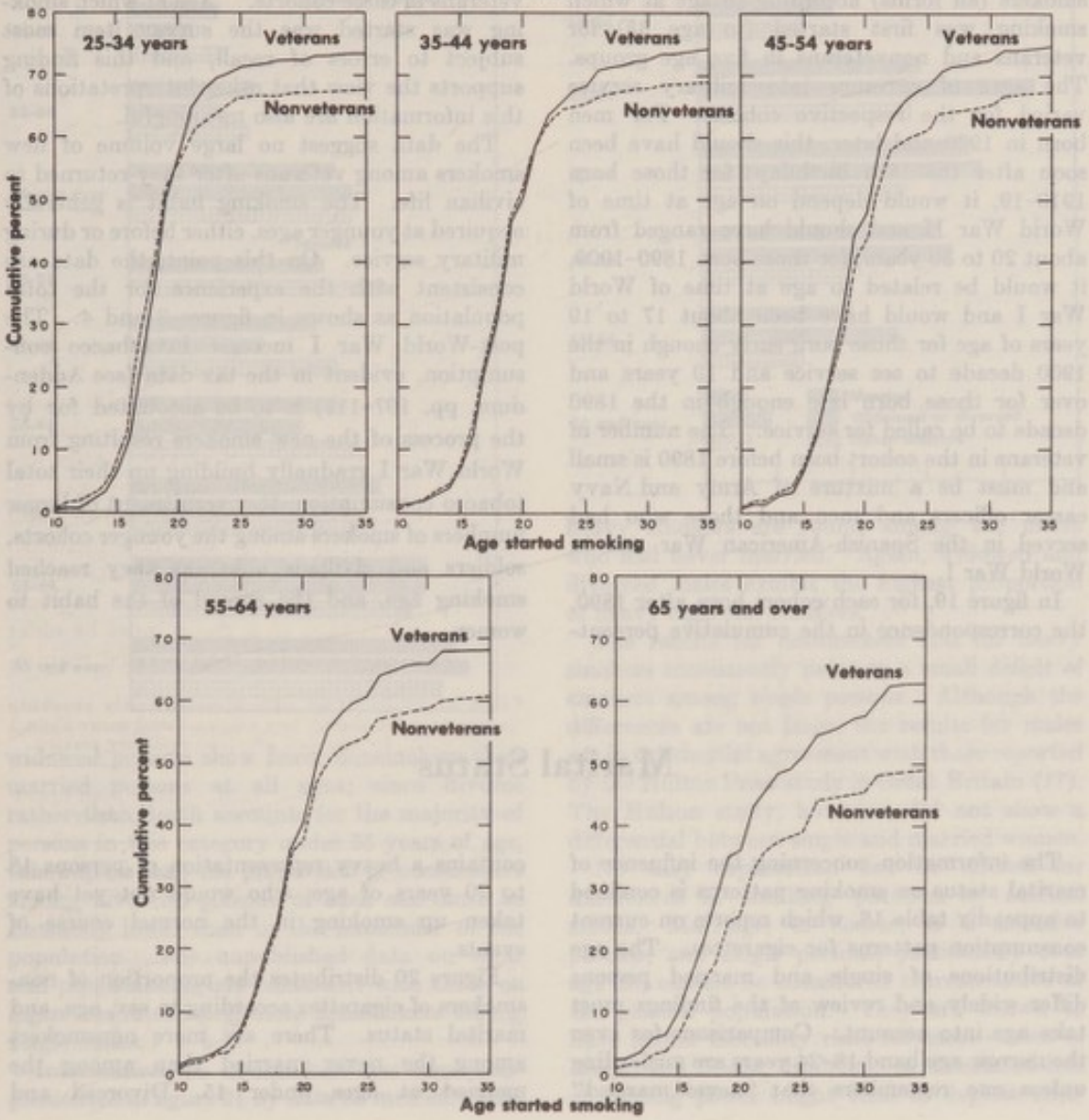
Age (years)[1]	Any amount		More than 1 pack daily	
	Military service	No military service	Military service	No military service
25–34	61. 1	57. 7	16. 1	12. 0
35–44	59. 7	57. 5	19. 3	16. 4
45–54	58. 4	52. 9	19. 1	15. 0
55–64	43. 4	40. 5	11. 9	9. 2
65 and over	28. 0	21. 0	5. 5	3. 1

[1] As of date of survey.

ments, whereas many of the nonveterans have only recently reached draft age. The proportion of males in the sample with previous military service varies with each cohort, ranging from a substantial majority of 74 percent at 25–34 years to 10 percent at 65 years and over.

The results, summarized for all current regular cigarette smokers and for that portion smoking more than one pack of cigarettes daily (table 28), support the impression of greater frequency of use among soldiers and of the persistence of the habit past the age of military service. The item on heavy smokers discriminates better between veterans and nonveterans than among "all regular smokers." No clear age pattern for the relationship emerges, except that the excess among veterans is most pronounced over age 65. The modest excess proportion of cigarette smokers among veterans should not prove too surprising. Military per-

Figure 19. Cumulative percentage of males becoming regular smokers (any form) prior to age specified, by age and previous military service.



sonnel are not insulated from the rest of the population, and forces acting on them carry over in some degree to civilians. Differences between veterans and nonveterans are only slightly enlarged when the percentage who never (or ever) smoked cigarettes regularly is the criterion for comparison.

Even though the differences in smoking patterns are not imposing in magnitude, there can be little doubt that they are directly traceable to military service. Figure 19 presents the cumulative percentage curves for regular smokers (all forms) according to age at which smoking was first started (to age 35) for veterans and nonveterans in five age groups. The ages of entrance into military service varied for the respective cohorts. For men born in 1920 and later, this should have been soon after the 18th birthday; for those born 1910–19, it would depend on age at time of World War II and should have ranged from about 20 to 30 years; for those born 1890–1909, it would be related to age at time of World War I and would have been about 17 to 19 years of age for those born early enough in the 1900 decade to see service and 19 years and over for those born late enough in the 1890 decade to be called for service. The number of veterans in the cohort born before 1890 is small and must be a mixture of Army and Navy career officers and men and those who had served in the Spanish-American War and in World War I.

In figure 19, for each cohort born after 1890, the correspondence in the cumulative percentage curves for smokers for veterans and nonveterans is good up to the age of probable entrance into military service. The curves diverge during the period of military service, and the disparity in favor of veterans is then maintained in subsequent years. There can be no doubt that if the data were rearranged by calendar year in which smoking was started the excess for veterans would be concentrated in war years. It is interesting that the smoking survey data were sensitive enough to detect and time correctly the differential effects for veterans in these cohorts. Age at which smoking was started was the survey item most subject to errors of recall, and this finding supports the view that other interpretations of this information are also meaningful.

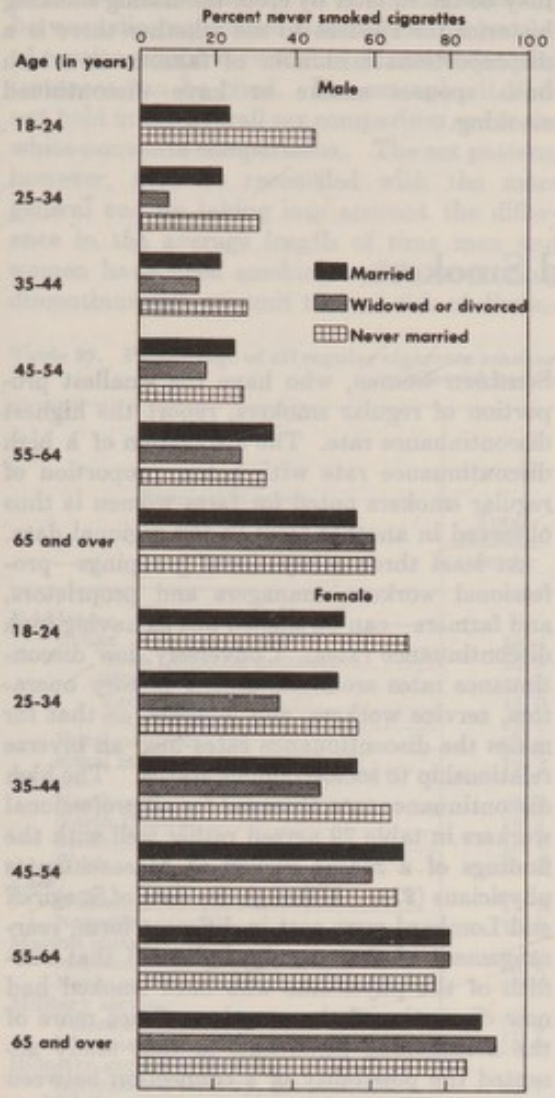
The data suggest no large volume of new smokers among veterans after they returned to civilian life. The smoking habit is generally acquired at younger ages, either before or during military service. On this point, the data are consistent with the experience for the total population as shown in figures 3 and 4. The post-World War I increase in tobacco consumption, evident in the tax data (see Addendum, pp. 107–111) is to be accounted for by the process of the new smokers resulting from World War I gradually building up their total tobacco consumption, the recruitment of larger numbers of smokers among the younger cohorts, soldiers and civilians alike, as they reached smoking age, and the spread of the habit to women.

Marital Status

The information concerning the influence of marital status on smoking patterns is confined to appendix table 18, which reports on current consumption patterns for cigarettes. The age distributions of single and married persons differ widely, and review of the findings must take age into account. Comparisons for even the narrow age band 18–24 years are misleading unless one remembers that "never married" contains a heavy representation of persons 18 to 20 years of age, who would not yet have taken up smoking in the normal course of events.

Figure 20 distributes the proportion of nonsmokers of cigarettes according to sex, age, and marital status. There are more nonsmokers among the never married than among the married at ages under 45. Divorced and

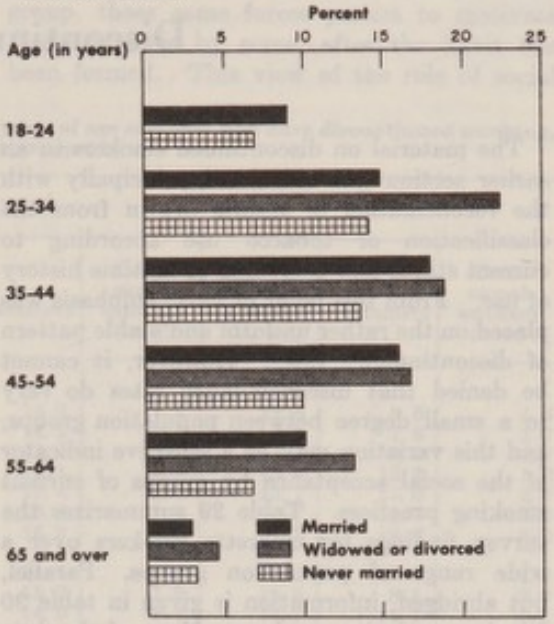
Figure 20. Percentage of persons who have never smoked cigarettes, by sex, age, and marital status.



widowed persons show fewer nonsmokers than married persons at all ages; since divorce rather than death accounts for the majority of persons in this category under 55 years of age, this implies that the proportion of nonsmokers among divorced persons of each sex must be distinctly lower than in the remainder of the population. The unpublished data on cigar and pipe smoking are consistent with those on cigarettes in showing more nonsmokers among single males.

Information on nonsmokers has been supplemented in figure 21 by data on men currently smoking more than one pack of cigarettes daily. Data on women are omitted since the sample did not yield stable estimates by marital status for older women although the fragmentary findings are not inconsistent with those presented for men. Up to age 65, fewer

Figure 21. Percentage of males currently smoking more than one pack of cigarettes daily, by age and marital status.



heavy smokers are found among the males who had never married. Again, widowed and divorced males exhibit the highest proportion of heavy cigarette smokers.

The results for nonsmokers and for heavy smokers consistently point to a small deficit of smokers among single persons. Although the differences are not large, the results for males are in substantial agreement with those reported by the Hulton Press study in Great Britain (*17*). The Hulton study, however, did not show a differential between single and married women.

No easy explanation can be offered for differences in smoking patterns by marital status. Marriage, of course, is a selective process, and single persons, particularly over age 25, cannot be considered representative of the general population. They are known to have higher mortality risks for most causes of death, and illness, disability, or associated loss of earning power might tend to depress their

smoking rates. Among married persons, the smoking habits of the spouse may have some influence; if one marital partner smokes, the other may tend to follow suit. This possible reinforcement of action between spouses might also account for the higher discontinuance rates noted among married persons. This hypothesis may be tested later by cross-tabulating smoking histories for spouses to see whether there is a disproportionate number of families in which both spouses smoke or have discontinued smoking.

Discontinued Smokers

The material on discontinued smokers in an earlier section was concerned principally with the reconciliation of results drawn from the classification of tobacco use according to current status and according to lifetime history of use. From this point of view, emphasis was placed on the rather uniform and stable pattern of discontinuance rates. However, it cannot be denied that discontinuance rates do vary to a small degree between population groups, and this variation may be a sensitive indicator of the social acceptance by groups of current smoking practices. Table 29 summarizes the survey findings for cigarette smokers over a wide range of population groups. Parallel, but abridged, information is given in table 30 for cigar and pipe smokers. None of the data are adjusted for age; adjustments were tried for selected categories with only minor changes in the rates, and the systematic application of this refinement was deemed unnecessary.

It has been pointed out that women as a group have lower cigarette discontinuance rates (not adjusted for time at risk of discontinuance) than men. This sex relationship holds for urban and rural nonfarm populations. The urban-rural gradient of discontinuance rates is more pronounced for women, so that the prevailing sex pattern is reversed in the farm population, in which the higher discontinuance rate occurs among women. This reversal suggests that social pressures against smoking among farm women may be so strong as to dissuade some from continuing, even after the practice has been adopted.

The highest discontinuance rate for men appears in the Western States, there being no significant differences among the other three regions. Southern women, who have the smallest proportion of regular smokers, report the highest discontinuance rate. The association of a high discontinuance rate with a low proportion of regular smokers noted for farm women is thus observed in another form in the regional data.

At least three occupational groupings—professional workers, managers and proprietors, and farmers—can be singled out as having high discontinuance rates. Conversely, low discontinuance rates are found among factory operators, service workers, and laborers, so that for males the discontinuance rates bear an inverse relationship to socioeconomic status. The high discontinuance rate observed for all professional workers in table 29 agreed rather well with the findings of a recent survey of Massachusetts physicians (*27*). Although the data of Snegireff and Lombard were cast in different form, rearrangement of their results indicated that one-fifth of the physicians who once smoked had now discontinued the practice. Since more of the nonsmoking physicians in that study accepted the possibility of a connection between heavy smoking and lung cancer than did their smoking confreres, it is a fair inference that at least some of the discontinuances in that group were prompted by fear of adverse effects of smoking on health.

Comparisons of occupational and industrial groupings for women reveal little variation. A sizable differential does appear in contrasting women by employment status; women not employed (mostly housewives) show higher discontinuance rates than employed women.

Male professional workers, farmers, and women not in the labor force also have a smaller proportion of regular cigarette smokers than

other comparable groups. These observations might tempt one to infer an inverse relationship between discontinuance rate and the proportion of regular smokers, even though nonconforming instances can be cited. For example, it does not hold in the overall sex comparison or for the white-nonwhite comparisons. The sex pattern, however, may be reconciled with the more general one by taking into account the difference in the average length of time men and women have been smoking and by measuring discontinuances per unit time at risk of discontinuance. When this is done, as for the discontinuance rates (5^{q}_{x}) in table 11, the sex difference virtually disappears. The white-nonwhite relationship could reflect differences between smokers in these two population groups in the values attached to the smoking habit.

The general hypothesis would then state that when social forces tend to militate against adoption of the smoking habit by members of a group, these same forces persist to motivate discontinuance by some, after the habit has been formed. This view of the role of social

Table 29. Percentage of all regular cigarette smokers 18 years of age and over who have discontinued smoking, selected population groups, by sex

Population group	Male			Female		
	Total discontinuances	Abstainers	Occasional smokers (now)	Total discontinuances	Abstainers	Occasional smokers (now)
Residence:						
Urban	16.4	15.0	1.4	11.5	9.6	1.8
Over 1,000,000 population	15.2	14.2	1.0	9.5	7.8	1.7
250,000–1,000,000	15.5	13.9	1.6	11.3	9.6	1.7
Under 250,000	18.0	16.1	1.9	13.6	12.6	1.0
Urban fringe	18.1	16.4	1.7	13.7	11.0	2.7
Rural nonfarm	17.9	16.1	1.8	16.7	14.7	2.0
Rural farm	19.2	16.9	2.3	21.2	19.0	2.2
Region:						
Northeast	16.2	14.6	1.6	12.0	10.1	1.9
North Central	16.5	15.2	1.3	10.8	8.5	2.3
South	16.4	14.6	1.7	16.1	14.7	1.4
West	21.3	19.4	1.9	14.2	12.1	2.1
Race:						
White	17.7	16.1	1.6	13.4	11.5	1.9
Nonwhite	10.8	8.5	2.3	9.7	7.5	2.2
Marital status:						
Married (over 45 years of age)	23.9	22.1	1.8	14.7	12.8	2.0
Never married (over 45 years of age)	19.7	15.4	4.3	19.5	17.1	2.4
Military service:						
Veteran	15.8	14.5	1.3	----	----	----
Nonveteran	18.0	16.1	1.9	----	----	----
Industry groups (selected):						
Agriculture, forestry, fisheries	18.7	16.5	2.2	----	----	----
Construction	13.8	12.3	1.5	----	----	----
Manufacturing	15.6	14.5	1.1	9.2	7.6	1.5
Wholesale trade	14.1	12.3	1.8	----	----	----
Retail trade	15.8	14.3	1.4	10.4	8.7	1.7
Public administration	18.1	17.2	.9	11.1	11.1	----
Major occupation groups (selected):						
Professional, technical, and kindred workers	21.6	20.4	1.2	10.1	8.6	1.6
Farmers, farm managers, managers, officials, and proprietors	21.2	19.8	1.4	----	----	----
Clerical and kindred workers	15.4	14.8	.7	9.0	7.7	1.3
Sales workers	14.7	12.7	2.0	10.0	7.1	2.9
Craftsmen, foremen, and kindred workers	16.6	15.1	1.5	----	----	----
Operatives and kindred workers	13.3	12.2	1.1	8.7	7.4	1.2
Service workers	13.7	12.1	1.5	8.1	7.2	.9
Laborers, except farm	13.8	11.6	2.2	----	----	----
Employed	----	----	----	9.5	8.0	1.5
Not employed	----	----	----	15.2	13.0	2.2

Table 30. Percentage of all regular male cigar and pipe smokers 18 years of age and over who have discontinued regular smoking, selected population groups

Population group	Cigars			Pipe		
	Total discontinuances	Abstainers	Occasional smokers (now)	Total discontinuances	Abstainers	Occasional smokers (now)
Residence:						
Urban	40.9	29.5	11.4	52.2	44.3	7.9
Rural nonfarm	45.3	33.4	11.9	52.9	45.8	7.1
Rural farm	48.4	38.4	9.9	45.8	40.4	5.4
Region:						
Northeast	36.1	25.1	11.0	48.3	40.0	8.3
North Central	44.6	33.2	11.4	50.1	43.7	6.4
South	39.0	28.4	10.6	51.8	43.4	8.3
West	58.6	45.1	13.4	59.7	53.9	5.8
Race:						
White	43.7	32.1	11.7	51.9	44.6	7.3
Nonwhite	28.0	19.9	8.1	43.1	35.6	7.5

forces differs from the one postulating internal drives predisposing some individuals to take up smoking. The inverse relationship noted between discontinuance rate and proportion of regular smokers in a group does not support a theory of smoker self-selection. If there were internal drives which lead individuals to take up smoking, as the total proportion of smokers increases through recruitment of marginal, less strongly motivated persons, the rate of discontinuance ought to rise also by means of a greater volume of withdrawals among these marginal smokers.

Since some variation in discontinuance rates may be found over a broad spectrum of population groups, this suggests the operation of multiple factors in conditioning the acceptance and discontinuance of smoking habits in these same groups. Questions on reasons for discontinuance did not form part of this inquiry, and no specific answers on motivation can be sought in the present data.

Summary

As a supplement to the United States Bureau of the Census Current Population Survey (CPS) for February 1955, smoking histories were collected from approximately 40,000 men and women 18 years of age and over. Persons in the survey were a representative cross-section of the population of the United States. The questionnaire regarding smoking history was similar to that used by the American Cancer Society and the National Cancer Institute in their forward studies on the association of smoking and lung cancer. The questions covered the use of cigarettes, cigars, and pipe tobacco, attempted to distinguish between occasional and regular smokers, and included items on age at which smoking was started and the maximum consumption rate ever attained. The CPS information on age, sex, residence, race, and other population characteristics was made available for analysis of these data.

The survey objective was to classify the population by broad smoking categories, and no attempt was made to validate verbal statements on rate of use by diaries or other means of recording consumption or purchases. Cur-

rent cigarette consumption as estimated from the survey data was checked against the national aggregate consumption determined from tax data, which indicated that the survey underestimated cigarette consumption by approximately 15 percent. Considering the different intent for which the questions on smoking were designed, the correspondence between the survey and tax estimate seems good.

Of the 49.6 million men and 55.1 million women 18 years of age and over in the civilian population outside institutions, 11 million men (23 percent) and 37 million women (67 percent) are reported to be nonsmokers, that is, they had never smoked tobacco occasionally or regularly in any form. Of the remaining 38 million men with a history of tobacco use, 31 million had smoked cigarettes regularly at some time during their lives. The corresponding numbers of lifetime male cigar and pipe smokers were 4.8 and 7.9 million, respectively. The numbers of current regular male smokers were as follows: cigarettes, 26 million; cigars, 2.8 million; pipes, 3.9 million. Of the 18 million women who reported some use of tobacco, 15 million had at one time or another been regular cigarette smokers. Thirteen million were regular smokers at the time of the survey. All of these estimates have been adjusted to take account of persons in the sample for whom no smoking histories were obtained.

Cigarettes are the major form of tobacco use. Trade sources estimate the number of cigarette smokers at about 60 million, and this figure probably includes "discontinued" smokers. If one adjusts the survey results to include occasional smokers, persons in military service and in institutions, and smokers presently under 18 years of age, the maximum resulting estimate of cigarette users to be derived from the survey is about 55 million. Some of this discrepancy between the survey estimate and that of trade sources may be traced to different estimates of the proportion of women smokers.

The profiles of age and sex differentials in tobacco use resulting from comparisons based on current practices or lifetime history of use are very similar, particularly for cigarettes. The highest percentage of current regular cigarette smokers appears at 25–34 years of age among both males and females and tapers off in successively older groups. The sex differentials in the proportion of smokers (or nonsmokers) is greatest at ages over 65. Among men over 65 years of age, pipe smoking rivals cigarette smoking in popularity. The differential in favor of cigarettes widens at the younger ages. Moreover, in the younger cohorts there seems to be a shift to "pure" cigarette smoking, accompanied by a lessened tendency to take up cigars or pipe exclusively.

A shift to an earlier age for starting to smoke is observed for younger persons. This has accompanied the rising proportion of regular smokers in these younger age groups. The smoking habit characteristics of a cohort become evident by a rather early age, around age 18 to 20 for men and at a slightly older age for women. Much of the decline in the number of cigar and pipe smokers may be traced to a failure of these forms of smoking to attract converts at an early age among persons born since 1900.

Aside from differences associated with age and sex, urban-rural residence is the population characteristic which differentiates smoking habits most sharply. Rural nonfarm persons closely resemble urban dwellers in smoking habits, and the sharp demarcation appears between the rural nonfarm and rural farm populations. The farm and nonfarm populations acquire the smoking habit at virtually the same ages as the urban population. Among men, the urban-rural differences are emphasized when comparison is restricted to cigarette smokers using more than one pack of cigarettes daily. Cigarette smoking has been increasingly accepted by urban women born after 1890. Sizable acceptance among farm women was delayed almost one generation, to women born after 1920.

Practically no differences in smoking patterns according to size of urban community were noted. This finding is at variance with earlier surveys and suggests that the events of the past 20 years have disposed of community size as an important determinant in shaping smoking habits.

For men, there is little variation in the distribution of smoking patterns in the four major regions of the country (Northeast, North Central, South, and West). The proportion of female nonsmokers is lower in the Northeastern

and Western regions than elsewhere. A higher proportion of heavy cigarette smokers was noted among Northeastern males and the excess cannot be accounted for solely by greater urbanization of that area.

The differences between whites and nonwhites with respect to the proportions of nonsmokers and of all regular cigarette smokers are trivial. However, the proportion of white male cigarette smokers who use more than one pack of cigarettes daily is almost double the proportion among the nonwhite males. A similar excess occurs among white females. Urban-rural gradients provide another distinction. Among white males, the rural nonfarm data on tobacco use resemble those for the urban population. Among nonwhites, the reverse is true, the nonfarm data resembling those for the farm population.

Smoking is less prevalent among farmers than among other male occupational groups. Among nonfarm workers, professional and technical personnel had the highest proportion of nonsmokers. There is evidence of some ordering by social class, the white-collar groups having more nonsmokers than craftsmen or operatives. This ordering by social class, noticeable for nonsmokers and for all regular cigarette smokers, tends to disappear when comparison is limited to smoking more than one pack of cigarettes daily.

The survey data support the observation that military life encourages the adoption of the smoking habit. The greater use of tobacco among soldiers persists after they leave military service.

The results by marital status point to a small deficit of smokers among single persons of both sexes. The proportion of nonsmokers among divorced persons of each sex is lower than for the remainder of the population.

Proportionately more men than women were reported as discontinued smokers at the time of the survey. When sex differences in the number of smokers and length of exposure to the habit are taken into account, the difference between men and women with respect to discontinuance disappears. For cigarettes particularly, discontinuance rates fall into consistent age patterns and show some stability over a variety of population groups, although in examining the data by population subgroups, a rather general inverse relationship between the proportion of regular smokers and the discontinuance rate does appear. It does not appear necessary to qualify group comparisons, based either on maximum or current rate of smoking, by taking into account the effect of discontinuance on duration of exposure to the habit.

The major purpose in collecting smoking histories was to investigate the meaning of the reported associations between smoking and lung cancer and to check whether the distribution of lung cancer deaths is consistent with estimates of the excess risk among smokers and the distribution of smokers in the population. The application of the data to test proposed models for lung cancer etiology has been reported elsewhere (*25*). This report is devoted solely to presentation of the CPS findings, and it is hoped that these data may prove useful to persons interested in the social, economic, and marketing aspects of tobacco use, as well as to investigators interested in lung cancer.

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Appendix I

Questionnaire Used in Tobacco Smoking Survey

SECTION I

CIGARETTES

(Answer all applicable questions in this Section; then continue with Section II)

A. Have you ever smoked any cigarettes?

1. ☐ No (Skip to Section II - Cigars)
2. ☐ Yes (Answer question below)

B. Do you smoke any cigarettes at the present time?

1. ☐ Yes (Answer questions below)
   - a. How many cigarettes do you smoke a day on the average?
     - (1) ☐ Smoke once in a while, not every day
     - (2) ☐ Less than 10 cigarettes (1/2 pack)
     - (3) ☐ Between 10 and 20 cigarettes (1/2-1 pack)
     - (4) ☐ Between 21 and 40 cigarettes (1-2 packs)
     - (5) ☐ Over 40 cigarettes (over 2 packs)
   - b. Did you ever regularly smoke more than this amount each day?
     - (1) ☐ No
     - (2) ☐ Yes: What was the **maximum** number of cigarettes you regularly smoked each day? _____
   - c. How old were you when you started smoking cigarettes regularly? _____
   - d. How many years have you been smoking cigarettes? _____

2. ☐ No (Answer questions below)
   - a. During your entire life, have you smoked as many as 5 to 10 packs of cigarettes?
     - (1) ☐ Yes (2) ☐ No (Skip to Section II)
   - b. How old were you when you started smoking cigarettes regularly? _____
   - c. How many years did you smoke cigarettes? _____
   - d. How long ago did you stop smoking cigarettes? _____
   - e. What was the **maximum** number of cigarettes you regularly smoked each day when you were a smoker?
     - (1) ☐ Smoked once in a while, not every day
     - (2) ☐ Less than 10 cigarettes (1/2 pack)
     - (3) ☐ Between 10 and 20 cigarettes (1/2-1 pack)
     - (4) ☐ Between 21 and 40 cigarettes (1-2 packs)
     - (5) ☐ Over 40 cigarettes (over 2 packs)

SECTION II

CIGARS

(Answer all applicable questions in this Section; then continue with Section III)

A. Have you ever smoked any cigars?

1. ☐ No (Skip to Section III - Pipe smoking)
2. ☐ Yes (Answer question below)

B. Do you smoke any cigars at the present time?

1. ☐ Yes (Answer questions below)
   - a. How many cigars do you smoke a day on the average?
     - (1) ☐ Smoke once in a while, not every day
     - (2) ☐ 1 or 2 cigars a day
     - (3) ☐ 3 or 4 cigars a day
     - (4) ☐ 5 to 8 cigars a day
     - (5) ☐ 9 or more cigars a day
   - b. Did you ever regularly smoke more than this amount each day?
     - (1) ☐ No
     - (2) ☐ Yes: What was the **maximum** number of cigars you regularly smoked each day? _____
   - c. How old were you when you started smoking cigars regularly? _____
   - d. How many years have you been smoking cigars? _____

2. ☐ No (Answer questions below)
   - a. During your entire life have you smoked as many as 50 to 75 cigars?
     - (1) ☐ Yes (2) ☐ No (Skip to Section III)
   - b. How old were you when you started smoking cigars regularly? _____
   - c. How many years did you smoke cigars? _____
   - d. How long ago did you stop smoking cigars? _____
   - e. What was the **maximum** number of cigars you regularly smoked each day when you were a smoker?
     - (1) ☐ Smoked once in a while, not every day
     - (2) ☐ 1 or 2 cigars a day
     - (3) ☐ 3 or 4 cigars a day
     - (4) ☐ 5 to 8 cigars a day
     - (5) ☐ 9 or more cigars a day

SECTION III

PIPE SMOKING

(Answer all applicable questions in this Section)

A. Have you ever smoked a pipe?

1. ☐ No (Do not answer any more questions)
2. ☐ Yes (Answer question below)

B. Do you smoke a pipe at the present time?

1. ☐ Yes (Answer questions below)
   - a. How many pipefuls do you smoke a day on the average?
     - (1) ☐ Smoke once in a while, not every day
     - (2) ☐ Less than 5 pipefuls
     - (3) ☐ Between 5 and 9 pipefuls
     - (4) ☐ Between 10 and 19 pipefuls
     - (5) ☐ 20 or more pipefuls
   - b. Did you ever regularly smoke more than this amount each day?
     - (1) ☐ No
     - (2) ☐ Yes: What was the **maximum** number of pipefuls you regularly smoked each day? _____
   - c. How old were you when you started smoking a pipe regularly? _____
   - d. How many years have you been smoking a pipe? _____

2. ☐ No (Answer questions below)
   - a. During your entire life, have you smoked as many as 3 to 5 packages of pipe tobacco?
     - (1) ☐ Yes (2) ☐ No (End of interview)
   - b. How old were you when you started smoking a pipe regularly? _____
   - c. How many years did you smoke a pipe? _____
   - d. How long ago did you stop smoking a pipe? _____
   - e. What was the **maximum** number of pipefuls you regularly smoked each day when you were a smoker?
     - (1) ☐ Smoked once in a while, not every day
     - (2) ☐ Less than 5 pipefuls
     - (3) ☐ Between 5 and 9 pipefuls
     - (4) ☐ Between 10 and 19 pipefuls
     - (5) ☐ 20 or more pipefuls

You should have at least one box checked in Sections I, II, and III.

Form CI-1 (12-21-54)
Comm-DC-44210

Control No. _____ Line No. of person _____ Line No. of respondent _____

Appendix II

Abstract from Enumerator's Instructions

All 3 sections of questionnaire must be filled

Each of the three sections of the questionnaire must be filled. A person who has never smoked cigars or pipes must check "No" in part A of sections II and III to inform us of this fact. If a section is left blank we may have to conclude that the respondent forgot to answer or skipped the question, when he really meant the answer to be "No."

What is meant by "present time" in question B of each section

There may be some uncertainty about the meaning of the term "present time" in question B. We did not want to pin the time period down to a specific day or week because that might exclude persons who usually smoked but did not do so during this interval. If the respondent asks for a definition of "present time" you may use the CPS survey week for convenience. In general, however, you should use any reasonable definition which the respondent offers. Obviously, we cannot accept the time of interview as "the present time" because that is too limited; but if the respondent uses the preceding day, week, or month, that will be acceptable.

Classification of persons who smoke
exactly *½ pack*
exactly *1 pack*
exactly *2 packs of cigarettes a day*

Note that in questions B–1 and B–2–c in section I, people who smoke *exactly one-half a pack or exactly one pack* of cigarettes a day are placed in group (3). Similarly, those who smoke *exactly two packs* a day are placed in group (4).

Age at which person started smoking regularly

In question B–1–c and B–2–b (of all three sections) we are *not* interested in the time the respondent took his first puff of tobacco. For example, children are sometimes permitted to puff their daddy's pipe on occasion. We are referring here to the age at which the respondent first began to use tobacco with some *regularity*. If, for example, at the age of 17 a boy or girl smoked a cigarette every Saturday night on a date, that would be the age at which smoking was started regularly. Some respondents may say they are not regular smokers. Our definition of a regular smoker is anyone who smokes at the present time or has smoked more than a given amount (5–10 packs of cigarettes, 50–75 cigars, or 3–5 packages of pipe tobacco) during his lifetime. Therefore, even people who smoke only occasionally, but not every day, would be regular smokers according to our definition of the term.

[NOTE: In the tables prepared for this monograph, only regular smokers as defined on p. 6 were tabulated as to age at which smoking was started regularly.]

Amount smoked each day *vs. amount smoked during lifetime*

Note that in question B–1–a we ask smokers about the number of cigarettes, cigars, or pipefuls they smoke *each day*, whereas in question B–2–a we are interested in the number smoked during a person's entire life. The reason for the latter question is that we want to separate the persons who do not smoke at present into two groups: (a) those who never did much smoking and (b) those who once smoked a substantial amount, but are no longer doing so.

Number of years person has been smoking, not necessarily difference between age at which started and present age

The answer to question B–1–d, "How many years have you been smoking? " is not necessarily the difference between the age at which a person started smoking and his present age. It may be less than this amount if there was a period of time during which the person stopped smoking. The same reasoning applies to question B–2–c, "How many years did you smoke? "

Numerical entries should be made to the nearest whole number

Entries for questions relating to age or number of years should be to the nearest whole number. Thus, if a person started smoking at the age of 15½, the entry should be 16. If a person stopped smoking at any time during the past year (say 2 months ago) enter "1" in B–2–d.

What you do in the field

This information will be obtained for all household members 18 years old or over for whom you get a basic schedule. This means, of course, that you will *not* get the information for members of the armed forces who are living in the household, but you will get it for veterans who are 18 years of age or over. As an aid to you in identifying household members 18 years of age and over, your office will have entered a check mark to the right of the printed line number in column 13 of the control card for each household member 18 years old and over, except in households in rotation groups 3 and 7, which are new or returning to sample this month. Wherever possible, obtain the information for form CI–1 for each person in the sample 18 years old or over *directly from the person himself.* In some cases it may be necessary to obtain the information from a respondent other than the person himself. For example, some housewives may insist on providing the information for their husbands because they do not want to bother them for the data; some people are too ill to be interviewed; illiterates, semiliterates, and persons who cooperate grudgingly are unlikely to return a mail questionnaire. In such cases, and others of a similar nature, it is permissible to obtain the information from some responsible household member other than the person himself. *In all such cases, you should provide at the bottom of the CI–1 a very brief explanation (5 or 6 words) for not interviewing the person himself.*

Treatment of individual noninterviews

If a person cannot be interviewed because he is not at home, or for some other reason, use the following procedure:

Personal visit

1. In the case of a *personal* visit, leave a questionnaire (with a District Office return envelope) for the person to fill and mail in to the District Office. Be sure to enter the sample person's name on the front of these forms and his control number and line number on the back.

Telephone interview

2. In a telephone household, try to find out when you can reach the person for an interview. If, after several telephone calls you still cannot reach one or more persons at a given address, inform the respondent that a questionnaire will be mailed to the address, which should be completed by the person to whom it is addressed and mailed to the District Office.

Appendix III

Table 1. Cumulative percentage of persons becomin g regular cigarette, cigar, and pipe smokers prior to age specified, by age and sex, United States, 1955

Age started smoking	Age at time of survey (years)					
	18–24 [1]	25–34 [1]	35–44	45–54	55–64	65 and over
ANY FORM OF TOBACCO (MEN)						
10	0.9	0.5	0.8	0.9	1.0	1.1
11	1.4	.9	1.5	1.8	1.9	1.8
12	1.6	1.0	1.8	2.0	2.2	2.0
13	2.4	2.2	3.1	3.3	3.4	3.4
14	3.5	3.3	4.1	4.0	4.0	4.2
15	6.5	6.5	7.3	6.8	6.9	6.5
16	11.9	12.5	13.4	12.1	12.0	11.0
17	23.6	25.0	24.3	20.9	18.9	15.5
18	34.0	36.0	32.3	28.4	23.9	19.3
19	45.3	51.1	46.9	41.3	35.8	26.0
20	50.6	56.5	51.3	45.0	38.5	26.9
21	54.8	63.1	59.1	54.6	47.8	34.3
22	56.7	66.2	62.9	59.3	52.3	37.2
23	58.0	67.8	65.0	61.0	54.3	39.0
24	58.8	68.8	66.1	62.2	55.5	39.8
25	.....	69.7	66.7	62.9	56.4	40.5
26	.....	70.2	68.6	65.4	59.4	45.0
27	.....	70.5	69.1	65.8	59.9	45.7
28	.....	70.6	69.4	66.0	60.5	46.0
29	.....	70.9	69.9	66.5	61.0	46.4
30	.....	71.0	70.0	66.8	61.1	46.7
31	.....	71.5	70.8	68.3	62.7	49.7
32	.....	71.5	70.8	68.4	62.7	50.0
33	.....	71.6	71.1	68.6	62.9	50.2
34	.....	71.8	71.3	68.8	63.0	50.3
35	.....	71.8	71.5	68.9	63.1	50.4
Future lifetime (maximum)	.....	75.9	75.8	73.5	69.4	58.1
CIGARETTES (MEN)						
10	0.9	0.5	0.7	0.9	0.9	0.7
11	1.4	.9	1.3	1.8	1.7	1.1
12	1.5	1.0	1.7	1.9	2.0	1.3
13	2.4	2.1	2.9	3.2	3.0	2.1
14	3.5	3.3	3.9	3.9	3.6	2.8
15	6.3	6.5	7.1	6.6	6.2	4.3
16	11.6	12.4	13.0	11.7	10.9	7.5
17	23.3	24.7	23.8	20.2	17.3	10.7
18	33.5	35.3	31.5	27.3	21.6	13.0
19	44.3	50.0	45.7	39.5	31.8	16.6
20	48.9	55.2	49.8	42.8	34.0	17.3
21	52.6	61.4	56.9	51.2	41.2	20.9
22	54.2	64.1	60.6	55.2	44.3	22.2
23	55.3	65.5	62.5	56.7	45.7	22.9
24	55.8	66.4	63.4	57.7	46.2	23.2
25	.....	67.0	64.0	58.2	46.9	23.4
26	.....	67.4	65.3	60.1	48.7	25.1
27	.....	67.6	65.8	60.4	49.1	25.6
28	.....	67.7	66.0	60.6	49.5	25.7
29	.....	67.9	66.4	61.1	50.0	26.0
30	.....	67.9	66.4	61.3	50.2	26.1
31	.....	68.2	66.9	62.2	51.2	27.5
32	.....	68.2	67.0	62.3	51.2	27.8
33	.....	68.4	67.1	62.5	51.2	28.0
34	.....	68.4	67.2	62.6	51.5	28.0
35	.....	68.4	67.3	62.7	51.6	28.1
Future lifetime (maximum)	.....	71.3	70.7	66.3	56.5	33.7
CIGARS (MEN)						
10	.....	.....	0.1	.....	0.1	0.1
11	.....	.....	.1	0.1	.1	.1
12	.....	.....	.1	.1	.1	.1
13	0.1	.....	.1	.1	.2	.3
14	.1	.....	.1	.1	.2	.3
15	.1	.....	.1	.2	.4	.7
16	.2	0.1	.2	.3	.7	1.3
17	.2	.3	.4	.6	1.2	2.2
18	.4	.5	.6	.9	1.6	2.9
19	1.0	.9	1.2	1.8	2.9	4.3
20	1.3	1.1	1.5	2.1	3.2	4.6
21	2.2	1.8	2.4	3.5	5.1	7.0
22	2.4	2.2	2.8	4.3	6.5	8.1
23	2.7	2.5	3.2	4.8	7.1	8.7
24	2.9	2.9	3.3	5.1	7.6	9.1
25	.....	3.2	3.5	5.3	7.7	9.4
26	.....	3.7	4.4	6.2	9.4	11.6
27	.....	3.9	4.5	6.2	9.6	11.7
28	.....	4.1	4.8	6.3	9.7	11.8
29	.....	4.1	4.9	6.4	9.8	11.9
30	.....	4.3	5.0	6.4	9.8	12.1
31	.....	4.7	5.4	7.2	11.0	13.5
32	.....	4.9	5.6	7.3	11.0	13.6
33	.....	5.1	5.8	7.4	11.1	13.6
34	.....	5.6	6.0	7.4	11.2	13.7
35	.....	5.6	6.0	7.4	11.2	13.7
Future lifetime (maximum)	.....	10.4	10.5	12.2	15.6	17.6
PIPES (MEN)						
10	.....	.....	0.1	0.1	0.1	0.4
11	.....	.....	.1	.1	.2	.7
12	.....	.....	.1	.1	.2	.7
13	0.1	0.1	.3	.2	.4	1.2
14	.1	.1	.3	.2	.5	1.3
15	.2	.1	.5	.4	.9	2.1
16	.4	.4	.8	.9	1.5	3.3
17	1.0	.8	1.3	1.4	2.5	4.4
18	1.5	1.5	1.8	2.0	3.4	5.9
19	2.4	2.7	3.1	3.2	5.3	8.1
20	3.8	3.3	3.7	3.6	5.8	8.6
21	4.7	4.9	5.0	5.2	8.8	12.9
22	5.8	5.8	5.6	6.4	10.1	14.2
23	6.9	6.5	6.2	7.0	10.8	15.1
24	7.9	7.0	6.5	7.2	11.4	15.4
25	.....	7.6	6.9	7.6	11.9	16.0
26	.....	8.1	8.2	9.0	13.6	18.5
27	.....	8.4	8.7	9.1	13.9	18.7
28	.....	8.8	8.8	9.4	14.2	18.9
29	.....	9.3	9.2	9.5	14.5	19.1
30	.....	9.5	9.3	9.6	14.5	19.3
31	.....	10.2	10.0	10.8	15.8	21.6
32	.....	10.5	10.2	10.9	15.9	21.7
33	.....	10.8	10.4	11.0	16.1	21.9
34	.....	11.1	10.5	11.1	16.1	21.9
35	.....	11.7	10.7	11.2	16.6	22.1
Future lifetime (maximum)	.....	18.8	17.5	18.1	22.4	29.4
CIGARETTES (WOMEN)						
10	0.1	0.1	.....	.....	0.1	.....
11	.2	.2	0.1	0.1	.2	.....
12	.2	.2	.1	.1	.2	.....
13	.3	.4	.2	.1	.2	.....
14	.9	.6	.3	.2	.2	0.1
15	2.1	1.5	.6	.3	.3	.1
16	4.8	3.2	1.7	.6	.6	.2
17	9.8	7.0	4.2	1.3	.7	.4
18	15.9	11.9	7.0	2.0	.8	.4
19	24.3	20.2	12.3	4.1	1.2	.6
20	28.9	23.9	14.5	4.7	1.4	.7
21	32.6	28.9	19.6	7.5	2.1	.9
22	34.7	31.9	22.0	8.7	2.3	1.0
23	35.8	34.0	23.4	9.7	2.4	1.0
24	36.2	35.3	24.8	10.3	2.6	1.1
25	.....	36.2	25.6	10.9	2.8	1.1
26	.....	37.6	28.0	13.4	3.4	1.4
27	.....	38.0	28.9	13.7	3.6	1.4
28	.....	38.7	29.6	14.1	3.7	1.4
29	.....	39.3	30.5	14.7	4.0	1.4
30	.....	39.8	30.8	14.9	4.1	1.4
31	.....	40.3	32.6	17.0	5.5	1.6
32	.....	40.7	32.7	17.3	5.6	1.7
33	.....	40.9	33.2	17.8	5.9	1.7
34	.....	41.2	33.4	18.2	5.9	1.7
35	.....	42.0	33.8	18.5	6.1	1.7
Future lifetime (maximum)	.....	47.8	40.5	26.7	12.6	4.6

[1] For ages under 35, adjusted by usual actuarial procedures to take account of population not exposed to risk for entire age span covered.

Table 2. Pattern of smoking (lifetime history), persons 18 years of age and over, by sex, age, and urban or rural residence, United States, 1955

[Numbers in thousands]

Age (years) and residence	Male												Female				
	Total	Never smoked	Smoked occasionally	Regular smoker									Total	Never smoked	Smoked occasionally	Regular smoker	Not reported
				Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, and pipes	Pattern not reported					
	NUMBER																
United States	49,581	10,704	2,448	33,566	23,411	1,354	1,760	1,280	3,712	808	888	353	2,863	55,096	35,785	2,259	14,953	2,099
18–24	5,405	1,869	315	2,878	2,487	34	65	47	187	2	30	26	343	7,460	4,391	376	2,346	347
25–34	11,063	2,097	402	7,920	6,442	119	143	197	761	67	121	70	646	12,196	6,254	606	4,843	493
35–44	10,755	1,846	400	7,850	5,936	221	163	293	929	81	154	73	659	11,484	6,455	506	4,090	433
45–54	9,100	1,584	432	6,545	4,549	289	240	322	786	96	192	71	539	9,399	6,292	423	2,362	322
55–64	6,934	1,464	366	4,719	2,701	332	323	251	629	200	218	65	385	7,296	5,881	234	933	248
65 and over	6,322	1,844	533	3,654	1,296	359	826	170	420	362	173	48	291	7,261	6,512	114	379	256
Urban	32,572	6,450	1,579	22,573	15,984	1,019	961	955	2,223	565	637	229	1,970	37,453	23,063	1,712	11,150	1,528
18–24	3,421	1,079	196	1,896	1,622	29	47	27	136	-------	24	11	250	4,966	2,687	261	1,765	253
25–34	7,430	1,335	279	5,386	4,419	75	92	146	492	42	81	39	430	8,220	3,950	453	3,470	347
35–44	7,125	1,127	261	5,283	4,011	146	112	223	572	52	117	50	454	7,832	4,035	392	3,085	320
45–54	6,076	1,012	295	4,401	3,133	238	110	235	437	72	134	42	368	6,481	4,093	331	1,826	231
55–64	4,680	882	238	3,288	1,930	257	176	195	377	142	160	51	272	5,036	3,928	192	720	196
65 and over	3,840	1,015	310	2,319	869	274	424	129	209	257	121	36	196	4,918	4,370	83	284	181
Rural nonfarm	10,456	2,299	512	7,092	4,890	215	400	253	911	164	172	87	553	11,473	7,616	421	3,051	385
18–24	1,116	408	60	598	520	5	15	16	27	2	4	9	50	1,651	1,039	94	447	71
25–34	2,535	502	98	1,779	1,412	29	32	39	195	20	27	25	156	2,876	1,536	109	1,120	111
35–44	2,414	433	96	1,743	1,301	52	26	54	246	27	23	14	142	2,372	1,379	89	827	77
45–54	1,766	277	75	1,316	897	40	55	72	181	14	34	23	98	1,787	1,248	75	423	41
55–64	1,285	263	74	885	511	42	68	44	136	34	38	12	63	1,283	1,056	33	164	30
65 and over	1,340	416	109	771	249	47	204	28	126	67	46	4	44	1,504	1,358	21	70	55
Rural farm	6,553	1,955	357	3,901	2,537	120	399	72	578	79	79	37	340	6,170	5,106	126	752	186
18–24	868	382	59	384	345	-------	3	4	24	-------	2	6	43	843	665	21	134	23
25–34	1,100	260	25	755	611	15	19	12	74	5	13	6	60	1,100	768	44	253	35
35–44	1,216	286	43	824	624	23	25	16	111	2	14	9	63	1,280	1,041	25	178	36
45–54	1,258	295	62	828	519	11	75	15	168	10	24	6	73	1,131	951	17	113	50
55–64	969	319	54	546	260	33	79	12	116	24	20	2	50	977	897	9	49	22
65 and over	1,142	413	114	564	178	38	198	13	85	38	6	8	51	839	784	10	25	20

Continued on page 58.

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Table 2. Pattern of smoking (lifetime history), persons 18 years of age and over, by sex, age, and urban or rural residence, United States, 1955—Con.

[Numbers in thousands]

Age (years) and residence	Male													Female				
	Total	Never smoked	Smoked occasionally	Regular smoker								Not reported	Total	Never smoked	Smoked occasionally	Regular smoker	Not reported
				Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, and pipes	Pattern not reported					
	PERCENT																
United States	100.0	21.6	4.9	67.7	47.2	2.7	3.5	2.6	7.5	1.6	1.8	.7	5.8	100.0	65.0	4.1	27.1	3.8
18–24	100.0	34.6	5.8	53.2	46.0	.9	1.2	.9	3.6		1.6	.5	6.3	100.0	58.9	5.0	31.4	4.7
25–34	100.0	19.0	3.6	71.6	58.2	1.1	1.3	1.8	6.9	.9	1.1	.7	5.6	100.0	51.3	5.0	39.7	4.0
35–44	100.0	17.2	3.7	73.0	55.2	2.1	1.5	2.7	8.6	.6	1.4	.7	6.1	100.0	56.2	4.4	33.6	3.8
45–54	100.0	17.4	4.7	71.9	50.0	2.3	2.6	3.5	8.6	1.1	2.1	.8	5.9	100.0	66.9	4.5	25.1	3.4
55–64	100.0	21.1	5.3	68.1	39.0	4.8	4.7	3.6	9.1	2.9	3.1	.9	5.6	100.0	80.6	3.2	12.8	3.4
65 and over	100.0	29.2	8.4	57.8	20.5	5.7	13.1	2.7	6.6	5.7	2.7	.8	4.6	100.0	89.7	1.6	5.2	3.5
Urban	100.0	19.8	4.8	69.3	49.1	3.1	3.0	2.9	6.8	1.7	2.0	.7	6.0	100.0	61.6	4.6	29.8	4.1
18–24	100.0	31.5	5.7	55.4	47.4	.8	1.4	.8	4.0		1.7	.5	7.3	100.0	54.1	5.3	35.5	5.1
25–34	100.0	18.0	3.8	72.5	59.5	1.0	1.2	2.0	6.6	.6	1.1	.5	5.8	100.0	48.1	5.5	42.2	4.2
35–44	100.0	15.8	3.7	74.1	56.3	2.0	1.6	3.1	8.0	.7	1.6	.7	6.4	100.0	51.5	5.0	39.4	4.1
45–54	100.0	16.7	4.9	72.4	51.6	2.9	1.8	3.9	7.2	1.2	2.2	.7	6.1	100.0	63.2	5.1	26.2	4.6
55–64	100.0	18.8	5.1	70.3	41.2	5.5	2.8	4.2	8.1	2.0	3.4	1.1	5.8	100.0	78.0	3.8	14.3	3.9
65 and over	100.0	26.4	8.1	60.1	22.6	7.1	11.0	3.4	5.4	6.7	3.2	.9	5.1	100.0	88.9	1.7	5.6	3.7
Rural nonfarm	100.0	22.0	4.9	67.8	46.8	2.1	3.8	2.4	8.7	1.6	1.6	.8	5.3	100.0	66.4	3.7	26.6	3.4
18–24	100.0	36.6	5.4	53.0	45.6	.4	1.3	1.4	2.4	.4	.4	.4	4.5	100.0	57.7	5.7	27.1	4.3
25–34	100.0	19.8	3.9	70.2	55.7	1.1	1.3	1.5	7.7	.8	1.1	1.0	5.2	100.0	53.4	3.8	38.9	3.9
35–44	100.0	17.9	4.0	72.2	53.9	2.2	1.1	2.2	10.2	1.1	1.6	1.0	5.9	100.0	58.1	3.8	34.9	3.2
45–54	100.0	15.7	4.2	74.5	50.8	2.3	3.1	4.1	10.2	1.8	1.3	1.9	5.5	100.0	69.8	4.2	23.7	2.3
55–64	100.0	20.5	5.6	68.9	39.8	2.3	5.3	3.4	10.6	2.6	3.0	.3	4.9	100.0	82.3	2.6	12.8	2.7
65 and over	100.0	31.0	8.1	57.5	18.6	3.5	15.2	2.1	9.4	5.0	3.4	.3	3.3	100.0	90.3	1.4	4.7	3.7
Rural farm	100.0	29.8	5.4	59.5	38.7	1.8	6.1	1.1	8.8	1.2	1.2	.6	5.2	100.0	82.8	2.0	12.2	3.0
18–24	100.0	44.0	6.8	44.2	39.7		.3	.5	2.8		.5	.7	5.6	100.0	76.9	2.5	15.9	2.7
25–34	100.0	23.6	2.3	68.6	55.6	1.4	2.1	1.3	6.7	.5	1.2	.5	5.3	100.0	68.8	4.0	23.0	3.2
35–44	100.0	23.5	3.5	67.8	51.3	1.9	2.1	1.3	9.1	.6	1.2	.7	5.8	100.0	81.3	2.0	13.9	2.8
45–54	100.0	23.4	4.9	65.8	41.3		6.0	1.2	13.4	.2	1.9	.5	5.2	100.0	84.1	1.5	10.0	4.4
55–64	100.0	32.9	5.6	56.3	26.8	2.4	8.2	1.2	12.0	2.5	2.1	.2	5.2	100.0	91.8	1.9	5.0	2.3
65 and over	100.0	36.2	10.0	49.4	15.6	2.3	17.3	1.1	7.4	3.3	.5	.7	4.5	100.0	93.4	1.2	3.0	2.4

Table 3. Pattern of smoking (lifetime history), persons 18 years of age and over, by sex, geographic region, and urban or rural residence, United States, 1955

[Numbers in thousands]

Region and residence	Male													Female				
	Total	Never smoked	Smoked occasionally	Regular smoker									Not reported	Total	Never smoked	Smoked occasionally	Regular smoker	Not reported
				Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, and pipes	Pattern not reported						
	NUMBER																	
United States	49,581	10,704	2,448	33,566	23,411	1,354	1,760	1,280	3,712	808	888	353	2,863	55,096	35,785	2,259	14,953	2,099
Urban	32,572	6,450	1,579	22,573	15,984	1,019	961	955	2,223	565	637	229	1,970	37,453	23,093	1,712	11,150	1,528
In urbanized areas	24,060	4,779	1,173	16,678	11,948	802	663	708	1,551	390	453	163	1,430	27,223	16,395	1,302	8,366	1,160
1 million or more	13,429	2,800	690	9,207	6,743	472	414	358	715	208	244	53	732	14,808	8,886	701	4,576	645
250,000 to 1 million	5,918	1,102	237	4,192	2,940	224	129	203	458	98	98	42	387	6,889	4,086	341	2,155	307
Under 250,000	4,713	877	246	3,279	2,265	106	120	147	378	84	111	68	311	5,526	3,423	260	1,635	208
Not in urbanized areas	8,512	1,671	406	5,895	4,036	217	298	247	672	175	184	66	540	10,230	6,668	410	2,784	368
Rural nonfarm	10,456	2,299	512	7,092	4,890	215	400	253	911	164	172	87	553	11,473	7,616	421	3,051	385
Rural farm	6,553	1,955	357	3,901	2,537	120	399	72	578	79	79	37	340	6,170	5,106	126	752	186
Northeast	13,663	2,715	664	9,371	6,362	490	511	404	970	283	283	71	913	15,434	9,337	774	4,581	742
Urban	10,902	2,095	542	7,511	5,200	426	374	340	659	240	216	56	754	12,483	7,429	682	3,754	618
Rural nonfarm	2,246	464	99	1,561	992	47	115	56	243	36	57	15	122	2,473	1,541	87	750	95
Rural farm	515	156	23	299	170	17	22	8	68	7	7	.....	37	478	367	5	77	29
North Central	15,269	3,474	745	10,228	6,944	383	633	358	1,292	245	271	102	822	16,256	10,937	563	4,176	580
Urban	9,939	2,036	466	6,866	4,828	259	322	266	773	157	189	72	571	10,984	6,887	419	3,262	416
Rural nonfarm	2,728	568	150	1,887	1,241	75	118	65	268	50	51	19	123	2,944	2,108	101	667	68
Rural farm	2,602	870	129	1,475	875	49	193	27	251	38	31	11	128	2,328	1,942	43	247	96
South	14,003	3,091	726	9,437	6,973	327	438	343	865	187	171	133	749	16,095	11,183	643	3,689	580
Urban	7,029	1,391	353	4,904	3,685	207	152	212	370	106	104	68	381	8,719	5,660	397	2,312	350
Rural nonfarm	3,996	913	192	2,690	1,953	84	121	101	288	51	38	44	201	4,446	3,052	170	1,055	169
Rural Farm	2,978	787	181	1,843	1,325	36	165	30	207	30	29	21	167	2,930	2,471	76	322	61
West	6,646	1,424	313	4,530	3,132	154	178	175	585	93	166	47	379	7,311	4,328	279	2,507	197
Urban	4,702	928	218	3,292	2,271	127	113	137	421	62	128	33	264	5,267	3,087	214	1,822	144
Rural nonfarm	1,486	354	71	954	694	9	46	31	112	27	26	9	107	1,610	915	63	579	53
Rural farm	458	142	24	284	167	18	19	7	52	4	12	5	8	434	326	2	106	.....
	PERCENT																	
United States	100.0	21.6	4.9	67.7	47.2	2.7	3.5	2.6	7.5	1.6	1.8	0.7	5.8	100.0	65.0	4.1	27.1	3.8
Urban	100.0	19.8	4.8	69.3	49.1	3.1	3.0	2.9	6.8	1.7	2.0	.7	6.0	100.0	61.6	4.6	29.8	4.1
In urbanized areas	100.0	19.9	4.9	69.3	49.7	3.3	2.8	2.9	6.4	1.6	1.9	.7	5.9	100.0	60.2	4.8	30.7	4.3
1 million or more	100.0	20.9	5.1	68.6	50.2	3.5	3.1	2.7	6.3	1.5	1.8	.4	5.5	100.0	60.0	4.7	30.9	4.4
250,000 to 1 million	100.0	18.6	4.0	70.8	49.7	3.8	2.2	3.4	7.7	1.7	1.7	.7	6.5	100.0	59.3	4.9	31.3	4.5
Under 250,000	100.0	18.6	5.2	69.6	48.1	2.2	2.5	3.1	8.0	1.8	2.4	1.4	6.6	100.0	61.9	4.7	29.6	3.8
Not in urbanized areas	100.0	19.6	4.8	69.3	47.4	2.5	3.5	2.9	7.9	2.1	2.2	.8	6.3	100.0	65.2	4.0	27.2	3.6
Rural nonfarm	100.0	22.0	4.9	67.8	46.8	2.1	3.8	2.4	8.7	1.6	1.6	.8	5.3	100.0	66.4	3.7	26.6	3.4
Rural farm	100.0	29.8	5.4	59.5	38.7	1.8	6.1	1.1	8.8	1.2	1.2	.6	5.2	100.0	82.8	2.0	12.2	3.0
Northeast	100.0	19.9	4.9	68.6	46.6	3.6	3.7	3.0	7.1	2.1	2.0	.5	6.7	100.0	60.5	5.0	29.7	4.8
Urban	100.0	19.2	5.0	68.9	47.7	3.9	3.4	3.1	6.0	2.2	2.0	.5	6.9	100.0	59.5	5.5	30.1	5.0
Rural nonfarm	100.0	20.7	4.4	69.5	44.2	2.1	5.1	2.5	10.8	1.6	2.5	.7	5.4	100.0	62.3	3.5	30.3	3.8
Rural farm	100.0	30.3	4.5	58.1	33.0	3.3	4.3	1.6	13.2	1.4	1.4	.....	7.2	100.0	76.8	1.0	16.1	6.1
North Central	100.0	22.8	4.9	67.0	45.5	2.5	4.1	2.3	8.5	1.6	1.8	.7	5.4	100.0	67.3	3.5	25.7	3.6
Urban	100.0	20.5	4.7	69.1	48.6	2.6	3.2	2.7	7.8	1.6	1.9	.7	5.7	100.0	62.7	3.8	29.7	3.8
Rural nonfarm	100.0	20.8	5.5	69.2	45.5	2.7	4.3	2.4	9.8	1.8	1.9	.7	4.5	100.0	71.6	3.4	22.7	2.3
Rural farm	100.0	33.4	5.0	56.7	33.6	1.9	7.4	1.0	9.6	1.5	1.2	.4	4.9	100.0	83.4	1.8	10.6	4.1
South	100.0	22.1	5.2	67.4	49.8	2.3	3.1	2.4	6.2	1.3	1.2	.9	5.3	100.0	69.5	4.0	22.9	3.6
Urban	100.0	19.8	5.0	69.8	52.4	2.9	2.2	3.0	5.3	1.5	1.5	1.0	5.4	100.0	64.9	4.6	26.5	4.0
Rural nonfarm	100.0	22.8	4.8	67.3	49.1	2.1	3.0	2.5	7.2	1.3	1.0	1.1	5.0	100.0	68.6	3.8	23.7	3.8
Rural farm	100.0	26.4	6.1	61.9	44.5	1.2	5.5	1.0	7.0	1.0	1.0	.7	5.6	100.0	84.3	2.6	11.0	2.1
West	100.0	21.4	4.7	68.2	47.1	2.3	2.7	2.6	8.8	1.4	2.5	.7	5.7	100.0	59.2	3.8	34.3	2.7
Urban	100.0	19.7	4.6	70.0	48.3	2.7	2.4	2.9	9.0	1.3	2.7	.7	5.6	100.0	58.6	4.1	34.6	2.7
Rural nonfarm	100.0	23.8	4.8	64.2	46.7	.6	3.1	2.1	7.5	1.8	1.7	.6	7.2	100.0	56.8	3.9	36.0	3.3
Rural farm	100.0	31.0	5.2	62.0	36.5	3.9	4.1	1.5	11.4	.9	2.6	1.1	1.7	100.0	75.1	.5	24.4	.....

Table 4. Pattern of smoking (lifetime history), persons 18 years of age and over, according to age, geographic region, and urban or rural residence, by sex and race, United States, 1955

[Numbers in thousands]

Region, race, age (years), and residence	Male													Female				
	Total	Never smoked	Smoked occasionally	Regular smoker									Not reported	Total	Never smoked	Smoked occasionally	Regular smoker	Not reported
				Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, and pipes	Pattern not reported						
	NUMBER																	
United States:																		
White	44,870	9,632	2,109	30,632	21,249	1,202	1,578	1,156	3,539	758	826	324	2,497	49,645	32,363	1,940	13,509	1,833
18–24	4,719	1,607	260	2,556	2,190	28	63	44	179	2	27	23	296	6,531	3,836	329	2,077	289
25–34	9,935	1,883	357	7,143	5,774	111	122	179	726	56	107	68	552	10,828	5,578	504	4,324	422
35–44	9,722	1,643	350	7,162	5,428	173	140	248	890	76	145	62	567	10,284	5,759	419	3,752	354
45–54	8,249	1,429	367	5,983	4,168	246	203	290	753	89	174	60	470	8,493	5,646	386	2,169	292
55–64	6,376	1,352	313	4,364	2,494	304	275	231	599	192	204	65	347	6,727	5,427	210	853	237
65 and over	5,869	1,718	462	3,424	1,195	340	775	164	392	343	169	46	265	6,782	6,117	92	334	239
Urban	29,228	5,729	1,379	20,409	14,328	894	885	848	2,118	535	587	214	1,711	33,417	20,634	1,482	9,989	1,322
Rural nonfarm	9,744	2,089	451	6,698	4,631	198	349	238	888	150	166	78	506	10,698	7,106	372	2,870	350
Rural farm	5,898	1,814	279	3,525	2,290	110	344	70	533	73	73	32	280	5,530	4,633	86	650	161
Nonwhite	4,711	1,072	339	2,934	2,162	152	182	124	173	50	62	29	366	5,451	3,422	319	1,444	266
18–24	686	262	55	322	297	6	2	3	8	-	3	3	47	929	555	47	269	58
25–34	1,130	214	45	777	668	8	21	18	35	11	14	2	94	1,368	676	102	519	71
35–44	1,033	203	50	688	508	48	23	45	39	5	9	11	92	1,200	696	87	338	79
45–54	851	155	65	562	381	43	37	32	33	7	18	11	69	906	646	37	193	30
55–64	558	112	53	335	207	28	48	20	30	8	14	-	38	569	454	24	80	11
65 and over	453	126	71	230	101	19	51	6	28	19	4	2	26	479	395	22	45	17
Urban	3,344	721	200	2,164	1,656	125	76	107	105	30	50	15	259	4,036	2,439	230	1,161	206
Rural nonfarm	712	210	61	394	259	17	51	15	23	14	6	9	47	775	510	49	181	35
Rural farm	655	141	78	376	247	10	55	2	45	6	6	5	60	640	473	40	102	25
North and West:																		
White	33,423	7,130	1,620	22,745	15,385	957	1,255	880	2,773	602	676	217	1,928	36,724	23,384	1,482	10,470	1,388
Urban	23,551	4,619	1,144	16,367	11,285	749	759	686	1,788	448	494	158	1,421	26,575	16,242	1,189	8,094	1,050
Rural nonfarm	6,329	1,355	306	4,328	2,893	124	262	152	616	106	132	43	340	6,939	4,527	245	1,954	213
Rural farm	3,543	1,156	170	2,050	1,207	84	234	42	369	48	50	16	167	3,210	2,615	48	422	125
Nonwhite	2,155	483	102	1,384	1,053	70	67	57	74	19	41	3	186	2,377	1,318	134	794	131
Urban	1,992	440	82	1,302	1,014	63	50	57	65	11	39	3	168	2,259	1,261	126	744	128
Rural nonfarm	131	31	14	74	34	7	17	0	7	7	2	-	12	88	37	6	42	3
Rural farm	32	12	6	8	5	-	-	0	2	1	-	-	6	30	20	2	8	-
South:																		
White	11,447	2,502	489	7,887	5,864	245	323	276	766	156	150	107	569	12,921	8,979	458	3,039	445
Urban	5,677	1,110	235	4,042	3,043	145	126	162	330	87	93	56	290	6,842	4,382	293	1,895	272
Rural nonfarm	3,415	734	145	2,370	1,738	74	87	86	272	44	34	35	166	3,759	2,579	127	916	137
Rural farm	2,355	658	109	1,475	1,083	26	110	28	164	25	23	16	113	2,320	2,018	38	228	36
Nonwhite	2,556	589	237	1,550	1,109	82	115	67	99	31	21	26	180	3,074	2,104	185	650	135
Urban	1,352	281	118	862	642	62	26	50	40	19	11	12	91	1,777	1,178	104	417	78
Rural nonfarm	581	179	47	320	225	10	34	15	16	7	4	9	35	687	473	43	139	32
Rural farm	623	129	72	368	242	10	55	2	43	5	6	5	54	610	453	38	94	25

PERCENT

| | | |

Table 5. Pattern of smoking (lifetime history), persons 18 years of age and over, by sex and major industry group, United States, 1955

[Numbers in thousands]

Major industry group (civilians)	Male													Female				
	Total	Never smoked	Smoked occasionally	Regular smoker									Not reported	Total	Never smoked	Smoked occasionally	Regular smoker	Not reported
				Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, and pipes	Pattern not reported						
	NUMBER																	
Total	49,581	10,704	2,448	33,566	23,411	1,354	1,760	1,280	3,712	808	888	353	2,863	55,096	35,785	2,259	14,953	2,099
Employed	40,260	8,122	1,871	27,922	20,262	1,073	1,011	1,072	3,054	479	694	277	2,345	18,082	10,763	852	5,550	917
Agriculture, forestry, and fisheries	4,412	1,315	268	2,620	1,739	60	261	51	383	38	58	30	209	459	384	4	56	15
Mining	761	114	41	562	438	12	15	14	63	10	8	2	44	19	12	.....	7	.....
Construction	3,375	571	121	2,461	1,845	76	84	100	244	29	60	23	222	119	60	2	52	5
Manufacturing	12,223	2,162	517	8,808	6,552	293	292	347	904	113	206	101	736	4,257	2,418	186	1,374	279
Durable goods	7,751	1,279	331	5,629	4,196	163	170	211	630	69	134	66	512	1,655	815	73	643	124
Nondurable goods	4,472	883	186	3,179	2,356	130	122	136	284	44	72	35	224	2,602	1,603	113	731	155
Transportation, communication, and other public utilities	3,720	658	141	2,733	2,010	107	71	105	304	44	75	17	188	762	417	57	251	37
Railroads	1,222	247	60	854	597	42	22	41	99	16	29	8	61	62	29	5	20	8
Other	2,498	411	81	1,879	1,413	65	49	64	205	28	46	9	127	700	388	52	231	29
Trade	7,452	1,449	321	5,230	3,867	266	102	232	505	81	124	53	452	4,147	2,355	170	1,403	219
Wholesale	2,020	333	67	1,510	1,124	85	38	53	146	15	31	18	110	510	304	12	168	26
Retail	5,432	1,116	254	3,720	2,743	181	64	179	359	66	93	35	342	3,637	2,051	158	1,235	193
Finance, insurance, and real estate	1,301	250	67	918	601	48	43	45	102	37	39	3	66	969	528	62	337	42
Business and repair services	1,212	200	62	852	639	33	14	32	90	20	15	9	98	367	139	9	99	20
Personal services	1,078	205	69	750	587	38	11	31	44	21	8	10	54	2,629	1,711	133	666	119
Entertainment and recreation services	315	56	13	226	175	8	2	17	20	.....	2	2	20	139	68	10	56	5
Professional and related services	2,339	697	139	1,383	871	60	91	43	225	49	39	5	120	3,520	2,226	181	978	135
Public administration	2,072	445	112	1,379	938	72	25	55	170	37	60	22	136	795	445	38	271	41
Not employed	9,321	2,582	577	5,644	3,149	281	749	208	658	329	194	76	518	37,014	25,022	1,407	9,403	1,182
	PERCENT																	
Total	100.0	21.6	4.9	67.7	47.2	2.7	3.5	2.6	7.5	1.6	1.8	0.7	5.8	100.0	64.9	4.1	27.1	3.8
Employed	100.0	20.2	4.6	69.4	50.3	2.7	2.5	2.7	7.6	1.2	1.7	.7	5.8	100.0	59.5	4.7	30.7	5.1
Agriculture, forestry, and fisheries	100.0	29.8	6.1	59.4	39.4	1.4	5.9	1.2	8.7	.9	1.3	.7	4.7	100.0	83.7	.9	12.2	3.3
Mining	100.0	15.0	5.4	73.9	57.6	1.6	2.0	1.8	8.3	1.3	1.1	.3	5.8	(1)	.....	.....	.....	.....
Construction	100.0	16.9	3.6	72.9	54.7	2.3	2.5	3.0	7.2	.9	1.8	.7	6.6	(1)	.....	.....	.....	.....
Manufacturing	100.0	17.7	4.2	72.1	53.6	2.4	2.4	2.8	7.4	.9	1.7	.8	6.0	100.0	56.8	4.4	32.3	6.6
Durable goods	100.0	16.5	4.3	72.6	54.1	2.1	2.2	2.7	8.0	.9	1.7	.9	6.6	100.0	49.2	4.4	38.9	7.5
Nondurable goods	100.0	19.7	4.2	71.1	52.7	2.9	2.7	3.0	6.4	1.0	1.6	.8	5.0	100.0	61.6	4.3	28.1	6.0
Transportation, communication, and other public utilities	100.0	17.7	3.8	73.5	54.0	2.9	1.9	2.8	8.2	1.2	2.0	.5	5.1	100.0	54.7	7.5	32.9	4.9
Railroads	100.0	20.2	4.9	69.9	48.9	3.4	1.8	3.4	8.1	1.3	2.4	.7	5.0	(1)	.....	.....	.....	.....
Other	100.0	16.5	3.2	75.2	56.6	2.6	2.0	2.6	8.2	1.1	1.8	.4	5.1	100.0	55.4	7.4	33.0	4.1
Trade	100.0	19.4	4.3	70.2	51.9	3.6	1.4	3.1	6.8	1.2	1.7	.7	6.1	100.0	56.8	4.1	33.8	5.3
Wholesale	100.0	16.5	3.3	74.8	55.6	4.2	1.9	2.6	7.2	.7	1.5	.9	5.4	100.0	59.6	2.4	32.9	5.1
Retail	100.0	20.5	4.7	68.5	50.5	3.3	1.2	3.3	6.6	1.2	1.7	.6	6.3	100.0	56.4	4.3	34.0	5.3
Finance, insurance, and real estate	100.0	19.2	5.1	70.6	46.2	3.7	3.3	3.5	7.8	2.8	3.0	.2	5.1	100.0	54.5	6.4	34.8	4.3
Business and repair services	100.0	16.5	5.1	70.3	52.7	2.7	1.2	2.6	7.4	1.7	1.2	.7	8.1	100.0	52.1	3.4	37.1	7.5
Personal services	100.0	19.0	6.4	69.6	54.5	3.5	1.0	2.9	4.1	1.9	.7	.9	5.0	100.0	65.1	5.1	25.3	4.5
Entertainment and recreation services	100.0	17.8	4.1	71.7	55.6	2.5	.6	5.4	6.3	.....	.6	.6	6.3	(1)	.....	.....	.....	.....
Professional and related services	100.0	29.8	5.9	59.1	37.2	2.6	3.9	1.8	9.6	2.1	1.7	.2	5.1	100.0	63.2	5.1	27.8	3.8
Public administration	100.0	21.5	5.4	66.6	45.3	3.5	1.2	2.7	8.2	1.8	2.9	1.1	6.6	100.0	56.0	4.8	34.1	5.2
Not employed	100.0	27.7	6.2	60.6	33.8	3.0	8.0	2.2	7.1	3.5	2.1	.8	5.6	100.0	67.6	3.8	25.4	3.2

[1] Percent not shown because of small number of observations.

Table 6. Pattern of smoking (lifetime history), persons 18 years of age and over, by sex and major occupational group, United States, 1955

[Numbers in thousands]

Major occupational group (civilians)	Male												Female				
	Total	Never smoked	Smoked occasionally	Regular smoker								Not reported	Total	Never smoked	Smoked occasionally	Regular smoker	Not reported
				Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, and pipes	Pattern not reported					
	NUMBER																
Total	49,581	10,704	2,448	33,566	23,411	1,354	1,760	1,280	3,712	808	888	353	2,863	55,096	35,785	2,259	14,953	2,099
Employed	40,260	8,122	1,871	27,922	20,262	1,073	1,011	1,072	3,054	479	694	277	2,345	18,082	10,763	852	5,550	917
Professional, technical, and kindred workers:																		
Total	3,671	1,019	211	2,277	1,508	103	118	87	326	64	59	12	164	2,314	1,473	112	640	89
Self-employed	689	173	35	454	295	21	15	31	49	20	23	.....	27	106	59	2	43	2
Salaried	2,982	846	176	1,823	1,213	82	103	56	277	44	36	12	137	2,208	1,414	110	597	87
Farmers and farm managers	3,293	1,023	201	1,930	1,229	51	214	31	305	32	49	19	139	79	63	2	14	.....
Managers, officials, and proprietors, except farm:																		
Total	5,300	1,012	247	3,745	2,533	270	85	217	397	85	124	34	296	984	582	43	319	40
Self-employed	3,014	576	171	2,101	1,398	182	35	133	217	59	54	23	166	598	377	32	175	14
Salaried	2,286	436	76	1,644	1,135	88	50	84	180	26	70	11	130	386	205	11	144	26
Clerical and kindred workers	2,775	589	138	1,888	1,373	55	50	67	219	29	71	24	160	5,463	2,909	309	1,940	305
Sales workers	2,305	435	89	1,662	1,194	66	51	73	191	29	40	18	119	1,255	741	65	378	71
Craftsmen, foremen, and kindred workers	7,710	1,230	310	5,718	4,231	177	177	239	630	67	146	51	452	164	99	5	55	5
Operatives and kindred workers	8,831	1,506	360	6,363	4,976	153	173	200	597	85	126	53	592	3,404	2,100	114	976	214
Private household workers	56	13	6	34	24	2	.....	3	3	.....	.....	2	3	1,623	1,123	87	358	55
Service workers, except private household	2,368	481	100	1,648	1,180	100	37	94	119	57	45	16	139	2,353	1,314	110	806	123
Farm laborers and foremen	910	236	51	563	427	5	37	8	65	4	3	14	60	346	303	.....	33	10
Laborers, except farm and mine	3,051	578	158	2,094	1,587	91	69	53	202	27	31	34	221	97	56	5	31	5
Not employed	9,321	2,582	577	5,644	3,149	281	749	208	658	329	194	76	518	37,014	25,022	1,407	9,403	1,182
Unemployed	2,371	371	91	1,752	1,318	27	86	61	193	22	30	15	157	940	525	57	322	36
Unable to work	1,294	394	77	742	393	40	110	26	84	45	27	17	81	834	661	27	83	63
Keeping house	74	29	3	40	17	.....	9	2	5	7	.....	.....	2	33,688	22,667	1,240	8,777	1,004
Other	5,582	1,788	406	3,110	1,421	214	544	119	376	255	137	44	278	1,552	1,169	83	221	79

Continued on p. 64.

Table 6. Pattern of smoking (lifetime history), persons 18 years of age and over by sex and major occupational group, United States, 1955—Con.

[Numbers in thousands]

Major occupational group (civilian)	Male													Female				
	Total	Never smoked	Smoked occasionally	Regular smoker									Not reported	Total	Never smoked	Smoked occasionally	Regular smoker	Not reported
				Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, pipes	Pattern not reported						
	PERCENT																	
Total	100.0	21.6	4.9	67.7	47.2	2.7	3.5	2.6	7.5	1.6	1.8	.7	5.8	100.0	65.0	4.1	27.1	3.8
Employed	100.0	20.2	4.6	69.3	50.3	2.7	2.5	2.7	7.6	1.2	1.7	.7	5.8	100.0	59.5	4.7	30.7	5.1
Professional, technical, and kindred workers	100.0	27.8	5.7	62.0	41.1	2.8	3.2	2.4	8.9	1.7	1.6	.3	4.5	100.0	63.6	4.8	27.7	3.8
Total	100.0	25.1	5.1	65.9	42.8	3.0	2.2	4.5	7.1	2.9	3.3	----	3.9	----	----	----	----	----
Self-employed	100.0	28.4	5.9	61.1	40.7	2.7	3.5	1.9	9.3	1.5	1.2	.4	4.6	100.0	64.0	5.0	27.0	3.9
Salaried	100.0	31.1	6.1	58.6	37.3	1.5	6.5	.9	9.3	1.0	1.5	.6	4.2	(1)	(1)	(1)	(1)	(1)
Farmers and farm managers	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----
Managers, officials, and proprietors, except farm:	100.0	19.1	4.7	70.7	47.8	5.1	1.6	4.1	7.5	1.6	2.3	.6	5.6	100.0	59.1	4.4	32.4	4.1
Total	100.0	19.1	5.7	69.7	46.4	6.0	1.2	4.4	7.2	2.0	1.6	.8	5.5	100.0	63.0	5.4	29.3	2.3
Self-employed	100.0	19.1	3.3	71.9	49.7	3.8	2.2	3.7	7.9	1.1	.5	.9	5.7	100.0	53.1	2.6	37.3	6.7
Salaried	100.0	21.2	5.0	68.0	49.5	2.0	1.8	2.4	7.9	1.0	3.1	2.6	5.8	100.0	53.2	5.7	35.5	5.6
Clerical and kindred workers	100.0	18.9	3.9	72.1	51.8	2.9	2.2	3.2	8.3	1.3	1.7	.8	5.2	100.0	59.0	5.2	30.1	5.7
Sales workers	100.0	16.0	4.0	74.2	54.9	2.3	2.3	3.1	8.2	.9	1.9	.7	5.9	(1)	----	----	----	----
Craftsmen, foremen, and kindred workers	100.0	17.1	4.1	72.1	56.4	1.7	2.0	2.3	6.8	1.0	1.4	.6	6.7	100.0	61.7	3.3	28.7	6.3
Operatives and kindred workers	----	----	----	----	----	----	----	----	----	----	----	----	----	100.0	69.2	5.4	22.1	3.4
Private household workers	(1)	----	----	----	----	----	----	----	----	----	----	----	----	100.0	55.8	4.7	34.3	5.2
Service workers, except private household	100.0	20.3	4.2	69.6	49.8	4.2	1.6	4.0	5.0	2.4	1.9	.7	5.9	(1)	(1)	(1)	(1)	(1)
Farm laborers and foremen	100.0	25.9	5.6	61.9	46.9	.5	4.1	.9	7.1	.4	.3	1.5	6.6	100.0	57.7	5.2	32.0	5.1
Laborers, except farm and mine	100.0	18.9	5.2	68.6	52.0	3.0	2.3	1.7	6.6	.9	1.0	1.1	7.2	100.0	57.6	3.8	25.4	3.2
Not employed	100.0	27.7	6.2	60.6	33.8	3.0	8.0	2.2	7.1	3.5	2.1	.8	5.6	100.0	67.6	5.1	24.3	3.8
Unemployed	100.0	15.6	2.8	73.9	55.6	1.1	3.6	2.6	8.1	.9	1.3	.6	6.6	100.0	55.9	6.1	34.3	7.6
Unable to work	100.0	30.4	6.0	57.3	30.4	3.1	8.5	2.0	6.5	3.5	2.1	1.3	6.3	100.0	79.3	3.2	10.0	3.0
Keeping house	(1)	----	----	----	----	----	----	----	----	----	----	----	----	100.0	67.3	3.7	26.1	3.0
Other	100.0	32.0	7.3	55.7	25.5	3.8	9.7	2.1	6.7	4.6	2.5	.8	5.0	100.0	75.3	3.3	14.2	3.1

[1] Percent not shown because of small number of observations.

Table 7. Pattern of smoking (lifetime history), regular smokers 18 years of age and over, current smoking status and age started smoking, by sex, United States, 1955

[Numbers in thousands]

Current smoking status and age started smoking (years)	Male									Female
	Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, and pipes	Not reported	
Total	33,566	23,411	1,354	1,760	1,280	3,712	808	888	353	14,953
Smoking pattern reported, total[1]	33,137	23,188	1,323	1,720	1,258	3,648	802	873	325	14,825
Smoker[2]	28,970	20,522	1,102	1,407	1,047	3,247	671	699	275	13,206
No interruption[3]	26,869	19,048	975	1,254	982	3,070	625	671	244	12,339
Interruption	2,101	1,474	127	153	65	177	46	28	31	867
Nonsmoker	4,167	2,666	221	313	211	401	131	174	50	1,619
Before age 14	1,888	1,279	20	63	94	303	19	90	20	230
Smoker	1,647	1,145	20	44	87	261	14	58	18	206
No interruption	1,527	1,069	17	39	77	245	14	53	13	192
Interruption	120	76	3	5	10	16		5	5	14
Nonsmoker	241	134		19	7	42	5	32	2	24
Between 14 and 16	4,194	3,123	40	86	148	597	41	124	35	862
Smoker	3,696	2,776	31	66	119	542	30	102	30	776
No interruption	3,467	2,594	26	57	109	521	28	102	30	721
Interruption	229	182	5	9	10	21	2			55
Nonsmoker	498	347	9	20	29	55	11	22	5	86
Between 16 and 18	8,630	6,674	80	149	290	1,059	89	208	81	2,644
Smoker	7,730	6,022	64	123	249	945	67	189	71	2,333
No interruption	7,302	5,671	54	113	234	910	67	182	71	2,193
Interruption	428	351	10	10	15	35		7		140
Nonsmoker	900	652	16	26	41	114	22	19	10	311
Between 18 and 20	8,089	6,040	179	248	339	849	144	200	90	3,518
Smoker	7,120	5,387	155	192	286	758	118	154	70	3,117
No interruption	6,583	4,987	126	164	272	724	106	144	60	2,897
Interruption	537	400	29	28	14	34	12	10	10	220
Nonsmoker	969	653	24	56	53	91	26	46	20	401
Between 20 and 22	5,430	3,661	304	418	208	464	201	135	39	2,590
Smoker	4,714	3,219	250	359	162	400	177	113	34	2,324
No interruption	4,312	2,950	227	320	155	358	168	107	27	2,163
Interruption	402	269	23	39	7	42	9	6	7	161
Nonsmoker	716	442	54	59	46	64	24	22	5	236
Between 22 and 25	1,646	962	122	175	75	164	92	44	12	1,240
Smoker	1,342	770	98	145	61	148	76	32	12	1,101
No interruption	1,224	699	87	135	59	131	74	32	7	1,040
Interruption	118	71	11	10	2	17	2		5	61
Nonsmoker	304	192	24	30	14	16	16	12		139
Between 25 and 30	1,548	743	249	202	50	124	115	48	17	1,452
Smoker	1,294	607	209	170	42	117	101	34	14	1,315
No interruption	1,160	539	181	154	37	112	91	34	12	1,228
Interruption	134	68	28	16	5	5	10		2	87
Nonsmoker	254	136	40	32	8	7	14	14	3	137
Between 30 and 35	757	319	157	125	36	55	44	14	7	922
Smoker	628	273	123	97	29	48	39	12	7	790
No interruption	554	241	115	75	27	43	34	12	7	735
Interruption	74	32	8	22	2	5	5			55
Nonsmoker	129	46	34	28	7	7	5	2		132
Between 35 and 40	358	152	75	68	10	15	24	7	7	572
Smoker	306	135	67	59	7	10	18	5	5	521
No interruption	275	120	59	55	7	8	16	5	5	478
Interruption	31	15	8	4		2	2			43
Nonsmoker	52	17	8	9	3	5	6	2	2	51
40 or later	597	235	97	186	8	18	33	3	17	826
Smoker	493	188	85	152	5	18	31		14	723
No interruption	465	178	83	142	5	18	27		12	692
Interruption	28	10	2	10			4		2	31
Nonsmoker	104	47	12	34	3	2	2	3	3	102
Smoking pattern not reported	429	223	31	40	22	64	6	15	28	128

[1] Excludes persons for whom data on current smoking status or age started smoking were not reported.
[2] Includes persons who are currently occasional smokers.
[3] See statement in text on p. 26.

Table 8. Regular smokers 18 years of age and over (any form of tobacco, lifetime history), by age, sex, urban or rural residence, and age started smoking, United States, 1955

[Numbers in thousands]

Age started smoking (years)	Age (years)													
	Male							Female						
	Total 18 and over	18–24	25–34	35–44	45–54	55–64	65 and over	Total 18 and over	18–24	25–34	35–44	45–54	55–64	65 and over
United States	33,566	2,878	7,920	7,850	6,545	4,719	3,654	14,953	2,346	4,843	4,090	2,362	903	379
Under 14	1,888	189	367	434	359	278	261	230	65	77	33	24	15	16
14–15	4,194	456	1,021	997	739	548	433	862	292	312	172	40	31	15
16–17	8,630	1,192	2,585	2,029	1,478	829	517	2,644	823	1,061	604	128	17	11
18–19	8,089	781	2,270	2,051	1,505	1,010	472	3,518	855	1,464	862	260	46	31
20–21	5,430	196	1,071	1,251	1,301	958	653	2,560	257	973	864	376	69	21
22–24	1,646	37	385	408	327	281	208	1,240	43	531	419	207	32	8
25–29	1,548	2	117	358	361	328	382	1,452	2	343	600	375	100	32
30–34	757	.....	32	155	194	141	235	922	.....	61	344	344	144	29
35–39	358	.....	.....	61	93	110	94	572	.....	2	145	275	114	36
40 or more	597	2	.....	13	115	168	299	825	.....	.....	22	290	348	165
Not reported	429	23	72	93	73	68	100	128	9	19	25	43	17	15
Urban	22,573	1,896	5,386	5,283	4,401	3,288	2,319	11,150	1,765	3,470	3,085	1,826	720	284
Under 14	1,197	122	224	267	235	192	157	151	54	38	26	17	2	14
14–15	2,727	301	691	639	470	367	257	619	206	222	135	26	24	6
16–17	5,717	783	1,764	1,328	954	563	325	1,934	619	734	452	94	15	.....
18–19	5,498	518	1,551	1,395	1,035	680	319	2,621	650	1,084	650	187	26	24
20–21	3,863	133	771	873	916	711	459	1,982	176	733	695	306	55	17
22–24	1,091	17	227	292	231	198	126	935	34	386	321	155	32	5
25–29	1,055	.....	79	259	252	232	233	1,049	2	221	419	302	80	25
30–34	497	.....	24	106	130	108	129	720	.....	40	253	284	118	25
35–39	234	.....	.....	44	56	81	53	425	.....	.....	97	214	89	25
40 or more	377	2	.....	8	69	103	195	620	.....	.....	10	208	270	132
Not reported	317	18	55	72	53	53	66	94	4	12	25	33	9	11
Rural	10,993	982	2,534	2,567	2,144	1,431	1,335	3,803	581	1,373	1,005	536	213	95
Under 14	691	67	143	167	124	86	104	79	11	39	7	7	13	2
14–15	1,467	153	330	358	269	181	176	243	86	90	37	14	7	9
16–17	2,913	409	821	701	524	266	192	710	184	327	152	34	2	11
18–19	2,591	263	719	656	470	330	153	897	205	380	212	73	20	7
20–21	1,567	63	300	378	385	247	194	578	81	240	169	70	14	4
22–24	555	20	158	116	96	81	82	305	9	145	96	52	.....	3
25–29	493	2	38	99	109	96	149	403	.....	122	181	73	20	7
30–34	260	.....	8	49	64	33	106	202	.....	21	91	60	26	4
35–39	124	.....	.....	17	37	29	41	147	.....	2	48	61	25	11
40 or more	220	.....	.....	5	46	65	104	205	.....	.....	12	82	78	33
Not reported	112	5	17	21	20	15	34	34	5	7	.....	10	8	4

Table 9. Former regular smokers 18 years of age and over now occasional or nonsmokers, according to pattern of smoking (lifetime history), by sex, urban or rural residence, current smoking status, and years stopped smoking, United States, 1955

[Numbers in thousands]

Current smoking status, and years stopped smoking	Male									Female
	Total	Cigarettes only	Cigars only	Pipes only	Cigarettes and cigars	Cigarettes and pipes	Cigars and pipes	Cigarettes, cigars, and pipes	Not reported	
TOTAL										
	4,869	3,097	287	399	232	455	158	183	58	1,900
Nonsmoker [1]	4,167	2,666	221	313	211	401	131	174	50	1,619
Less than 1½	1,172	795	43	39	67	148	25	45	10	559
1½–2½	469	321	14	24	20	52	14	17	7	171
2½–4½	484	316	13	31	19	55	23	23	4	219
4½–9½	743	488	42	48	31	71	12	40	11	361
9½ or more	1,289	739	109	168	74	75	57	49	18	291
Not reported	10	7		3						18
Occasional smoker	702	431	66	86	21	54	27	9	8	281
URBAN										
	3,139	2,028	205	174	173	275	106	134	44	1,253
Nonsmoker	2,712	1,770	151	134	159	245	84	127	42	1,049
Less than 1½	783	551	20	13	60	87	17	25	10	367
1½–2½	311	207	9	18	15	30	10	17	5	107
2½–4½	333	218	11	14	12	35	18	21	4	134
4½–9½	473	314	32	22	19	48	5	25	8	241
9½ or more	807	475	79	67	53	45	34	39	15	186
Not reported	5	5								14
Occasional smoker	427	258	54	40	14	30	22	7	2	204
RURAL										
	1,730	1,069	82	225	59	180	52	49	14	647
Nonsmoker	1,455	896	70	179	52	156	47	47	8	570
Less than 1½	389	244	23	26	7	61	8	20		192
1½–2½	158	114	5	6	5	22	4		2	64
2½–4½	151	98	2	17	7	20	5	2		85
4½–9½	270	174	10	26	12	23	7	15	3	120
9½ or more	482	264	30	101	21	30	23	10	3	105
Not reported	5	2		3						4
Occasional smoker	275	173	12	46	7	24	5	2	6	77

[1] Former regular smokers of 2 or more forms of tobacco are classified as nonsmokers only if they have discontinued regular or occasional smoking of all forms of tobacco.

Table 10a. Former regular male *cigarette* smokers, 18 years of age and over, according to current smoking status and years stopped smoking *cigarettes*, United States, 1955

[Numbers in thousands]

Years stopped smoking	Total	Non-smoker at present	Occasional cigar or pipe smoker	Regular smoker				Not reported
				Total	Cigars only	Pipes only	Cigars and pipes	
Total [1]	*4,597*	*3,600*	*233*	*757*	*507*	*580*	*70*	*7*
Less than 1½	1,351	1,039	74	235	81	131	23	3
1½–2½	533	416	27	90	32	47	11	----
2½–4½	514	428	21	65	40	22	3	----
4½–9½	814	654	47	111	47	59	5	2
9½ or more	1,356	1,046	59	249	104	117	28	2
Not reported	29	17	5	7	3	4	----	----

[1] Excludes persons who are now occasional cigarette smokers.

Table 10b. Former regular male *cigar* smokers, 18 years of age and over, according to current smoking status and years stopped smoking *cigars*, United States, 1955

[Numbers in thousands]

Years stopped smoking	Total	Non-smoker at present	Occasional cigarette or pipe smoker	Regular smoker				Not reported
				Total	Cigarettes only	Pipes only	Cigarettes and pipes	
Total [1]	*1,416*	*796*	*29*	*591*	*484*	*78*	*29*	----
Less than 1½	231	148	5	78	61	8	9	----
1½–2½	94	58	7	29	22	5	2	----
2½–4½	110	72	----	38	33	5	----	----
4½–9½	211	123	2	86	66	12	8	----
9½ or more	731	387	12	332	277	45	10	----
Not reported	39	8	3	28	25	3	----	----

[1] Excludes persons who are now occasional cigar smokers.

Table 10c. Former regular male *pipe* smokers, 18 years of age and over, according to current smoking status and years stopped smoking *pipes*, United States, 1955

[Numbers in thousands]

Years stopped smoking	Total	Non-smoker at present	Occasional cigarette or cigar smoker	Regular smoker				Not reported
				Total	Cigarettes only	Cigars only	Cigarettes and cigars	
Total [1]	*3,299*	*1,083*	*112*	*2,098*	*1,831*	*203*	*73*	----
Less than 1½	442	171	17	254	221	25	8	----
1½–2½	257	92	9	156	135	21	----	----
2½–4½	296	110	10	176	151	22	3	----
4½–9½	555	182	15	358	293	35	29	----
9½ or more	1,637	522	48	1,067	941	96	30	----
Not reported	112	12	13	87	80	2	5	----

[1] Excludes persons who are now occasional pipe smokers.

Table 11. Former regular smokers, 18 years of age and over, now occasional or nonsmokers, classified as cigarette smoker only or as other smoker (lifetime history), by sex, age, urban or rural residence, current smoking status, and years stopped smoking, United States, 1955

[Numbers in thousands]

Residence, current smoking status, and years stopped smoking	All smokers, 18 years and over			18–24 years			25–34 years			35–44 years			45–54 years			55–64 years			65 years and over		
	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker
	MALE																				
Total	4,869	3,097	1,772	140	131	9	853	666	187	947	715	232	911	642	269	985	561	424	1,033	382	651
Nonsmoker	4,167	2,666	1,501	116	111	5	715	562	153	804	607	197	787	557	230	872	500	372	873	329	544
Less than 1½	1,172	795	377	89	86	3	314	247	67	273	191	82	197	131	66	168	88	80	131	52	79
1½–2½	469	321	148	10	10	.....	113	91	22	93	80	13	86	52	34	100	53	47	67	35	32
2½–4½	484	316	168	10	8	2	129	96	33	93	69	24	90	77	13	84	42	42	78	24	54
4½–9½	743	488	255	7	7	.....	118	92	26	177	136	41	161	110	51	154	92	62	126	51	75
9½ or more	1,289	739	550	.....	.....	.....	41	36	5	163	126	37	253	187	66	366	225	141	466	165	301
Not reported	10	7	3	.....	.....	.....	.....	.....	.....	5	5	.....	.....	.....	.....	.....	.....	.....	5	2	3
Occasional smoker	702	431	271	24	20	4	138	104	34	143	108	35	124	85	39	113	61	52	160	53	107
Urban	3,139	2,028	1,111	83	79	4	550	429	121	584	427	157	594	428	166	682	409	273	646	256	390
Nonsmoker	2,712	1,770	942	72	72	.....	461	368	93	511	377	134	511	367	144	615	368	247	542	218	324
Less than 1½	783	551	232	57	57	.....	225	182	43	190	138	52	127	80	47	116	65	51	68	29	39
1½–2½	311	207	104	8	8	.....	62	54	8	54	45	9	72	43	29	69	33	36	46	24	22
2½–4½	333	218	115	5	5	.....	89	63	26	61	44	17	76	65	11	56	29	27	46	12	34
4½–9½	473	314	159	2	2	.....	62	46	16	117	86	31	84	63	21	127	79	48	81	38	43
9½ or more	807	475	332	.....	.....	.....	23	23	.....	84	59	25	152	116	36	247	162	85	301	115	186
Not reported	5	5	.....	.....	.....	.....	.....	.....	.....	5	5	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Occasional smoker	427	258	169	11	7	4	89	61	28	73	50	23	83	61	22	67	41	26	104	38	66
Rural	1,730	1,069	661	57	52	5	303	237	66	363	288	75	317	214	103	303	152	151	387	126	261
Nonsmoker	1,455	896	559	44	39	5	254	194	60	293	230	63	276	190	86	257	132	125	331	111	220
Less than 1½	389	244	145	32	29	3	89	65	24	83	53	30	70	51	19	52	23	29	63	23	40
1½–2½	158	114	44	2	2	.....	51	37	14	39	35	4	14	9	5	31	20	11	21	11	10
2½–4½	151	98	53	5	3	2	40	33	7	32	25	7	14	12	2	28	13	15	32	12	20
4½–9½	270	174	96	5	5	.....	56	46	10	60	50	10	77	47	30	27	13	14	45	13	32
9½ or more	482	264	218	.....	.....	.....	18	13	5	79	67	12	101	71	30	119	63	56	165	50	115
Not reported	5	2	3	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	5	2	3
Occasional smoker	275	173	102	13	13	.....	49	43	6	70	58	12	41	24	17	46	20	26	56	15	41

Continued on page 70.

Table 11. Former regular smokers, 18 years of age and over, now occasional or nonsmokers, classified as cigarette smoker only or as other smoker (lifetime history), by sex, age, urban or rural residence, current smoking status, and years stopped smoking, United States, 1955—Con.

[Numbers in thousands]

Residence, current smoking status, and years stopped smoking	All smokers, 18 years and over			18–24 years			25–34 years			35–44 years			45–54 years			55–64 years			65 years and over		
	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker	Total	Cigarette smoker only	Other smoker
	FEMALE [1]																				
Total	1,900			230			651			477			286			150			106		
Nonsmoker	1,619			188			548			411			255			127			90		
Less than 1½	559			123			181			128			75			43			9		
1½–2½	171			26			61			51			19			7			7		
2½–4½	219			26			80			53			29			21			10		
4½–9½	361			8			172			90			53			20			18		
9½ or more	291			3			44			87			77			36			44		
Not reported	18			2			10			2			2						2		
Occasional smoker	281			42			103			66			31			23			16		
Urban	1,253			151			372			329			209			110			82		
Nonsmoker	1,049			120			301			277			189			96			66		
Less than 1½	367			76			108			85			58			36			4		
1½–2½	107			18			32			32			17			5			3		
2½–4½	134			20			38			30			19			19			8		
4½–9½	241			6			95			73			39			15			13		
9½ or more	186						20			55			54			21			36		
Not reported	14						8			2			2						2		
Occasional smoker	204			31			71			52			20			14			16		
Rural	647			79			279			148			77			40			24		
Nonsmoker	570			68			247			134			66			31			24		
Less than 1½	192			47			73			43			17			7			5		
1½–2½	64			8			29			19			2			2			4		
2½–4½	85			6			42			23			10			2			2		
4½–9½	120			2			77			17			14			5			5		
9½ or more	105			3			24			32			23			15			8		
Not reported	4			2			2														
Occasional smoker	77			11			32			14			11			9					

[1] Data by type of smoking not shown separately for females.

Table 12. Current rate of cigarette smoking, persons 18 years of age and over, by sex, age, and identity of respondent, United States, 1955

[Numbers in thousands]

Identity and age (years) of respondent	Total	Nonsmoker of cigarettes				Cigarette smoker											
						Occasional			Regular								
										Regularly smokes cigarettes only					Regularly smokes cigarettes and cigars or pipe (by number of cigarettes)				
		Total	Never smoked cigarettes	Previously smoked cigarettes		Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily
				Occasionally	Regularly														
	MALE																		
Self-respondent	26,844	11,539	8,041	411	3,087	1,135	824	311	14,170	12,939	1,915	7,400	3,321	303	1,231	380	592	229	30
18–24	2,163	796	694	28	74	88	70	18	1,279	1,191	241	750	185	15	88	31	33	24	----
25–34	5,507	1,734	1,157	62	515	213	160	53	3,560	3,305	360	1,999	887	59	255	55	147	50	3
35–44	5,643	1,845	1,148	67	630	214	140	74	3,584	3,292	386	1,823	970	113	292	54	162	65	11
45–54	4,945	1,840	1,119	78	643	212	165	47	2,893	2,647	357	1,446	763	81	246	78	120	46	2
55–64	4,161	2,115	1,371	67	677	187	137	50	1,859	1,646	301	923	398	24	213	78	93	38	4
65 and over	4,425	3,209	2,552	109	548	221	152	69	995	858	270	459	118	11	137	84	37	6	10
Response given by another member of family	19,492	8,434	6,785	173	1,476	849	689	160	10,209	9,654	1,712	5,691	2,089	162	555	175	290	80	10
18–24	2,829	1,360	1,280	11	69	170	155	15	1,299	1,268	358	728	179	3	31	11	15	5	----
25–34	4,796	1,613	1,263	23	327	153	115	38	3,030	2,936	450	1,866	581	29	104	18	52	31	3
35–44	4,370	1,564	1,151	42	371	182	141	41	2,624	2,482	361	1,419	643	59	142	37	88	17	----
45–54	3,577	1,466	1,104	47	315	166	127	39	1,945	1,779	239	1,028	444	48	166	53	96	12	5
55–64	2,336	1,246	964	30	252	123	103	20	967	887	179	502	189	17	80	34	32	12	2
65 and over	1,584	1,185	1,023	20	142	55	48	7	344	312	105	148	53	6	32	22	7	3	----
	FEMALE																		
Self-respondent	43,730	30,730	28,778	404	1,548	1,807	1,548	259	11,193	11,145	3,952	6,099	1,038	56	48	18	27	3	----
18–24	5,054	3,097	2,869	38	190	284	253	31	1,673	1,664	698	853	113	----	9	4	5	----	----
25–34	10,096	5,891	5,249	115	527	543	442	101	3,662	3,648	1,169	2,115	341	23	14	4	7	3	----
35–44	9,464	5,870	5,365	104	401	388	322	66	3,206	3,197	1,068	1,809	306	14	9	4	5	----	----
45–54	7,700	5,605	5,276	79	250	309	283	26	1,786	1,776	609	942	211	14	10	2	8	----	----
55–64	5,928	5,067	4,907	30	121	194	173	21	667	661	301	296	59	5	6	3	3	----	----
65 and over	5,488	5,199	5,112	29	58	89	75	14	200	200	107	84	9	----	----	----	----	----	----
Response given by another member of family	8,947	6,981	6,820	51	110	269	249	20	1,697	1,693	659	861	148	25	4	2	2	----	----
18–24	2,024	1,515	1,494	7	14	82	77	5	427	427	200	199	28	----	----	----	----	----	----
25–34	1,539	1,008	967	11	30	42	42	----	489	485	177	247	51	10	4	2	2	----	----
35–44	1,565	1,109	1,070	10	29	76	68	8	380	380	133	213	31	3	----	----	----	----	----
45–54	1,313	1,011	981	15	15	43	38	5	259	259	98	128	24	9	----	----	----	----	----
55–64	1,066	948	933	5	10	19	19	----	99	99	29	55	12	3	----	----	----	----	----
65 and over	1,441	1,392	1,376	3	13	7	5	2	42	42	22	18	2	----	----	----	----	----	----

Table 13a. Current rate of *cigarette* smoking, persons 18 years of age and over, by sex, age, and urban or rural residence, United States, 1955

[Numbers in thousands]

Residence and age (in years)	Total	Nonsmoker of cigarettes				Cigarette smoker														Not reported
						Occasional			Regular											
										Regularly smokes cigarettes only					Regularly smokes cigarettes and cigars or pipes (by amount of cigarettes) [2]					
		Total	Never smoked cigarettes	Previously smoked cigarettes		Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly [1]	Total [1]	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	
				Occasionally	Regularly [1]															
	MALE																			
United States	49,581	20,219	15,030	592	4,597	2,005	1,523	482	24,667	22,852	3,657	13,238	5,481	476	1,815	559	900	317	39	2,690
18–24	5,405	2,202	2,019	39	144	262	229	33	2,605	2,486	601	1,496	370	19	119	44	46	29	.....	336
25–34	11,065	3,404	2,465	88	851	377	280	97	6,655	6,289	812	3,910	1,479	88	366	76	202	82	6	629
35–44	10,755	3,437	2,314	108	1,015	398	281	117	6,286	5,846	760	3,275	1,632	179	440	89	260	81	10	634
45–54	9,100	3,325	2,241	127	957	379	291	88	4,899	4,477	623	2,500	1,225	129	422	130	219	66	7	497
55–64	6,934	3,392	2,355	100	937	312	243	69	2,865	2,571	486	1,442	598	45	294	111	127	50	6	365
65 and over	6,322	4,459	3,636	130	693	277	199	78	1,357	1,183	375	615	177	16	174	109	46	9	10	229
Urban	32,572	12,616	9,250	349	3,017	1,312	1,025	287	16,791	15,618	2,391	8,953	3,920	354	1,173	356	566	223	28	1,853
18–24	3,421	1,302	1,186	28	88	161	141	20	1,711	1,634	377	1,000	247	10	77	29	24	24	.....	247
25–34	7,430	2,182	1,582	40	560	262	201	61	4,571	4,345	517	2,680	1,086	62	226	45	123	52	6	415
35–44	7,125	2,156	1,445	72	639	242	181	61	4,294	4,008	497	2,220	1,151	140	286	51	165	62	8	433
45–54	6,076	2,136	1,459	73	604	260	205	55	3,336	3,041	419	1,651	875	96	295	100	141	49	5	344
55–64	4,680	2,214	1,489	55	670	206	161	45	1,995	1,806	322	1,018	434	32	189	68	88	29	4	265
65 and over	3,840	2,626	2,089	81	456	181	135	45	884	784	259	384	127	14	100	63	25	7	5	149
Rural nonfarm	10,456	4,339	3,172	150	1,017	403	286	117	5,187	4,785	753	2,831	1,121	80	402	114	223	56	9	827
18–24	1,116	471	440	4	27	53	45	8	542	522	134	304	84	.....	20	5	10	5	.....	50
25–34	2,535	848	596	35	217	75	55	20	1,460	1,367	183	850	310	24	93	25	49	19	.....	152
35–44	2,414	836	544	28	264	94	54	40	1,340	1,239	152	715	344	28	101	21	64	14	2	144
45–54	1,766	639	401	26	212	59	41	18	978	895	110	543	225	17	83	12	59	10	2	90
55–64	1,285	598	404	28	166	66	53	13	562	504	108	261	127	8	58	23	26	7	2	59
65 and over	1,340	947	787	29	131	56	38	18	305	258	66	158	31	3	47	28	15	1	3	32
Rural farm	6,553	3,264	2,608	93	563	290	212	78	2,689	2,449	513	1,454	440	42	240	89	111	38	2	310
18–24	868	429	393	7	29	48	43	5	352	330	90	192	39	9	22	10	12	.....	.....	39
25–34	1,100	374	287	13	74	40	24	16	624	577	112	380	83	2	47	6	30	11	.....	62
35–44	1,216	445	325	8	112	62	46	16	652	599	111	340	137	11	53	17	31	5	.....	57
45–54	1,258	550	381	28	141	60	45	15	585	541	94	306	125	16	44	18	19	7	.....	63
55–64	969	580	462	17	101	40	29	11	308	261	56	163	38	4	47	20	13	14	.....	41
65 and over	1,142	886	760	20	106	40	25	15	168	141	50	73	18	.....	27	18	6	1	2	48

FEMALE

United States	55,096	38,048	35,927	457	1,664	2,086	1,803	283	12,986	12,930	4,640	7,013	1,196	81	.....	.....	.....	1,976
18–24	7,460	4,651	4,403	44	204	369	330	39	2,118	2,110	901	1,666	143	.....	.....	.....	.....	322
25–34	12,196	6,950	6,261	127	562	589	487	102	4,192	4,172	1,364	2,378	396	32	.....	.....	.....	465
35–44	11,484	7,020	6,476	114	630	466	390	76	3,596	3,584	1,206	2,029	330	19	.....	.....	.....	402
45–54	9,399	6,674	6,316	94	264	353	323	30	2,667	2,657	709	1,684	241	23	.....	.....	.....	305
55–64	7,296	6,080	5,904	44	132	212	192	30	767	761	330	351	73	7	.....	.....	.....	237
65 and over	7,261	6,673	6,567	34	72	97	81	16	246	246	130	105	11	.....	.....	.....	.....	245
Urban	37,453	24,542	23,148	221	1,073	1,601	1,396	205	9,858	9,817	3,489	5,305	955	68	.....	.....	.....	1,452
18–24	4,966	2,845	2,699	30	126	269	241	28	1,614	1,610	652	837	121	.....	.....	.....	.....	288
25–34	8,220	4,335	3,953	94	305	433	364	69	3,093	3,089	1,007	1,734	313	26	.....	.....	.....	339
35–44	7,822	4,412	4,045	77	290	374	314	60	2,759	2,729	691	1,561	270	17	.....	.....	.....	296
45–54	6,481	4,377	4,113	70	194	275	235	30	1,646	1,601	569	921	190	21	.....	.....	.....	221
55–64	5,036	4,073	3,959	32	102	177	165	12	601	595	271	266	54	4	.....	.....	.....	185
65 and over	4,918	4,480	4,399	28	53	73	57	16	192	192	99	86	7	.....	.....	.....	.....	173
Rural nonfarm	11,473	8,210	7,648	112	450	369	308	61	2,542	2,532	917	1,398	204	13	.....	.....	.....	332
18–24	1,651	1,124	1,039	21	64	80	71	9	385	381	182	183	16	.....	.....	.....	.....	62
25–34	2,876	1,757	1,538	23	196	116	88	28	903	901	295	533	77	6	.....	.....	.....	100
35–44	2,372	1,619	1,387	28	104	71	61	10	711	710	265	395	48	2	.....	.....	.....	71
45–54	1,757	1,325	1,250	22	53	61	53	8	394	391	99	215	45	2	.....	.....	.....	37
55–64	1,262	1,092	1,041	12	19	36	30	6	133	133	50	66	14	3	.....	.....	.....	32
65 and over	1,504	1,383	1,373	6	14	15	15	.....	46	46	26	16	4	.....	.....	.....	.....	30
Rural farm	6,170	5,296	5,131	24	141	116	99	17	586	581	234	310	37	.....	.....	.....	.....	172
18–24	843	682	665	3	14	20	18	2	119	119	67	46	6	.....	.....	.....	.....	22
25–34	1,100	838	770	10	58	40	35	5	196	191	62	121	8	.....	.....	.....	.....	26
35–44	1,280	1,089	1,044	9	36	21	15	6	135	135	60	73	12	.....	.....	.....	.....	35
45–54	1,131	972	953	2	17	17	15	2	95	95	41	48	6	.....	.....	.....	.....	47
55–64	977	915	904	.....	11	9	7	2	33	33	9	19	5	.....	.....	.....	.....	20
65 and over	839	800	795	.....	5	9	9	.....	8	8	5	3	.....	.....	.....	.....	.....	22

1 Due to slight difference in classification by lifetime history and current status for the 3 forms of tobacco, the totals for regular smokers for each form in appendix table 13 do not agree exactly with those in table 2 of text.

* Number and percent not shown for females because of small number of observations.

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Table 13b. Current rate of *cigarette* smoking, persons 18 years of age and over, according to sex, age, and urban or rural residence, United States, 1955

[Percent]

Residence and age (years)	Total	Nonsmoker of cigarettes				Cigarette smoker																Not reported
		Total	Never smoked cigarettes	Previously smoked cigarettes		Total	Occasional			Regular												
				Occasionally	Regularly		Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Cigarettes only						Cigarettes and cigars or pipes (by number of cigarettes) [1]					
										Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	
MALE																						
United States	100.0	40.8	30.3	1.2	9.3	4.0	3.1	1.0	49.8	46.1	7.4	26.7	11.1	1.0	3.7	1.1	1.8	0.6	0.1	5.4
18–24	100.0	40.7	37.4	.7	2.7	4.8	4.2	.6	48.2	46.0	11.1	27.7	6.8	.4	2.2	.8	.9	.5	.....	6.2
25–34	100.0	30.8	22.3	.8	7.7	3.4	2.5	.9	60.1	56.8	7.3	35.3	13.4	.8	3.3	.7	1.8	.7	.1	5.7
35–44	100.0	22.0	21.5	1.0	9.4	3.7	2.6	1.1	58.4	54.4	7.1	30.5	15.2	1.7	4.1	.8	2.4	.8	.1	5.9
45–54	100.0	26.5	24.6	1.4	10.5	4.2	3.2	1.0	53.8	49.2	6.8	27.5	13.5	1.4	4.6	1.4	2.4	.7	.1	5.5
55–64	100.0	48.9	34.0	1.4	12.5	4.5	3.5	1.0	41.3	37.1	7.0	20.8	8.6	1.6	4.2	1.6	1.9	.7	.1	5.3
65 and over	100.0	70.5	57.5	2.1	11.0	4.4	3.1	1.2	21.5	18.7	6.9	9.7	2.8	.3	2.8	1.7	.7	.1	.2	3.6
Urban	100.0	38.7	28.4	1.1	9.3	4.0	3.1	.9	51.6	47.9	7.3	27.5	12.0	1.1	3.6	1.1	1.7	.7	.1	5.7
18–24	100.0	38.1	34.7	.6	2.6	4.7	4.1	.6	50.0	47.8	11.0	29.2	7.2	.3	2.3	.6	.7	.7	.....	7.2
25–34	100.0	29.4	21.3	.5	7.5	3.5	2.7	.8	61.5	58.5	7.0	36.1	14.6	.8	3.0	.6	1.7	.7	.1	5.6
35–44	100.0	20.3	20.3	1.0	9.0	3.4	2.5	.9	60.3	56.3	7.0	31.2	16.2	2.0	4.0	.7	2.3	.9	.1	6.1
45–54	100.0	25.2	24.0	1.2	9.9	4.3	3.4	.9	54.9	50.0	6.9	27.2	14.4	1.6	4.9	1.6	2.3	.8	.1	5.7
55–64	100.0	47.3	31.8	1.2	14.3	4.4	3.4	1.0	42.6	38.6	6.9	21.8	9.3	1.7	4.0	1.5	1.9	.7	.1	5.7
65 and over	100.0	68.4	54.4	2.1	11.9	4.7	3.5	1.2	23.0	20.4	6.7	10.0	3.3	.4	2.6	1.6	.7	.2	.....	3.9
Rural nonfarm	100.0	41.5	30.3	1.4	9.7	3.9	2.7	1.1	49.6	45.8	7.2	27.1	10.7	.8	3.8	1.1	2.1	.5	.1	5.0
18–24	100.0	42.2	39.4	.4	2.4	4.7	4.0	.7	48.6	46.8	12.6	27.2	7.5	.4	1.8	.4	.9	.4	.....	4.6
25–34	100.0	33.5	23.5	1.4	8.6	3.0	2.2	.8	57.6	53.9	7.2	33.6	12.2	.9	3.7	1.0	1.9	.7	.1	6.0
35–44	100.0	24.6	22.5	1.2	10.0	3.9	2.8	1.1	55.5	51.3	6.3	29.6	14.3	1.2	4.2	.9	2.7	.6	.1	6.0
45–54	100.0	36.2	32.7	1.5	12.0	3.3	2.3	1.0	55.4	50.7	6.2	30.7	12.7	1.0	4.7	.7	2.3	.6	.1	5.1
55–64	100.0	46.5	31.4	2.2	12.9	5.1	4.1	1.0	43.7	39.2	6.4	20.3	9.9	.6	4.5	1.8	2.0	.5	.2	4.7
65 and over	100.0	70.7	58.7	2.2	9.8	4.2	2.8	1.3	22.8	19.3	4.9	11.8	2.3	.3	3.5	2.1	1.1	.1	.2	2.4
Rural farm	100.0	49.8	39.8	1.4	8.6	4.4	3.2	1.2	41.0	37.4	7.8	22.2	6.7	.6	3.7	1.4	1.7	.6	.....	4.7
18–24	100.0	49.4	45.3	.8	2.3	5.5	5.0	.6	40.6	38.0	10.4	22.1	4.5	1.0	2.5	1.2	1.4	.....	.....	4.5
25–34	100.0	34.0	26.1	1.2	6.7	3.6	2.8	1.5	56.7	52.5	10.2	34.5	7.5	.2	4.3	.5	2.7	1.0	.....	5.6
35–44	100.0	26.6	26.7	.7	9.2	5.1	3.8	1.3	53.6	49.3	9.1	28.0	11.3	1.3	4.4	1.4	2.6	.4	.....	4.7
45–54	100.0	43.7	30.3	2.2	11.2	4.8	3.6	1.2	46.5	43.0	7.5	24.3	9.9	1.3	3.5	1.4	1.6	.6	.....	5.0
55–64	100.0	59.9	47.7	1.8	10.4	4.1	3.0	1.1	31.8	28.9	5.8	16.8	5.9	.4	2.9	2.1	1.3	1.4	.....	4.2
65 and over	100.0	77.6	66.5	1.8	9.3	3.5	2.2	1.3	14.7	12.3	4.4	6.4	1.6	.....	2.4	1.6	.5	.1	.2	4.2

| | FEMALE | | |

Table 13c. Current rate of *cigar* smoking, males 18 years of age and over, by age and urban or rural residence, United States, 1955

[Numbers in thousands]

Residence and age (years)	Total	Nonsmoker of cigars				Cigar smokers						Not reported
						Occasional			Regular			
		Total	Never smoked cigars	Previously smoked cigars		Total	Never smoked cigars regularly	Previously smoked cigars regularly	Total	Cigars only	Cigars and cigarettes or pipes	
				Occasionally	Regularly							
United States	49,581	36,807	34,100	1,291	1,416	7,391	6,872	519	2,636	1,560	1,076	2,747
18–24	5,405	4,504	4,449	30	25	481	453	28	70	41	29	350
25–34	11,065	8,257	7,965	160	132	1,841	1,743	98	326	163	163	641
35–44	10,755	7,925	7,451	253	221	1,727	1,629	98	456	294	162	647
45–54	9,100	6,569	6,043	283	243	1,415	1,307	108	619	357	262	497
55–64	6,934	4,914	4,339	239	336	1,024	950	74	625	373	252	371
65 and over	6,322	4,638	3,853	326	459	903	790	113	540	332	208	241
Urban	32,572	23,828	22,055	786	987	4,886	4,506	380	1,975	1,164	811	1,883
18–24	3,421	2,813	2,779	18	16	300	283	17	53	34	19	255
25–34	7,430	5,539	5,360	98	81	1,247	1,178	69	223	102	121	421
35–44	7,125	5,262	4,923	173	166	1,106	1,028	78	318	205	113	439
45–54	6,076	4,283	3,962	179	142	950	871	79	499	285	214	344
55–64	4,680	3,227	2,835	135	257	713	659	54	475	293	182	265
65 and over	3,840	2,704	2,196	183	325	570	487	83	407	245	162	159
Rural nonfarm	10,456	7,841	7,205	350	286	1,593	1,491	102	469	277	192	553
18–24	1,116	952	940	5	7	97	89	8	13	7	6	54
25–34	2,535	1,838	1,762	47	29	456	432	24	81	44	37	16
35–44	2,414	1,757	1,659	62	36	407	389	18	100	67	33	150
45–54	1,766	1,310	1,165	79	66	267	248	19	96	56	40	91
55–64	1,285	944	826	69	49	184	168	16	96	47	49	65
65 and over	1,340	1,040	853	88	99	182	165	17	83	56	27	33
Rural farm	6,553	5,138	4,840	155	143	912	875	37	192	119	73	311
18–24	868	739	730	7	2	84	81	3	4	------	4	41
25–34	1,100	880	843	15	22	138	133	5	22	17	5	60
35–44	1,216	906	869	18	19	214	212	2	38	22	16	58
45–54	1,258	975	916	25	35	198	188	10	24	16	8	60
55–64	969	743	678	35	30	127	123	4	54	33	21	45
65 and over	1,142	894	804	55	35	151	138	13	50	31	19	47

Table 13d. Current rate of *pipe* smoking, males 18 years of age and over, by age and urban or rural residence, United States, 1955

[Numbers in thousands]

Residence and age (years)	Total	Nonsmoker of pipes				Pipe smoker						Not reported
						Occasional			Regular			
		Total	Never smoked pipes	Previously smoked pipes		Total	Never smoked pipes regularly	Previously smoked pipes regularly	Total	Pipes only	Pipes and cigars or cigarettes	
				Occasionally	Regularly							
United States	49,581	38,553	33,245	2,009	3,299	4,630	4,081	549	3,645	1,979	1,666	2,753
18–24	5,405	4,472	4,317	64	91	408	375	33	174	88	86	351
25–34	11,065	8,735	7,740	439	556	1,206	1,087	119	483	195	288	641
35–44	10,755	8,296	7,080	576	640	1,200	1,083	117	622	269	353	637
45–54	9,100	7,011	5,953	427	631	938	818	120	635	291	344	516
55–64	6,934	5,337	4,413	266	658	540	444	96	672	359	313	385
65 and over	6,322	4,702	3,732	237	733	338	274	64	1,059	777	282	223
Urban	32,572	25,401	22,044	1,327	2,030	3,086	2,724	362	2,191	1,131	1,060	1,894
18–24	3,421	2,794	2,686	46	62	251	229	22	128	66	62	248
25–34	7,430	5,881	5,234	281	366	826	746	80	297	130	167	426
35–44	7,125	5,488	4,707	376	405	806	718	88	393	162	231	438
45–54	6,076	4,762	4,087	300	375	619	541	78	342	127	215	353
55–64	4,680	3,611	3,007	182	422	375	323	52	419	218	201	275
65 and over	3,840	2,865	2,323	142	400	209	167	42	612	428	184	154
Rural nonfarm	10,456	8,051	6,788	465	798	1,040	916	124	822	460	362	543
18–24	1,116	934	897	16	21	98	89	9	27	16	11	57
25–34	2,535	1,973	1,714	115	144	281	251	30	127	51	76	154
35–44	2,414	1,852	1,538	151	163	268	249	19	153	70	83	141
45–54	1,766	1,297	1,096	78	123	222	193	29	148	69	79	99
55–64	1,285	1,008	799	58	151	92	72	20	124	72	52	61
65 and over	1,340	987	744	47	196	79	62	17	243	182	61	31
Rural farm	6,553	5,101	4,413	217	471	504	441	63	632	388	244	316
18–24	868	744	734	2	8	59	57	2	19	6	13	46
25–34	1,100	881	792	43	46	99	90	9	59	14	45	61
35–44	1,216	956	835	49	72	126	116	10	76	37	39	58
45–54	1,258	952	780	49	123	97	84	13	145	95	50	64
55–64	969	718	607	26	85	73	49	24	129	69	60	49
65 and over	1,142	850	665	48	137	50	45	5	204	167	37	38

Table 14a. Current rate of *cigarette* smoking, persons 18 years of age and over, by sex, geographic region, and urban or rural residence, United States, 1955

[Numbers in thousands]

Geographic region and residence	Total	Nonsmoker of cigarettes				Cigarette smoker													Not reported
						Occasional			Regular										
										Cigarettes only					Cigarettes and cigars or pipes (by number of cigarettes) [1]				
		Total	Never smoked cigarettes	Previously smoked cigarettes		Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	40 or more daily	
				Occasionally	Regularly														
	MALE																		
United States	49,581	20,219	15,030	592	4,597	2,005	1,523	482	24,667	22,852	3,657	13,238	5,481	476	1,815	559	900	317	39	2,690
Urban	32,572	12,616	9,250	349	3,017	1,312	1,025	287	16,791	15,618	2,391	8,953	3,920	354	1,173	356	566	223	28	1,853
In urbanized areas	24,060	9,243	6,811	272	2,160	951	755	196	12,513	11,695	1,781	6,658	2,969	287	818	252	397	150	19	1,353
1 million or more	13,429	5,291	3,979	155	1,157	559	479	80	6,888	6,514	980	3,620	1,746	168	374	135	157	70	12	691
250,000 to 1 million	5,918	2,165	1,582	60	523	205	146	59	3,176	2,930	480	1,735	651	64	246	72	130	42	2	372
Under 250,000	4,713	1,787	1,250	57	480	187	130	57	2,449	2,251	321	1,303	572	55	198	45	110	30	5	290
Not in urbanized areas	8,512	3,373	2,439	77	857	361	270	91	4,278	3,923	610	2,295	951	67	355	104	169	73	9	500
Rural nonfarm	10,456	4,339	3,172	150	1,017	403	286	117	5,187	4,785	753	2,831	1,121	80	402	114	223	56	9	527
Rural farm	6,553	3,264	2,608	93	563	290	212	78	2,689	2,449	513	1,454	440	42	240	89	111	38	2	310
Northeast	13,663	5,420	4,091	144	1,185	573	439	134	6,806	6,279	906	3,351	1,845	177	527	180	235	104	8	864
Urban	10,902	4,258	3,198	101	959	477	379	98	5,450	5,073	727	2,636	1,553	157	377	139	167	63	8	717
Rural nonfarm	2,246	925	696	33	196	71	42	29	1,138	1,026	149	606	256	15	112	34	52	26	----	112
Rural farm	515	237	197	10	30	25	18	7	218	180	30	109	36	5	38	7	16	15	----	35
North Central	15,269	6,438	4,889	184	1,365	560	439	121	7,495	6,904	1,160	4,161	1,507	136	591	171	339	74	7	776
Urban	9,939	3,847	2,871	110	866	344	279	65	5,211	4,826	750	2,881	1,091	104	385	98	228	52	7	537
Rural nonfarm	2,723	1,167	841	44	282	110	86	24	1,334	1,226	178	749	276	23	108	29	67	12	----	117
Rural farm	2,602	1,424	1,177	30	217	106	74	32	950	852	172	531	140	9	98	44	44	10	----	122
South	14,003	5,587	4,165	176	1,246	599	450	149	7,122	6,639	1,160	3,886	1,477	116	483	131	240	98	14	695
Urban	7,029	2,625	1,923	73	624	297	227	70	3,749	3,506	562	2,078	810	56	243	60	111	67	5	358
Rural nonfarm	3,996	1,599	1,191	56	352	160	115	45	2,043	1,892	317	1,097	441	37	151	40	84	18	9	194
Rural farm	2,978	1,363	1,046	47	270	142	108	34	1,330	1,241	281	711	226	23	89	31	45	13	----	143
West	6,646	2,774	1,885	88	801	273	195	78	3,244	3,030	491	1,840	652	47	214	77	86	41	10	355
Urban	4,702	1,886	1,253	65	568	194	140	54	2,381	2,213	352	1,358	466	37	168	59	60	41	8	241
Rural nonfarm	1,486	648	444	17	187	62	43	19	672	641	109	379	148	5	31	11	20	----	----	104
Rural farm	458	240	188	6	46	17	12	5	191	176	30	103	38	5	15	7	6	----	2	10

	FEMALE																		
United States	55,096	38,048	35,927	457	1,664	2,086	1,803	283	12,986	12,930	4,640	7,013	1,196	81	.....	.....	.....	.....	.....	1,976
Urban	37,453	24,542	23,148	321	1,073	1,601	1,396	205	9,858	9,817	3,489	5,305	955	68	.....	.....	.....	.....	.....	1,452
In urbanized areas	27,223	17,461	16,464	232	765	1,212	1,081	131	7,444	7,414	2,526	4,070	763	55	.....	.....	.....	.....	.....	1,106
1 million or more	14,808	9,372	8,899	119	354	666	588	78	4,131	4,122	1,357	2,272	451	42	.....	.....	.....	.....	.....	639
250,000 to 1 million	6,889	4,361	4,095	59	207	320	284	36	1,911	1,906	683	1,041	175	7	.....	.....	.....	.....	.....	297
Under 250,000	5,526	3,728	3,470	54	204	226	209	17	1,402	1,386	486	757	137	6	.....	.....	.....	.....	.....	170
Not in urbanized areas	10,230	7,081	6,684	89	308	389	315	74	2,414	2,403	963	1,235	192	13	.....	.....	.....	.....	.....	346
Rural nonfarm	11,473	8,210	7,648	112	450	369	308	61	2,542	2,532	917	1,398	204	13	.....	.....	.....	.....	.....	332
Rural farm	6,170	5,296	5,131	24	141	116	99	17	586	581	234	310	37	.....	.....	.....	.....	.....	.....	172
Northeast	15,434	9,941	9,351	123	467	743	657	86	4,049	4,035	1,455	2,031	517	32	.....	.....	.....	.....	.....	701
Urban	12,483	7,912	7,441	108	363	640	576	64	3,343	3,329	1,194	1,655	453	27	.....	.....	.....	.....	.....	588
Rural nonfarm	2,473	1,643	1,541	12	90	97	78	19	647	647	237	349	56	5	.....	.....	.....	.....	.....	86
Rural farm	478	386	369	3	14	6	3	3	59	59	24	27	8	.....	.....	.....	.....	.....	.....	27
North Central	16,256	11,460	10,961	144	355	515	421	94	3,720	3,708	1,362	2,066	259	21	.....	.....	.....	.....	.....	561
Urban	10,384	7,228	6,901	101	226	397	320	77	2,947	2,940	1,067	1,641	216	16	.....	.....	.....	.....	.....	412
Rural nonfarm	2,944	2,236	2,111	36	89	77	65	12	572	567	222	312	28	5	.....	.....	.....	.....	.....	59
Rural farm	2,328	1,996	1,949	7	40	41	36	5	201	201	73	113	15	.....	.....	.....	.....	.....	.....	90
South	16,095	11,950	11,284	129	537	558	507	51	3,065	3,037	1,129	1,630	261	17	.....	.....	.....	.....	.....	522
Urban	8,719	6,082	5,716	71	295	357	327	30	1,968	1,948	707	1,056	168	17	.....	.....	.....	.....	.....	312
Rural nonfarm	4,446	3,294	3,081	44	169	136	122	14	860	857	318	458	81	.....	.....	.....	.....	.....	.....	156
Rural farm	2,930	2,574	2,487	14	73	65	58	7	237	232	104	116	12	.....	.....	.....	.....	.....	.....	54
West	7,311	4,697	4,331	61	305	270	218	52	2,152	2,150	694	1,286	159	11	.....	.....	.....	.....	.....	192
Urban	5,267	3,320	3,090	41	189	207	173	34	1,600	1,600	521	953	118	8	.....	.....	.....	.....	.....	140
Rural nonfarm	1,610	1,037	915	20	102	59	43	16	463	461	140	279	39	3	.....	.....	.....	.....	.....	51
Rural farm	434	340	326	.....	14	4	2	2	89	89	33	54	2	.....	.....	.....	.....	.....	.....	1

[1] Not shown for females because of small number of observations.

Table 14b. Current amount of *cigarette* smoking, persons 18 years of age and over, by sex, geographic region, and urban or rural residence, United States, 1955

[Percent]

Geographic region and residence	Total	Nonsmoker			Cigarette smoker										Not reported
						Occasional			Regular						
		Total	Never smoked cigarettes	Previously smoked cigarettes	Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Cigarettes only				Cigarettes and cigars or pipes (by amount of cigarettes)[1]			
									Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	
MALE																			
United States	100.0	40.8	30.3	10.5	4.0	3.1	1.0	49.8	46.1	7.4	26.7	11.1	1.0	3.7	1.1	1.8	0.6	0.1	5.4
Urban	100.0	38.7	28.4	10.3	4.0	3.1	.9	51.5	47.9	7.3	27.5	12.0	1.1	3.6	1.1	1.9	.7	.1	5.7
In urbanized areas	100.0	38.4	28.3	10.1	4.0	3.1	.8	52.0	48.6	7.4	27.7	12.3	1.2	3.4	1.0	1.7	.6	.1	5.6
1 million or more	100.0	39.4	29.6	9.8	4.2	3.6	.6	51.3	48.5	7.3	27.0	13.0	1.1	2.8	1.0	1.2	.5	.1	5.1
250,000 to 1 million	100.0	36.6	26.7	9.8	3.5	2.5	1.0	53.7	49.5	8.1	29.3	11.0	1.1	4.2	1.2	2.2	.7	.1	6.3
Under 250,000	100.0	37.9	26.5	10.2	4.0	2.8	1.2	52.0	47.8	6.8	27.6	12.1	1.2	4.2	1.0	2.3	.8	.1	6.2
Not in urbanized areas	100.0	39.6	28.7	10.1	4.2	3.2	1.1	50.3	46.1	7.2	27.0	11.2	.8	4.2	1.2	2.0	.9	.1	5.9
Rural nonfarm	100.0	41.5	30.3	9.7	3.9	2.7	1.1	49.6	45.8	7.2	27.1	10.7	.8	3.8	1.1	2.1	.5	.1	5.0
Rural farm	100.0	49.8	39.8	8.6	4.4	3.2	1.2	41.0	37.4	7.8	22.2	6.7	.6	3.7	1.4	1.7	.6	.....	4.7
Northeast	100.0	39.7	29.9	8.7	4.2	3.2	1.0	49.8	46.0	6.6	24.5	13.5	1.3	3.9	1.3	1.7	.8	.1	6.3
Urban	100.0	39.1	29.3	8.8	4.4	3.5	.9	50.0	46.5	6.7	24.2	14.2	1.4	3.5	1.3	1.5	.6	.1	6.6
Rural nonfarm	100.0	41.2	31.0	8.7	3.2	1.9	1.3	50.7	45.7	6.6	27.0	11.4	.7	5.0	1.5	2.3	1.2	.....	5.0
Rural farm	100.0	46.0	38.3	5.8	4.9	3.5	1.4	42.3	35.0	5.8	21.1	7.0	1.0	7.4	1.4	3.1	2.9	.....	6.8
North Central	100.0	42.2	32.0	8.9	3.7	2.9	.8	49.0	45.2	7.2	27.3	9.9	.9	3.9	1.1	2.2	.5	.....	5.1
Urban	100.0	38.7	28.9	8.7	3.5	2.8	.7	52.4	48.6	7.5	29.0	11.0	1.0	3.9	1.0	2.3	.5	.1	5.4
Rural nonfarm	100.0	42.6	30.8	10.3	4.0	2.2	.9	48.9	44.9	6.5	27.5	10.1	.8	4.0	1.1	2.5	.4	.....	4.3
Rural farm	100.0	54.7	45.2	8.3	4.1	2.8	1.2	36.5	32.7	6.6	20.4	5.4	.3	3.8	1.7	1.7	.4	.....	4.7
South	100.0	39.9	29.7	8.9	4.3	3.2	1.1	50.9	47.4	8.3	27.8	10.5	.8	3.4	.9	1.7	.7	.1	5.0
Urban	100.0	37.3	27.4	8.9	4.2	3.2	1.0	53.3	49.9	8.0	29.6	11.5	.8	3.5	.9	1.6	1.0	.1	5.1
Rural nonfarm	100.0	40.0	29.8	8.8	4.0	2.9	1.1	51.1	47.3	7.9	27.5	11.0	.9	3.8	1.0	2.1	.5	.2	4.9
Rural farm	100.0	45.8	35.1	9.1	4.8	3.6	1.1	44.7	41.7	9.4	23.9	7.6	.8	3.0	1.0	1.5	.4	.....	4.8
West	100.0	41.7	28.4	12.1	4.1	2.9	1.2	48.8	45.6	7.4	27.7	9.8	.7	3.2	1.2	1.3	.6	.2	5.3
Urban	100.0	40.1	28.6	12.1	4.1	3.0	1.1	50.6	47.1	7.5	28.9	9.9	.8	3.6	1.3	1.3	.9	.2	5.1
Rural nonfarm	100.0	43.6	29.9	12.6	4.2	2.9	1.3	45.2	43.1	7.3	25.5	10.0	.3	2.1	.7	1.3	.....	.....	7.0
Rural farm	100.0	52.4	41.0	10.1	3.7	2.6	1.1	41.7	38.4	6.6	22.5	8.3	1.1	3.3	1.5	1.3	.....	.4	2.2

FEMALE

| | | |

Table 14c. Current rate of *cigar* smoking, males 18 years of age and over, by region and urban or rural residence, United States, 1955

[Numbers in thousands]

Geographic region and residence	Total	Nonsmoker of cigars				Cigar smoker						Not reported
						Occasional			Regular			
		Total	Never smoked cigars	Previously smoked cigars		Total	Never smoked cigars regularly	Previously smoked cigars regularly	Total	Cigars only	Cigars and cigarettes or pipes	
				Occasionally	Regularly							
United States	49,581	36,807	34,100	1,291	1,416	7,391	6,872	519	2,636	1,560	1,076	2,747
Urban	32,572	23,828	22,055	786	987	4,886	4,506	380	1,975	1,164	811	1,883
In urbanized areas	24,060	17,653	16,354	589	710	3,549	3,283	266	1,490	905	585	1,368
1 million or more	13,429	10,000	9,332	311	357	1,880	1,750	130	850	546	304	699
250,000 to 1 million	5,918	4,224	3,906	134	184	922	853	69	398	231	167	374
Under 250,000	4,713	3,429	3,116	144	169	747	680	67	242	128	114	295
Not in urbanized areas	8,512	6,175	5,701	197	277	1,337	1,223	114	485	259	226	515
Rural nonfarm	10,456	7,841	7,205	350	286	1,593	1,491	102	469	277	192	553
Rural farm	6,553	5,138	4,840	155	143	912	875	37	192	119	73	311
Northeast	13,663	9,758	9,036	341	381	2,071	1,904	167	971	572	399	863
Urban	10,902	7,742	7,180	256	306	1,620	1,482	138	828	513	315	712
Rural nonfarm	2,246	1,649	1,516	65	68	366	342	24	115	49	66	116
Rural farm	515	367	340	20	7	85	80	5	28	10	18	35
North Central	15,269	11,305	10,466	397	442	2,434	2,282	152	738	426	312	792
Urban	9,939	7,284	6,739	240	305	1,585	1,482	104	518	284	234	551
Rural nonfarm	2,728	1,979	1,799	108	72	484	446	38	146	97	49	119
Rural farm	2,602	2,042	1,928	49	65	364	354	10	74	45	29	122
South	14,003	10,625	10,036	278	311	1,976	1,860	116	668	417	251	734
Urban	7,029	5,195	4,928	101	166	1,029	959	70	426	258	168	379
Rural nonfarm	3,996	3,058	2,851	116	91	554	526	28	173	110	63	211
Rural farm	2,978	2,372	2,257	61	54	393	375	18	69	49	20	144
West	6,646	5,119	4,562	275	282	910	826	84	259	145	114	358
Urban	4,702	3,607	3,208	189	210	651	583	68	203	109	94	241
Rural nonfarm	1,486	1,155	1,039	61	55	189	177	12	35	21	14	107
Rural farm	458	357	315	25	17	70	66	4	21	15	6	10

Table 14d. Current rate of *pipe* smoking, males 18 years of age and over, by region and urban or rural residence, United States, 1955

[Numbers in thousands]

Geographic region and residence	Total	Nonsmoker of pipes				Pipe smoker						Not reported
						Occasional			Regular			
		Total	Never smoked pipes	Previously smoked pipes		Total	Never smoked pipes regularly	Previously smoked pipes regularly	Total	Pipes only	Pipes and cigars or cigarettes	
				Occasionally	Regularly							
United States	49,581	38,553	33,245	2,009	3,299	4,630	4,081	549	3,645	1,979	1,666	2,753
Urban	32,572	25,401	22,044	1,327	2,030	3,086	2,724	362	2,191	1,131	1,060	1,894
In urbanized areas	24,060	18,812	16,439	964	1,409	2,358	2,081	277	1,509	781	728	1,381
1 million or more	13,429	10,734	9,532	529	673	1,186	1,057	129	807	438	369	702
250,000 to 1 million	5,918	4,500	3,947	201	352	640	556	84	398	178	220	380
Under 250,000	4,713	3,578	2,960	234	384	532	468	64	304	165	139	299
Not in urbanized areas	8,512	6,589	5,605	363	621	728	643	85	682	350	332	513
Rural nonfarm	10,456	8,051	6,788	465	798	1,040	916	124	822	460	362	543
Rural farm	6,553	5,101	4,413	217	471	504	441	63	632	388	244	316
Northeast	13,663	10,365	9,048	472	845	1,331	1,155	176	1,091	569	522	876
Urban	10,902	8,329	7,375	361	593	1,039	902	137	808	431	377	726
Rural nonfarm	2,246	1,670	1,379	86	205	232	195	37	228	120	108	116
Rural farm	515	366	294	25	47	60	58	2	55	18	37	34
North Central	15,289	11,621	9,849	668	1,104	1,588	1,426	162	1,263	697	566	797
Urban	9,939	7,664	6,539	427	698	1,016	917	99	702	350	352	557
Rural nonfarm	2,728	2,021	1,636	162	223	332	305	27	258	147	111	117
Rural farm	2,602	1,936	1,674	79	183	240	204	36	303	200	103	123
outh	14,003	11,225	9,989	460	776	1,188	1,039	149	862	479	383	728
Urban	7,029	5,630	5,053	249	328	641	563	78	385	198	187	373
Rural nonfarm	3,996	3,189	2,798	135	256	366	313	53	237	132	105	204
Rural farm	2,978	2,406	2,138	76	192	181	163	18	240	149	91	151
West	6,646	5,342	4,359	409	574	523	461	62	429	234	195	352
Urban	4,702	3,778	3,077	290	411	390	342	48	296	152	144	238
Rural nonfarm	1,486	1,171	975	82	114	110	103	7	99	61	38	106
Rural farm	458	393	307	37	49	23	16	7	34	21	13	8

Table 15a. Current rate of *cigarette* smoking, persons 18 years of age and over, by sex, race, geographic region, and urban or rural residence, United States, 1955

[Numbers in thousands]

Race, geographic region, and residence	Total	Nonsmoker of cigarettes				Cigarette smoker											Not reported
						Occasional		Regular									
		Total	Never smoked cigarettes	Previously smoked cigarettes		Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Cigarettes only				Cigarettes and cigars or pipes (by number of cigarettes)[1]			
				Occasionally	Regularly					Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	40 or more daily	
								MALE												
United States:																				
White	44,870	18,452	13,535	537	4,380	1,691	1,267	424	22,357	20,707	3,047	12,696	5,293	454	1,650	520	827	294	39	2,349
Urban	29,228	11,449	8,274	320	2,855	1,118	872	246	15,059	13,976	1,972	7,969	3,674	341	1,083	333	519	206	25	1,622
Rural nonfarm	9,744	4,003	2,873	144	984	337	234	103	4,922	4,548	673	2,697	1,104	74	374	111	202	52	9	482
Rural farm	5,898	3,000	2,386	73	541	236	161	75	2,376	2,183	402	1,029	422	39	223	79	106	36	2	256
Nonwhite	4,711	1,767	1,465	55	217	314	256	58	2,280	2,145	610	1,232	281	22	135	39	73	33		350
Urban	3,344	1,167	976	29	162	194	153	41	1,732	1,642	419	964	246	13	90	26	47	17		251
Rural nonfarm	712	336	297	6	33	66	53	14	265	237	80	134	17	6	28	3	21	4		43
Rural farm	655	264	222	20	22	54	51	3	283	265	111	134	18	3	17	10	5	2		54
North and West:																				
White	33,423	13,884	10,244	396	3,244	1,277	965	312	16,444	15,169	2,276	8,696	3,863	349	1,275	408	636	206	25	1,815
Urban	23,551	9,326	6,770	259	2,297	916	718	198	11,977	11,103	1,619	6,226	2,956	297	877	276	433	143	23	1,332
Rural nonfarm	6,329	2,672	1,924	94	654	217	147	70	3,115	2,866	429	1,716	678	43	249	74	157	38		325
Rural farm	3,543	1,886	1,550	43	293	144	100	44	1,352	1,203	228	742	214	19	149	56	45	25	2	161
Nonwhite	2,155	748	621	30	107	129	108	21	1,101	1,044	221	656	156	11	57	20	24	13		177
Urban	1,992	665	552	17	96	99	80	19	1,065	1,012	210	637	154	11	53	18	22	13		163
Rural nonfarm	131	68	57		11	26	24	2	29	27	7	18	2		2		2			8
Rural farm	32	15	12	3		4	4		7	5	4	1			2	2				6
South:																				
White	11,447	4,568	3,291	141	1,136	414	302	112	5,943	5,538	771	3,310	1,352	105	495	112	191	88	14	522
Urban	5,677	2,123	1,594	61	528	202	154	48	3,082	2,876	353	1,751	718	54	206	52	86	63	5	270
Rural nonfarm	3,415	1,331	951	30	330	120	87	33	1,807	1,682	244	981	436	31	125	37	65	14	9	157
Rural farm	2,355	1,114	830	30	248	92	61	31	1,054	980	174	578	198	20	74	23	40	11		95
Nonwhite	2,596	1,019	874	25	110	185	146	37	1,179	1,101	369	576	125	11	78	19	49	10		173
Urban	1,352	502	424	12	66	95	73	22	667	630	209	327	92	2	37	6	25	4		88
Rural nonfarm	581	208	240	6	22	40	28	12	236	210	73	116	15	6	26	3	19	4		37
Rural farm	663	309	210	17	22	50	47	3	276	261	107	133	18	3	13	8	5	2		48

FEMALE

| | | |

Table 15b. Current rate of *cigarette* smoking, persons 18 years of age and over, by sex, race, geographic region, and urban or rural residence, United States, 1955

[Percent]

Race, geographic region, and residence	Total	Nonsmoker of cigarettes				Cigarette smoker													Not reported
						Occasional			Regular										
		Total	Never smoked cigarettes	Previously smoked cigarettes		Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Cigarettes only					Cigarettes and cigars or pipes (by number of cigarettes) [1]				
				Occasionally	Regularly					Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	40 or more daily
	MALE																		
United States:																			
White	100.0	41.1	30.2	1.2	9.8	3.8	2.8	0.9	49.9	46.1	6.8	26.8	11.6	1.0	3.7	1.2	1.8	0.7	0.1	5.2
Urban	100.0	39.2	28.3	1.1	9.8	3.8	3.0	.8	51.5	47.8	6.7	27.3	12.6	1.2	3.7	1.1	1.8	.7	.1	5.5
Rural nonfarm	100.0	41.1	29.5	1.5	10.1	3.5	2.4	1.1	50.5	46.7	6.9	27.7	11.3	.8	3.8	1.1	2.1	.5	.1	4.9
Rural farm	100.0	50.9	40.5	1.2	9.2	4.0	2.7	1.3	40.8	37.0	6.8	22.4	7.2	.7	3.8	1.3	1.8	.6	.....	4.3
Nonwhite	100.0	37.5	31.7	1.2	4.6	6.7	5.4	1.2	48.4	45.5	12.9	26.2	6.0	.5	2.9	.8	1.5	.5	.....	7.4
Urban	100.0	34.9	29.2	.9	4.8	5.8	4.6	1.2	51.8	49.1	12.5	28.8	7.4	.4	2.7	.8	1.4	.5	.....	7.5
Rural nonfarm	100.0	47.2	41.7	.8	4.6	9.3	7.3	2.0	37.2	33.3	11.2	18.8	2.4	.8	3.9	.4	2.9	.6	.....	6.3
Rural farm	100.0	40.3	33.9	3.1	3.4	8.2	7.8	.5	43.2	40.6	16.9	20.5	2.7	.5	2.6	1.5	.8	.3	.....	8.2
North and West:																			
White	100.0	41.5	30.6	1.2	9.7	3.8	2.9	.9	49.2	45.4	6.8	26.0	11.5	1.0	3.8	1.2	1.9	.6	.1	5.4
Urban	100.0	39.6	28.7	1.1	9.8	3.9	3.0	.8	50.9	47.1	6.9	26.5	12.6	1.2	3.7	1.2	1.8	.6	.1	5.7
Rural nonfarm	100.0	42.2	30.4	1.5	10.3	3.4	2.3	1.1	49.2	45.3	6.8	27.1	10.7	.7	3.9	1.2	2.2	.6	.....	5.1
Rural farm	100.0	53.2	43.7	1.2	8.3	4.1	2.8	1.2	38.2	34.0	6.4	20.9	6.0	.5	4.2	1.6	1.9	.7	.1	4.5
Nonwhite	100.0	34.7	28.8	.9	5.0	6.0	5.0	1.0	51.1	48.4	10.3	30.4	7.2	.5	2.6	.9	1.1	.6	.....	8.2
Urban	100.0	33.4	27.7	.9	4.8	5.0	4.0	1.0	53.5	50.8	10.5	32.0	7.7	.6	2.7	.9	1.1	.7	.....	8.2
Rural nonfarm	100.0	51.9	43.5	.....	8.4	19.8	18.3	1.5	22.1	20.6	5.3	13.7	1.5	.....	1.5	.....	1.5	.....	.....	6.1
Rural farm	100.0	46.9	37.5	9.4	.....	12.5	12.5	.....	21.9	15.6	12.5	3.1	.....	.....	6.3	6.3	.....	.....	.....	18.8
South:																			
White	100.0	39.9	28.7	1.2	9.9	3.6	2.6	1.0	51.9	48.4	6.7	28.9	11.8	.9	3.5	1.0	1.7	.8	.1	4.6
Urban	100.0	37.4	26.5	1.1	9.8	3.6	2.7	.8	54.3	50.7	6.2	30.8	12.6	1.0	3.6	.9	1.5	1.1	.1	4.8
Rural nonfarm	100.0	39.0	27.8	1.5	9.7	3.5	2.5	1.0	53.0	49.3	7.1	28.7	12.5	.9	3.7	1.1	1.9	.4	.3	4.6
Rural farm	100.0	47.3	35.5	1.3	10.5	3.9	2.6	1.3	44.8	41.6	7.4	24.5	8.8	.8	3.1	1.0	1.7	.5	.....	4.0
Nonwhite	100.0	39.9	34.1	1.4	4.3	7.2	5.8	1.4	46.1	43.1	15.2	22.5	4.9	.4	3.1	.7	1.9	.4	.....	6.7
Urban	100.0	37.1	31.4	.9	4.9	7.0	5.4	1.6	49.3	46.6	15.5	24.2	6.8	.1	2.7	.6	1.8	.3	.....	6.5
Rural nonfarm	100.0	46.1	41.3	1.0	3.8	6.9	4.8	2.1	40.6	36.1	12.6	20.0	2.6	1.0	4.5	.5	3.3	.7	.....	6.4
Rural farm	100.0	40.0	33.7	2.7	3.5	8.0	7.5	.5	44.3	41.9	17.2	21.3	2.9	.5	2.4	1.3	.8	.3	.....	7.7

| | FEMALE | | |

Table 15c. Current rate of *cigar* smoking, males 18 years and over, by race, geographic region, and urban or rural residence, United States, 1955

[Numbers in thousands]

Race, geographic region, and residence	Total	Nonsmoker of cigars				Cigar smoker						Not reported
						Occasional			Regular			
		Total	Never smoked cigars	Previously smoked cigars		Total	Never smoked cigars regularly	Previously smoked cigars regularly	Total	Cigars only	Cigars and cigarettes or pipes	
				Occasionally	Regularly							
United States:												
White	44,870	33,371	30,792	1,244	1,335	6,772	6,286	486	2,343	1,364	979	2,384
Urban	29,228	21,390	19,696	771	923	4,468	4,117	351	1,745	1,005	740	1,625
Rural nonfarm	9,744	7,318	6,704	339	275	1,493	1,395	98	430	258	172	503
Rural farm	5,898	4,663	4,392	134	137	811	774	37	168	101	67	256
Nonwhite	4,711	3,436	3,308	47	81	619	586	33	293	196	97	363
Urban	3,344	2,438	2,359	15	64	418	389	29	230	159	71	258
Rural nonfarm	712	523	501	11	11	100	96	4	39	19	20	50
Rural farm	655	475	448	21	6	101	101	.....	24	18	6	55
North and West:												
White	33,423	24,630	22,569	996	1,065	5,120	4,738	382	1,835	1,057	778	1,838
Urban	23,551	17,194	15,739	672	783	3,583	3,294	289	1,431	827	604	1,343
Rural nonfarm	6,329	4,693	4,270	230	193	1,020	946	74	282	160	122	334
Rural farm	3,543	2,743	2,560	94	89	517	498	19	122	70	52	161
Nonwhite	2,155	1,552	1,495	17	40	295	274	21	133	86	47	175
Urban	1,992	1,439	1,388	13	38	274	253	21	118	79	39	161
Rural nonfarm	131	90	84	4	2	19	19	.....	14	7	7	8
Rural farm	32	23	23	.....	.....	2	2	.....	1	.....	1	6
South:												
White	11,447	8,741	8,223	248	270	1,652	1,548	104	508	307	201	546
Urban	5,677	4,196	3,957	99	140	885	823	62	314	178	136	282
Rural nonfarm	3,415	2,625	2,434	109	82	473	449	24	148	98	50	169
Rural farm	2,355	1,920	1,832	40	48	294	276	18	46	31	15	95
Nonwhite	2,556	1,884	1,813	30	41	324	312	12	160	110	50	188
Urban	1,352	999	971	2	26	144	136	8	112	80	32	97
Rural nonfarm	581	433	417	7	9	81	77	4	25	12	13	42
Rural farm	623	452	425	21	6	99	99	.....	23	18	5	49

Table 15d. Current rate of *pipe* smoking, males 18 years of age and over, by race, geographic region, and urban or rural residence, United States, 1955

[Numbers in thousands]

Race, geographic region, and residence	Total	Nonsmoker of pipes				Pipe smoker						Not reported
						Occasional pipe smoker			Regular pipe smoker			
		Total	Never smoked pipes	Previously smoked pipes		Total	Never smoked pipes regularly	Previously smoked pipes regularly	Total	Pipes only	Pipes and cigars or cigarettes	
				Occasionally	Regularly							
United States:												
White	44,870	34,706	29,741	1,928	3,127	4,310	3,797	513	3,370	1,800	1,570	2,394
Urban	29,228	22,686	19,484	1,275	1,927	2,857	2,511	346	2,041	1,043	998	1,644
Rural nonfarm	9,744	7,497	6,267	455	775	1,000	886	114	758	414	344	489
Rural farm	5,898	4,613	3,990	198	425	453	400	53	571	343	228	261
Nonwhite	4,711	3,757	3,504	81	172	320	284	36	275	179	96	359
Urban	3,344	2,715	2,560	52	103	229	213	16	150	88	62	250
Rural nonfarm	712	554	521	10	23	40	30	10	64	46	18	54
Rural farm	655	488	423	19	46	51	41	10	61	45	16	55
North and West:												
White	33,423	25,615	21,655	1,510	2,450	3,290	2,907	383	2,670	1,434	1,236	1,848
Urban	23,551	18,161	15,486	1,041	1,634	2,308	2,037	271	1,719	881	838	1,363
Rural nonfarm	6,329	4,779	3,914	328	537	662	595	67	562	314	248	326
Rural farm	3,543	2,675	2,255	141	279	320	275	45	389	239	150	159
Nonwhite	2,155	1,713	1,601	39	73	152	135	17	113	66	47	177
Urban	1,992	1,610	1,505	37	68	137	124	13	87	52	35	158
Rural nonfarm	131	83	76	2	5	12	8	4	23	14	9	13
Rural farm	32	20	20	-------	-------	3	3	-------	3	-------	3	6
South:												
White	11,447	9,181	8,086	418	677	1,020	890	130	700	366	334	546
Urban	5,677	4,525	3,998	234	293	549	474	75	322	162	160	281
Rural nonfarm	3,415	2,718	2,353	127	238	338	291	47	196	100	96	163
Rural farm	2,355	1,938	1,735	57	146	133	125	8	182	104	78	102
Nonwhite	2,556	2,044	1,903	42	99	168	149	19	162	113	49	182
Urban	1,352	1,105	1,055	15	35	92	89	3	63	36	27	92
Rural nonfarm	581	471	445	8	18	28	22	6	41	32	9	41
Rural farm	623	468	403	19	46	48	38	10	58	45	13	49

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Table 16. Current rate of cigarette smoking, persons 18 years of age and over, by sex and major industry group, United States, 1955

[Numbers in thousands]

Major industry group (Civilians)	Total	Nonsmoker of cigarettes			Cigarette smoker											Not reported
		Total	Never smoked cigarettes	Previously smoked cigarettes	Occasional			Regular								
						Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Cigarettes only				Cigarettes and cigars or pipes (by number of cigarettes) [1]		
										Total	Less than 10 daily	10 to 20 daily	21 to 40 daily	41 or more daily	Total	Less than 10 daily	10 to 20 daily	21 to 40 daily	40 or more daily	
	MALE																			
Total	49,581	20,219	15,030	592	4,597	2,005	1,523	482	24,967	22,852	3,657	12,238	5,481	476	1,815	559	900	317	39	2,690
Employed	40,260	15,170	10,950	420	3,800	1,694	1,218	376	21,397	19,706	2,900	11,421	4,958	427	1,541	439	776	290	36	2,249
Agriculture, forestry and fisheries	4,412	2,158	1,714	67	377	205	155	50	1,857	1,692	374	1,035	251	32	165	65	79	19	2	192
Mining	761	257	162	17	78	33	16	7	440	412	70	280	105	7	26	5	13	10	----	41
Construction	3,375	1,053	759	15	279	144	110	34	1,955	1,813	199	1,049	533	32	142	39	59	44	----	223
Manufacturing	12,223	4,235	2,950	108	1,177	418	327	91	6,856	6,395	905	3,789	1,598	103	461	120	220	80	11	714
Durable goods	7,751	2,993	1,754	65	774	253	197	56	4,406	4,101	557	2,469	1,001	74	307	79	167	53	6	497
Nondurable goods	4,472	1,642	1,196	43	403	165	130	35	2,448	2,294	348	1,320	597	29	154	41	83	25	5	217
Transportation, communication, and other public utilities	3,720	1,206	907	22	367	135	95	40	2,103	1,945	229	1,150	529	47	148	43	74	15	16	186
Railroads	1,222	472	357	7	128	36	43	15	632	569	79	330	143	17	63	16	31	5	11	60
Other	2,498	734	550	15	239	77	52	25	1,471	1,376	150	820	386	30	83	27	43	10	5	126
Trade	7,452	2,669	1,935	94	660	273	200	73	4,061	3,758	495	2,109	1,036	118	303	88	148	62	5	429
Wholesale	2,020	676	481	26	169	64	40	24	1,177	1,069	126	616	305	40	88	13	50	22	3	103
Retail	5,432	2,013	1,454	68	491	209	160	49	2,884	2,689	367	1,493	731	78	215	75	98	40	2	326
Finance, insurance, and real estate	1,301	533	384	15	134	57	47	10	648	593	86	306	178	23	55	21	24	10	----	63
Business and repair services	1,212	421	275	12	134	60	45	15	636	596	82	375	139	10	40	7	18	13	2	95
Personal services	1,078	434	289	21	114	53	40	13	552	526	84	393	129	10	26	11	15	----	----	49
Entertainment and recreation services	315	86	66	----	20	20	13	7	188	178	35	92	51	----	10	2	6	2	----	21
Professional and related services	2,839	1,184	913	27	244	119	94	25	924	844	201	431	193	19	80	15	45	20	----	112
Public administration	2,072	834	596	22	216	87	76	11	1,027	944	140	552	226	26	83	23	43	15	----	124
Not employed	9,321	5,049	4,080	172	797	411	305	106	3,430	3,146	757	1,817	533	49	274	120	124	27	3	441

	FEMALE																			
Total	55,096	38,048	35,927	457	1,664	2,066	1,803	263	12,986	12,930	4,640	7,013	1,196	81	------	------	------	------	------	1,976
Employed	18,062	11,399	10,799	136	444	777	696	81	5,028	4,996	1,819	2,682	461	34	------	------	------	------	------	878
Agriculture, forestry, and fisheries	459	405	388	4	13	------	------	------	40	40	13	27	------	------	------	------	------	------	------	14
Mining	19	12	12	------	------	------	------	------	7	7	------	7	------	------	------	------	------	------	------	------
Construction	119	62	60	------	2	2	2	------	50	50	17	31	2	------	------	------	------	------	------	5
Manufacturing	4,257	2,565	2,426	34	105	173	152	21	1,250	1,227	483	637	110	7	------	------	------	------	------	260
Durable goods	1,655	879	818	17	44	63	56	7	387	387	187	334	61	5	------	------	------	------	------	126
Nondurable goods	2,602	1,686	1,608	17	61	110	96	14	863	840	296	303	49	2	------	------	------	------	------	143
Transportation, communication, and other public utilities	762	446	417	14	15	42	42	------	238	238	60	139	35	4	------	------	------	------	------	36
Railroads	62	31	29	------	2	5	5	------	20	30	------	16	2	2	------	------	------	------	------	6
Other	700	415	388	14	13	37	37	------	218	218	60	123	33	2	------	------	------	------	------	30
Trade	4,147	2,502	2,362	27	113	173	145	28	1,264	1,250	380	732	124	14	------	------	------	------	------	208
Wholesale	510	313	306	2	5	14	7	7	156	156	45	96	15	------	------	------	------	------	------	27
Retail	3,637	2,189	2,056	25	108	159	138	21	1,108	1,094	335	636	109	14	------	------	------	------	------	181
Finance, insurance, and real estate	969	573	528	15	30	47	47	------	311	309	126	159	22	------	------	------	------	------	------	38
Business and repair services	267	151	139	2	10	9	7	2	88	88	20	58	10	------	------	------	------	------	------	19
Personal services	2,629	1,797	1,723	27	47	113	106	7	598	598	262	274	60	2	------	------	------	------	------	121
Entertainment and recreation services	139	70	68	2	------	11	8	3	54	54	10	32	12	------	------	------	------	------	------	4
Professional and related services	3,530	2,337	2,229	29	79	172	152	20	887	887	371	447	64	5	------	------	------	------	------	124
Public administration	795	479	447	2	30	35	35	------	241	238	75	139	22	2	------	------	------	------	------	40
Not employed	37,014	26,649	25,128	301	1,220	1,309	1,107	202	7,958	7,934	2,821	4,331	735	47	------	------	------	------	------	1,098

[1] Not shown for females because of small number of observations.

Table 17. Current rate of cigarette smoking, persons 18 years of age and over, by sex and major occupational group, United States, 1955

[Numbers in thousands]

Major occupational group (civilians)	Total	Nonsmoker of cigarettes				Cigarette smoker										Not reported
		Total	Never smoked cigarettes	Previously smoked cigarettes		Occasional		Regular								
				Occasionally	Regularly	Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Cigarettes only			Cigarettes and cigars or pipes by number of cigarettes[1]			
										Total	Less than 10 daily	10 to 21 daily	21 to 40 daily	41 or more daily	Total	Less than 10 daily	10 to 21 daily	21 to 40 daily	41 or more daily	
	MALE															
Total	49,581	30,219	15,030	592	4,597	2,005	1,523	482	24,667	22,852	3,657	13,258	5,481	476	1,815	559	900	317	39	2,660
Employed	40,260	15,170	10,950	420	3,900	1,594	1,218	376	21,247	19,706	2,900	11,421	4,908	427	1,541	439	776	290	36	2,349
Professional, technical, and kindred workers:																				
Total	3,671	1,775	1,324	43	408	170	145	25	1,898	1,434	228	803	384	19	134	47	63	17	7	158
Self-employed	689	338	234	7	97	30	20	10	295	208	41	146	79	2	27	8	12	5	2	26
Salaried	2,982	1,437	1,090	36	311	140	125	15	1,273	1,166	187	657	305	17	107	39	51	12	5	132
Farmers and farm managers	3,293	1,732	1,369	55	305	142	96	46	1,300	1,176	255	715	177	29	124	52	60	10	2	119
Managers, officials, and proprietors, except farm:																				
Total	5,300	2,206	1,496	54	656	196	151	47	2,616	2,380	263	1,296	735	86	298	72	115	42	7	280
Self-employed	3,014	1,300	887	27	386	134	111	23	1,427	1,288	132	674	414	48	139	45	65	27	2	153
Salaried	2,286	906	609	27	270	64	40	24	1,189	1,092	111	622	321	38	97	27	50	15	5	127
Clerical and kindred workers	2,775	1,043	746	38	259	100	88	12	1,464	1,391	187	851	324	29	93	32	39	20	2	146
Sales workers	2,305	791	567	30	194	96	68	30	1,298	1,214	162	657	365	30	84	24	44	16		118
Craftsmen, foremen, and kindred workers	7,710	2,574	1,696	79	799	282	200	82	4,422	4,084	516	2,333	1,159	86	338	91	165	77	5	432
Operatives and kindred workers	8,921	2,763	1,964	72	727	315	249	66	5,167	4,854	645	2,965	1,154	90	313	65	159	76	13	576
Private household workers	56	20	17		3	4	4		28	23	7	9	7		5	5				4
Service workers, except private household	2,368	890	691	23	176	88	66	22	1,252	1,165	229	612	295	29	87	23	51	13		138
Farm laborers and foremen	910	343	252	12	49	44	40	4	464	432	103	267	59	3	32	13	14	5		69
Laborers, except farm and mine	3,051	1,033	798	14	221	133	111	22	1,648	1,553	305	923	299	26	95	30	61	14	3	217
Not employed	9,321	5,049	4,080	172	797	411	305	106	3,430	3,146	757	1,817	533	49	274	120	124	27		441
Unemployed	2,371	677	486	44	147	98	70	28	1,449	1,366	232	818	278	18	83	22	59	2		147
Unable to work	1,294	781	628	10	145	43	38	7	365	365	115	183	51	16	30	9	11	8	2	73
Keeping house	74	46	46			5	3	2	22	19	7	10	2		3	3				1
Other	5,582	3,545	2,922	118	505	263	194	69	1,554	1,396	383	806	192	15	156	86	54	17	1	229

	FEMALE																		
Total	55,096	38,048	35,927	457	1,664	2,086	1,803	283	12,986	12,930	4,640	7,013	1,196	81	.....	.....	.....	.....	.....	1,976
Employed	18,082	11,399	10,799	156	444	777	696	81	5,028	4,996	1,819	2,682	461	34	.....	.....	.....	.....	.....	878
Professional, technical, and kindred workers:																				
Total	2,314	1,542	1,475	12	55	110	100	10	578	578	252	269	55	2	.....	.....	.....	.....	.....	84
Self-employed	106	64	59	.....	5	5	2	3	36	36	5	21	8	2	.....	.....	.....	.....	.....	1
Salaried	2,208	1,478	1,416	12	50	105	98	7	542	542	247	248	47	.....	.....	.....	.....	.....	.....	83
Farmers and farm managers	79	67	63	2	2	.....	.....	.....	12	12	2	10	.....	.....	.....	.....	.....	.....	.....	.....
Managers, officials, and proprietors, except farm:																				
Total	984	628	582	13	33	40	32	8	278	276	59	172	35	10	.....	.....	.....	.....	.....	38
Self-employed	598	401	377	7	17	30	25	5	153	151	32	93	21	5	.....	.....	.....	.....	.....	14
Salaried	386	227	205	6	16	10	7	3	125	125	27	79	14	5	.....	.....	.....	.....	.....	24
Clerical and kindred workers	5,463	3,119	2,917	53	149	278	252	26	1,768	1,759	610	1,012	131	6	.....	.....	.....	.....	.....	298
Sales workers	1,255	786	743	16	27	63	52	11	343	333	114	192	27	.....	.....	.....	.....	.....	.....	63
Craftsmen, foremen, and kindred workers	164	104	99	.....	5	5	5	.....	52	52	16	34	2	.....	.....	.....	.....	.....	.....	3
Operatives and kindred workers	3,404	2,194	2,102	19	73	105	93	12	897	888	352	440	89	7	.....	.....	.....	.....	.....	208
Private household workers	1,623	1,182	1,132	20	30	74	67	7	310	310	160	118	30	2	.....	.....	.....	.....	.....	57
Service workers, except private household	2,353	1,402	1,323	21	58	97	90	7	742	740	232	413	90	5	.....	.....	.....	.....	.....	112
Farm laborers and foremen	346	317	307	.....	10	.....	.....	.....	19	19	7	12	.....	.....	.....	.....	.....	.....	.....	10
Laborers, except farm and mine	97	58	56	.....	2	5	5	.....	29	29	15	10	2	2	.....	.....	.....	.....	.....	5
Not employed	37,014	26,649	25,128	301	1,220	1,309	1,107	202	7,958	7,934	2,821	4,331	735	47	.....	.....	.....	.....	.....	1,098
Unemployed	940	554	529	15	10	49	42	7	303	299	116	147	36	.....	.....	.....	.....	.....	.....	34
Unable to work	834	718	678	10	30	15	15	.....	42	42	15	23	2	2	.....	.....	.....	.....	.....	59
Keeping house	33,688	24,163	22,744	263	1,156	1,168	978	190	7,423	7,403	2,691	4,075	692	45	.....	.....	.....	.....	.....	934
Other	1,552	1,214	1,177	13	24	77	72	5	190	190	99	86	5	.....	.....	.....	.....	.....	.....	71

[1] Not shown for females because of small number of observations.

Table 18. Current rate of cigarette smoking, persons 18 years of age and over, by sex, age, and marital status, United States, 1955

[Numbers in thousands]

Age and marital status	Total	Nonsmoker of cigarettes				Cigarette smokers													Not reported
						Occasions			Regular										
										Cigarettes only					Cigarettes and cigars or pipes (by number of cigarettes)[1]					
		Total	Never smoked cigarettes	Smoked cigarettes occasionally	Smoked cigarettes regularly	Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	
	MALE																		
All ages	49,581	20,219	15,030	592	4,597	2,005	1,523	482	24,667	22,852	3,657	13,238	5,481	476	1,815	559	900	317	39	2,690
Married, spouse present	37,844	15,091	10,636	463	3,992	1,429	1,073	356	19,552	18,034	2,627	10,583	4,430	394	1,518	450	764	272	32	1,772
Married, spouse absent	1,006	286	223	8	55	46	28	18	561	533	114	284	122	13	28	3	17	6	2	113
Widowed or divorced	2,934	1,434	1,156	37	241	136	97	39	1,081	982	163	523	277	19	90	42	47	5	5	283
Never married	7,797	3,408	3,015	84	309	394	325	69	3,473	3,303	753	1,848	652	50	170	64	72	34	----	522
18–24 years	5,405	2,202	2,019	39	144	262	229	33	2,605	2,486	601	1,496	370	19	119	44	46	29	----	336
Married, spouse present	1,826	528	434	12	82	55	43	12	1,022	954	166	635	148	5	68	17	37	14	----	221
Married, spouse absent	80	17	15	2	----	3	3	----	46	46	16	25	5	----	----	----	----	----	----	14
Widowed or divorced	63	8	8	----	----	----	----	----	19	19	5	12	2	----	----	----	----	----	----	36
Never married	3,436	1,649	1,562	25	62	204	183	21	1,518	1,467	414	824	215	14	51	27	9	15	----	65
25–34 years	11,065	3,404	2,465	88	851	377	280	97	6,655	6,289	812	3,910	1,479	88	366	76	202	82	6	629
Married, spouse present	8,941	2,756	1,899	80	777	315	234	81	5,456	5,135	624	3,262	1,176	73	321	65	174	76	6	414
Married, spouse absent	236	43	33	----	10	4	2	2	162	159	25	94	38	2	3	3	----	----	----	27
Widowed or divorced	269	19	16	----	3	5	5	----	119	106	7	52	44	3	13	8	5	----	----	66
Never married	1,679	586	517	8	61	53	39	14	918	889	156	502	221	10	29	----	23	6	----	122
35–44 years	10,755	3,437	2,314	108	1,015	398	281	117	6,286	5,846	760	3,275	1,632	179	440	89	260	81	10	634
Married, spouse present	9,249	2,960	1,954	95	911	356	246	110	5,515	5,120	627	2,912	1,428	153	395	78	231	76	10	418
Married, spouse absent	218	47	36	----	11	3	3	----	142	135	42	48	40	5	7	----	7	----	----	26
Widowed or divorced	290	57	45	----	12	10	7	3	156	148	21	72	50	5	8	3	5	----	----	67
Never married	998	373	279	13	81	29	25	4	473	443	70	243	114	16	30	8	17	5	----	123
45–54 years	9,100	3,325	2,241	127	957	379	291	88	4,899	4,477	623	2,500	1,225	129	422	130	219	66	7	497
Married, spouse present	7,946	2,976	1,982	118	876	318	247	71	4,324	3,948	551	2,188	1,090	119	376	118	195	58	5	328
Married, spouse absent	187	38	31	----	7	14	9	5	114	107	10	66	27	4	7	----	3	2	2	21
Widowed or divorced	328	92	56	4	32	13	8	5	171	155	12	88	49	6	16	3	13	----	----	52
Never married	639	219	172	5	42	34	27	7	280	267	50	158	59	----	23	9	8	6	----	96
55–64 years	6,934	3,392	2,355	100	937	312	243	69	2,865	2,571	486	1,442	598	45	294	111	127	50	6	365
Married, spouse present	5,627	2,861	1,951	75	835	217	176	41	2,308	2,070	391	1,158	489	32	238	90	99	43	6	241
Married, spouse absent	160	74	58	3	13	16	7	9	55	50	16	23	9	2	5	----	5	----	----	15
Widowed or divorced	612	227	162	9	56	30	22	8	317	288	43	169	71	5	29	9	15	5	----	38
Never married	535	230	174	13	43	49	38	11	185	163	36	92	29	6	22	12	8	2	----	71
65 years and over	6,322	4,459	3,636	130	693	277	199	78	1,357	1,183	375	615	177	16	174	109	46	9	10	229
Married, spouse present	4,255	3,010	2,406	83	521	168	127	41	927	807	268	428	99	12	120	82	28	5	5	100
Married, spouse absent	125	67	50	3	14	6	4	2	42	36	5	28	3	----	6	----	2	4	----	10
Widowed or divorced	1,432	1,031	869	24	138	78	55	23	299	266	75	130	61	----	33	19	9	----	5	24
Never married	510	351	311	20	20	25	13	12	89	74	27	29	14	4	15	8	7	----	----	45

FEMALE

| | | |

Table 19. Current rate of cigarette smoking, males 18 years of age and over, by age and previous military service, United States, 1955

[Numbers in thousands]

Military service and age (years)	Total [1]	Nonsmoker of cigarettes			Cigarette smoker											Not reported
		Total	Never smoked cigarettes	Previously smoked cigarettes	Occasional			Regular								
					Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Cigarettes only				Cigarettes and cigars or pipes (by number of cigarettes)			
									Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	
With previous military service	19,120	6,384	4,298	192	706	538	168	10,961	10,188	1,269	6,039	2,654	226	773	187	406	168	12	1,069
18–24	1,455	370	304	12	59	49	10	905	838	132	566	153	7	67	12	21	14	------	121
25–34	8,103	2,418	1,681	89	267	210	57	4,651	4,666	540	2,896	1,163	67	285	58	156	66	5	467
35–44	6,685	1,492	967	48	199	140	59	2,916	2,714	338	1,470	806	98	202	29	133	38	2	276
45–54	1,700	561	336	10	50	38	12	993	892	79	512	271	30	101	24	53	21	3	95
55–64	2,352	1,141	717	47	105	83	22	1,021	907	144	513	233	17	114	43	42	28	2	85
65 and over	825	402	290	17	27	18	9	176	149	36	82	25	6	27	22	2	3	------	23
No military service	30,136	13,645	10,573	393	1,286	977	309	13,618	12,575	2,361	7,151	2,814	249	1,043	373	495	148	27	1,587
18–24	3,887	1,800	1,685	25	199	176	23	1,677	1,605	459	921	214	11	72	31	27	14	------	211
25–34	2,916	971	770	29	109	70	39	1,683	1,603	267	1,061	314	21	80	18	46	16	------	153
35–44	5,836	1,933	1,337	60	198	142	56	3,333	3,113	421	1,790	821	81	240	62	126	44	8	332
45–54	7,351	2,742	1,885	115	332	254	78	3,880	3,564	537	1,977	952	98	325	107	168	45	6	388
55–64	4,550	2,233	1,631	50	196	153	43	1,942	1,660	341	927	366	26	182	69	85	23	5	277
65 and over	5,594	2,966	2,266	113	249	182	67	1,172	1,029	335	535	145	11	143	86	41	6	10	207

[1] Males for whom information on previous military service was not reported have been excluded.

Table 20a. Current rate of *cigarette* smoking, males 18 years of age and over, by pattern of smoking (lifetime history), United States, 1955

[Numbers in thousands]

Pattern of smoking (lifetime history)	Total	Nonsmoker of cigarettes				Cigarette smoker												Not reported
		Total	Never smoked cigarettes	Previously smoked cigarettes		Occasional			Regular									
				Occasionally	Regularly	Total	Never smoked cigarettes regularly	Previously smoked cigarettes regularly	Total	Cigarettes only					Cigarettes and cigars or pipes (by number of cigarettes)					
										Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Less than 10 daily	10–20 daily	21–40 daily	40 or more daily	
Total	49,581	20,219	15,030	592	4,597	2,005	1,523	482	24,667	22,852	3,657	13,238	5,481	476	1,815	559	900	317	39	2,690
Never smoked	10,704	10,704	10,704																	
Occasional smoker	2,448	1,368	993	375		1,080	1,071	9												
Regular smoker	[1]33,213	7,863	3,267	186	4,410	908	445	463	24,437	22,705	3,642	13,133	5,459	471	1,732	542	858	293	39	5
Cigarettes only	23,411	2,845			2,845	252		252	20,314	20,314	3,304	11,798	4,814	398						
Cigars only	1,354	1,227	1,163	64		127	124	3												
Pipes only	1,760	1,518	1,435	83		242	242													
Cigarettes and cigars	1,280	448			448	36		36	796	357	54	215	76	12	439	116	216	88	19	
Cigarettes and pipes	3,712	769			769	149		149	2,794	1,746	247	965	478	56	1,048	331	550	150	17	
Cigars and pipes	808	720	669	39	12	83	79	4												5
Cigarettes, cigars, and pipes	888	336			336	19		19	533	288	37	155	91	5	245	95	92	55	3	
Not reported	3,216	284	66	31	187	17	7	10	230	147	15	105	22	5	83	17	42	24		2,685

[1] Excludes the 353,000 regular smokers classified in appendix table 2 as "pattern not reported." In appendix table 20, this group is classified as "not reported."

Table 20b. Current rate of *cigar* smoking, males 18 years of age and over, by pattern of smoking (lifetime history), United States, 1955

[Numbers in thousands]

Pattern of smoking (lifetime history)	Total	Nonsmoker of cigars				Cigar smoker						Not reported
		Total	Never smoked cigars	Previously smoked cigars		Occasional			Regular			
				Occasionally	Regularly	Total	Never smoked cigars regularly	Previously smoked cigars regularly	Total	Cigars only	Cigars and cigarettes or pipes	
Total	49,581	36,807	34,100	1,291	1,416	7,391	6,872	519	2,636	1,560	1,076	2,747
Never smoked	10,704	10,704	10,704									
Occasional smoker	2,448	1,470	1,331	139		978	968	10				
Regular smoker	[1] 33,213	24,296	21,832	1,136	1,328	6,312	5,813	499	2,600	1,545	1,055	5
Cigarettes only	23,411	19,297	18,608	689		4,114	4,044	70				
Cigars only	1,354	233			233	54		54	1,067	1,067		
Pipes only	1,760	1,174	1,068	106		586	586					
Cigarettes and cigars	1,280	462			462	131		131	687	250	437	
Cigarettes and pipes	3,712	2,517	2,156	341	20	1,190	1,183	7				5
Cigars and pipes	808	187			187	89		89	532	155	377	
Cigarettes, cigars and pipes	888	426			426	148		148	314	73	241	
Not reported	3,216	337	233	16	88	101	91	10	36	15	21	2,742

[1] Excludes the 353,000 regular smokers classified in appendix table 2 as "pattern not reported." In appendix table 20 this group is classified as "not reported."

Table 20c. Current rate of *pipe* smoking, males 18 years of age and over, by pattern of smoking (lifetime history), United States, 1955

[Numbers in thousands]

Pattern of smoking (lifetime history)	Total	Nonsmoker of pipes				Pipe smoker						Not reported
		Total	Never smoked pipes	Previously smoked pipes		Occasional			Regular			
				Occasionally	Regularly	Total	Never smoked pipes regularly	Previously smoked pipes regularly	Total	Pipes only	Pipes and cigarettes or cigars	
Total	49,581	38,553	33,245	2,009	3,299	4,630	4,081	549	3,645	1,979	1,666	2,753
Never smoked	10,704	10,704	10,704									
Occasional smoker	2,448	1,961	1,777	182	2	487	478	9				
Regular smoker	[1] 33,213	25,465	20,567	1,808	3,090	4,112	3,577	535	3,629	1,971	1,658	7
Cigarettes only	23,411	20,244	18,735	1,509		3,167	3,101	66				
Cigars only	1,354	1,106	978	128		248	246	2				
Pipes only	1,760	350			350	48		48	1,362	1,362		
Cigarettes and cigars	1,280	1,040	854	171	15	233	230	3				7
Cigarettes and pipes	3,712	1,914			1,914	291		291	1,507	458	1,049	
Cigars and pipes	808	273			273	39		39	496	117	379	
Cigarettes, cigars and pipes	888	538			538	86		86	264	34	230	
Not reported	3,216	423	197	19	207	31	26	5	16	8	8	2,746

[1] Excludes the 353,000 regular smokers classified in appendix table 2 as "pattern not reported." In appendix table 20 this group is classified as "not reported."

Table 21. Duration of cigarette smoking, current regular cigarette smokers 18 years of age and over, by sex, age, and urban or rural residence, United States, 1955

[Numbers in thousands]

Age and residence	Duration of cigarette smoking (years)							
	Total	Less than 5	5–9	10–14	15–19	20–24	25 or more	Not reported
MALE								
All ages:								
Smokes cigarettes only	22,852	1,497	2,456	3,673	3,093	3,095	8,801	317
Urban	15,618	957	1,693	2,567	2,132	2,035	5,993	241
Rural	7,234	540	763	1,106	961	961	2,808	76
Smokes cigarettes and cigars or pipes	1,815	81	104	215	208	241	920	46
Urban	1,173	50	70	141	127	155	602	28
Rural	642	31	34	74	81	86	318	18
18–24 years:								
Smokes cigarettes only	2,486	1,274	1,041	114	32	----	----	25
Urban	1,634	809	703	91	18	----	----	13
Rural	852	465	338	23	14	----	----	12
Smokes cigarettes and cigars or pipes	119	52	53	12	----	----	----	2
Urban	77	30	34	12	----	----	----	1
Rural	42	22	19	----	----	----	----	1
25–34 years:								
Smokes cigarettes only	6,289	140	1,302	3,143	1,463	124	41	76
Urban	4,345	93	913	2,206	975	73	21	64
Rural	1,944	47	389	937	488	51	20	12
Smokes cigarettes and cigars or pipes	366	21	44	184	100	11	----	6
Urban	226	13	31	119	52	8	----	3
Rural	140	8	13	65	48	3	----	3
35–44 years:								
Smokes cigarettes only	5,846	25	51	295	1,408	2,491	1,497	79
Urban	4,008	20	44	203	1,016	1,667	1,000	58
Rural	1,838	5	7	92	392	824	497	21
Smokes cigarettes and cigars or pipes	440	3	----	14	93	193	119	18
Urban	286	3	----	10	65	124	69	15
Rural	154	----	----	4	28	69	50	3
45–54 years:								
Smokes cigarettes only	4,477	32	36	69	137	279	3,859	65
Urban	3,041	19	20	44	89	201	2,621	47
Rural	1,436	13	16	25	48	78	1,238	18
Smokes cigarettes and cigars or pipes	422	----	3	----	10	23	381	5
Urban	295	----	3	----	10	16	266	----
Rural	127	----	----	----	----	7	115	5
55–64 years:								
Smokes cigarettes only	2,571	12	16	36	32	81	2,350	44
Urban	1,806	5	8	18	23	59	1,662	31
Rural	765	7	8	18	9	22	688	13
Smokes cigarettes and cigars or pipes	294	3	2	----	5	9	273	2
Urban	189	3	----	----	----	5	181	----
Rural	105	----	2	----	5	4	92	2
65 years and over:								
Smokes cigarettes only	1,183	14	10	16	21	40	1,054	28
Urban	784	11	5	5	11	35	689	28
Rural	399	3	5	11	10	5	365	----
Smokes cigarettes and cigars or pipes	174	2	2	5	----	5	147	13
Urban	100	1	2	----	----	2	86	9
Rural	74	1	----	5	----	3	61	4
FEMALE								
All ages	12,986	1,886	2,588	2,786	2,185	1,774	1,671	96
Urban	9,858	1,381	1,966	2,034	1,725	1,388	1,294	70
Rural	3,128	505	622	752	460	386	377	26
18–24 years	2,118	1,263	785	38	20	----	----	12
Urban	1,614	946	618	33	10	----	----	7
Rural	504	317	167	5	10	----	----	5
25–34 years	4,192	354	1,374	1,789	591	47	17	20
Urban	3,093	238	1,043	1,291	473	33	8	7
Rural	1,099	116	331	498	118	14	9	13
35–44 years	3,596	120	243	607	1,182	1,137	281	26
Urban	2,750	86	174	447	938	883	226	26
Rural	846	34	69	160	274	254	55	----
45–54 years	2,067	106	123	248	283	414	869	24
Urban	1,608	87	82	182	234	329	671	23
Rural	459	19	41	66	49	85	198	1
55–64 years	767	31	56	83	92	127	365	13
Urban	601	17	42	63	83	101	288	7
Rural	166	14	14	20	9	26	77	6
65 years and over	246	12	7	21	17	49	139	1
Urban	192	7	7	18	17	42	101	----
Rural	54	5	----	3	----	7	38	1

Table 22a. Regular *cigarette* smokers 18 years of age and over, cross-classified by maximum rate of cigarette smoking (lifetime history) and by current rate of cigarette smoking, by sex and age, United States, 1955

[Numbers in thousands]

Age and maximum rate (number cigarettes daily)	Total	Previous regular cigarettes smokers			Current regular cigarette smoker										
						Regularly smokes—									
						Cigarettes only							Cigarettes and cigars or pipes [1]		
								Once smoked more (by current number)							
		Total	Non-smoker of cigarettes now	Occasional smoker of cigarettes now	Total	Total	Now smoking at maximum	Total	More than 10 daily	10–20 daily	21–40 daily	41 or more daily	Total	Now smoking cigarettes at maximum	Once smoked more
	MALE														
All ages	29,746	5,079	4,597	482	24,667	22,852	20,500	2,352	780	1,358	211	3	1,815	1,380	435
Less than 10	4,263	1,012	959	53	3,251	2,877	2,877						374	364	10
10–20	15,994	2,602	2,270	332	13,392	12,495	11,880	615	615				897	710	187
21–40	8,124	1,053	983	70	7,071	6,610	5,270	1,340	128	1,212			461	276	185
41 or more	1,055	247	243	4	808	744	473	271	8	82	181		64	30	34
Not reported	310	165	142	23	145	126		126	29	64	30	3	19		19
18–24 years	2,782	177	144	33	2,605	2,486	2,289	197	67	114	16		119	90	29
Less than 10	642	70	60	10	572	534	534						38	38	
10–20	1,562	82	62	20	1,480	1,432	1,382	50	50				48	33	15
21–40	502	15	15		487	461	354	107	9	98			26	19	7
41 or more	54	5	5		49	44	19	25		9	16		5		5
Not reported	22	5	2	3	17	15		15	8	7			2		2
25–34 years	7,603	948	851	97	6,655	6,289	5,618	671	181	426	64		366	281	85
Less than 10	816	141	123	18	675	631	631						44	44	
10–20	4,386	564	500	64	3,822	3,634	3,484	150	150				188	155	33
21–40	2,125	186	176	10	1,939	1,812	1,415	397	23	374			127	78	49
41 or more	222	36	36		186	179	88	91	3	34	54		7	4	3
Not reported	54	21	16	5	33	33		33	5	18	10				
35–44 years	7,418	1,132	1,015	117	6,286	5,846	5,298	548	167	344	37		440	325	115
Less than 10	849	202	188	14	647	593	593						54	52	2
10–20	3,852	559	472	87	3,293	3,067	2,931	136	136				226	196	30
21–40	2,362	289	273	16	2,073	1,937	1,595	342	28	314			136	70	66
41 or more	281	39	39		242	224	179	45		15	30		18	7	11
Not reported	74	43	43		31	25		25	3	15	7		6		6
45–54 years	5,944	1,045	957	88	4,899	4,477	4,010	467	144	248	72	3	422	326	96
Less than 10	756	194	186	8	562	479	479						83	83	
10–20	3,121	515	455	60	2,606	2,370	2,252	118	118				236	180	56
21–40	1,718	232	217	15	1,486	1,398	1,153	245	20	225			88	58	30
41 or more	277	66	66		211	201	126	75	3	10	62		10	5	5
Not reported	72	38	33	5	34	29		29	3	13	10	3	5		5
55–64 years	3,871	1,006	937	69	2,865	2,571	2,280	291	119	163	9		294	240	54
Less than 10	650	202	199	3	448	367	367						81	77	4
10–20	2,018	519	468	51	1,499	1,364	1,279	85	85				135	111	24
21–40	1,024	196	187	9	828	761	589	172	29	143			67	45	22
41 or more	136	67	63	4	69	60	45	15		9	6		9	7	2
Not reported	43	22	20	2	21	19		19	5	11	3		2		2

| | | |

Table 22b. Regular male *cigar* smokers 18 years of age and over, cross-classified by maximum rate of cigar smoking (lifetime history) and by current rate of cigar smoking, United States, 1955

[Numbers in thousands]

Maximum rate (number cigars daily)	Total	Previous regular cigar smoker			Current regular cigar smoker						
		Total	Non-smoker of cigars now	Occasional smoker of cigars now	Total	Cigars only			Cigars and cigarettes or pipes		
						Total	Now smoking at maximum	Once smoked more cigars	Total	Now smoking cigars at maximum	Once smoked more cigars
All cigar smokers	4,571	1,935	1,416	519	2,636	1,560	1,333	227	1,076	911	165
1-2	2,408	951	547	404	1,457	762	596	166	695	588	107
3-4	1,221	442	425	17	779	510	458	52	269	232	37
5-8	594	281	281	----	313	240	240	----	73	71	2
9 or more	126	65	65	----	61	41	39	2	20	20	----
Not reported	222	196	98	98	26	7	----	7	19	----	19

Table 22c. Regular male *pipe* smokers 18 years of age and over, cross-classified by maximum rate of pipe smoking (lifetime history) and by current rate of pipe smoking, United States, 1955

[Numbers in thousands]

Maximum rate (number pipefuls daily)	Total	Previous regular pipe smoker			Current regular pipe smoker						
		Total	Non-smoker of pipes now	Occasional smoker of pipes now	Total	Pipes only			Pipes and cigarettes or cigars		
						Total	Now smoking at maximum	Once smoked more	Total	Now smoking pipes at maximum	Once smoked pipes more
Total	7,493	3,848	3,299	549	3,645	1,979	1,809	170	1,666	1,484	182
Under 5	3,457	1,791	1,478	313	1,666	736	680	56	930	863	67
5-9	2,354	1,100	965	135	1,254	732	658	74	522	422	100
10-19	1,072	521	515	6	551	389	370	19	162	156	6
20 or more	284	137	137	----	147	104	101	3	43	43	----
Not reported	326	299	204	95	27	18	----	18	9	----	9

Table 23. Maximum rate of cigarette smoking, regular cigarette smokers 18 years of age and over (lifetime history), by sex, age, and urban or rural residence, United States, 1955

[Numbers in thousands]

Residence and age (years)	Male													Female					
	Total	Regularly smoked cigarettes only (lifetime history)						Regularly smoked cigarettes and cigars or pipes (lifetime history; by number of cigarettes)						Regularly smoked cigarettes only (lifetime history)					
		Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Not reported	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Not reported	Total	Less than 10 daily	10–20 daily	21–40 daily	41 or more daily	Not reported
	NUMBER																		
Urban	20,095	16,280	2,206	8,778	4,577	528	-191	3,815	547	1,882	1,127	242	17	11,136	3,614	6,005	1,324	116	77
18–24	1,819	1,633	353	946	298	25	11	186	34	90	48	14	.....	1,768	648	915	187	10	8
25–34	5,192	4,478	475	2,576	1,260	120	47	714	58	332	284	40	.....	3,470	1,014	1,973	426	55	2
35–44	4,994	4,081	471	2,092	1,317	155	46	913	80	465	309	59	.....	3,100	947	1,760	353	18	22
45–54	3,995	3,178	385	1,654	973	139	27	817	114	423	206	57	17	1,822	601	926	263	26	6
55–64	2,710	1,981	294	1,047	551	63	26	729	134	348	204	43	.....	715	284	333	85	7	6
65 and over	1,385	929	228	463	178	26	34	456	127	224	76	29	.....	261	120	98	10	.....	33
Rural nonfarm	6,321	4,956	708	2,740	1,360	133	15	1,365	192	719	357	57	40	3,053	1,020	1,633	333	38	29
18–24	577	522	144	271	103	4	.....	55	8	32	6	.....	9	458	192	231	54	2	8
25–34	1,697	1,437	161	860	365	44	7	260	23	162	66	9	.....	1,127	337	641	131	10	8
35–44	1,644	1,308	130	696	442	40	.....	336	33	184	83	18	18	825	275	471	68	9	2
45–54	1,208	918	113	486	286	27	6	290	42	153	78	15	2	425	127	226	57	12	3
55–64	741	517	106	284	117	10	.....	224	33	97	76	10	8	158	62	75	18	3	.....
65 and over	454	254	54	143	47	8	2	200	53	91	48	5	3	60	27	19	5	2	7
Rural farm	3,330	2,601	463	1,495	499	67	47	729	117	380	204	28	.....	744	294	353	74	12	11
18–24	386	350	97	203	41	9	.....	36	8	22	6	.....	.....	135	75	49	8	3	.....
25–34	714	616	92	401	113	8	2	98	5	53	38	2	.....	259	80	147	24	3	5
35–44	780	640	111	340	170	11	8	140	24	75	41	.....	.....	177	67	84	22	4	.....
45–54	741	536	78	299	116	23	20	205	24	106	59	16	.....	114	48	50	12	.....	4
55–64	420	272	54	161	42	5	10	148	29	81	33	5	.....	46	15	21	8	2	.....
65 and over	289	187	61	91	17	11	7	102	27	43	27	5	.....	13	9	2	.....	.....	2
	PERCENT [1]																		
Urban	61.7	50.0	6.8	26.9	14.1	1.6	0.6	11.7	1.7	5.8	3.5	0.7	0.1	29.7	9.7	16.0	3.5	0.3	0.2
18–24	53.2	47.7	10.3	27.7	8.7	.7	.3	5.4	1.0	2.6	1.4	.4	.....	35.6	13.0	18.4	3.8	.2	.2
25–34	69.9	60.3	6.4	34.7	17.0	1.6	.6	9.6	.8	4.5	3.8	.5	.....	42.2	12.3	24.0	5.2	.7	.9
35–44	70.1	57.3	6.6	29.4	18.5	2.2	.6	12.8	1.1	6.5	4.3	.8	.....	39.6	12.1	22.5	4.5	.2	.3
45–54	65.8	52.3	6.3	27.2	16.0	2.2	.4	13.4	1.9	7.0	3.4	.9	.3	28.1	9.3	14.3	4.1	.4	.1
55–64	57.9	42.3	6.3	22.4	11.8	1.3	.6	15.6	2.9	7.4	4.4	.9	.....	14.2	5.6	6.6	1.7	.1	.1
65 and over	36.1	24.2	5.9	12.1	4.6	.7	.9	11.9	3.3	5.8	2.0	.8	.....	5.3	2.4	2.0	.2	.....	.7
Rural nonfarm	60.5	47.4	6.8	26.2	13.0	1.3	.1	13.1	1.8	6.9	3.4	.5	.4	26.6	8.9	14.2	2.9	.3	.3
18–24	51.7	46.8	12.9	24.3	9.2	.4	.....	4.9	.7	2.9	.5	.....	.8	27.7	11.6	12.2	3.3	.1	.5
25–34	66.9	56.7	6.4	33.9	14.4	1.7	.3	10.3	.9	6.4	2.6	.4	.....	39.2	11.7	22.3	4.6	.3	.3
35–44	68.1	54.2	5.4	28.8	18.3	1.7	.....	13.9	1.4	7.6	3.4	.7	.7	34.8	11.6	19.9	2.9	.4	.1
45–54	68.4	52.0	6.4	27.5	16.2	1.5	.3	16.4	2.4	8.7	4.4	.8	.1	23.8	7.1	12.6	3.2	.7	.2
55–64	57.6	40.2	8.2	22.1	9.1	.8	.....	17.4	2.6	7.5	5.9	.8	.6	12.3	4.8	5.8	1.4	.2	.....
65 and over	33.9	19.0	4.0	10.7	3.5	.6	.1	14.9	4.0	6.8	3.6	.4	.2	4.0	1.8	1.3	.3	.1	.5
Rural farm	50.8	39.7	7.5	22.8	7.6	1.0	.7	11.1	1.8	5.8	3.1	.4	.....	12.1	4.8	5.7	1.2	.2	.2
18–24	44.5	40.3	11.1	23.4	4.7	1.0	.....	4.1	.9	2.5	.7	.....	.....	16.0	8.9	5.8	.9	.4	.....
25–34	64.9	56.0	8.3	36.5	10.3	.7	.2	8.9	.5	4.8	3.5	.2	.....	23.5	7.3	13.4	2.2	.3	.5
35–44	64.1	52.6	9.1	28.0	14.0	.9	.7	11.5	2.0	6.2	3.4	.....	.....	13.8	5.2	6.6	1.7	.3	.....
45–54	58.9	42.6	6.2	23.8	9.2	1.8	1.6	16.3	1.9	8.4	4.7	1.3	.....	10.1	4.2	4.4	1.1	.....	.4
55–64	43.3	28.1	5.6	16.6	4.3	.5	1.0	13.3	3.0	8.4	3.4	.5	.....	4.7	1.5	2.1	.8	.2	.....
65 and over	25.3	16.4	5.3	8.0	1.5	1.0	.6	8.9	2.4	3.8	2.4	.4	.....	1.5	1.1	.2	.....	.....	2

[1] Based on total population in each sex, age, and residence group; see appendix table 2.

Table 24. Maximum rate of cigarette smoking, regular cigarette smokers 18 years of age and over (lifetime history), by sex, age, and age started smoking, United States, 1955

[Numbers in thousands]

Current age and age started smoking (years)[1]	Male (number cigarettes smoked daily)						Female (number cigarettes smoked daily)					
	Total respondents	Less than 10	10–20	21–40	41 or more	Not reported	Total respondents	Less than 10	10–20	21–40	41 or more	Not reported
All ages	29,766	4,263	15,994	8,124	1,055	310	14,933	4,928	7,991	1,731	166	117
Under 18	14,114	1,543	7,514	4,409	621	27	3,709	839	2,171	606	86	7
18–19	7,499	1,157	4,175	1,946	210	11	3,498	1,146	1,920	408	24	-------
20–21	4,492	738	2,543	1,066	140	5	2,554	867	1,428	233	26	-------
22–24	1,247	230	675	307	32	3	1,239	454	659	126	-------	-------
25 and over	1,857	527	965	324	41	-------	3,741	1,595	1,777	349	20	-------
Starting age not reported [1]	537	68	122	72	11	264	192	27	36	9	10	110
18–24 years	2,782	642	1,562	502	54	22	2,361	914	1,164	246	15	22
Under 18	1,819	363	1,035	368	51	2	1,181	372	639	153	10	7
18–19	733	204	418	107	2	2	853	386	388	79	-------	-------
20–21	168	60	84	24	-------	-------	257	130	113	14	-------	-------
22–24	29	12	17	-------	-------	-------	44	24	20	-------	-------	-------
Starting age not reported	33	3	8	3	1	18	26	2	4	-------	5	15
25–34 years	7,603	816	4,386	2,125	222	54	4,856	1,430	2,756	577	69	24
Under 18	3,930	293	2,242	1,255	133	7	1,449	249	897	257	46	-------
18–19	2,211	266	1,300	592	50	3	1,464	461	825	165	13	-------
20–21	977	151	607	180	36	3	973	303	570	90	10	-------
22–24	305	65	165	72	3	-------	531	220	264	47	-------	-------
25 and over	90	35	40	15	-------	-------	407	189	200	18	-------	-------
Starting age not reported	90	6	32	11	-------	41	32	8	-------	-------	-------	24
35–44 years	7,418	849	3,852	2,362	281	74	4,102	1,288	2,308	443	31	32
Under 18	3,421	291	1,732	1,237	159	2	803	166	479	138	20	-------
18–19	1,967	223	1,071	606	61	6	861	217	545	97	2	-------
20–21	1,162	171	636	312	43	-------	862	272	509	74	7	-------
22–24	354	36	192	117	9	-------	417	132	245	40	-------	-------
25 and over	381	112	200	61	8	-------	1,113	499	520	92	2	-------
Starting age not reported	133	16	21	29	1	66	46	2	10	2	-------	32
45–54 years	5,944	756	3,121	1,718	277	72	2,361	774	1,190	333	37	27
Under 18	2,522	233	1,308	820	153	8	185	24	112	39	10	-------
18–19	1,411	181	787	370	73	-------	257	64	129	55	9	-------
20–21	1,125	165	594	327	39	-------	376	121	198	48	9	-------
22–24	281	38	165	75	-------	3	208	63	115	30	-------	-------
25 and over	504	124	257	111	12	-------	1,279	493	621	158	7	-------
Starting age not reported	101	15	10	15	-------	61	56	9	15	3	2	27
55–64 years	3,871	650	2,018	1,024	136	43	919	359	429	110	13	8
Under 18	1,545	191	758	514	76	6	61	11	35	15	-------	-------
18–19	890	184	466	223	17	-------	41	15	14	12	-------	-------
20–21	731	115	432	165	17	2	66	29	30	7	-------	-------
22–24	185	43	100	31	11	-------	31	12	10	9	-------	-------
25 and over	436	101	232	88	15	-------	698	286	338	65	9	-------
Starting age not reported	84	16	30	3	-------	35	22	6	2	3	3	8
65 years and over	2,128	550	1,055	393	85	45	334	163	144	21	2	4
Under 18	877	172	439	215	49	2	30	17	9	4	-------	-------
18–19	287	99	133	48	7	-------	22	3	19	-------	-------	-------
20–21	329	76	190	58	5	-------	20	12	8	-------	-------	-------
22–24	93	36	36	12	9	-------	8	3	5	-------	-------	-------
25 and over	446	155	236	49	6	-------	244	128	98	16	2	-------
Starting age not reported	96	12	21	11	9	43	10	-------	5	1	-------	4

[1] Distribution by age started smoking does not agree completely with data presented in appendix table 7 because of minor differences in tabulation processing and control.

Table 25. Regular smokers 18 years of age and over (any form, lifetime history), by sex, age, geographic region, and age started smoking, United States, 1955

[Numbers in thousands]

Geographic region, and age started smoking (years)	Male							Female						
	Total	18–24 years	25–34 years	35–44 years	45–54 years	55–64 years	65 years and over	Total	18–24 years	25–34 years	35–44 years	45–54 years	55–64 years	65 years and over
United States	33,566	2,878	7,920	7,850	6,545	4,719	3,654	14,953	2,346	4,843	4,090	2,362	933	379
Under 14	1,888	189	367	434	359	278	261	230	65	77	33	24	15	16
14–15	4,194	456	1,021	997	739	548	433	862	292	312	172	40	31	15
16–17	8,630	1,192	2,585	2,029	1,478	829	517	2,644	823	1,061	604	128	17	11
18–19	8,089	781	2,270	2,051	1,505	1,010	472	3,518	855	1,464	862	260	46	31
20–21	5,430	196	1,071	1,251	1,301	958	653	2,560	257	973	864	376	69	21
22–24	1,646	37	385	408	327	281	208	1,240	43	531	419	207	32	8
25–29	1,548	2	117	358	361	328	382	1,452	2	343	600	375	100	32
30–34	757	-------	32	155	194	141	235	922	-------	61	344	344	144	29
35–39	358	-------	-------	61	93	110	94	572	-------	2	145	275	114	36
40 or more	597	2	-------	13	115	168	299	825	-------	-------	22	260	348	165
Not reported	429	23	72	93	73	68	100	128	9	19	25	43	17	15
Northeast	9,371	769	2,221	2,178	1,635	1,483	1,094	4,581	676	1,332	1,298	828	316	131
Under 14	468	36	85	115	51	87	94	59	22	13	12	7	5	-------
14–15	1,128	118	286	241	169	177	137	219	62	81	50	10	12	4
16–17	2,537	333	799	615	369	270	151	828	256	268	224	66	5	9
18–19	2,283	199	609	576	384	341	174	1,129	248	452	276	116	26	11
20–21	1,513	48	278	328	359	292	208	774	71	268	270	132	22	11
22–24	412	14	98	101	79	75	45	372	13	146	117	77	17	2
25–29	427	-------	30	88	100	115	94	435	2	83	188	124	27	11
30–34	205	-------	5	49	51	30	70	270	-------	17	110	99	40	4
35–39	114	-------	-------	22	27	39	26	149	-------	-------	37	77	29	6
40 or more	134	2	-------	-------	25	44	63	310	-------	-------	5	106	128	71
Not reported	150	10	31	43	21	13	32	36	2	4	9	14	5	2
North Central	10,228	826	2,548	2,287	1,962	1,495	1,110	4,176	727	1,438	1,080	616	242	73
Under 14	398	33	72	80	103	65	45	45	8	17	10	5	2	3
14–15	1,150	113	282	263	207	154	131	242	91	79	48	12	8	4
16–17	2,554	353	815	579	417	237	153	802	294	327	145	29	7	-------
18–19	2,715	250	840	693	474	329	129	1,008	241	445	251	64	2	5
20–21	1,796	64	354	399	407	345	227	718	81	314	252	86	19	6
22–24	495	13	118	107	90	98	69	303	7	137	107	47	5	-------
25–29	471	-------	44	89	122	89	127	369	-------	95	140	101	29	4
30–34	199	-------	8	24	55	42	70	273	-------	15	110	98	34	16
35–39	105	-------	-------	14	28	33	30	191	-------	-------	38	90	47	16
40 or more	194	-------	-------	5	29	64	96	170	-------	-------	10	62	81	17
Not reported	151	-------	15	34	30	39	33	55	5	9	9	22	8	2
South	9,437	950	2,228	2,291	1,898	1,150	920	3,689	590	1,271	951	545	215	117
Under 14	729	96	168	182	132	81	70	83	22	29	7	9	6	10
14–15	1,459	174	360	403	240	158	124	238	85	116	27	16	7	7
16–17	2,538	380	705	605	489	230	129	644	205	287	130	20	-------	2
18–19	2,008	239	541	480	422	218	108	773	193	318	173	61	18	10
20–21	1,303	43	277	323	316	213	131	670	70	233	241	109	16	1
22–24	431	5	122	102	86	70	46	305	15	148	97	35	7	3
25–29	400	-------	32	114	82	76	96	389	-------	119	157	75	29	9
30–34	208	-------	13	48	51	36	60	198	-------	14	65	84	26	9
35–39	84	-------	-------	15	17	20	32	133	-------	2	42	51	24	14
40 or more	184	-------	-------	5	42	32	105	214	-------	-------	5	79	80	50
Not reported	93	13	10	14	21	16	19	22	-------	5	7	6	2	2
West	4,530	342	923	1,094	1,050	591	530	2,507	353	802	761	373	160	58
Under 14	293	24	42	57	73	45	52	43	13	18	4	3	2	3
14–15	457	51	93	90	123	59	41	143	54	36	47	2	4	-------
16–17	1,001	126	266	230	203	92	84	370	68	179	105	13	5	-------
18–19	1,083	93	280	302	225	122	61	608	173	249	162	19	-------	5
20–21	818	41	162	201	219	108	87	398	35	158	141	49	12	3
22–24	308	5	47	98	72	38	48	260	8	100	98	48	3	3
25–29	250	2	11	67	57	48	65	259	-------	46	115	75	15	8
30–34	145	-------	6	34	37	33	35	181	-------	15	59	63	44	-------
35–39	55	-------	-------	10	21	18	6	99	-------	-------	28	57	14	-------
40 or more	85	-------	-------	3	19	28	35	131	-------	-------	2	43	59	27
Not reported	35	-------	16	2	1	-------	16	15	2	1	-------	1	2	9

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ADDENDUM

Tobacco Consumption in the United States, 1880 to 1955

Benno K. Milmore, M.D., M.P.H., and Arthur G. Conover, M.A.

Estimates of consumption of cigarettes and other tobacco products given in this article are derived from two sources: records of production, imports, tax-paid and tax-free removal for domestic consumption of tobacco products, as compiled by the Internal Revenue Service, and information on consumption of tobacco products published recently by the Agricultural Marketing Service, United States Department of Agriculture (*1*). These data were adjusted to obtain comparable estimates of the consump-

NOTE: With figures through 1954 only, this article appeared in Agricultural Economic Research for January 1956, vol. 8, pp. 9–13.

Table 1. Tobacco products, unstemmed-processing weight: Consumption per person over 14 years of age, United States, 1880–1955 [1]

Year	Tobacco						Year	Tobacco					
	Cigarettes	Cigars	Smoking	Chewing	Snuff	Total		Cigarettes	Cigars	Smoking	Chewing	Snuff	Total
	Lb.	*Lb.*	*Lb.*	*Lb.*	*Lb.*	*Lb.*		*Lb.*	*Lb.*	*Lb.*	*Lb.*	*Lb.*	*Lb.*
1880	0.047	1.36	0.73	3.15	0.12	5.41	1918	1.70	2.26	2.03	2.63	0.50	9.12
1881	.053	1.50	.82	3.62	.13	6.12	1919	1.98	2.19	1.70	2.47	.46	8.80
1882	.055	1.60	.83	3.13	.14	5.75	1920	1.89	2.45	1.50	2.36	.47	8.67
1883	.069	1.66	.91	3.80	.15	6.59	1921	2.07	2.00	1.56	2.13	.45	8.21
1884	.085	1.64	.84	3.20	.16	5.93	1922	2.14	2.16	1.67	2.13	.48	8.58
1885	.10	1.61	.94	3.82	.17	6.64	1923	2.51	2.21	1.59	2.19	.48	8.98
1886	.12	1.67	.97	3.73	.17	6.66	1924	2.69	2.06	1.62	1.97	.47	8.81
1887	.14	1.70	1.04	3.88	.20	6.96	1925	2.96	1.99	1.61	1.97	.45	8.98
1888	.16	1.68	.99	3.50	.13	6.46	1926	3.17	1.99	1.53	1.90	.44	9.03
1889	.17	1.69	1.05	4.04	.21	7.16	1927	3.42	1.93	1.42	1.73	.46	8.96
1890	.18	1.78	1.12	3.99	.22	7.29	1928	3.58	1.91	1.34	1.64	.46	8.93
1891	.20	1.83	1.20	4.13	.24	7.60	1929	3.91	1.86	1.38	1.62	.44	9.21
1892	.22	1.86	1.15	4.11	.25	7.59	1930	3.84	1.67	1.44	1.47	.43	8.85
1893	.22	1.72	1.14	3.53	.26	6.87	1931	3.63	1.53	1.59	1.28	.42	8.45
1894	.22	1.56	1.22	3.71	.25	6.96	1932	3.21	1.24	1.73	1.08	.38	7.64
1895	.25	1.56	1.20	3.77	.21	6.99	1933	3.49	1.23	1.69	1.00	.38	7.79
1896	.27	1.51	1.17	3.40	.26	6.61	1934	3.94	1.29	1.71	1.02	.38	8.34
1897	.25	1.60	1.18	3.93	.27	7.23	1935	4.11	1.30	1.52	.91	.37	8.21
1898	.21	1.75	1.20	3.42	.27	6.85	1936	4.61	1.40	1.52	.91	.38	8.82
1899	.17	1.90	1.36	3.49	.28	7.20	1937	4.81	1.40	1.47	.90	.37	8.95
1900	.16	1.99	1.42	3.56	.30	7.43	1938	4.76	1.31	1.51	.80	.37	8.75
1901	.16	2.08	1.50	3.47	.32	7.53	1939	4.95	1.32	1.45	.74	.37	8.83
1902	.18	2.20	1.57	3.41	.33	7.69	1940	5.16	1.36	1.50	.74	.36	9.12
1903	.18	2.29	1.78	3.75	.35	8.35	1941	5.95	1.42	1.33	.71	.37	9.78
1904	.19	2.26	2.00	3.74	.36	8.55	1942	7.01	1.41	1.13	.75	.39	10.69
1905	.19	2.31	2.18	3.56	.37	8.61	1943	7.99	1.28	1.02	.77	.40	11.46
1906	.23	2.40	2.14	3.60	.38	8.75	1944	8.04	1.22	.82	.74	.40	11.22
1907	.26	2.37	2.15	3.51	.37	8.66	1945	9.15	1.26	.96	.68	.41	12.46
1908	.28	2.19	2.12	3.39	.39	8.37	1946	9.24	1.37	.60	.63	.36	12.20
1909	.32	2.20	2.17	3.56	.45	8.70	1947	9.16	1.29	.59	.56	.35	11.95
1910	.41	2.19	2.17	3.35	.47	8.59	1948	9.35	1.31	.58	.52	.36	12.12
1911	.48	2.28	2.20	3.25	.43	8.64	1949	9.33	1.16	.59	.49	.36	11.93
1912	.60	2.27	2.20	3.21	.46	8.74	1950	9.36	1.18	.59	.49	.34	11.96
1913	.71	2.36	2.08	3.06	.47	8.68	1951	9.98	1.19	.53	.45	.33	12.48
1914	.74	2.27	2.06	2.88	.43	8.38	1952	10.41	1.26	.49	.43	.33	12.92
1915	.82	2.17	2.13	2.84	.44	8.40	1953	10.46	1.26	.44	.42	.32	12.90
1916	1.13	2.32	2.05	3.00	.47	8.97	1954	9.73	1.22	.43	.41	.32	12.11
1917	1.58	2.42	1.96	3.03	.46	9.45	1955 [2]	9.56	1.22	.40	.40	.32	11.90

[1] 1940–55 data include armed forces overseas.
[2] Provisional.

tion of each of the five principal classes of these products in the United States during the last 75 years. Table 1 shows these estimates in terms of unstemmed-processing weight of tobacco per person over 14 years of age.

Unstemmed-processing weight represents weight of tobacco before stems are removed. It does not include any nontobacco materials, such as sugar, honey, and licorice, that may be added during manufacturing operations. Farm production and total supplies of tobacco, including stocks, are generally stated in terms of farm-sales weight. The unstemmed-processing weight of tobacco averages about 10 percent less than its equivalent farm-sales weight.

The actual weight of the tobacco in cigarettes and cigars in their finished form is of course less than the unstemmed-weight equivalent as shown in tables 1 and 4. By the time the unstemmed tobacco has reached the product-making stage, there has been about a 25-percent loss in weight due to stemming or removal of the midrib from the leaves. Over a period of many years, this percentage loss in weight probably has varied because of differences in physical characteristics of tobacco and modifications in the processing and manufacturing operations. Therefore, the 25-percent weight difference should be regarded only as a rough approximation. Sufficient information is not available as to what allowances should be made for losses in the final leaf cleaning and cutting operations, or for differences in moisture content, but they would be small compared with the stemming factor. Recently, processes have been developed by which stems, scraps, and other tobacco are ground up and reconstituted into thin sheets for use in tobacco products. If these processes should be adopted on a broad scale, the difference between the actual weight of manufactured products and the unstemmed weight equivalent will tend to become smaller.

To facilitate conversion of unstemmed-processing weight of tobacco in cigarettes and cigars into numbers of cigarettes and cigars, appropriate conversion factors were used (table 2). Recent changes in the number of cigarettes per pound of tobacco are principally attributable to the increased popularity of filter tip and of king-size cigarettes. It has been estimated that in 1955 about 27 percent of domestically consumed cigarettes were king-size, non-filter tips—an increase of 44 percent since 1952. These estimates also indicated that filter tip cigarettes, both regular and king-size, accounted for about 19 percent of the total domestic consumption—about 13 times as many as in 1952.

Table 2. Number of cigarettes and cigars per pound of tobacco, unstemmed-processing weight; averages 1880–1954, annual 1950–55

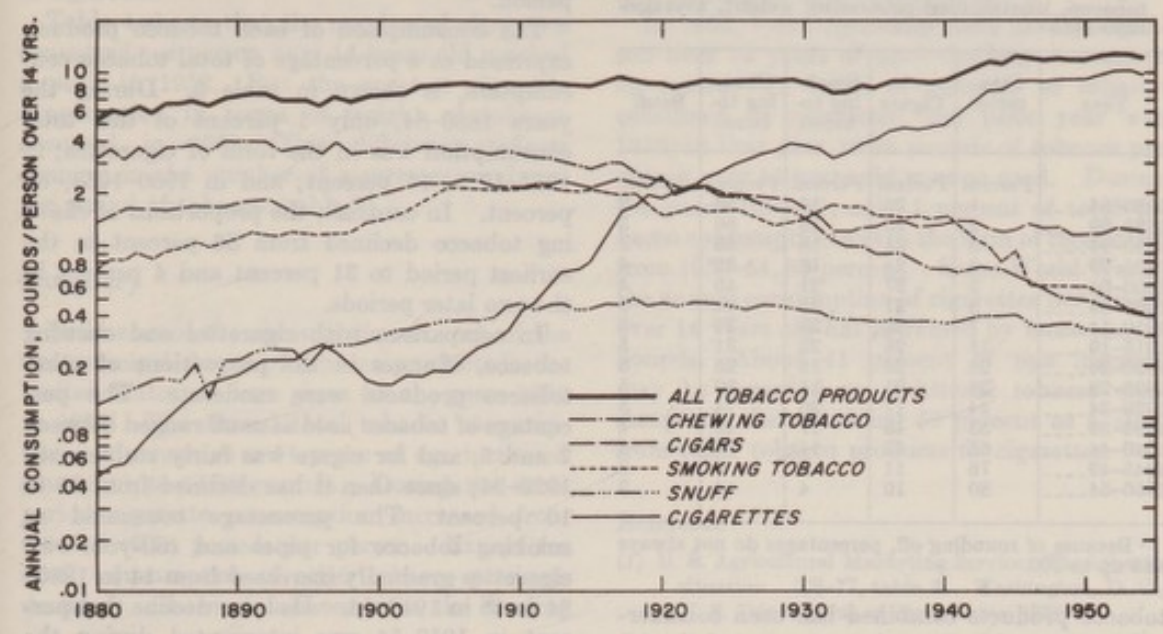
Period	Cigarettes	Cigars [1]
	Average	
1880–99	333	59. 9
1900–09	334	63. 5
1910–19	332	56. 3
1920–29	338	47. 3
1930–39	353	43. 6
1940–44	348	42. 1
1945–49	351	42. 1
1950–54	349	43. 1
	Annual	
1950	355	43. 0
1951	353	43. 7
1952	352	42. 8
1953	340	42. 9
1954	343	43. 3
1955 [2]	354	43. 2

[1] Small cigars (weighing not more than 3 pounds per 1,000) represented 6 to 14 percent of all cigars from 1900 to 1929 but had diminished to about 1 percent by 1950 to 1954.
[2] Provisional.

Estimates of the unstemmed-processing weights consumed separately as smoking and chewing tobacco are only approximate and mainly reflect their respective proportions on a manufactured-weight basis. Consumption figures given here do not include the home use of natural leaf for chewing and smoking. This indeterminate quantity probably was noteworthy, particularly in tobacco-growing areas in the earlier years of the period covered, but undoubtedly it dwindled to a minor quantity with the passage of time.

Cigarettes that are represented in the data here given are those produced in manufacturing plants; they do not include roll-your-own cigarettes. Tobacco used in these cigarettes is included in the smoking tobacco category and is not separately available. In the years 1933-40, consumption of roll-your-own cigarettes was

Figure 1. Tobacco products, unstemmed-processing weight: Consumption per person over 14 years of age, United States, 1880–1954



considerably larger than during later years and was probably above the pre-1933–40 period. It is estimated that during 1933–40, the tobacco used in the roll-your-own cigarettes comprised 46 percent of the smoking tobacco category, but by 1950–54 this proportion had dropped to around 30 percent.

For the purpose of computing average annual tobacco consumption per person, the tobacco-consuming population is arbitrarily considered as all persons 15 or more years old. Although this appears more realistic than use of the total population, it obviously does not adequately represent the tobacco-consuming segment of the population. Little is known concerning how much of the increase or decrease in consumption of various tobacco products per person over 14 years old is attributable to changes in the proportion of such people who use tobacco rather than to changes in the average quantities consumed by users of tobacco.

Variation in Trend, by Product

Trends of consumption of tobacco, based on table 1, are portrayed on a semilogarithmic scale in figure 1. The consumption of all

Figure 2. Tobacco products, unstemmed-processing weight: Consumption per person over 14 years of age, United States, selected 5-year periods, 1880–1954.

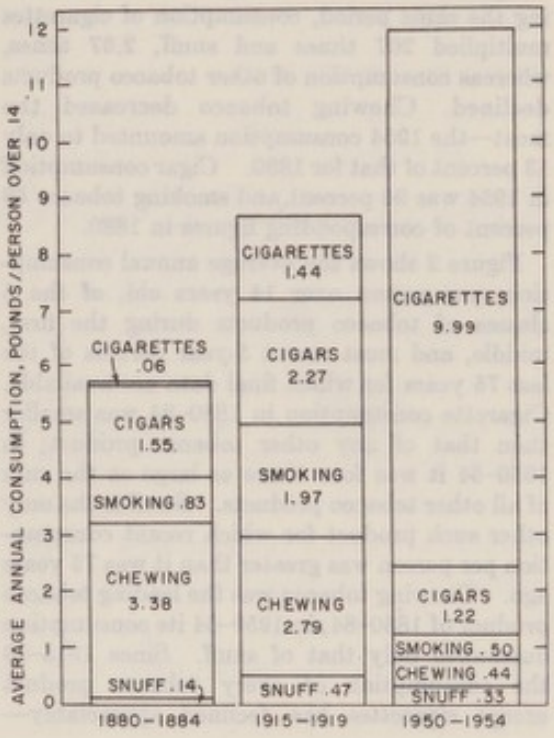


Table 3. Consumption of each tobacco product expressed as a percentage of total consumption of tobacco, unstemmed-processing weight, averages 1880–1954 [1]

Year	Cigarettes	Cigars	Smoking tobacco	Chewing tobacco	Snuff
	Percent	*Percent*	*Percent*	*Percent*	*Percent*
1880–84	1	26	14	56	2
1885–89	2	25	15	56	3
1890–94	3	24	16	53	3
1895–99	3	24	18	52	4
1900–04	2	27	21	45	4
1905–09	3	27	25	41	5
1910–14	7	26	25	37	5
1915–19	16	25	22	31	5
1920–24	26	25	18	25	5
1925–29	38	21	16	20	5
1930–34	44	17	20	14	5
1935–39	53	15	17	10	4
1940–44	65	13	11	7	4
1945–49	76	11	5	5	3
1950–54	80	10	4	4	3

[1] Because of rounding off, percentages do not always add up to 100.

tobacco products combined has been considerably more stable than that of some of the individual tobacco products. In 1954, total tobacco consumption per person over 14 years of age was 2.24 times as large as in 1880. During the same period, consumption of cigarettes multiplied 207 times and snuff, 2.67 times, whereas consumption of other tobacco products declined. Chewing tobacco decreased the most—the 1954 consumption amounted to only 13 percent of that for 1880. Cigar consumption in 1954 was 90 percent and smoking tobacco 59 percent of corresponding figures in 1880.

Figure 2 shows the average annual consumption, per person over 14 years old, of the 5 classes of tobacco products during the first, middle, and most recent 5-year periods of the last 75 years for which final data are available. Cigarette consumption in 1880–84 was smaller than that of any other tobacco product; in 1950–54 it was four times as large as the sum of all other tobacco products. Snuff is the only other such product for which recent consumption per person was greater than it was 75 years ago. Chewing tobacco was the leading tobacco product of 1880–84; in 1950–54 its consumption outranked only that of snuff. Since 1915–19 the consumption of every tobacco product except cigarettes has declined appreciably—from an aggregate of 7.50 to 2.49 pounds per person.

The consumption of each tobacco product, expressed as a percentage of total tobacco consumption, is shown in table 3. During the years 1880–84, only 1 percent of this total consumption was in the form of cigarettes; in 1915–1919, 16 percent; and in 1950–1954, 80 percent. In contrast, the proportions of chewing tobacco declined from 56 percent in the earliest period to 31 percent and 4 percent in the two later periods.

In comparison with cigarettes and chewing tobacco, changes in the proportions of other tobacco products were moderate. The percentage of tobacco used as snuff ranged between 2 and 5, and for cigars was fairly stable until 1920–24; since then it has declined from 25 to 10 percent. The percentage consumed as smoking tobacco for pipes and roll-your-own cigarettes gradually increased from 14 in 1880–84 to 25 in 1910–14. Its later decline to 4 percent in 1950–54 was interrupted during the depression years 1930–34.

Cigarettes Lead Increases

The consumption of cigarettes per person over 14 years of age was 8.55 pounds greater in 1950–54 than in 1915–19. During the same interval, consumption of all other tobacco products declined 5.01 pounds. Thus, 3.54 pounds, or 41 percent of the increase in cigarette consumption since World War I, may be considered as additional tobacco consumption. The other 59 percent of the increase may be

Table 4. Consumption of cigarettes per person over 14 years of age, United States, 1950–55

Year	Cigarettes	
	Quantity	Weight [1]
	Number	*Pounds*
1950	3, 320	9. 36
1951	3, 527	9. 98
1952	3, 661	10. 41
1953	3, 558	10. 46
1954	3, 339	9. 73
1955 [2]	3, 386	9. 56

[1] Unstemmed-processing weight.
[2] Provisional.

regarded as a shift from other tobacco products to cigarettes.

Table 4 shows that the number of cigarettes consumed per person over 14 years old reached a peak in 1952. But the greatest cigarette consumption in terms of weight of tobacco occurred in 1953. This difference reflects changes in the number of cigarettes produced per pound of tobacco (table 2).

Summary

Estimates of the consumption of each of the five principal classes of tobacco products in the United States are here given for the years 1880 to 1955. The annual total tobacco consumption per person over 14 years of age increased from 5.41 to 11.90 pounds. During the same period cigarette consumption increased from 0.047 to 9.56 pounds per person. Except for snuff, consumption of other tobacco products declined. Chewing tobacco was the leading tobacco product in 1880; in 1955 its consumption outranked only that of snuff.

In 1952, 3,661 cigarettes were used per person over 14 years of age—the largest number on record. In terms of quantity of tobacco consumed as cigarettes, the peak year was 1953; in that year 10.46 pounds of tobacco per person over 14 years old were so used. During the years 1880–84, only 1 percent of total tobacco consumption was in the form of cigarettes; from 1950–54, 80 percent. Since World War I, the annual consumption of cigarettes per person over 14 years old has increased by more than 8 pounds. About 41 percent of this increase may be regarded as additional tobacco consumption, the remaining 59 percent as a shift from other tobacco products to cigarettes.

Reference

(*1*) U. S. Agricultural Marketing Service: The tobacco situation. TS–77, table 3. Washington, D. C., U. S. Department of Agriculture, 1956.

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EXHIBIT 8A

[For release in p. m. newspapers, Friday, July 12, 1957]

UNITED STATES DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

PUBLIC HEALTH SERVICE

Washington 25, D. C.

The Public Health Service said today there is increasing evidence that excessive cigarette smoking is one of the factors which can cause lung cancer.

The statement by Surgeon General Leroy E. Burney modified somewhat the previous official position taken by the Public Health Service in 1954 on the question of cigarette smoking and lung cancer.

At that time, the Service noted there was some evidence of a statistical association between cigarette smoking and lung cancer but left open the question of whether there was a cause and effect relationship.

In today's statement, Dr. Burney said many independent studies have confirmed beyond reasonable doubt that there is a high degree of statistical association between lung cancer and heavy and prolonged cigarette smoking.

Although this evidence is still largely statistical in nature, Dr. Burney said, some laboratory studies also support the concept of a cause and effect relationship.

Dr. Burney noted that many important public health advances have been developed on the basis of statistical or epidemiological information.

Copies of the Surgeon General's statement, together with supplementary data, will be made available to State health officers and others in the health professions as a further step in bringing the matter of smoking and lung cancer to public attention.

Dr. Burney said it is clear that cigarette smoking is not the only causative factor in lung cancer. Further, he said, the precise factors in cigarette smoking which can cause lung cancer are not known.

He said more research is needed to identify and try to eliminate these factors. He also called for more research into other probable causes of lung cancer, including air pollution.

The United States now has more than 25,000 deaths each year from lung cancer. How many of these persons were heavy cigarette smokers is not known. Altogether, deaths from cancer make up about 250,000 of the 1.6 million total deaths in the United States each year.

The Public Health Service issued the statement after completing its review of the report of a study group on smoking and health. The study group was established 1 year ago to appraise available scientific evidence and recommend further needed research.

Dr. F. M. Strong, of the University of Wisconsin Medical School, was chairman of the seven-man study group, which was organized jointly by the Public Health Service's National Cancer Institute and National Heart Institute, and the voluntary private organizations, the American Cancer Society and the American Heart Association.

The report of the study group was made public in New York on March 23, and was published in full in the June 7 issue of Science.

Dr. Burney said the Public Health Service, in making its first formal public statement on smoking and health since 1954, also took into account other recent data, including the report last month by Dr. E. C. Hammond and Dr. Daniel Horn of the American Cancer Society to the American Medical Association in New York.

Although there are naturally some differences of opinion in interpreting the data on lung cancer and cigarette smoking, Dr. Burney said, the Public Health Service feels the weight of the evidence is increasingly pointing in one direction: that there is an association between excessive smoking and lung cancer.

Dr. Burney said the Public Health Service statement has been discussed with the executive committee of the State and Territorial Health Officers' Association.

The text of today's Public Health Service statement follows:

"STATEMENT BY SURG. GEN. LEROY E. BURNEY OF THE PUBLIC HEALTH SERVICE, DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, ON EXCESSIVE CIGARETTE SMOKING AND HEALTH

"The Public Health Service is, of course, concerned with broad factors which substantially affect the health of the American people. The Service also has a

responsibility to bring health facts to the attention of the health professions and the public.

"In June 1956, units of the Public Health Service joined with two private voluntary health organizations to establish a scientific study group to appraise the available data on smoking and health. We have now reviewed the report of this study group and other recent data, including the report of Dr. E. C. Hammond and Dr. Daniel Horn on June 5, to the American Medical Association in New York.

"In the light of these studies, it is clear that there is an increasing and consistent body of evidence that excessive cigarette smoking is one of the causative factors in lung cancer.

"The study group, appraising 18 independent studies, reported that lung cancer occurs much more frequently among cigarette smokers than among nonsmokers, and there is a direct relationship between the incidence of lung cancer and the amount smoked. This finding was reinforced by the more recent report to the AMA by Drs. Hammond and Horn.

"Many independent studies thus have confirmed beyond reasonable doubt that there is a high degree of statistical association between lung cancer and heavy and prolonged cigarette smoking.

"Such evidence, of course, is largely epidemiological in nature. It should be noted, however, that many important public health advances in the past have been developed upon the basis of statistical or epidemiological information. The study group also reported that in laboratory studies on animals at least five independent investigators have produced malignancies by tobacco smoke condensates. It also reported that biological changes similar to those which take place in the genesis of cancer have been observed in the lungs of heavy smokers. Thus, some laboratory and biological data provide contributory evidence to support the concept that excessive smoking is one of the causative factors in the increasing incidence of lung cancer.

"At the same time, it is clear that heavy and prolonged cigarette smoking is not the only cause of lung cancer. Lung cancer occurs among nonsmokers, and the incidence of lung cancer among various population groups does not always coincide with the amount of cigarette smoking.

"The precise nature of the factors in heavy and prolonged cigarette smoking which can cause lung cancer is not known. The Public Health Service supports the recommendation of the study group that more research is needed to identify, isolate and try to eliminate the factors in excessive cigarette smoking which can cause cancer.

"The Service also supports the recommendation that more research is needed into the role of air pollution and other factors which may also be causes of lung cancer in man.

"To help disseminate the facts, the Public Health Service is sending copies of this statement, the study group report and the report of Drs. Hammond and Horn to State health officers and to the American Medical Association with the request that they consider distributing copies to local health officers, medical societies and other health groups.

"While there are naturally differences of opinion in interpreting the data on lung cancer and cigarette smoking, the Public Health Service feels the weight of the evidence is increasingly pointing in one direction: that excessive smoking is one of the causative factors in lung cancer.

"The Service notes that the study group found that more study is needed to determine the meaning and significance of any statistical association between smoking and heart disease. The study group reported there is no convincing biological or clinical evidence to date to indicate that smoking per se is one of the causative factors in heart disease. Although the report by Drs. Hammond and Horn has since provided additional data on this subject, the Service feels that more statistical and biological data is needed to establish a definite position on this matter."

EXHIBIT 8B

[Immediate release, Friday, July 12, 1957]

From: Hill and Knowlton, Inc., 1000 Vermont Ave. NW., Washington, D. C. Sterling 3–0270.
For: Tobacco Industry Research Committee.

HARTNETT CITES SCIENTISTS DOUBTING SMOKING-CANCER THEORY

Commenting on a release by the Surgeon General in Washington today, Timothy V. Hartnett, chairman of the Tobacco Industry Research Committee, said:

The Surgeon General, in his statement today, places full reliance on the recent study group report which failed to acknowledge the considerable work in the field of lung cancer which does not coincide with the study group position.

Actually, in recent years many doctors and scientists have publicly expressed their doubts or disbelief in the theory that smoking causes lung cancer. Their statements, usually made in medical and scientific journals, were nonsensational in nature and therefore have been generally overlooked by much of the press and public.

The study group report and this latest statement based on it failed to give recognition to the views of the many scientists working on the lung-cancer problem.

For example, Dr. Joseph Berkson, head of the section of biometry and medical statistics of the Mayo Clinic, was quoted April 1, 1957, by a national magazine as saying in reference to the study group report, "it is my personal opinion, and I know as much about it as anyone else, that smoking does not cause cancer of the lung."

On July 27, 1955, Dr. Berkson published a paper in which, according to an official Mayo Clinic summary he questioned "the validity of the statistics of the report of the American Cancer Society claiming to prove an association between cigarette smoking and lung cancer."

Dr. Harry S. N. Greene, chairman of the department of pathology, Yale University School of Medicine, has challenged the theory that smoking causes lung cancer in his introduction to a book just published, Science Looks at Smoking.

Dr. Greene said he doubted that available statistical and experimental evidence can "be interpreted as conveying a suggestion of a causal relationship between tobacco smoking and lung cancer."

"The case against tobacco is derived mostly from statistical associations and some experimental work with animals," Dr. Greene wrote.

"The mere fact of a coincident increase or decrease in the frequency of given happenings does not necessarily mean that one is causally related to the other."

Dr. Greene said that he and other investigators had conducted laboratory experiments into smoking and lung cancer and found no causal relationship. He said he himself experimented with tobacco tar and embryonic human tissue without getting any cancers.

In April 1957, Dr. R. H. Rigdon, a noted cancer researcher and director of the laboratory of experimental pathology, University of Texas medical branch, Galveston, wrote an article in the Southern Medical Journal that concluded with this section:

"In summary, it may be said that cancer of the lung has been recognized for approximately 150 years. During the past 100 years an almost continuous controversy has been carried on—is the increase in the frequency of lung cancer only apparent, due to better diagnoses, increase in the age of the population, etc., or is the disease actually progressively increasing, due to some specific etiologic agent or agents?

"Vital statistics are too crude to establish accurately the frequency of cancer of the lung. It is my opinion, based on the available data, that cancer of the lung has not actually increased in proportion to all other neoplasms.

"Tobacco products, especially cigarettes, have been suggested frequently since the beginning of the 20th century as an etiologic agent in cancer of the lungs. The present controversy is only a flareup of this problem.

"A demonstration of a carcinogen in cigarette tars for the skin of a mouse and a rabbit cannot be accepted scientifically as a carcinogen for the lung of man; such has been known for 25 years or longer. The frequency of the habit of smoking by peoples throughout the world during the past several hundred years and the relative infrequency of cancer of the lung should make us critical of the observations suggesting an association between cigarette smoking and cancer of the lung as cause and effect. Diseases other than lung cancer occur in individuals who are heavy smokers without anyone suggesting cause and effect. Furthermore, many individuals have died with cancer of the lung that did not smoke. Scientific studies on this problem should be encouraged because facts may be discovered about the pathogenesis of cancer which may serve as the basis for treatment."

In their 1956 book, Cancer of the Lung, Drs. Milton Rosenblatt and James Lisa, said, "the reports on lung cancer and smoking are concerned with only two variables, whereas a great many are undoubtedly involved. As statistical correlation does not imply a cause and effect relationship * * *."

They also said that the relationship between the increased incidence of lung cancer and the rise in cigarette consumption "is purely speculative. Both in this country and in England the death rate from lung cancer has increased at a far greater pace than has the consumption of tobacco. Analysis of international reports reveals little correlation between the per capita consumption of cigarettes and the death rate from lung cancer. The incidence of lung cancer appears to be related far more to the diagnostic facilities available * * *"

Edward A. Lew, actuary and statistician of the Metropolitan Life Insurance Co., wrote in 1954 that approximately half the increase in respiratory cancer deaths from 1930 to 1953 "reflects merely the growth and aging of the population, and a considerable part of the remainder represents improved diagnosis and more complete case finding. Nevertheless, there does appear to be an appreciable real rise in the incidence of respiratory cancer, but data are not available to show how much of it can reasonably be attributed to the effect of specific factors."

Today, Dr. Clarence Cook Little, chairman of the scientific advisory board to the Tobacco Industry Research Committee, reiterated the board's position on this important question, stating:

"The statement issued today by the Surgeon General adds nothing new to what has been known about the case of lung cancer. It reflects the opinions of some statisticians and the relatively few experimental scientists which have actively charged that cigarette smoking is a cause of lung cancer.

"No new evidence has been produced since the scientific advisory board to the Tobacco Industry Research Committee last stated its position on this question on May 1, 1957. At that time, I said that although anyone has the right to state an opinion on cancer causation, 'the scientific advisory board questions the existence of sufficient definitive evidence to establish a simple cause and effect explanation of the complex problem of lung cancer.'

"This is most definitely our position today."

EXHIBIT 9

INTERVIEW **with Dr. John R. Heller, Director, National Cancer Institute, U. S. Public Health Service**

THE TRUTH ABOUT SMOKING AND CANCER

WHAT IS KNOWN AND UNKNOWN

Millions of Americans continue to smoke—even though many medical authorities agree that a heavy smoker of cigarettes stands 1 chance in 10 of dying of lung cancer.

What are the facts about the relationship between smoking and lung cancer? Are cigarettes more suspect than cigars and pipes?

Do doctors recommend giving up smoking? What about smoking in moderation?

To get the truth about these and other questions on this subject, "U. S. News & World Report" interviewed in its conference room a leading health authority.

Dr. John R. Heller, director of the National Cancer Institute, of the U. S. Public Health Service, tells here what physicians in this and other countries have learned about smoking and the perils of lung cancer.

Q Dr. Heller, is cancer among smokers a serious problem?

A Well, let's put it this way: The problem first came to the attention of the medical profession when it was found that we had an increasing number of people who were dying of lung cancer. It was found that many of those dying from lung cancer were also heavy and prolonged smokers—cigarette smokers.

We don't know why people get lung cancer. We do know that men get lung cancer to a much greater extent than women.

Q If smoking is the cause, why doesn't it show up in women?

A It is showing up in women. There are several reasons, perhaps, why it is more prevalent in men. There is a sex difference, certainly. Men have a greater risk of lung cancer than do women, whether they smoke or not. That's first. Probably more men than women smoke, although we're speaking of proportional figures here. Men have been smoking longer than women, as a rule—for greater lengths of time. Therefore, our data are more complete on men.

However, data which have been reported recently indicate that, as one adjusts the length of time of smoking, of ages and what not, the death rate from lung cancer in women is beginning to come closer to that of men.

Q Does every heavy smoker stand a chance of getting lung cancer?

A He stands a chance.

Q Is that chance very slight?

A It's reported from the data in this country that, if one does not smoke, his chances are 1 in 275 of acquiring lung cancer. If one is a heavy cigarette smoker—two packages of cigarettes or more a day—his chances of dying from lung cancer are 1 in 10.

Q How many cases of lung cancer are there in this country in a year?

A Of reported lung cancer, there are about 25,000 deaths a year.

Q Out of how many deaths from all causes?

A The figure is about 1.6 million, I believe. Lung cancer is not a great factor but, when added up, year after year, the chances of one dying from this—if he is a man, if he is above 45, if he is a heavy smoker—his chances of dying from that particular condition are almost as good as his chances of being hit by an automobile.

Q This person you just described is 1 in 10 of these?

A His chances of acquiring lung cancer are 1 in 10.

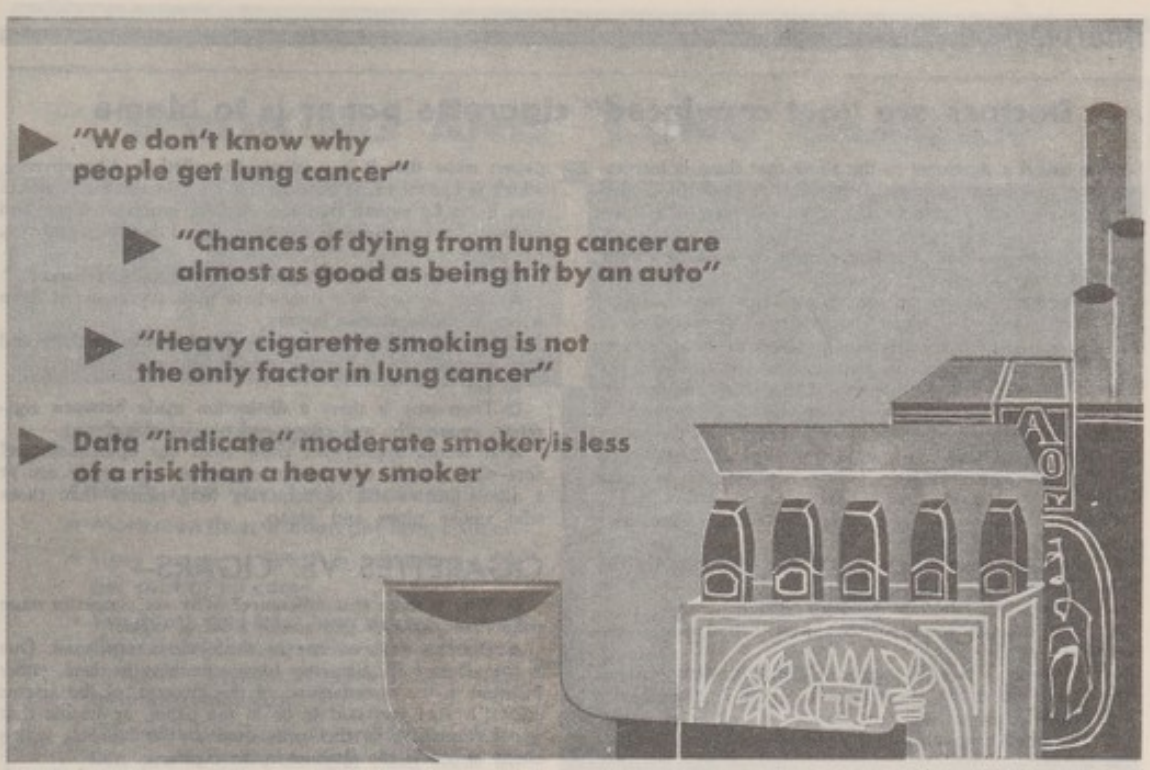
Q Is lung cancer always fatal?

A Almost invariably fatal, unless found very early. The rate of survivors from lung cancer is less than 5 per cent. In other words, of all those who were diagnosed and sur-

John Roderick Heller is recognized as one of the foremost authorities in the U. S. on the subject of cancer. At 52, Dr. Heller has been director of the National Cancer Institute, financed by the Government, for nine years. He directs research into causes of cancer and methods of controlling it.

Dr. Heller was educated as a medical scientist at Clemson College and Emory University School of Medicine, receiving his M.D. degree in 1929. He is a member of the scientific advisory board of Sloan-Kettering Institute for Cancer Research.

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gery undertaken, less than 5 per cent survived for a period of 5 years.

Q If only 25,000 people die yearly of lung cancer, how can one's chances be 1 in 10 if he is a heavy smoker? Aren't there enough heavy smokers so that 1 in 10 would produce far more than 25,000?

A His chances of dying of lung cancer from the time he starts smoking until he dies are 1 in 10.

Q How does that compare with deaths from all cancer?

A There are about 250,000 deaths a year in this country from all cancer. It's about one tenth of the cancer deaths.

Q So you could escape lung cancer and still have nine other chances of dying from some other form of cancer?

A Cancers cause 13 per cent of all the deaths in this country.



Q Then lung cancer causes about 1 per cent?

A Yes, slightly more than 1 per cent of all the deaths.

Q Is the proportion of deaths caused by all types of cancer increasing and the proportion of deaths caused by lung cancer increasing?

A Yes. In other words, as we are surviving typhoid and gastroenteritis, malaria and so forth, we are living longer. We must die of something. We're most likely to die of heart disease—cardiovascular disease. But if we don't die of that, the next chance is cancer, and the next, I believe, is accidents.

The chances of any particular individual dying of any given disease—unless he has been exposed to an infectious disease or something on that order—can be mathematically computed, and it doesn't mean that he needs to worry about it particularly. A person who is a heavy smoker looks at these figures and says, "Well, I enjoy smoking. My chances of dying of lung cancer are pretty remote. I'm not going to worry about it." And maybe he goes on and lives to the age of 85 or 90 and dies of heart disease.

Q What would be the advice of the Public Health Service?

A The job of the Public Health Service is to present the facts, its best judgment or interpretation of the facts, to the health professions and the public generally. We don't, of course, generally act as a physician in giving medical care or advice to individual patients.

Q Have you noticed that the British Government has issued a warning about the dangers of lung cancer from cigarettes?

A Yes, we have read that in the newspapers.

Q What is the U. S. Government's position on that?

A You will recall that on July 12 the Public Health

(Continued on next page)

Interview

. . . Doctors are "not convinced" cigarette paper is to blame

Service issued a statement to the effect that there is increasing and consistent evidence that heavy and prolonged cigarette smoking is one of the causative factors in lung cancer.

Q Why do you put your finger right on smoking as the cause of lung cancer?

A I wouldn't say we put our finger "right on smoking." We simply say the signs are pointing increasingly to smoking as one of the factors involved in lung cancer. It's one of the common denominators we find around the world. It's been the British experience, Scandinavian experience, Austrian experience—the experience of anybody, I think, whose evidence we can rely upon. On the other hand, we know that heavy cigarette smoking certainly is not the only factor in lung cancer. Nonsmokers get lung cancer, too.

Q Were tests carried out in all those other countries?

A Yes.

Q What you are saying now is a new viewpoint for the Public Health Service, isn't it?

A Well, it brings our position up to date.

Q Is it because of some recent evaluation you've received?

A We believe that the increasing and consistent material which has come to our attention warrants this viewpoint.

Q Is that based on your own studies, as well as on others?

A The studies throughout the world that we have access to.

Q How recent is this conclusion? Six months ago would you have made the same statement?

A Well, we didn't. It has been arrived at gradually.

Q How does the incidence of lung cancer in the United States compare with the incidence in some of these other countries that made studies?

A The rate has been increasing in this country. It is not as great as the incidence of lung cancer in Great Britain, in Austria, or in Scandinavia.

Q Do those people smoke more than we do?

A They do in Austria, I'm told. In Great Britain it's very difficult because their smoking habits are slightly different. Their total cigarette consumption is less than ours, but they smoke a cigarette right down to the bare nub in Britain. They smoke different types of cigarettes in other countries.

Q But there is some common factor in there?

A There's some common factor, apparently.

ROLE OF TARS IN SMOKE—

Q Haven't there been some reports that substances in the tobacco smoke had been isolated which are known to be cancer-causing chemicals?

A Oh, yes, some tars.

Q Specific things in the tars themselves?

A Yes, there have been some investigators who contend that it is substances called aromatic amines but, as far as my knowledge goes, the exact chemical structure or the exact material at which one can point a finger with suspicion and say it is this and nothing else has not been identified.

Q Has the paper been suspect?

A Yes, the cigarette papers have been suspect. Many investigators have contended that it is something in the burned paper. Others contend that it is arsenic impregnated in the paper; some that it is a substance called 3, 4-benzpyrene, which is known to be cancerigenic to animals. But I would sum it up by saying that the medical group involved and interested in this problem is not convinced that the agent is in the paper.

Q But they do feel that there is a criminal in tobacco?

A They do feel that somewhere in heavy smoking there is one of the causative factors.

Q They feel there is a relationship between smoking and lung cancer?

A Yes.

Q Then why is there a distinction made between cigarettes, apparently, and cigars and pipe smoking?

A It would appear—we know from our epidemiological data—that people who smoke cigarettes excessively are at a much greater risk of acquiring lung cancer than those who smoke pipes and cigars.

CIGARETTES VS. CIGARS—

Q Why is there that difference? Why are cigarettes more risky than cigars—if you smoke a lot of cigars?

A There have been two or three views expressed. One is that, first of all, cigarettes have something in them, either inherent in the manufacture, of the glycerin, of the arsenic originally that was said to be in the paper, or arsenic that was incorporated in the spray used on the tobacco, which ought to obtain the same as in the cigars.

There are those who believe there are chemical changes resulting from the higher burning point in cigarettes than in a cigar. For example, it has been stated that the critical temperature is about 800 degrees Fahrenheit at the end of a cigarette, and in anything less than that probably one has much less risk of acquiring cancer.

It may be a function of the packing of the tobacco in the cigarettes as contrasted with cigars and pipes. It may be the curing of the tobacco—the incorporation of certain chemicals involved in the processing, perhaps.

Q Could it be that cigarette smokers inhale, whereas many pipe and cigar smokers do not inhale?

A That has been advanced as certainly a cause of difference. It sounds very logical to me.

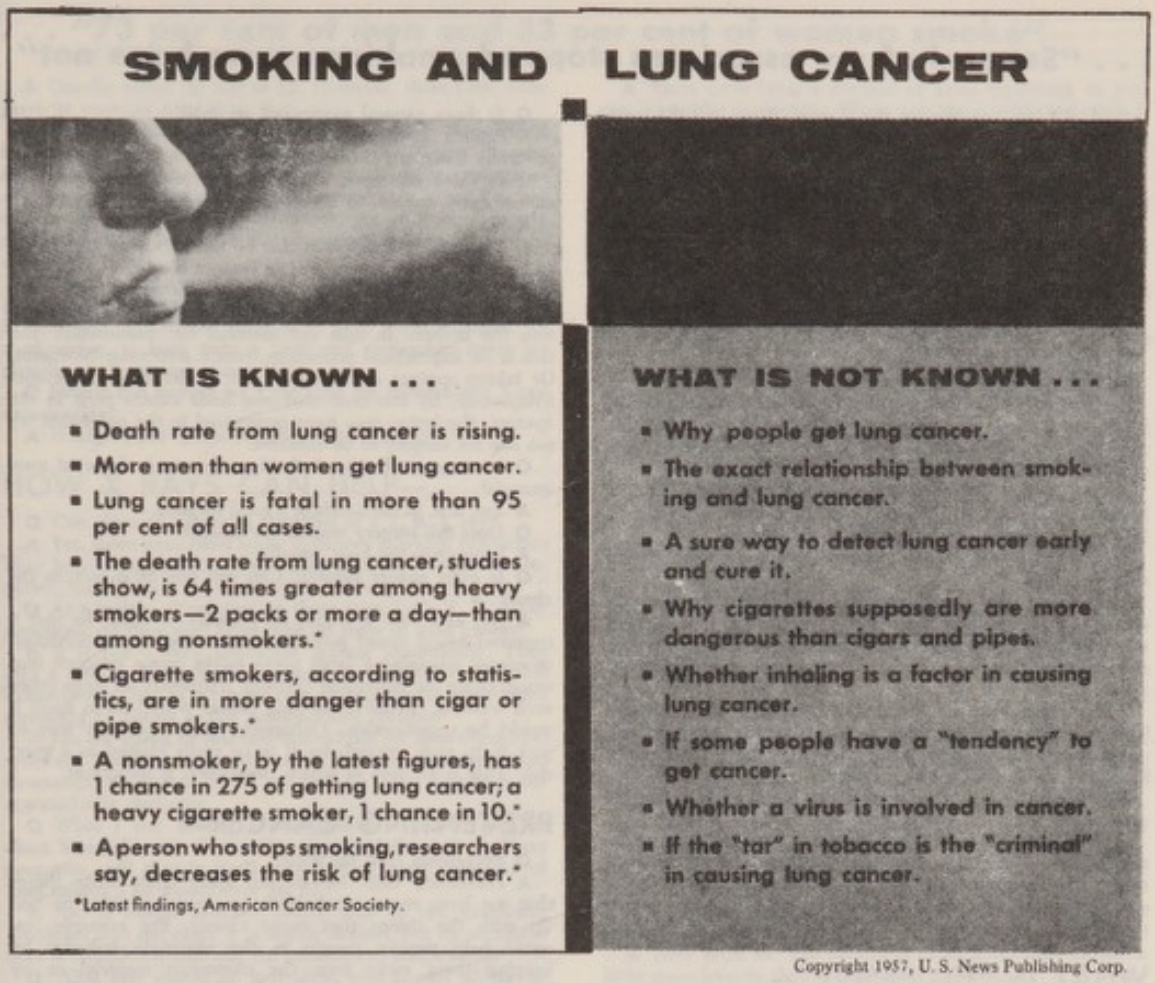
Q But then, at the same time, cancer of the mouth and of the throat is higher in cigarette smokers. Cigar smokers, I should think, would have the same danger, would they not?

A One might think so.

Q Doctor, in this relationship between cigarettes and lung cancer, is there any difference between filter cigarettes and nonfilter cigarettes?

A I don't know whether you have kept up with the current discussion concerning the contents of the several kinds of cigarettes—king size and regular size, tar content and nicotine content, etc. It seems to be factually correct that the presence of filters cuts down only slightly the amount of tars in cigarettes.

Originally, as I understand it, the filter really filtered out a lot of the tars, but it filtered out the taste of the cigarette as well. Then the manufacturers, attempting to market their product, very understandably wanted to retain the best features of their filter, I suppose, and at the same time allow taste, too, and presumably loosened the cellulose acetate of which the filter is composed, or otherwise made it less effective. Or



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maybe they have changed the type of tobacco they use so that more taste comes through.

Anything that will cut down the amount of smoke that comes in will automatically cut down the amount of tar. Filters help to some extent, but not nearly as much as some people might think.

Q Can a filter be designed that would help?

A I think it can, and the consensus among those who are in a position to know, with whom I have talked, believe that it is possible to design a filter that will cut down the tar appreciably.

Q Would that take out the taste as well?

A So far, the experience has been that it probably would take out the taste, but there are those who feel that it is possible, with the ingenuity that can be brought to bear, to retain a good bit of the taste and, at the same time, effectively filter out the smoke and, therefore, the tars.

Q What about the filters that are made to put in cigarette holders? Are they more effective than those built into the cigarettes themselves?

A I really don't know. Again, when we go back to the fundamental concept that if this filter—or such as you mentioned, the kind that is not built in—would diminish the amount of smoke, or strain out the amount of smoke that enters the lungs, certainly one could see that it would be a useful and effective tool in preventing the amount of tars that enter.

Q Is nicotine itself dangerous?

A As far as the production of lung cancer is concerned, it doesn't seem to have any bearing.

Q Is nicotine the thing that a person seeks in a cigarette—the effects of nicotine?

A No, apparently not. Apparently it's the tars that give him that sense of satisfaction. I'm using tars in the broad sense. They seem to be the important constituent in the satisfaction to the user.

Q How is that determined? How can you tell such a thing?

A For one thing, when you use filters, when you remove the tar, the cigarette is said to be tasteless. People simply will not stand for it. It doesn't give them satisfaction.

(Continued on next page)

Interview

. . . "Several of my associates stopped smoking; some have not"

Q And when you remove the nicotine there isn't the same reaction?

A Not the same reaction.

Q Does nicotine stunt your growth the way we used to hear?

A I've always heard that, but I don't know any reason why it would stunt one's growth.

Q If you had a perfect filter you really wouldn't be smoking, would you?

A Taken to its ultimate conclusion, that's correct. With a perfect filter no smoke would get through—just hot air.

Q Doctor, are you a smoker?

A I am not. I have never smoked.

Q How about some of the people you are associated with, people who have the same knowledge of these matters as you? Do you notice that a good many of those people have stopped smoking?

A Yes. Several of my associates in the National Cancer Institute in the study of this problem stopped smoking as long as 2 to 2½ years ago. Some have not stopped.

Q Would you recommend, Doctor, that any smoker should stop smoking?

A I would say this: I believe the weight of the evidence, from the data we have at hand, is that a man who is a heavy and prolonged smoker is at a much greater risk of acquiring lung cancer than a man who does not smoke.

Nobody has ever contended that smoking is good for one from a health point of view. But, if a person gets pleasure out of it and desires to smoke, knowing the facts, then, in the final analysis that's his determination.

Q Would you say, "Smoke what you like, but do it in moderation"?

A If a patient said to me, "Doctor, I just can't stop smoking," I'd say, "Well, the data that we have at hand indicate that a moderate smoker is less of a risk than a heavy smoker, and that a man who smokes a pipe or cigar is less of a risk than a cigarette smoker."

WHAT IS "EXCESSIVE"—

Q What do you regard as excessive?

A Two packs a day or more.

Q What do you regard as moderate?

A Certainly less than a pack a day.

Q What if you smoke moderately?

A You have less risk of acquiring lung cancer.

Q Is there any risk?

A There is still a risk. One may acquire lung cancer even though one doesn't smoke, I hasten to add. However, the risk of acquiring lung cancer begins to climb appreciably as one smokes more—more cigarettes, particularly—and to some extent with pipes and cigars, but much, much less.

Q But your advice would be moderation, certainly?

A That would be my personal advice as a physician.

Q Or would you advise complete abstinence?

A If an individual is up to it, I think as a physician I would say to him if he was sufficiently afraid—if he was in the age range and was sufficiently worried about lung cancer—I'd say, "If you can quit, quit."

Q Would you say, "Switch to a pipe"?

A Switch to a pipe or a cigar.

Q Is there general agreement on that?

A Among many students of the problem, yes, although naturally there are also some differences of opinion.

Q Is there any sure way that a person can detect lung cancer early enough to be almost sure of curing it?

A No.

Q You can't tell a person that he can go ahead and smoke and rely on a certain regimen to protect him—

A Not to my knowledge. The only dependence we've got is X ray and, of course, the introduction of a bronchoscope into the bronchi to take out material for examination. But this is an impractical procedure from a screening viewpoint. Or taking sputum and examining it under the microscope. Frequently, by the time that one finds cancer cells in the sputum, the lesion may have progressed to the point that it's too late for surgery to be effective.

Q Is surgery effective if the lung cancer is detected soon enough?

A Yes, it is, in the hands of a good surgeon.

Q Does this surgery remove the cancer?

A It removes the lung, usually.

Q Is early cancer painful? Can you rely on a pain in the chest?

A No, as a rule it would be painless. It could be that a ruptured blood vessel possibly would result in hemorrhage, or cancer adjoining a large nerve might create pressure that would cause the individual to cough frequently, or some other such warning. Usually, the lung cancer would be so small it would be unnoticeable. Unhappily, there's no easy way to pick it up early enough for it to be really classed as a good risk. This is an area in which prevention is important.

PREVENTING CANCER—

Q Prevention calls for what?

A Prevention calls for staying away from those things that we have reason to believe are cancer-causing or tied up with the things that cause cancer. For example, we could help protect people in the chromate industry by keeping them away from the chromium material or by having them wear masks. People who are out in the sun a great deal—and therefore much more likely to get skin cancer—should cover their heads, bodies; or, if it's arsenic with which they come in contact, we should protect them by suitable clothing or keep arsenic out of the industrial process—or whatever it may be. Those are preventive measures.

In this instance there seems to be reason to believe that cigarette smoking is increasingly important in the scheme of things. Then, to prevent lung cancer, you simply say, "O. K., let's tell the people what we know or believe so they can stop smoking excessively if they want to in the light of the facts." Or you can take out the thing in the cigarette that's causing it, or whatever preventive measures might be appropriate.

Q They have no idea yet just what they could take out—what the real cause is, have they?

A Some investigators believe it's the tars in the smoke. We refer to "tars" simply as the inconclusive term for all the "gunk" that's in cigarette smoke. They may be polycyclic hydrocarbons, chemically—a host of different chemicals.

Q At what age does lung cancer occur?

. . . "73 per cent of men and 33 per cent of women smoke"

A Usually above the age of 50. However, there have been cases at younger ages than that.

Q One thing that the ordinary person begins to think about in connection with what you have said is that if you discover it early you might be able to stop it. Are there any recognizable symptoms? Is there any advice you can give? For example: "If you get a persistent cough, get an X ray," or any other advice you can give to the average person on how to detect this thing?

A The best advice that can be given to anybody—about lung cancer, or anything else for that matter, but particularly in detecting lung cancer early—is to have an examination by a physician, at least once a year and preferably every six months when you are above 45.

Q Should this include an X ray, like the tuberculosis X-ray examination?

A It might, but that would be up to the physician.

HOW X RAYS CAN HELP—

Q Can signs of cancer be detected in a T. B. X ray?

A Yes, there have been many instances in which physicians have been searching for tuberculosis and have found the so-called "coin" lesion or other lesions of lung cancer.

Q As a matter of practice, in these large-scale T. B. X-ray programs, do they very often pick up signs of lung cancer?

A It is not a very important finding numerically. They find a few in these huge programs of 200,000 or 300,000 examinations, but as a cancer case-finding device the routine screening of chests is not economically feasible.

If you are going to examine individuals for something else, well and good. But to set out to examine thousands and thousands of persons by X ray just for lung cancer is not economically desirable.

Q What I was getting at was this: If a person gets his chest X-rayed every six months on one of these T. B. X-ray programs, will that in itself take care of early detection of lung cancer if he should ever get it?

A No, it would indicate pretty well to the examining physician that the chest is probably normal, but by no means does it completely rule out the presence of lung cancer.

Q What else should an individual who is past 45 do for his own protection against lung cancer?

A He should report to his physician. The physician probably will ask him questions which would be related to the presence of cough, weakness, a lot of other signs and symptoms which are of significance to the physician in the screening of a possible chest condition, whether it be cancer, tuberculosis, or what-have-you.

Q And what about all those roentgens from X ray? Don't they give you cancer?

A With these small machines—the little 70-millimeter or the 35-millimeter photofluorographic examination—you probably wouldn't get over a tenth of a roentgen at a time—not enough to make one uneasy about it becoming a cause of cancer.

Q Is that the sort of equipment that is in most of these T. B. X-ray trailers?

A Yes.

Q Have you given any consideration to writing off this generation of people who are now smoking, and directing advice against excessive smoking to the younger generation and people who have not yet started smoking?

A There have been a number of ideas expressed, as you can probably appreciate. There are those who feel that a proper course is, first of all, to put the facts from an authoritative source before the public, to advise moderation for those who are now smoking. If they wish to continue to smoke excessively, that's their business. Advise moderation, but advise young people of the risk and suggest that they do not start smoking.

Q Smoking cigarettes, or any thing?

A Smoking any way, but particularly cigarettes.

Q Is there any reason to believe that a heavy smoker improves his own chances by stopping smoking?

A Yes, there are data to indicate that one who stops smoking as long as a year, for example, improves his chances. Let me put it this way: He decreases the risk of acquiring lung cancer if he stops smoking.

Q Take a man who has been a heavy smoker for 25 years. If he stops smoking, wouldn't he be beyond redemption by that time?

A I'm not sure at what age the damage is irrevocable. The data that Dr. E. Cuyler Hammond of the American Cancer Society has produced would suggest that, if an individual stops smoking, his longevity increases generally; he's not so likely to die regardless of how long he has been smoking. There seems to be a correlation between excessive smoking and earlier death. The death rate in individuals who are heavy smokers is excessive in comparison with nonsmokers. Now, I personally would like to see that worked out much more precisely. I don't know what it means quite yet in measurable terms.

It may well be that the sort of person who is impelled to smoke heavily is the sort who has the body build or the other characteristics that lead him to take unnecessary risks. Maybe he is the sort who is geared up at a fast rate and is much more likely to have coronary heart disease, or maybe he is much more likely to acquire diabetes—he may be a heavy eater. All of these things have to be woven into the blanket we're talking about.

Q Do people who live in cities get lung cancer more often than people who don't?

A They seem to. The people in cities apparently smoke a little more heavily than do people in the country.

SHIFT IN SMOKING HABITS—

Q Smoking has been going on quite a time. Do you think there's any chance of a change?

A Possibly, if one goes by the experience that I've had in my own immediate circle of friends. A third to half of them have stopped smoking, but that is an unusual sample. Among the young people with whom I come into contact, less than half of them are taking up smoking.

Now, I don't know whether that is generally true throughout the country. I think the figures show that approximately 73 per cent of all men smoke and about 33 per cent of ~~women~~ smoke.

Q And yet the over-all cigarette consumption is on the increase, isn't it?

A It is on the increase, but there are almost 3 million more of us each year.

Q What can you tell us of the research being conducted by the Public Health Service on lung cancer and smoking?

(Continued on next page)

Interview

A We have undertaken research both in the laboratory and in the field. Statistical data usually result from field research. And, while the research we have been undertaking hasn't been the spectacular, earth-stirring sort, nevertheless it has been designed to get some of the answers we are anxious to have.

Q Has that research been going on some time?

A Yes, the study with the veterans' group with which you may be familiar has been going since 1953. It was started as a co-operative project with the Veterans' Administration and is designed to find out what happens to men in a certain age group—this particular group happened to be veterans of World War I—on whom we obtained smoking histories. And since they have National Service Life Insurance, their deaths come to the attention of the veterans' facility. Many of them, of course, die in veterans' hospitals, which have accurate records and data, so we know what happens to them. And they die at an almost predictable rate.

In this instance we started out with 290,000, I believe it was, and we have been able to get smoking histories and follow-up data on something over 260,000. That's the largest sample in existence, I believe.

THE AGE GROUP TESTED—

Q Is your group more representative than the American Cancer Society group?

A Perhaps. It must be remembered that these are all men in the age group from 55 upward. We have some of the same ones that the American Cancer Society has. I would say that, since we have more and they are veterans, it probably might be considered to be a more representative group.

Q Well, yours covers the entire nation, doesn't it?

A Yes.

Q There has been some criticism of the Cancer Society group because they were, in effect, selected from a particular economic level. Would that influence the statistical results?

A I don't think it would influence it very much. It might. I'm not a statistician, but I think the Cancer Society has a sufficiently large sample well enough based geographically to refute any undue criticism of the statistical sample. I do believe that the study of the 290,000 which we inaugurated in 1953 may be a better cross section. First of all, there are more of them, and they were soldiers and sailors who went back to all parts of the country and lived under all sorts of conditions. I think one of the principal advantages will be that we will have better autopsy records of these individuals.

Q Have you had any results yet?

A No results that can be indicated as anything more than a sample to show us which way things are going. So far, preliminary data roughly parallel those on which the Cancer Society has already reported. However, these are small samples so far and we are involved in coding all of the records. It takes a good while to get a record back from the veterans' hospital or the place at which death occurred. These records go first to the veterans' facility, then to us for coding and recording. We haven't been attempting urgently to process these data and get them out because we would rather do a good, thorough job and study them completely.

Q Doctor, have you set a time for this study? Will it go on for a given period of years?

(Continued on page 64)

WHAT THE HEALTH SERVICE SAYS ABOUT SMOKING

Following is full text of a statement issued July 12, 1957, by Surgeon General Leroy E. Burney of the Public Health Service in Washington, D.C.:

The Public Health Service is, of course, concerned with broad factors which substantially affect the health of the American people. The Service also has a responsibility to bring health facts to the attention of the health professions and the public.

In June, 1956, units of the Public Health Service joined with two private voluntary health organizations to establish a scientific study group to appraise the available data on smoking and health. We have now reviewed the report of this study group and other recent data, including the report of Dr. E. C. Hammond and Dr. Daniel Horn on June 5 to the American Medical Association in New York.

In the light of these studies, it is clear that there is an increasing and consistent body of evidence that excessive cigarette smoking is one of the causative factors in lung cancer.

The study group, appraising 18 independent studies, reported that lung cancer occurs much more frequently among cigarette smokers than among nonsmokers, and there is a direct relationship between the incidence of lung cancer and the amount smoked. This finding was



—Harris & Ewing

DR. LEROY BURNEY is the U. S. Surgeon General. In the statement above, he warns the public of possible danger in "excessive cigarette smoking."



—United Press

DR. C. C. LITTLE is chairman of the tobacco industry's Scientific Advisory Board. He says, in the statement at right, that there is "no new evidence" on lung cancer.

reinforced by the more recent report to the AMA by Drs. Hammond and Horn.

Many independent studies thus have confirmed beyond reasonable doubt that there is a high degree of statistical association between lung cancer and heavy and prolonged cigarette smoking.

Such evidence, of course, is largely epidemiological in nature. It should be noted, however, that many important public-health advances in the past have been developed upon the basis of statistical or epidemiological information. The study group also reported that, in laboratory studies on animals, at least five independent investigators have produced malignancies by tobacco-smoke condensates. It also reported that biological changes similar to those which take place in the genesis of cancer have been observed in the lungs of heavy smokers. Thus, some laboratory and biological data provide contributory evidence to support the concept that excessive smoking is one of the causative factors in the increasing incidence of lung cancer.

At the same time, it is clear that heavy and prolonged cigarette smoking is not the only cause of lung cancer. Lung cancer occurs among nonsmokers, and the incidence of lung cancer among various population groups does not always coincide with the amount of cigarette smoking.

The precise nature of the factors in heavy and prolonged cigarette smoking which can cause lung cancer is not known. The Public Health Service supports the recommendation of the study group that more research is needed to identify, isolate and try to eliminate the factors in excessive cigarette smoking which can cause cancer.

The Service also supports the recommendation that more research is needed into the role of air pollution and other factors which may also be causes of lung cancer in man.

To help disseminate the facts, the Public Health Service is sending copies of this statement, the study-group report and the report of Drs. Hammond and Horn to State health officers and to the American Medical Association with the request that they consider distributing copies to local health officers, medical societies and other health groups.

While there are naturally differences of opinion in interpreting the data on lung cancer and cigarette smoking, the Public Health Service feels the weight of the evidence is increasingly pointing in one direction: that excessive smoking is one of the causative factors in lung cancer.

The Service notes that the study group found that more study is needed to determine the meaning and significance of any statistical association between smoking and heart disease. The study group reported there is no convincing biological or clinical evidence to date to indicate that smoking per se is one of the causative factors in heart disease. Although the report by Drs. Hammond and Horn has since provided additional data on this subject, the Service feels that more statistical and biological data is needed to establish a definite position on this matter.

TOBACCO-INDUSTRY REPLY

Following is full text of a statement by Dr. Clarence Cook Little, chairman of the Scientific Advisory Board to the Tobacco Industry Research Committee, issued in Washington, D. C., July 12, 1957:

The statement issued today by the Surgeon General adds nothing new to what has been known about the cause of lung cancer. It reflects the opinions of some statisticians and the relatively few experimental scientists who have actively charged that cigarette smoking is a cause of lung cancer.

No new evidence has been produced since the Scientific Advisory Board of the Tobacco Industry Research Committee last stated its position on this question on May 1, 1957. At that time, I said that, although anyone has the right to state an opinion on cancer causation, "the Scientific Advisory Board questions the existence of sufficient definitive evidence to establish a simple cause-and-effect explanation of the complex problem of lung cancer."

That is most definitely our position today.

The Surgeon General's own statement makes clear that "lung cancer occurs among nonsmokers and the incidence of lung cancer among various population groups does not always coincide with the amount of cigarette smoking."

The Public Health Service also supports the recommendation that more research is needed into the role of air pollution and other factors.

For the past three years, the Scientific Advisory Board has had the matter of tobacco use and human health under continuous review and consideration, both in the Board's regular meetings and in individual endeavors. We have had the responsibility of guiding a research program through which the Tobacco Industry Research Committee already has provided 2.2 million dollars for grants to independent scientists working in the fields of cancer and other challengers of human survival.

This research, thus far, has produced no evidence that cigarette smoking or other tobacco use contributes to the origin of lung cancer.

Many experiments on inhalation of cigarette smoke in animals have failed to produce a single cancer similar to the most prevalent type of lung cancer in humans. This and other facts show the need for continued unbiased research into the causes of cancer and other diseases.

Statisticians have so far failed to consider adequately many variables in human habits, environments and constitution, such as biological susceptibility to cancer, the effects of previous lung disease, hormonal influences and many other factors. It should be remembered that statistical association does not prove cause and effect.

In advising and educating the public, the Scientific Advisory Board believes that one should be as cautious in accepting a claim that a cause has been found for cancer as they have found it wise to be in the past in accepting a claim of a cure for cancer.

The Scientific Advisory Board intends to continue expansion of its program of making grants-in-aid to qualified scientists who propose to explore those areas of human health where the basic research problems appear most compelling and the prospect of results most promising.

Interview

. . . "Of course, we don't know what causes cancer"

A We propose that the study should go on as long as we can continue to get these records back, which means, theoretically, as long as any of these original people are alive.

Q Let me put it another way: Do you have any estimate of how long it will be before you do have some results from it that you feel confident enough of to report?

A There has been a tentative timetable set. By next spring we hope to have some preliminary data.

Q Do you feel that you have enough results so that you are sure it's going to run along the lines of the Cancer Society study?

A No, I can't say that. Merely, when you dip into the stream of information coming in, the samples tend to indicate that it was something in the same order of magnitude of happenings that the Cancer Society studied.

AIM IN CANCER RESEARCH—

Q What about your research studies—actual scientific studies—on the subject? Have they been carried forward?

A We have not undertaken some of the same sorts of studies that the late Dr. Evarts Graham, Dr. Ernest L. Wynder and others have undertaken, although we have supported—and are still supporting—many projects of this kind under our grants program. However, many years ago some of our investigators started studies subjecting mice to smoke. But the results were somewhat inconclusive. There is not much point in some of our scientists attempting to duplicate unnecessarily something that has been pretty well done by someone else. We feel that our skills and resources probably can best be utilized by studies in the field, studies someone else would find very difficult to undertake.

Some of our scientists are working in the laboratory attempting to find out some of the morphologic changes that occur in the lung structure when exposed to things like tobacco smoke and irritants of other kinds.

Q Have you found anything?

A Nothing of any spectacular nature as yet. You are probably aware of the work that Drs. Graham and Wynder and others have done in painting the condensates of tobacco tar on the skin of animals over a continued period. They have found that skin cancers will occur in a certain percentage of these mice and rabbits.

Q How high a percentage?

A They'll go as high, I believe, as about 40 per cent.

Q Aren't they using mice that are especially susceptible to cancer, and when you use ordinary mice you don't get the same results?

A That is true. There have been differences observed, utilizing the same techniques that Drs. Wynder and Graham set forth, but at least five investigators in this country have duplicated the Wynder and Graham results, using their same techniques.

Q If you use mice that are especially susceptible, how can you jump to any conclusions that smoke is causing the cancer?

A Controls are used.

Q Just because it is caused on the back of a mouse doesn't mean that the smoke would cause it in a human lung, does it?

A No, you are quite right. That's been one of the criticisms, of course, that mice aren't men, and certainly one cannot extrapolate from mice to men. However, there has been more or less of a truism, or a rule of thumb, set forth that any compound or any material which can definitely be incriminated as causing cancer in an animal is looked at with considerable suspicion as a possible cancerigenic agent in humans. In other words, it is suspect until we can prove unequivocally that it does not cause cancer in humans.

Q Do you suspect that there is in individuals a tendency toward getting cancer?

A Probably there is something in that premise. Why are some individuals cancer-prone and others not? First of all, of course, we don't know what causes cancer. We don't know whether it is a combination of things; whether the particular combination of atmospheric pollution, cigarette smoking, a person's sex—all of these things happen to come together and one triggers the others; whether certain individuals are born with cancer cells quiescent and something in time triggers them. We just don't know these things.

There are those who are convinced that individuals with certain body builds are much more likely to acquire cancer. We know, for instance, that an obese person has a slightly greater risk of acquiring any sort of cancer than a man who isn't quite so obese.

Q What if you knew? It wouldn't do you any good, would it?

A You could be examined every three or six months.

Q Wouldn't you create a nation of people with phobias?

A One certainly might be confronted with that, but, if it is approached in a common-sense fashion, I don't think that necessarily we would have people with phobias. People who acquire phobias—cancer phobia, whatever the phobia may be—are the sort of people who have got to be afraid of something anyway.

Q Are you getting closer to finding the causes of cancer?

A I think we are. I don't know whether it is right around the corner or many corners away from us.

THEORY ABOUT VIRUSES—

Q Is it possible that a virus or organism might be a factor?

A We do not know whether viruses can cause human cancer. We do know that viruses can cause certain cancers in animals—leukemias and certain other tumors. There are many investigators in the cancer field who are convinced that many human cancers have a viral origin. That thesis has not been demonstrated conclusively, and whether or not it is true is simply in the future.

Q Is there anything to indicate any connection between alcohol and cancer?

A Not enough for one to speak with any degree of assurance. The Roswell Park group in New York noticed some correlation between alcohol and the occurrence of cancer of the urinary bladder. There have been some studies on alcohol in the occurrence of oral cancer, but not enough that anyone feels confident about it.

Q Doctor, you say that you don't know what the cause of

. . . "We don't know what happens" in a cancer cell

cancer is, but you seem to imply at the same time that you feel almost 100 per cent sure that there is some type of cause-and-effect relationship between excessive smoking and lung cancer. You haven't found out just what it is today, but someday you'll find it and it will be there in that relationship, is that right?

A When we speak of a "cause" of cancer, we don't know what happens in that cell to cause it to cease being a normal cell and become an abnormal, wild, berserk organism. What happens in there chemically, we just don't know. Whether it's something that normally resides in that cell or whether it's something that comes in from the outside and changes the chemical constituents in the cell or whether it's a combination of these things, we don't know. All these things we have to find out.

Q You don't know when the breakthrough will come?

A I don't know when it will come. I believe it will come.

Q Soon?

A I wouldn't stick my neck out that much. I believe it's coming. I think that a lot of good work has been done in many areas and many aspects of cancer research.

Q When you break through, will that cover the range of cancers or just one or two of them?

A No, I suspect that it will be in one site [location in the body] of cancer. It might be in leukemia or in Hodgkin's disease or in lymphosarcoma or in that class of so-called cancers.

For example, there's one very important ray of hope: A rather rare cancer called choriocarcinoma, occurs in the uterus of women who have just given birth to a youngster—only about 300 cases a year occur in this country, so you can see how rare it is. Using a drug called methotrexate, which is an antifolic compound, five cases have been treated at the National Cancer Institute and, to the best of my knowledge, all of them are doing well, even though the cancer had spread. Now, we're not contending that's a cure. I'm merely mentioning it here because of its pertinence. We are hoping for a breakthrough in this particularly rare tumor. If we can cure one human tumor with a chemical compound, that's a handhold and we will try to find out how it works and why it works and apply it to others. It's likely that a breakthrough will occur tumor by tumor or site by site. **[END]**

A Chemical in Tobacco—Can It Cause Cancer?

What follows is one more phase in the dispute over cigarette smoking and cancer.

A news story by the United Press, distributed on July 15, said:

U. S. Surgeon General Leroy E. Burney said Government scientists have found a chemical agent in cigarette smoke which they suspect may cause lung cancer.

Burney, in an interview with United Press, identified the substance as benzpyrene. He said it is formed when the aromatic oils in tobacco are burned at a high temperature and "cracked" much like crude oil is cracked to make gasoline.

The chief of the Public Health Service emphasized that "no one has definitely isolated the cause of lung cancer." But, he said, researchers believe "excessive and prolonged" exposure to benzpyrene may be it.

Burney said benzpyrene was found in significant amounts only in cigarette smoke because only cigarettes burn at high-enough temperatures—800 to 815 degrees Fahrenheit—to crack the aromatic oils. Pipe tobacco and cigars burn at much lower temperatures, he said.

Dr. Robert C. Hockett, associate scientific director of the Tobacco Industry Research Committee, replied on the same day, July 15, to the statements attributed to Dr. Burney. Excerpts from Dr. Hockett's statement follow:

The question of the presence of benzpyrene in tobacco smoke has been under investigation for a number of years, and it has been widely discounted as a significant factor in connection with lung cancer by scientists familiar with the work.

There are a number of reasons for this.

Scientists have not actually succeeded in isolating the substance from tobacco smoke. Some feel that they have identified the substance by means of extremely sensitive, indirect measurements as probably present in smoke. Several chemists have not been able to detect benzpyrene in smoke at all, so elusive is this substance.

The general conclusion is that, if benzpyrene is actually present in cigarette smoke, it occurs in such minute quantities it could not even account for such biologic activity as has been reported for tobacco smoke in some experiments on sensitive mouse skin.

* * *

The substance frequently is produced in minute quantities in the burning of any organic compound and is present in varying degrees in city air. Recent reports in England show that the daily intake of benzpyrene from breathing London air is equivalent in total volume to the intake from smoking about 100 cigarettes a day.

After seeing the press reports, the Surgeon General on July 16 issued the following statement:

There are some scientific studies which indicate that benzpyrene has been identified in cigarette tars. There is no evidence to indicate, however, that benzpyrene of itself is present in sufficient quantities in cigarette tars to cause human lung cancer. More research is needed on this question and the effects produced by other chemical agents, as well as temperature gradients and various specific materials in the original tobacco.

WHAT BRITONS ARE TOLD ABOUT LUNG CANCER AND TOBACCO

At the same time that Americans are being told officially of a link between smoking and cancer, the British Government is carrying the same process a step further.

In Britain, posters are being distributed that carry a warning from the Government about the risk involved in cigarettes.

What decided the British Government upon taking this unprecedented step were the findings of a new study on smoking and cancer made by a panel of independent scientists for that country's Medical Research Council.

On these pages is the study that brought official action in Britain.

Following is full text of a report, "Cancer of the Lung—Recent Knowledge of Causative Factors," released in England by the Medical Research Council on June 27, 1957:

In their Annual Report for 1948-50 the Council drew attention to the very great increase that had taken place in the death rate from lung cancer in Britain over the previous 25 years, and they referred to the early results of an investigation into the possible causes of this increase, which had been undertaken by Prof. Bradford Hill and Dr. R. Doll of the Statistical Research Unit.

Since that time the death rate has continued to rise, so that in 1955 it reached a level more than double that recorded only 10 years previously—388 deaths per million persons of all ages in 1955 compared with 188 in 1945. Among males, the disease is now responsible for approximately 1 in 18 of the deaths at all ages, while of those aged 45 to 64 years the proportion is as high as 1 in 9. The corresponding figures for females are 1 in 103 and 1 in 42.

Knowledge of the causation of the disease is still incomplete but, as a result of the great amount of work carried out in the last few years, much light has been thrown upon what now appears to be the principal factor, namely, the smoking of tobacco, particularly in the form of cigarettes.

The final results of the retrospective investigation referred to above were published by Doll and Hill in 1952. In the course of that study very nearly 5,000 hospital patients, including almost 1,500 suffering from lung cancer, were interviewed by highly trained workers.

Analysis of the histories and habits of the patients with various diseases revealed only one striking contrast—the difference in the smoking habits of those with and those without lung cancer.

The proportion of cigarette smokers and heavy smokers was larger in the lung-cancer group than in the "control" group, and the proportion of nonsmokers, light smokers and pipe smokers was smaller. Thus, of the men with lung cancer, 25 per cent reported that they had been smoking an average of 25 grams (nearly an ounce) of tobacco, or more, a day, in cigarettes or pipes; for the male "control" patients the proportion was only 13 per cent. The corresponding figures for women were 11 per cent and 1 per cent.

Many similar investigations have now been reported from this country and elsewhere—from Finland, Germany, Holland, Norway, Switzerland and the U.S.A. In every case the principal results have been much the same.

Since then, the evidence in this country has been greatly strengthened by the results of a forward-looking inquiry—prospective as opposed to retrospective—reported by Doll and Hill in 1956.

In this study, a questionnaire was sent at the end of 1951 to all members of the medical profession in the United Kingdom—men and women—asking for brief details of their smoking habits. Over 40,000 doctors replied and, on the basis of their answers, were classified into a few broad groups according to the amount of tobacco they smoked, their method of smoking it, and whether they had given up smoking or were, at that time, continuing to smoke.

These groups have now been followed up over the subsequent four and a half years, information being obtained from the Registrars-General and other sources about the deaths that have occurred among them.

Analysis of the data relating to men has shown a marked and steady increase in the mortality from lung cancer as the amount smoked increases. Thus, at ages 35 and over, the death rate per year rose from 0.07 per 1,000 in nonsmokers—based upon the observation of one death only—to 0.47 per 1,000 in smokers of 14 grams a day, to 0.86 per 1,000 in smokers of 15 to 24 grams a day, and finally to 1.66 per 1,000 in smokers of 25 grams or more a day—one gram of tobacco is approximately the amount contained in one cigarette.

The death rate of the heavy smokers was therefore some 20 times the rate in the nonsmokers. In cigarette smokers the rate was substantially higher than in pipe smokers, while the rate for smokers by both methods fell in between.

Among men who had given up smoking within the previous 10 years the rate was lower than among men who, at the time of completing their questionnaire, were continuing to smoke, and among men who had given up smoking for more than 10 years it was lower still.

It follows that the highest mortalities were found among men who were continuing to smoke cigarettes, and among heavy smokers in this group the death rate was nearly 40 times the rate among nonsmokers—that is, an annual rate of 2.76 per 1,000 against an annual rate of 0.07 per 1,000.

Similar results have been reported from the U.S.A. by Drs.

E. C. Hammond and D. Horn of the American Cancer Society, and in total the statistical evidence, from one or other form of inquiry is now very considerable. It is further strengthened by the observation from several sources that the extent of the relationship with smoking differs for different types of lung cancer. For the squamous, oat-cell and anaplastic cancers, which constitute the great majority of the cases, the relationship is close, but for one relatively uncommon type—adenocarcinoma—the relationship is weak or nonexistent. These forms can be distinguished only by microscopic examination of the tumors. So far, no adequate explanation of all this statistical evidence has been advanced except that of direct cause and effect—that smoking is, indeed, the principal factor in the causation of the disease.

From the physical and chemical point of view there is nothing inherently improbable in this interpretation. Tobacco smoke consists largely of microscopic oily droplets held in suspension in air, and these droplets are of a suitable size to be taken into the lungs and retained. The smoke may be condensed to a yellowish-brown tar, which is formed by partial combustion of the tobacco and by chemical changes produced in its constituents by brief submission to high temperatures.

Other materials formed by similar high-temperature treatments (for example, coal tar) are known to be responsible for certain forms of industrial cancer, and they have been shown to produce malignant skin tumors in laboratory animals. Dr. E. L. Wynder, of the Sloan-Kettering Institute for Cancer Research in New York, and his collaborators showed in 1953 that skin cancer in mice could be produced similarly by application of the tobacco tar formed by smoking large numbers of cigarettes in machines designed to reproduce as closely as possible the conditions of human smoking. Human skin, however, at least that on the fingers, does not seem to be susceptible to this action of the tar, at least in the amounts to which the smoker is exposed.

More recently, Wynder has shown that cancer may be induced in other strains of mice than those used in his first experiments, and also that the active material is contained in a chemically neutral fraction of the tar. Other workers, including Dr. D. L. Woodhouse in the University of Birmingham and Prof. R. D. Passey at the Chester Beatty Research Institute, London, have failed to elicit tumors in mice by application of cigarette smoke tar produced in a similar way.

Discrepant results in such experiments are not altogether surprising. Tobacco smoke is a most complex mixture. About a hundred constituents have been reported as having been identified with greater or lesser degrees of certainty. There must be many more not yet identified, and the composition of the smoke may be expected to vary with the type of tobacco, the method of smoking and the temperatures attained by the burning tobacco; these temperatures, in their turn, may be influenced by the tightness of packing and the degree of humidity of the tobacco. Cigarette smoke is strongly acid, unlike the smoke from most cigars and pipes, but it is not known whether the enhanced liability to lung cancer of cigarette smokers, as compared with cigar and pipe smokers, is related to such a difference.

Complex aromatic hydrocarbons are commonly formed by submitting organic materials to very high temperatures; such compounds have been shown to be present in cigarette smoke by several workers, notably Dr. A. J. Lindsey of the Sir John Cass College, London, with his collaborators Mr. R. L. Cooper and Mr. R. E. Walker. One of these compounds is 3,4-benzpyrene, a constituent of coal tar which is very potent in producing malignant skin tumors in mice. Other workers, including Prof. G. R. Clemo at Newcastle and Dr. A. I. Kosak and his collaborators at the Institute of Industrial Medicine of New York University, have failed to find 3,4-benzpyrene in cigarette smoke, but Prof. H. Lettre in Germany has reported its presence in the material extracted from cigar stubs.

In view of these experimental observations, it may be supposed that a case is made out incriminating the carcinogenic substance 3,4-benzpyrene in tobacco smoke as a causative agent in lung cancer. But a closer examination leads to the conclusion that the case is not proven. The amount of 3,4-benzpyrene in the smoke from 100 cigarettes has been estimated to be about one thirtieth of a millionth of an ounce; about a fifteenth of this probably comes from the cigarette paper and the remainder from the tobacco. Even though this substance is known to be a powerful cancer-producing agent, there is no certainty that it is harmful in such low concentration.

A neutral fraction of cigarette-smoke tar, used in experiments reported by Wynder in collaboration with Prof. F. G. Wright of the University of Toronto in 1956, was found to contain 3,4-benzpyrene, but the amount present was considered to be much too small to account for the skin tumors in mice induced with this fraction; some unknown agent was therefore held to be responsible.

Another weakness in the evidence lies in the nature of the biological test. Apart from the methods, reported by Lisco and Finkel and by A. J. Vorwald, of producing lung cancer in rats by exposure to radioactive cerium and to beryllium salts respectively, there is no method of inducing in laboratory animals cancers arising from the bronchial tubes similar to those which constitute most of the lung cancers in man. The fact that a given material will produce skin cancer in mice or in rabbits is far from being presumptive evidence that the same material can cause lung cancer of the type with which we are concerned—experimentally induced lung cancer has usually been of quite another type.

A new biological test is required. Toward this end some

HOW THE BRITISH ARE BEING WARNED ABOUT SMOKING

The British Government, acting on the findings made by the Medical Research Council, now is distributing posters through local health authorities that read as follows:

SMOKING AND HEALTH

It is my duty to warn all cigarette smokers that there is now conclusive evidence that they are running a greater risk of contracting lung cancer than nonsmokers. The risk mounts with the number of cigarettes smoked. Giving up smoking reduces the risk.

Medical Officer of Health

TO ALL SMOKERS

There are now the strongest reasons to believe that smokers—particularly of cigarettes—run a greater risk of lung cancer than nonsmokers. The more cigarettes smoked, the greater the risk.

. . . "Many factors other than tobacco smoke" can produce cancer

promising tissue-culture experiments were reported in 1956 by Dr. I. Lasnitzki of the Strangeways Research Laboratory, Cambridge. She showed that small quantities of 3,4-benzpyrene induced hyperplasia of epithelial cells in human foetal lung tissue grown *in vitro*. If this technique can be suitably developed, it will help materially in the investigation of tobacco smoke.

Many factors other than tobacco smoke are undoubtedly also capable of producing lung cancer in man. At least five industrial hazards have already been recognized, and there is evidence that there may be others. For instance, in the last few years Dr. Lesley Bidstrup of the Department for Research in Industrial Medicine, London Hospital, and Dr. R. A. M. Case of the Institute of Cancer Research, London, have shown an enhanced risk in men engaged in this country in the manufacture of chromates from chromite ore; the extent of the risk for men heavily exposed to asbestos dust has also been demonstrated by Doll.

Dangers in Polluted Air

Moreover, studies of atmospheric pollution which suggest an association with lung cancer have been reported. Thus Dr. D. F. Eastcott found that immigrants into New Zealand—most of whom came from Britain—showed a higher mortality from lung cancer than native-born white New Zealanders. The mortality was particularly high among those who had immigrated late in life. Total tobacco consumption has been approximately the same in the two countries for the last 50 years, and Eastcott therefore suggested that differences in smoking habits were unlikely to be the underlying cause of the difference. However, cigarette consumption appears to have been much greater in Britain than in New Zealand; this may account for the difference, since the risk of developing lung cancer is greater with cigarette smoking than with smoking tobacco in other forms.

Some part of the difference may, perhaps, also be attributable to differences in the degree of pollution of the air; this additional factor has been suggested as the explanation of the higher mortality rates which have been consistently observed in the most densely populated parts of Britain and other countries.

A difficulty in assessing the effects of atmospheric pollution is that the smoking habits of persons resident in town and country are by no means identical—and may in past years have differed still more markedly. Dr. P. Stocks and Dr. J. M. Campbell have endeavored to overcome this to some extent by collecting data from persons dying of lung cancer in Liverpool, in mixed urban and rural areas in Cheshire, and in rural areas in North Wales. They have compared the smoking histories thus obtained with those given by hospital patients suffering from other diseases resident in the same areas.

By such means they have estimated the lung-cancer death rates for men in various smoking categories in each of the three types of area. They conclude that the great majority of cases in the semiurban and rural areas outside Liverpool can be attributed to smoking, but that in Liverpool itself about three eighths of the cases may be due to some other factor, which, they suggest, is likely to be a general atmospheric pollutant. Doll and Hill's data, on the other hand, failed to show any evidence of a substantial difference in the risk among nonsmokers in Greater London and in rural areas.

The particular kinds of atmospheric pollution which come under suspicion as causes of lung cancer are exhaust fumes from petrol engines and diesel engines as well as smoke from chimneys. It has long been known that extracts of soot will produce skin cancer in mice, and it has also been shown that soot particles contain firmly bound, 3,4-benzpyrene. Moreover, soot is deposited in the lungs of town dwellers.

Recently Dr. P. Kotin, of the University of Southern California, Los Angeles, showed, with his collaborators, the presence of 3,4-benzpyrene in the exhaust smoke from a petrol engine and from a diesel engine under conditions of inefficient operation, although they found none when the engine was operating efficiently.

The presence of 3,4-benzpyrene in diesel-engine exhaust was also shown by Dr. P. R. Peacock in Glasgow, but Mr. B. T. Commins, Mr. R. E. Waller and Dr. P. J. Lawther in an investigation of smoke in a London bus garage concluded that the exhaust smoke from the buses in operation contains very little. Dr. Kotin and Dr. H. L. Falk obtained cancerous skin tumors in mice by application of an extract, freed from aromatic hydrocarbons, of a town atmosphere. The material was thought to contain oxidized products of petrol, emitted by internal-combustion engines.

Indirect negative evidence has been submitted by Dr. P. A. B. Raffle, senior medical officer to the London Transport Executive, who has found that in recent years the drivers, conductors and maintenance men in its employ—men who may be presumed to have had unusually heavy exposure to engine fumes—have suffered a death rate from lung cancer somewhat lower than the rate for the whole country.

Mr. R. L. Cooper suggested in 1953 that the solvent action of some of the known constituents of tobacco smoke might remove the 3,4-benzpyrene from the soot deposited in the lung and bring it into intimate contact with the tissues. In this way the tobacco smoke and the atmospheric pollution could be complementary to one another. Or it may be that the additive effect of these various sources of cancer-producing substance—which is not necessarily entirely, or even mainly, 3,4-benzpyrene—is sufficient to turn the scale and produce cancer when one of these sources alone would be harmless. These are some of the many questions that have been raised by recent studies.

Britain's Research Program

In their endeavor to answer them the Council have set up two committees to co-ordinate the experimental studies now being undertaken. Much of this work is being financed from a substantial benefaction by the British Tobacco Manufacturers' Association for research into the causes of lung cancer. With assistance from this fund the Council have provided laboratory facilities and staff in the University of Exeter, where the new Carcinogenic Substances Research Group has been established under the honorary direction of Dr. J. W. Cook; and they have set up two further research groups, on Atmospheric Pollution at St. Bartholomew's Hospital, London, under the direction of Dr. P. J. Lawther, where experiments on the effects of inhaling atmospheric pollutants, including tobacco smoke, are being undertaken, and for Epidemiological Research on Respiratory Diseases in the University of Sheffield, under the direction of Dr. J. Pemberton.

In addition, grants have been made to individual workers in universities, hospitals and elsewhere for personal remunera-

. . . "Amount of tar in cigarette smoke can be controlled"

tion, for scientific and technical assistance, or for special research expenses in connection with many different aspects of the problem.

The Council are therefore supporting an expanding program on lung cancer which is being directed toward elucidation of the various factors involved, and particularly that of tobacco smoke. The work is taking two main directions:

First, further investigations are being undertaken to follow up existing suggestions. In this category the greater part of the work is being directed (1) toward further statistical surveys bearing upon possible causative factors other than smoking, particularly occupational hazards in certain industries, (2) toward further tests on laboratory animals of the crude products of combustion of tobacco, and (3) toward investigations on the interrelationships that have been suggested between smoking, atmospheric pollution, chronic bronchitis and cancer of the lung.

Secondly, more fundamental work is being done on the chemical analysis of tobacco and its products of combustion, and the separate substances so detected are being tested biologically for cancer-producing properties. Much of this work is still in its comparatively early stages. In addition to these investigations, work is in progress to evaluate the results of different methods of treatment of lung cancer.

What a U. S. Scientist Says About Making "A Safer Cigarette" in Future

Following is text of a statement to the press, released by the Sloan-Kettering Institute, July 19, 1957:

"A safer cigarette can be made today by using an effective filter plus the proper blend of tobacco," said Dr. Ernest L. Wynder of the Sloan-Kettering Institute for Cancer Research.

Some tobacco companies, however, have taken advantage of the public's desire for filtered cigarettes by marketing increasingly ineffective cigarette filters placed on cigarettes containing tobacco which yields greater quantities of cancer-producing tar, he reported. Recent laboratory studies indicate that the majority of filtered cigarettes on the market have a tar and nicotine content which is at least as high, if not higher, than that of unfiltered regular-sized cigarettes. This is current despite the fact that the amount of tar (cancer-causing material) in cigarette smoke can be controlled without further delay with the knowledge about filters and types of tobaccos already at hand.

Dr. Wynder was testifying on July 19, by invitation, before the House of Representatives Legal and Monetary Affairs Subcommittee of the Committee on Government Operations.

In outlining the magnitude of the problem, Dr. Wynder pointed out that 25,000 deaths from lung cancer will occur this year and that at least 80 per cent of these deaths could have been prevented had these patients not smoked tobacco, and in particular cigarettes. Because it is such a firmly entrenched habit, however, more practical solutions than elimination of tobacco smoking have to be found, said Dr. Wynder.

The general use of an effective filter was one of the suggestions made by Dr. Wynder. He defined an "effective filter" as one which removed at least 40 per cent of the tar and nicotine from tobacco smoke of the average regular-sized cigarette. This is based on Dr. Wynder's laboratory findings and statistical studies which indicate that "the value of a filtered cigarette in reducing cancer risk is directly related to the decrease in tar content of the smoke over that of unfiltered cigarettes."

Dr. Wynder emphasized that to make a safer cigarette would require the addition of the effective filter to an average regular-size cigarette containing the proper blend of tobaccos. Some cigarette manufacturers have increased the tar production of their cigarettes by using high-tar-yielding tobaccos; the use of this tobacco plus an ineffective filter has actually increased the tar yield of some presently marketed "filtered cigarettes." "However," testified Dr. Wynder, "in a recent laboratory study, attention was called to a new development in the filtered-cigarette industry which seems to be encouraging—a filtered cigarette with a good pressure drop and satisfactory tobacco taste can be produced which will yield 40 per cent less nicotine and tar than the average regular-sized unfiltered cigarette." According to Dr. Wynder, "uniform acceptance of a filter in this range will be a partial answer to the present problem, provided, of course, that the smoker does not decide to smoke twice as many cigarettes, and provided, too, that the tobacco selection, cut or packing is not altered in such a way as to yield increasingly more tar."

Other recommendations made by Dr. Wynder for the solution of the cigarette problem were: (1) the possible lowering of burning temperature of cigarettes, since virtually no cancer-causing substances are produced when tobacco is burned at 620 degrees or less (present cigarette burning temperature is 880 degrees); (2) "dry-cleaning" the tobacco leaf to remove the waxy coating, shown by Dr. Wynder's group to be a major source of most of the cancer-causing substances.

Dr. Wynder gave an extensive review of the accumulated statistical and laboratory evidence of cigarette smoking as the primary cause of lung cancer. He also summarized the evaluation of the evidence by responsible organizations such as the public health services of the United States, Great Britain, Sweden and the Netherlands, the American Cancer Society, and leading scientific journals.

An American study group convened, at the request of the National Cancer Institute, National Heart Institute, American Cancer Society and the American Heart Association, concluded that: "The sum total of scientific evidence establishes beyond reasonable doubt that cigarette smoking is a causative factor in the rapidly increasing incidence of human epidermoid carcinoma of the lung."

The Medical Research Council of Great Britain summarized that: "Evidence from many investigations in different countries indicates that a major part of the increase is associated with tobacco smoking, particularly in the form of cigarettes. In the opinion of the Council, the most reasonable interpretation of this evidence is that the relationship is one of direct cause and effect. The identification of several carcinogenic substances in tobacco smoke provides a rational basis for such a causal relationship." [END]

EXHIBIT 10

NATIONAL CANCER INSTITUTE RESEARCH PROJECTS

RELATING TO LUNG CANCER AND TO TOBACCO

The following table lists pertinent intramural research projects and extramural research grants supported by the National Cancer Institute during the 5 years, July 1, 1952, to June 30, 1957. Many of these studies were initiated before the beginning of this period and most of them will continue active in the future.

A total of 82 grants and projects are included. Of these, 11 (4 projects, 7 grants) are directly concerned with lung cancer and tobacco, and 42 (21 projects, 21 grants) with lung cancer and other substances. Another group of 27 (13 projects, 14 grants) are devoted to studies on the etiology of cancer, including lung, without necessarily being concerned with a specific relationship between any potential etiological agent and lung cancer. Two grants are concerned with oral cancer and tobacco.

The 44 extramural grants to university and other nongovernmental laboratories represent an actual or planned expenditure of $1,137,000 over the 5-year period. Grants for the study of lung cancer and tobacco have received $212,430; those for the study of lung cancer and other suspect etiological agents, $330,742; those for general etiological studies involving lung cancer, $558,024; and those for oral cancer and tobacco, $35,804.

Equivalent figures for the intramural research projects of the National Cancer Institute are not available for the 5-year period since the Institute's accounts are based on organizational units rather than individual projects. We roughly estimate the cost of these 38 projects at $440,000. The total investment of our research effort in the field of lung cancer etiology is therefore about $1,577,000 over the period 1952–57.

Research projects relating to lung cancer and to tobacco, July 1, 1952–June 30, 1957

LUNG CANCER AND TOBACCO

Project or grant No.	Investigator and project or grant title	Date	Amount of grant
	Biometry Branch, National Cancer Institute, Bethesda, Md.:		
NCI-61	William Haenszel and Michael G. Shimkin: Survey of smoking patterns in the United States.	July 1, 1956, to June 30, 1957	
NCI-63	William Haenszel and Michael B. Shimkin: Retrospective study on the association of smoking and lung cancer in women.	do	
NCI-65	William Haenszel, I. M. Moriyami, and M. G. Sirken: Association of smoking history, residence history, and occupation history with lung cancer mortality.	do	
	Field investigation and demonstrations branch, National Cancer Institute, Bethesda, Md.:		
NCI-125	H. F. Dorn, R. F. Kaiser, M. D., and W. S. Baum, M. D.: The incidence of respiratory cancer and other major diseases among World War I veterans in relation to their smoking habits.	Dec. 1, 1953, to June 30, 1957	
	FIELD INVESTIGATION GRANTS		
	Health Research, Inc., Buffalo, N. Y.:		
CS-9408	Abraham Lilienfeld, Wendell Ames, and Morton Levin: Field study of the distribution of tobacco habits and other selected characteristics associated with various types of cancer in the population of Erie County, N. Y.	Sept 1, 1955, to Aug. 31, 1957	$23,304
	Utah State Department of Health, Salt Lake City, Utah:		
CS-9499	John W. Wright: Cancer mortality in a closed population, Utah State Department of Health.	June 1, 1957, to May 31, 1958	6,912

Research projects relating to lung cancer and to tobacco, July 1, 1952–June 30, 1957—Continued

LUNG CANCER AND TOBACCO—Continued

Project or grant No.	Investigator and project or grant title	Date	Amount of grant
	FIELD INVESTIGATION GRANTS—con.		
	Washington University, St. Louis, Mo.:		
CS-9158	E. A. Graham: Experimental production of cancer by tobacco products	Mar. 1, 1952, to Feb. 28, 1953	$17,000
		Mar. 1, 1953, to Feb. 28, 1954	18,572
		Mar. 1, 1954, to Feb. 28, 1955	18,800
	Total		54,372
	RESEARCH GRANTS		
	College of Medical Evangelists School of Medicine, Loma Linda, Calif.:		
C-2615	Lester H. Lonergan: The incidence of malignant neoplasms in nutritionally depleted mice exposed to cigarette smoke.	June 1, 1956, to May 31, 1957	6,808
	University of Louisville, Louisville, Ky.:		
C-2335	A. J. Miller and W. M. Christopherson: The possible carcinogenic properties of cigarette smoke, tars, and cigarette ingredients.	Sept. 1, 1954, to Aug. 31, 1955	8,067
		Sept. 1, 1955, to Aug. 31, 1956	5,240
		Sept. 1, 1956, to Aug. 31, 1957	7,056
	Total		20,363
	University of Texas, Southwestern Medical School, Dallas, Tex.:		
C-3036	R. H. Holland: Effects of cigarette smoke on the respiratory passages of the rabbit.	July 1, 1956, to Aug. 31, 1957	2,300
	University of Washington, St. Louis, Mo.:		
C-2640	E. A. Graham (deceased) and E. V. Cowdry (1957–58): Experimental production of cancer by tobacco products.	Mar. 1, 1953, to Feb. 28, 1954	16,543
		Mar. 1, 1954, to Feb. 28, 1955	18,227
		Mar. 1, 1955, to Feb. 28, 1956	17,977
		Mar. 1, 1956, to Feb. 28, 1957	23,894
		Mar. 1, 1957, to Feb. 28, 1958	23,000
	Total		98,641
	LUNG CANCER AND SUSPECT ETIOLOGICAL AGENTS OTHER THAN TOBACCO		
	Office of the Associate Director in Charge of Research, National Cancer Institute, Bethesda, Md.:		
OADR-206c	M. Kuratsune: Polycyclic aromatic hydrocarbons in roasted coffee and coffee soots.	1952–57	
		July 1, 1956, to June 30, 1957	
OADR-209	W. C. Hueper: Experimental studies in metal cancerigenesis.	1952–57	
		July 1, 1956, to June 30, 1957	
OADR-211	H. J. Cahnmann: Evaluation of various adsorbents elution methods for the chromatographic fractionation of polynuclear aromatic hydrocarbons.	1952–56	
		July 1, 1955, to June 30, 1956	
OADR-210a	W. C. Hueper: Studies on occupational and environmental respiratory cancer hazards caused by the inhalation of carbonaceous air pollutants (coal tar, petroleum asphalt, cutting oil, coffee-roast soot).	1952–57	
		July 1, 1956, to June 30, 1957	
	Biometry Branch, National Cancer Institute, Bethesda, Md.:		
NCI-056	Gilbert W. Beebe and Michael B. Shimkin: Relationship of mustard gas and influenza among veterans of World War I, and later development of lung cancer. (Additional support through contract SA-ph-1404 with the National Research Council.)	July 1, 1956, to June 30, 1957	
	Field Investigations and Demonstrations Branch, National Cancer Institute, Bethesda, Md.:		
NCI-052	C. D. McClure, M. D.: Epidemiology of cancer of the lung, Pittsburgh field study.	Jan. 1, 1954, to June 30, 1957	
NCI-053	A. G. Gilliam, M. D., B. K. Milmore, M. D., and Robert L. Smith, M. D.: Cancer in a closed population.	Oct. 1, 1953, to June 30, 1957	

Research projects relating to lung cancer and to tobacco, July 1, 1952–June 30, 1957—Continued

LUNG CANCER AND SUSPECT ETIOLOGICAL AGENTS OTHER THAN TOBACCO—Con.

Project or grant No.	Investigator and project or grant title	Date	Amount of grant
	RESEARCH GRANTS—continued		
NCI-080	Clarence Salsbury, M. D., William J. Pendergast, M. D., B. K. Milmore, MD., and A. G. Gilliam, M. D.: Studies in geographic and racial distribution of cancer.	June 1, 1954, to June 30, 1957	
NCI-119	Ruth Taylor, M. D., and Benjamin E. Carroll: Cancer mortality in several selected population groups in New England.	Apr. 1, 1956, to June 30, 1957	
NCI-123 (c)	Victor, E. Archer, M. D., James W. Egan, M. D., and Marian Erjavec, R. N.: Study of occupational backgrounds of hospitalized cases of lung and bowel cancer, leukemias, and primary malignant tumors of bone, in males over the age of 25 years.	Dec. 1, 1954, to June 30, 1957	
NCI-124 (c)	Victor E. Archer, M. D., James W. Egan, M. D., and Pope A. Lawrence (in cooperation with occupational health program, PHS): Cancer occurence in uranium miners of the Colorado Plateau.	Sept. 1, 1949, to June 30, 1957	
NCI-129 (c)	Pope A. Lawrence and Victor E. Archer, M. D.: Health survey of personnel employed in baths using radioactive water.	Dec. 1, 1952, to June 30, 1957	
NCI-132	Pope A. Lawrence, Benjamin E. Carroll, and P. Morgan, M. D.: Cancer experience in community population exposed to unusual radon gas concentration.	do	
NCI-130	Pope A. Lawrence, Ruth Taylor, M. D., and Marian Erjavec, R. N.: Survey of cancer mortality in West Virginia.	Oct. 1, 1953, to June 30, 1957	
NCI-184	John E. Dunn, Jr., M. D.: Occupation and lung cancer.	July 1, 1956, to June 30, 1957	
NCI-185	John E. Dunn, Jr., M. D.: Relation of smog to lung cancer.	do	
NCI-176	Victor E. Archer, M. D. (in cooperation with radiological health program of PHS): External radiation exposure of uranium miners as related to cancer occurrence.	Dec. 1, 1956, to June 30, 1957	
NCI-163	Victor E. Archer, M. D., James W. Egan, M. D., Marian Erjavec, R. N., and Pope A. Lawrence: Study of the cancer experience of nonuranium hard-rock miners in the Rocky Mountain area.	Dec. 1, 1953, to June 30, 1957	
NCI-128	Pope A. Lawrence and Benjamin E. Carroll: Cancer among railroad workers in certain occupations retrospective phase.	Dec. 1, 1954, to June 30, 1957	
NCI-174	Pope A. Lawrence and Benjamin E. Carroll: Types of cancer among railroad employees. Forward-going phase beginning January 1956.	Jan. 1, 1956, to June 30, 1957	
NCI-120	Benjamin E. Carroll and Pope A. Lawrence: Cancer as a cause of death and disability in railroad workers.	June 1, 1957, to June 30, 1957	
	FIELD INVESTIGATION GRANTS		
	Colorado State Department of Public Health, Denver, Colo.:		
CS-9422	R. L. Cleere: A study to develop analytical techniques to be used in correlating radioactivity in urine and the respiratory exposures of miners to radon and radon-daughter products.	Sept. 1, 1956, to June 30, 1957	$3,888
CS-9274	Joseph E. Cannon: Study of the effects from internal radiation in nonuranium miners.	Sept. 1, 1953, to Aug. 31, 1954 Sept. 1, 1954, to Aug. 31, 1955	13,880 13,990
	Total		27,870

Research projects relating to lung cancer and to tobacco, July 1, 1952–June 30, 1957—Continued

LUNG CANCER AND SUSPECT ETIOLOGICAL AGENTS OTHER THAN TOBACCO—Con.

Project or grant No.	Investigator and project or grant title	Date	Amount of grant
	FIELD INVESTIGATION GRANTS—con.		
CS-9118	R. L. Cleere: Study of workers and community inhabitants who are exposed to radioactive substances from uranium mines and processing industry in the Colorado Plateau.	July 1, 1952, to Sept. 30, 1954	$19,500
	University of Oklahoma, School of Medicine, Oklahoma City, Okla.:		
CS-9351	John Spencer Felton: Incidence of occupational cancer in an industrial-agricultural State with emphasis on the occurrence within the petroleum industry.	Sept. 1, 1954, to Aug. 31, 1955	15,253
		Sept. 1, 1955, to Aug. 31, 1956	14,445
		Sept. 1, 1956, to Aug. 31, 1957	16,170
	Total		45,868
	University of Pennsylvania, Philadelphia, Pa.:		
CS-9246	Douglas P. Murphy: The occurrence of cancer in the male members of families in which the incidence of cancer in the female members has been determined.	Dec. 1, 1952, to Nov. 30, 1953	19,817
		Dec. 1, 1953, to Nov. 30, 1954	20,913
		Dec. 1, 1954, to July 31, 1955	14,733
		Aug. 1, 1955, to July 31, 1957	12,108
	Total		67,571
	University of Utah, Salt Lake City, Utah:		
CS-9161	John Z. Bowers: Cancer hazards associated with the mining and milling of uranium ores and other radioactive materials.	Apr. 1, 1952, to Mar. 31, 1953	16,997
		Apr. 1, 1953, to Mar. 31, 1954	20,850
		Apr. 1, 1954, to June 30, 1955	11,819
	Total		49,666
	University of Southern California, Los Angeles, Calif.:		
CS-9399	Paul Kotin, John W. Mehl, and Hugh A. Edmonson: Experimental pulmonary carcinogenesis using vehicular exhausts, occupational, and atmospheric metal and inorganic dust particles and study of factors associated with sexual difference in cancer incidence.	Sept. 1, 1955, to Aug. 31, 1956	15,363
CS-9397	Paul Kotin, John W. Mehl, and Hugh A. Edmonson: Identification of atmospheric aliphatic hydrocarbons including epoxides and their use in experimental tumorigenesis using painting and inhalation.	do	16,416
CS-9398	Paul Kotin, John W. Mehl, and Hugh A. Edmonson: The fate of carcinogenic polycyclic aromatic hydrocarbons in soot following their emission into the atmosphere and deposition in the lungs of humans and experimental species.	do	16,254
CS-9156	Paul Kotin, John W. Mehl, and Hugh A. Edmonson: Investigation of potential carcinogenic environmental compounds.	June 1, 1952, to May 31, 1953	22,176
		June 1, 1953, to May 31, 1954	22,939
		June 1, 1954, to May 31, 1955	22,063
		June 1, 1955, to May 31, 1956	22,842
		June 1, 1956, to May 31, 1957	42,276
	Total		139,296
	RESEARCH GRANTS		
	University of California, San Francisco, Calif.:		
C-2379	David A. Wood and Seymour Farber: The evaluation of the cellular alterations following enzymatic aerosol treatment in mice and human beings.	Sept. 1, 1954, to Aug. 31, 1955	14,362
		Sept. 1, 1955, to Aug. 31, 1956	13,603
	Total		27,965
	College of Osteopathic Physicians and Surgeons, Los Angeles, Calif.:		
C-3077	M. H. Simmers: Carcinogenicity of paving petroleum asphalt.	Apr. 1, 1957, to Mar. 31, 1958	10,373

Research projects relating to lung cancer and to tobacco, July 1, 1952–June 30, 1957—Continued

LUNG CANCER AND SUSPECT ETIOLOGICAL AGENTS OTHER THAN TOBACCO—Con.

Project or grant No.	Investigator and project or grant title	Date	Amount of grant
	RESEARCH GRANTS—continued		
	Duke University, Durham, N. C.:		
C–1741	Stanfield Rogers: An investigation into the conditionality of tumor production (induction of lung tumors by urethane).	Sept. 1, 1952, to Aug. 31, 1953	$9,558
		Sept. 1, 1953, to Aug. 31, 1954	8,100
		Sept. 1, 1954, to Aug. 31, 1955	10,744
		Sept. 1, 1955, to Aug. 31, 1956	5,740
		Sept. 1, 1956, to Aug. 31, 1957	11,479
	Total		45,621
	Johns Hopkins University, Baltimore, Md.:		
C–603	A. M. Baetjer, C. H. Steffee, and J. H. Clark: Relation of hexavalent chromate dust to brochogenic carcinoma	Apr. 1, 1953, to Mar. 31, 1954	8,892
		Apr. 1, 1954, to Mar. 31, 1955	14,969
		Apr. 1, 1955, to Mar. 31, 1956	7,569
	Total		31,430
	University of Mississippi Medical Center, Jackson, Miss.:		
C–3263	T. D. Norman: Aerial metastasis of tumors in the lung (effect of vitamin E deficiency on lung epithelium).	Apr. 1, 1957, to Mar. 31, 1958	7,992
	Medical College of South Carolina, Charleston, S. C.:		
C–474	Kenneth M. Lynch: Lung tumors (induced by asbestos "floats").	Jan. 1 to Dec. 31, 1953	8,799
		Jan. 1 to Dec. 31, 1954	9,262
		Jan. 1 to Dec. 31, 1955	9,461
		Jan. 1 to Dec. 31, 1956	7,257
		Jan. 1 to Dec. 31, 1957	4,970
			39,749
	New York University, New York, N. Y.:		
C–3311	M. Kushner, N. Nelson, and S. Laskin: The experimental induction of bronchogenic carcinoma by compounds of chromium.	Apr. 1, 1957, to Mar. 31, 1958	8,038
	University of Texas, M. D. Anderson Hospital, Houston, Tex.:		
C–1542	Arthur Kirschbaum: Intrinsic susceptibility of mice to carcinogenic stimuli (differences in pulmonary tumor incidences in different genetic strains of mice after urethane treatment).	Sept. 1, 1952, to Aug. 31, 1953	3,672
		Sept. 1, 1953, to Aug. 31, 1954	3,760
			7,432
	Wayne University, Detroit, Mich.:		
C–2507	A. J. Vorwald and E. C. J. Urban: Experimental pulmonary cancer in monkeys exposed to beryllium compounds.	Dec. 1, 1954, to Nov. 30, 1955	24,437
		Dec. 1, 1955, to Nov. 30, 1956	8,209
		Dec. 1, 1956, to Nov. 30, 1957	15,972
			48,618
	Research Laboratories, National Cancer Institute, Bethesda, Md.:		
NCI–417	W. E. Heston, M. K. Deringer, T. B. Dunn, D. Uphoff, and G. Vlahakis: Investigation of the role of genes and their relationship to nongenetic factors in the development of cancer. (Response of different genetic strains to urethane.)	1952–57; July 1, 1956, to June 30, 1957.	
NCI–506	C. H. Steffee: Pulmonary tumors. (Induced by beryllium, cigarette tars, methylcolanthrene.)	1952–55; July 1, 1954, to June 30, 1955.	

LUNG CANCER, GENERAL ETIOLOGICAL STUDIES

	Field Investigation and Demonstrations Branch, National Cancer Institute, Bethesda, Md.:		
NCI–116 (c)	Miriam D. Manning, M. D.: Clinical and environmental study of mothers of children with malignancy other than hemotopoietic.	Apr. 1, 1953, to June 30, 1957	

Research projects relating to lung cancer and to tobacco, July 1, 1952–June 30, 1957—Continued

LUNG CANCER, GENERAL ETIOLOGICAL STUDIES—Continued

Project or grant No.	Investigator and project or grant title	Date	Amount of grant
	RESEARCH GRANTS—continued		
NCI-118	Pope A. Lawrence, Benjamin E. Carroll, and Ruth Taylor, M. D.: Geographical incidence of cancer of major sites in upstate New York.	Feb. 1, 1953, to June 30, 1957	
NCI-131	Pope A. Lawrence, K. Beck, Ruth Taylor, M. D., and Wm. Lloyd: Geographic distribution of respiratory cancer mortality in 15 selected States: West Virginia, Virginia, Maryland, Pennsylvania, Ohio, Kentucky, North Carolina, Missouri, Arkansas, Louisiana, Arizona, Utah, Colorado, New Mexico, and Montana.	Apr. 1, 1954, to June 30, 1957	
NCI-135	John Vander, M. D., and H. F. Dorn: Frequency of occurrence of cancer and other major diseases among a group covered by a statewide unemployment insurance.	July 1, 1955, to June 30, 1957	
NCI-137	Philip Morgan, M. D., W. S. Baum, M. D., P. A. Lawrence, and Marian Erjavec, R. N.: Etiology of lung, laryngeal, and bladder cancer in New Orleans, La.	Dec. 1, 1953, to June 30, 1957	
NCI-142	John E. Dunn, Jr.: Evaluation of death certificate reliability as source of environmental cancer information.		
NCI-146	Pope A. Lawrence, Henry Greenville, and Wm. Chen, M. D.: Geographic and environmental factors in cancer mortality in Washington County, Md.	May 1, 1955, to June 30, 1957	
NCI-150	Pope A. Lawrence: Geographic distribution of cancer in the United States.	Nov. 1, 1954, to June 30, 1957	
NCI-182	Lester Grinspoon, M. D. and John Dunn, Jr., M. D.: Excess lung cancer among Mexican females.	July 1, 1956, to June 30, 1957	
NCI-183	Lester Grinspoon, M. D.: Study of nonneoplastic and neoplastic changes in the respiratory mucosa of man, and the external factors which may have a relationship to these changes.	do	
NCI-186	B. E. Carroll and William Haenszel: Cancer mortality among Catholic Brothers and Sisters in several teaching orders in the United States.	Aug. 1, 1956, to June 30, 1957	
NCI-050	B. K. Milmore, M. D. and A. G. Gilliam, M. D.: Research in epidemiologic method.	Continuing	
NCI-051	A. G. Gilliam, M. D., B. K. Milmore, M. D., and Robert L. Smith, M. D.: Stimulation of epidemiological research in nonservice agencies and development of sources of field data suitable to NCI for answering specific epidemiological questions.	do	
	FIELD INVESTIGATION GRANTS		
	Arizona State Department of Health, Phoenix, Ariz.:		
CS-9489	C. G. Salsbury: Navaho cancer research project for 1957.	May 1 ,1957 to Dec. 31, 1957	$34, 400
	California State Department of Public Health, San Francisco, Calif.:		
CS-9267	Lester Breslow: Investigation of occupational aspects of lung cancer.	May 1, 1953, to Apr. 30, 1954	23, 400
		May 1, 1954, to Apr. 30, 1955	27, 270
		May 1, 1955, to Apr. 30, 1956	29, 123
	Total		79, 793
	The Chicago Medical School, division of oncology, Chicago, Ill.:		
CS-9212	Philippe Shubik: Methodology for studying environmental hazards.	Sept. 1, 1952, to Sept. 31, 1953	22, 518
	9212 (C)	Sept. 1, 1953, to Aug. 31, 1954	20, 866
	9212 (C2)	Sept. 1, 1954, to Aug. 31, 1955	20, 866

Research projects relating to lung cancer and to tobacco, July 1, 1952–June 30, 1957—Continued

LUNG CANCER, GENERAL ETIOLOGICAL STUDIES—Continued

Project or grant No.	Investigator and project or grant title	Date	Amount of grant
	FIELD INVESTIGATION GRANTS—CON.		
CS-9212	9212 (C3)	Sept. 1, 1955, to Aug. 31, 1956	$20,866
	9212 (C4)	Sept. 1, 1956, to Aug. 31, 1957	35,985
	Total		121,101
	University of Cincinnati, Cincinnati, Ohio:		
CS-9076	Edward J. McGrath: A coordinated clinical, pathological and roentgenographic study of carcinoma of the lung.	Sept. 1, 1952, to Aug. 31, 1953	4,374
		Sept. 1, 1953, to Aug. 31, 1954	4,698
		Sept. 1, 1954, to Aug. 31, 1955	4,968
	Total		14,040
	Committee for the Study of Lung Cancer, Philadelphia, Pa.:		
CS-9451	A Reynolds Crane and Walter H. Maloney: A clinical study of lung cancer.	May 1, 1956, to Apr. 30, 1957	4,615
		May 1, 1957, to Apr. 30, 1958	6,000
	Total		10,615
	Connecticut State Department of Health, Hartford, Conn.:		
CS-9347	Matthew H. Griswold: Cancer in Connecticut 1935–50 a comprehensive statistical report.	Sept. 1, 1954, to May 31, 1956	8,000
CS-9437	Matthew H. Griswold: Studies in cancer epidemiology based on Connecticut cancer register.	Dec. 1, 1955, to Nov. 30, 1956	15,000
		Dec. 1, 1956, to Nov. 30, 1957	15,000
	Total		30,000
CS-9146	Matthew H. Griswold: Environmental cancer study project	June 1, 1952, to May 31, 1953	22,295
		June 1, 1953, to Aug. 31, 1954	17,550
	Total		39,845
	President and Fellows of Harvard University, Cambridge, Mass.:		
CS-9141	Leonid S. Snegireff and Herbert L. Lombore: Epidemiological aspects of cancer of the lung.	Jan. 1 to Dec. 31, 1952	5,322
		Jan. 1 to Dec. 31, 1953	5,322
		Jan. 1, 1954, to Mar. 31, 1955	5,574
	Total		16,218
	State of Montana State Board of Health, Helena, Mont.:		
CS-9185	Kenneth E. Markuson and G. D. Carlyle Thompson: Environmental and occupational cancer survey.	Jan. 1, 1952, to Dec. 31, 1953	17,966
	National Academy of Sciences, National Research Council, Washington, D. C.:		
CS-9222	W. U. Gardner: Conference on the etiology of cancer of the lungs.	June 16, 1952, to June 30, 1953	3,000
CS-9409	Bernard M. Cohen, Paul Steiner, Seymour Jablon, and Robert Schrek: A study of cancer incidence in veterans.	Sept. 1, 1955, to Dec. 31, 1956	22,853
		Jan. 1 to Dec. 31, 1957	16,070
	Total		38,923
	University of Pittsburgh, Pittsburgh, Pa.:		
CS-9187	A. G. Kammer and W. E. Poel: Environmental cancer research unit:		
	9187 (C)	Dec. 1, 1952, to Nov. 30, 1953	20,320
	9187 (C2)	Dec. 1, 1953, to Nov. 30, 1954	19,989
	9187 (C3)	Dec. 1, 1954, to Nov. 30, 1955	26,337
	9187 (C4)	Dec. 1, 1955, to Nov. 30, 1956	24,000
	9187 (C5)	Dec. 1, 1956, to Nov. 30, 1957	37,893
	Total		128,539
	University of Utah, College of Medicine, Salt Lake City, Utah:		
CS-9336	Russell S. Jones: Incidence of lung cancer.	June 1, 1954, to May 31, 1955	8,024
		June 1, 1955, to Nov. 30, 1956	7,560
	Total		15,584

Research projects relating to lung cancer and to tobacco, July 1, 1952–June 30, 1957—Continued

ORAL CANCER AND TOBACCO

Project or grant No.	Investigator and project or grant title	Date	Amount of grant
	RESEARCH GRANTS		
	Indian Cancer Research Center, Bombay, India:		
C-1648	V. R. Khanolkar: Chemical analysis of varieties of Indian tobacco. Biological testing of purified fractions, or isolates for carcinogenicity (oral cancer).	Sept. 1, 1953, to Aug. 31, 1954	$7,750
		Sept. 1, 1954, to Aug. 31, 1955	7,329
		Sept. 1, 1955, to Aug. 31, 1956	4,015
		Sept. 1, 1956, to Aug. 31, 1957	8,137
			27,231
	Northwestern University, Chicago, Ill.:		
C-2775	J. C. Calandra and R. W. Tiecke: Role of tobacco in oral cancer.	do	8,573

EXHIBIT 11

[The New York Times, July 21, 1957]

HUGE TOBACCO INDUSTRY AGAIN ON DEFENSIVE

By William M. Blair

WASHINGTON, July 20.—After 2 years of relative calm, the cigarette-cancer controversy has been rekindled by the United States Public Health Service.

The Service's carefully worded announcement that there was "increasing and consistent evidence" that excessive smoking was a factor in cancer aroused the tobacco industry into a counterattack at a time when it believed the issue was dying from lack of nourishment.

A followup congressional inquiry bids fair to giving longer life to sharply differing views on the subject. The Senate received a bill this week to cut off Federal farm price supports and soil-bank payments to tobacco growers. It was spurred by the belief that there was a ridiculous element in the Public Health Service's cautioning Americans about smoking while the Government spent money to support tobacco production.

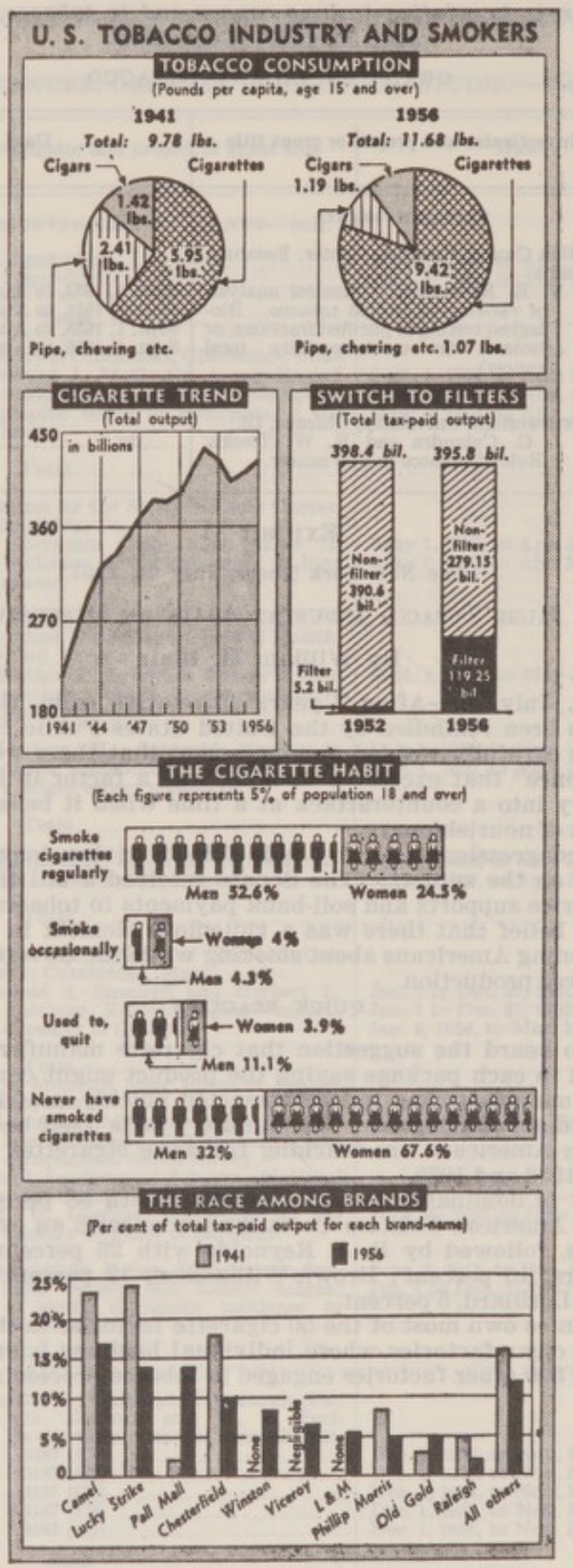
QUICK REACTION

Congress also heard the suggestion that cigarette manufacturers be required to paste a label in each package saying the product might cause cancer.

The tobacco manufacturing industry reacted quickly, matching statement for statement in and out of Congress. The industry finds itself back on the defensive at a time when Americans are reaching for more cigarettes after reaching for fewer between 1953 and 1955.

The industry is dominated by six companies with 98 percent of the tobacco business. The American Tobacco Co. is the leader with an estimated 30 percent of the business, followed by R. J. Reynolds with 26 percent. The others are Liggett & Myers, 16 percent; Brown-Williamson, 12 percent; Philip Morris, 9 percent, and P. Lorillard, 5 percent.

These companies own most of the 50 cigarette factories in the country. There are some 2,000 cigar factories whose individual business is small. In addition, there are about 300 other factories engaged in tobacco processing.



Data on filters and brands from Harry M. Wootten, consultant on tobacco industry. Cigarette smoking pattern from 1955 United States Census-National Cancer Institute survey.

INDUSTRY EMPLOYMENT

These factories employ about 500,000 persons in processing products with a retail value of $5,600 million and a value to some 3,000,000 persons on 700,000 farms of $1,200 million. The trade estimates 1,300,000 persons are engaged in wholesale and retail outlets, but this covers all types of sellers, including employes of drugstores and other establishments dispensing other products as well as tobacco.

About four-fifths of the tobacco crop goes into the making of cigarettes. Tobacco ranks near the top in cash value among farm crops, but near the bottom in acreage. Grown from New Hampshire to Florida and as far west as Minnesota, tobacco comes in many different kinds and varieties of leaf. Nearly all leading brands of cigarettes now are blends, with mixtures varying in the amount of flue-cured leaf, burley, and Turkish tobacco. They also contain a moistener and some a flavoring.

Cigarette manufacturers, engaged in a hot competitive fight among themselves as well as warding off the cancer issue, are reluctant to disclose their secrets.

Nevertheless, a dramatic change is taking place in the industry. Filter cigarettes are using less tobacco. A new process of using stems and pieces that formerly were thrown away is gaining in the trade. This means more profits as sales soar.

In its latest tobacco report, the Agriculture Department noted that while cigarette output was running 3 percent above 1955–56, indications were that the utilization of flue-cured and burley tobaccos had not increased.

"More cigarettes are being manufactured per pound of leaf tobacco than formerly," the Department said. "Filter-tip cigarettes, which continue to gain, take less tobacco per cigarette than those without filters. The use of processed tobacco sheet and stems and more efficient machinery also make it possible to get more cigarettes from a given quantity of leaf tobacco."

The new process involves grinding stems, fragments and broken and inferior leaves. The particles are mixed with a binder and rolled into a sheet. The sheet is dried and shredded and blended with higher-grade tobaccos for cigarettes. The process has become known as "homogenization." Some estimates place the cut in tobacco costs as high as 50 percent. Some trade spokesmen contend that this manmade leaf is milder and more favorable than natural leaf because the blending can be controlled.

MAKERS RETICENT

Tobacco growers' fears over the increasing use of "homogenized" tobacco brought a congressional hearing last year, but manufacturers were extremely reticent about the new process.

According to estimates, three-quarters of the cost to the manufacturer of a cigarette goes for tobacco. Since filter-tips take less tobacco and can be made with processed sheets, but sell for more per pack than non-filters, the manufacturers stand to save considerable money.

The filter elements cost only a fraction of the same amount of tobacco.

The Agriculture Department also reported there had been a reduction in the length and circumference of cigarettes with the introduction of filter-tips. It estimated that based on lengths and sizes manufacturers saved from 7 to 14 percent in tobacco in a filter-tip.

In 20 years the price of cigarettes has risen from 13 cents a pack to 25 cents on the average. Taxes are taking a bigger slice than before. The Federal excise tax is 8 cents a pack and States add an average of nearly 4 cents. The industry estimates tobacco companies pay $2,200 million annually in direct taxes and $500 million in corporate and other taxes.

TOP IN ADVERTISING

Contrary to public belief, the industry insists it spends less than a fourth as much for advertising as for tobacco, but the "Big Six" of the industry rank at the top of leading national advertisers in all media.

The advertising has shifted with the public winds over the years. Previously the emphasis was on health, with such slogans as "Guard against throat scratch" and "Safe for your T-zone." Those catch-phrases led to conflict with the Federal Trade Commission, which 2 years ago laid down a guide to its staff in judging cigarette advertising. The guide included cautions against claims of medical approval, references to the effect of smoking on nerves, noses and other parts of the human body and claims on nicotine and tar content.

This guide followed the first impact of the cancer controversy. The manufacturers shifted tactics. Now the emphasis is on pleasure and taste. Some students of the business say that the cancer problem threw a scare into the industry from which it has not yet fully recovered.

EXHIBIT 12

Submitted with Dr. Rigdon's statement
to the Subcommittee on Legal and Monetary Affairs
of the House Committee on Government Operations
Friday, July 19, 1957

Consideration of the Relationship of Smoking to Lung Cancer: With a Review of the Literature

R. H. RIGDON, M.D.
Galveston, Tex.

Consideration of the Relationship of Smoking to Lung Cancer:

With a Review of the Literature*

R. H. RIGDON, M.D.,† *Galveston, Tex.*

The argument over the place of cigarette smoking in the etiology of bronchogenic cancer continues to be waged and is far from settled. The author believes no good proof of the importance of smoking has been brought forth.

Historical Background

CANCER OF THE LUNG is not a new disease. Agricola in 1521 to 1527[1] described a pulmonary lesion in European miners which has been considered to be cancer. The illness suffered by some of the miners was characterized by an eating away of the hand, feet and lungs. In certain mines the mountain women were found to have married seven husbands, "all of whom this terrible consumption has carried off to a premature death."[2]

John Baptist Morgagni[3] in 1761 described a "cancerous ulcer" that occurred in "a man about sixty-six years of age, who for a long time had expectorated mucus, was seized, in consequence of exposure to cold, with an acute pain in the left side, accompanied with cough and fever. From the commencement of these symptoms he almost incessantly spat a thick and yellow matter streaked with blood. At length, however, this excretion was suppressed; and he died the seventh day of the disease." At autopsy "the left cavity of the thorax contained a fluid which resembled whey, and the lung on that side was not only very hard, but firmly adhered to the mediastinum, and also to the pleura investing the ribs. The lung on the right side had formed similar attachments, but especially to that part of the pleura which lines the anterior portion of the upper ribs; and the corresponding lobule of this viscus was the seat of a cancerous ulcer."

Bayle,[4] a Frenchman, in 1810 reported three cases of cancer of the lung, two of which were metastatic. The third has been accepted[5,6] as the first reported case of primary lung cancer; it occurred in the year 1805 in a man 72 years of age. One of the first cases of primary cancer of the lung reported in America was published by Storer in 1851.[7]

Interest in cancer of the lung has increased progressively since 1810. The difficulties encountered in the clinical diagnosis were emphasized by Laennec[8] in 1823. In 1837, Stokes[9] commented on the fact that "lung tumors are by no means as rare either in England or in Ireland as was generally assumed." Pepper,[10] in discussing this problem in 1851, had this to say: "It is not until quite recently that this subject has fully engaged the attention of the profession; hitherto, such cases were viewed as mere matters of medical curiosity, 'not known to be in any degree influenced by medicine, and too rare to be of much practical importance.' There is good reason to believe, however, this disease is of much more frequent occurrence than is commonly supposed, and that in a vast majority of cases it entirely escapes detection, owing to the great difficulty which attends its diagnosis." Rokitansky,[11] in 1854, considered cancer of the lung to be exceedingly rare. Pässler,[12] in 1896, accepted only 70 cases from 132 previously reported since "no cases without a postmortem report can be considered." Adler,[6] in 1912, was able to find in the literature 374 cases of lung tumors. Many of these apparently were erroneously diagnosed and others no doubt

*Presented to the Wisconsin Chapter of the American Academy of General Practice, Green Bay, Wisconsin, June 28, 1956. The experimental studies included in this paper are part of the program aided by the Medical College of Virginia.

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were metastatic lesions. Sweany,[13] in 1934, stated, "During the last half century primary lung cancer has sprung from obscurity into a rather common disease." It is evident, according to Sweany,[13] that part or perhaps much of this increase may be attributed to: (1) an increase in life expectancy; (2) increased knowledge in the course of certain diseases such as tuberculosis and bronchiectasis; (3) the tremendous effect on diagnosis brought about by the x-ray and the bronchoscope; (4) increased zeal on the part of the medical profession and laity as indicated by better hospitalization and better transportation; and (5) the changing attitude in the pathologic interpretation of lung cancer referable to metastases and sarcoma.

Review of Literature

There are many problems associated with the establishment of the frequency of any disease. One of the earliest critical analyses of this problem was made by King and Newsholme[14] and published in 1893. It was pointed out by these men that "the increase in cancer is only apparent and not real, and is due to improvement in diagnosis and more careful certification of the causes of death. This is shown by the fact that the whole of the increase has taken place in inaccessible cancer difficult of diagnosis, while accessible cancer easily diagnosed has remained practically stationary." In 1917, Willcox,[15] in discussing cancer, had this to say: "The question whether the disease is increasing can be answered only by a well-developed and trustworthy system of morbidity statistics. No country or city in the world now has such a system, because none has a good registration of sickness in the entire population." In 1931, it was pointed out by Downes[16] that "the general limitations of official mortality statistics as scientific data are already so well-known that none save the tyro in statistics will fail to go behind the published figures in order to take into account their grosser faults. The full extent of their limitations will not be appreciated adequately by anyone, however, until he makes the attempt to ascertain what the death rate actually is from a given disease in a given area." A report in 1955 from the Vital Statistics office of the United Nations[17] pointed out: "All medically certified cancers are not equal in quality. The knowledge and skill of the physician in making the diagnosis, his willingness to report completely, and the existence of facts for reporting certain diseases all affect the ultimate results."

A recent experience referable to cancer of the lung will illustrate one of these problems in vital statistics. A man had clinically what all of us thought was a bronchogenic carcinoma,—a biopsy was positive. However, at autopsy the patient had cirrhosis of the liver with a primary hepatic cell carcinoma that had metastasized to the lung and invaded the wall of a medium-sized bronchus near the hilus. Without an autopsy this case would have been recorded as a primary carcinoma of the lung.

In 1929, Dr. Weller[18] of the University of Michigan, warned us all of the difficulties encountered in the diagnosis of primary cancer of the lung. He said: "Only those reputed examples of primary carcinoma of the lung are of value on which a complete autopsy has been done. . . . There must be microscopic verification of each case, if the results are to be fully accepted. . . . Every experienced prosector can recall autopsies which painstaking search failed to disclose a primary site of malignant growth although abundant secondaries were present in the lungs or in the liver. . . . Cases are encountered too, in regard to which positive conclusion cannot be drawn even with thorough microscopic study. . . . In studies which are only statistical, the reader is at a loss to know how critical the compiler may have been in selecting his original units."

Recently at a clinicopathologic conference a most capable clinician presented a case of mediastinal tumor in a middle-aged white male. The x-ray confirmed the presence of the tumor. The resident on the case was asked about the smoking history. His answer was "Yes, two packs per day for 20 years." With this information the diagnosis of primary bronchogenic carcinoma was made. As pathologist I pointed out the fact that I also thought it was a case of lung cancer until I studied the histologic sections. They were typical of Hodgkin's disease. Without an autopsy this case would have been recorded as bronchogenic carcinoma. I am sure that every physician has one or more such diagnostic problems each year. If each one of us either fails to recognize only one case of cancer of the lung or makes an erroneous certifi-

cation of death, think what this would mean to our statistics on cancer of the lung.

Cancer of the lung has been considered by many to have increased since the time Bayle[4] first reported his case in 1810. Among these may be mentioned Hampeln[19] 1875, Menetrier[20] 1906, Karrenstein[21] 1908, Adler[6] 1912, Staehelin and Sachs[22] 1914, Kikuth[23] 1925, Konrad and Franke[24] 1929, Biberfeld[25] 1926, Pekelis[26] 1931, Zacherl[27] 1931, Loizaga and Vivoli[28] 1934, and Menne and Anderson[29] in 1941.

Perfillieff[30] in 1926 stated: "There has been a great increase in the number of cases of primary cancer of the lung during recent years. . . . In Russia this increase first became apparent in 1910 and was unusually high in 1923 and 1924." Wahl,[31] in 1927, found that cancer of the lung had increased 6.97 per cent during the preceding five years and stated that "we cannot suppress the impression . . . that just in the last years the lung carcinoma shows an enormous increase." Sweany,[13] in 1934, stated that the increase was tenfold during the past 50 years and that the diagnosis was not over five per cent correct at the beginning of the century. As late as 1925 the percentage of correct diagnoses had barely risen to 47. Rosenblatt and associates,[32] in 1956, stated cancer in the lung had increased 500 per cent since 1930. Dorn's[33] statistical studies indicate that the increase in mortality from lung cancer is slowing down. Between 1940 and 1950 the rate of increase in mortality declined and was only about one-half that of the previous decade. Among white males the death rate of ages under 35 was no higher between 1949 and 1951 than it had been two decades before. There was no recorded increase among white females from 1939 to 1949, except among those over 35 years of age. In the nonwhite population the rise in death rate from lung cancer was confined to persons over 30 years of age. Gilliam[34] reported in 1955: "I have previously stated that the rate of increase in recorded mortality was greatest in this country between 1914 and 1930 and it has been declining since."

The question has been asked frequently during the past 50 years—Is the increase in frequency of lung cancer real or only apparent? Many have considered it only apparent;[35-38] others have considered the increase to be real.[39-42] The U. S. Public Health Service[43] in 1950-54 reported a study on the incidence rate for cancer of the bronchus and lung per 100,000 population in ten cities for the years 1938 and 1948. The findings are as follow:

City	*1938*	*1948*	*Increase*
Atlanta	2.9	8.9	3.06
Dallas	3.6	16.3	4.52
Birmingham	4.7	13.6	2.89
Denver	6.6	14.8	2.24
New Orleans	7.6	20.8	2.60
Detroit	7.6	19.0	2.50
Pittsburgh	8.1	15.7	1.93
Chicago	9.1	15.9	1.74
Philadelphia	9.1	18.3	2.01
San Francisco	9.8	20.8	2.12

It is difficult to account for the wide variations in the frequency of this lesion in these ten cities. It is of considerable interest, however, to observe the terrific increase in the frequency of the disease within a period of ten years. It should be remembered that the bases of classification for cancer of the lung in Vital Statistics were changed in 1939. This may be a significant factor in this increase.

In attempting to determine the frequency of cancer of the lung during the past 100 years, it may be advantageous to establish its frequency referable to all malignancies as observed in large series of autopsies. Such studies have been compiled by Karczag,[44] Rigdon and Kirchoff,[45] and by Steiner.[46] The latter found "a general constancy of lung cancer in the different institutions provided the series are excluded which deal only with the early years of the present century and those confined to very recent years. On the average lung cancer comprised about 1, 7, and 8 per cent respectively of all necropsies, all malignant neoplasms, and all carcinomas." Many statisticians object to opinions based upon autopsies because the number of cases is small and the method of sampling unsatisfactory. The question must be answered as to whether the conclusions drawn from clinical diagnoses and Vital Statistics are preferable to those from autopsies for establishing the frequency of cancer of the lung.

Theories on Etiology

Many correlative studies have been made on the frequency of cancer of the lung in an attempt to establish the etiology of this lesion. Klotz[47] in 1927 said, "It is true that the cancer incidence of the lungs shows a parallelism with the increased use of gasoline engines." Kennaway and Kennaway[48] in 1936 reported that men engaged in the preparation and sale of tobacco show an increased prevalence of pulmonary cancer. Frommel[49] found a correlation between tarred roads and cancer of the lung. According to Doll and Hill,[50] Stocks, who is an internationally recognized statistician, showed in 1952, a direct relation between the size of a town, assessed by the number of occupied dwellings, and the mortality from lung cancer. Furthermore, Stocks[51] pointed out an inverse relation between the incidence of cancer of the lung and the amount of sunshine in different cities in England. The correlation receiving the most attention is that which relates the per capita consumption of tobacco with the increased frequency of lung cancer.[52-60] McNally,[52] in 1932, and Ochsner,[56] in 1941, were among the first to point out the graphic relation between the production of tobacco products and the increase in frequency of cancer of the lung. However, in 1948, seven years after the original paper,[56] Ochsner[61] and his group stated that "although we previously were of the opinion that the chronic irritation resulting from excessive cigarette smoking was a factor, this cannot be proved. However, the fact that there is a parallelism between the number of cigarettes sold in the United States and the increased incidence of bronchogenic carcinoma is interesting. . . . In our series we have not been able to show that there has been a higher incidence of smokers than in the average population as a whole." Data recently reported by Dr. Emma Moss[62] from Charity Hospital in New Orleans were interpreted by her as showing no increase in the frequency of cancer of the lung in the autopsies at Charity Hospital between 1947 and 1954.

Some of the clinicians at the John Sealy Hospital, which is the teaching hospital for the Medical Branch of the University of Texas, recently were greatly impressed by the tremendous increase in the number of cases of lung cancer they were finding. When these cases were checked for the county of residence, it was found that the cases were being referred to this hospital by physicians from many counties throughout the state. It was pointed out by Rigdon[63] at that time that better cooperation between doctors, better facilities for diagnosis and treatment, and better highways and improved means of transportation, all contributed to the increased number of cases of lung cancer being seen in the John Sealy Hospital.

In 1952, a study was made by Rigdon and Kirchoff[64] on the relation of death from cancer of the lung to the number of physicians and the number of hospital beds in each of the 48 states. In this study it was shown that the reported death rate from cancer of the lung is higher in those states which have a larger number of physicians relative to the size of the population, than it is in states which have a relatively smaller number of physicians. Likewise, the same death rate is higher in states which have a large number of hospital beds in proportion to the population, than it is in states with a relatively low number of beds. Based on this statistical study, it may be said that if you do not wish to be recorded as dying from cancer of the lung move to a state with few physicians and few hospital beds and you will be more likely to die from something other than cancer of the lung.

Time does not permit a discussion of the many agents which have been suggested as the etiology of lung cancer. Among those considered have been tuberculosis,[65] syphilis,[66] influenza,[67] diphtheria and measles,[68] chronic pulmonary inflammation,[69] pulmonary scarring,[70] road dust,[31] dust from grooming horses,[71] motor exhaust,[72] war gas,[73] cigar makers and sorters,[74] arsenic[75] chromium,[76] asbestosis,[77] silicosis,[78] metaplasia,[79] tar,[39] heredity,[80] trauma,[81] air pollution,[82] cigarette smoking,[55] and inhalation of printers ink while reading the morning newspaper.[83]

Tobacco smoking has been considered for many years as a cause of lung cancer. In 1888 Pennell[84] quoted Bultin as saying, "There is no evidence with which I am acquainted which will prove that carcinoma (of the tongue) is really much more common among adult males who smoke than among adult males who do not smoke; yet I think it is not improbable that smoking does to a certain

extent predispose to the disease." As early as the end of the 19th century Soemmering[85] suspected smoking to be associated with pulmonary cancer. Brosch[86] in 1900 was one of the first to produce malignant proliferation in the skin of guinea pigs by smearing the area with "tobacco juice." Tobacco has been implicated frequently as an etiologic agent in cancer of the lung since 1900. Among those who have mentioned this as a possible etiologic agent are Adler[6] 1912, Joannovic[87] 1923, Seyfarth[88] 1924, Staehelin[89] 1925, Philippson[90] 1926, Tylecote[91] 1927, Lipschitz[92] 1929, Lickint[93] 1930, Hoffman[94] 1931, McNally[52] 1932, Ferrari[95] 1933, Thys[96] 1935, Arkin and Wagner[97] 1936, Cramer[98] 1937, and Muller[99] in 1939.

The most recent "flare-up" of this perennial problem followed the studies of Doll and Hill,[50] Ochsner and associates,[100] Wynder and Graham,[55] and Hammond and Horn.[54] A careful study of these reports reveals some interesting facts. Figure 4, in the paper by Hammond and Horn,[54] shows that the death rate per 100,000 population for coronary artery disease by current amount of cigarette smoking is higher in males who smoke one-half to one pack of cigarettes per day than in those who smoke one pack or more a day. Furthermore, there is little difference at age 65 to 69 between those who smoke one pack or more a day and those that never smoked. In the same paper figure 6 shows death rates from cancer (all sites) to be greater when less than one-half a pack of cigarettes is smoked than it is when one smokes one-half to one pack a day. Regardless of these specific observations, these authors concluded, "These findings prove that there is a definite association between smoking habits and death rates, at least in white men between the ages of 50 and 69. . . . The associations found between regular cigarette smoking and death rates from diseases of the coronary arteries and between regular cigarette smoking and death rates from lung cancer reflect cause and effect relationships."

Study on Smoking Habits and Disease

In our attempt to evaluate the relation of smoking to disease we interviewed the following groups of people to obtain their smoking histories:[101]

University of Texas Medical Branch Personnel	1,000
John Sealy Hospital and Clinic Patients	12,905
Visitors to above patients	783
A Veterans Administration Hospital and a U.S.P.H.S. Hospital	550
College students	6,374
Total	21,612

The clinical diagnosis on 12,050 hospital and clinic patients has been correlated with their smoking history; this will be reported more fully in a subsequent publication.

The smoking data are based upon the average number of cigarettes smoked a day and the length of time this number has been used. It includes data on males and females, white and colored, for the following age groups: 15-19, 20-24, 25-29, 30-39, 40-49, 50-59, 60-69, and 70 years of age and over. The number of cigarettes smoked daily is classified as follows:

1.	Less than 5 cigarettes	
2.	One-half pack	5-14 cigarettes
3.	One pack	15-24 cigarettes
4.	One and one-half packs	25-34 cigarettes
5.	Two packs	35-44 cigarettes
6.	More than 2 packs	45 or more cigarettes

In this study pipe and cigar smokers are not converted into cigarette smokers by estimating one cigar to be equivalent to five cigarettes and a pipeful of tobacco equivalent to two and one-half cigarettes as Doll and Hill[53] have done, or a cigar equivalent to 10 cigarettes as Sadowsky and associates[57] did in their statistical study. Based on this method of converting cigars to cigarettes, a man who smokes three cigars a day would be considered statistically a heavy smoker since he would fall into the pack and one-half group. Should this same man also smoke 10 cigarettes, he would be classified as a "chain smoker."[60]

Observations on smoking have suggested to us many pitfalls that may occur in such a study. Among these may be mentioned the length to which a cigarette is smoked. In this connection Hilding[102] from Duluth, Minnesota, points out that the butts of cigarettes left by smokers give only information as to how much of the cigarette was burned—not how much was burned into the mouth. Important also is whether the cigarettes are "rolled" or machine made, and the memory accuracy of the respondent when the smoking

TABLE 1

THE PER CENT DISTRIBUTION OF 7,873 WHITE MEN ACCORDING TO AGE GROUPS AND SMOKING HABITS

		Per Cent			Per Cent Smoking		
Age	Total	Present	Past	Non-smokers	Cigarettes	Pipes	Cigars
15-19	1552	36.8	5.6	57.6	34.2	7.5	5.9
20-24	1746	57.7	5.1	37.2	53.5	12.6	10.5
25-29	941	70.5	8.0	21.5	65.6	12.4	10.6
30-39	858	77.7	9.1	13.2	72.8	9.2	10.8
40-49	850	79.2	8.7	12.1	74.7	5.1	6.3
50-59	865	78.6	9.7	11.7	71.0	7.5	8.9
60-69	752	66.7	17.2	16.1	58.8	8.8	7.3
70+	509	55.6	21.4	23.0	36.1	18.7	11.4
All ages	7873	63.2	9.0	27.8	57.3	10.0	8.8

history is taken. Other problems referable to statistical studies on smoking and lung cancer are discussed more fully by Kirchoff and Rigdon,[101] Berkson,[103] Lew,[104] Gilliam,[34] Macdonald,[105] and Hilding.[102]

Table 1 gives the total number of white males in the different age groups, the percentage of present, past and nonsmokers, and the percentage smoking cigarettes, pipes and/or cigars in our survey.[101] From our data the white men 40-49 years of age have the following smoking history:

79.2 per cent are present smokers, 8.7 per cent are past smokers, and 12.1 per cent have never smoked.

74.7 per cent now smoke cigarettes, 5.1 per cent pipes, and 6.3 per cent cigars.

82.8 per cent smoke or have smoked cigarettes.

15.9 per cent of these present cigarette smokers smoke less than one pack daily, 54.2 per cent smoke one pack, and 29.9 per cent more than one pack.

1.9 per cent of these present cigarette smokers have smoked 5 years or less, 1.4 per cent for 6 to 10 years, 4.7 per cent for 11 to 15 years, and 92.0 per cent have smoked 16 years or longer.

12.8 per cent of those who have smoked for 16 years or longer smoke one-half a pack daily, 46.9 per cent smoke one pack, 17.8 per cent smoke one and one-half packs, and 11.0 per cent smoke two or more packs.

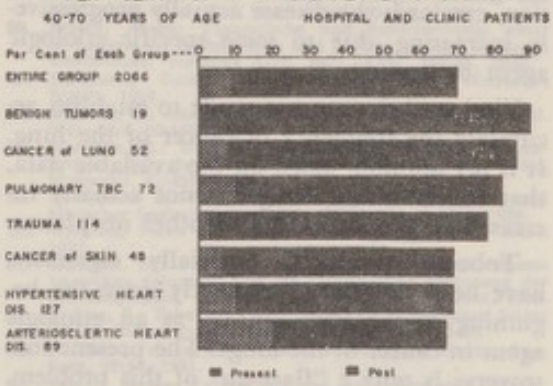
Our average cigarette smoker would be considered a maximum smoker in the classification of Hammond and Horn,[54] Mills and Porter,[100] and Schrek and associates.[60]

According to the smoking history and the medical diagnoses on the people in our survey[101] we have found that a high percentage of those with benign tumors, traumatic lesions and tuberculosis smoke cigarettes (Fig. 1). In a group of 409 pregnant white females 20 to 29 years of age, 46.5 per cent are present or past smokers; we do not wish to imply a "cause and effect" relation in this group of smokers.

In our group of 2,066 white men 40 to 70 years of age (Fig. 1), 90 per cent of those with benign tumors smoke cigarettes, 75 per cent of those with cancer of the skin and 79 per cent of those with hypertensive heart disease now smoke or have smoked cigarettes. Of the 114 cases that were seen because of a traumatic lesion, 81 per cent have smoked or now smoke cigarettes. Of the 52 white men with cancer of the lung, 94 per cent now smoke or have smoked cigarettes. Of those with pulmonary tuberculosis, 92 per cent have smoked or now smoke cigarettes. In our study the percentage of cigarette smokers among the patients with tuberculosis is only slightly less than that among the patients having lung cancer.

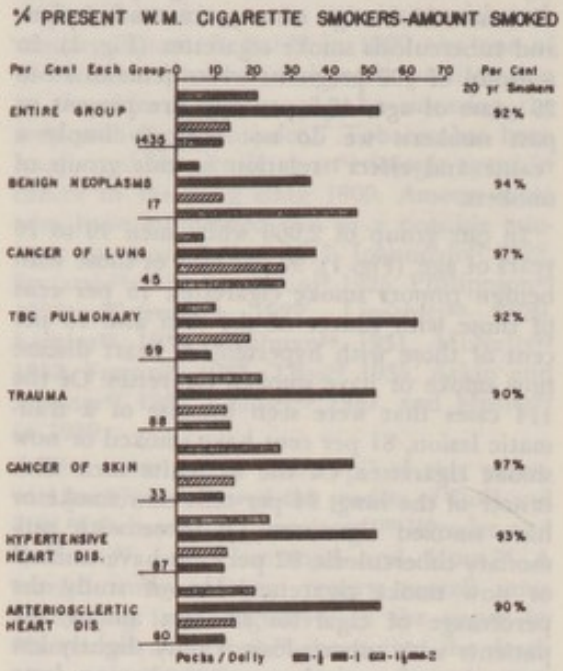
In figure 2 we find that the percentage of white male present cigarette smokers smoking two packs or more of cigarettes a day is much higher in the group with benign tumors (47.1 per cent) than it is in the group with cancer of the lung (28.9 per cent). Furthermore, the per cent distribution of the amount of cigarettes smoked daily by the people in the hypertensive heart disease group and in the arteriosclerotic heart disease group is es-

FIG. 1



The percentage of present and past cigarette smokers is shown according to diagnosis, in a group of white men between 40 and 70 years of age.

FIG. 2



Shows the percentage of white male present cigarette smokers, according to diagnosis and amount smoked, in a group of white men 40 to 70 years of age; also the percentage, according to diagnosis, smoking for 20 years or longer.

sentially the same as the distribution for the entire group.

Summary

In summary, it may be said that cancer of the lung has been recognized for approximately 150 years. During the past 100 years an almost continuous controversy has been carried on,—Is the increase in the frequency of lung cancer only apparent, due to better diagnoses, increase in the age of the population, etc., or is the disease actually progressively increasing, due to some specific etiologic agent or agents?

Vital statistics are too crude to establish accurately the frequency of cancer of the lung. It is my opinion, based on the available data, that cancer of the lung has not actually increased in proportion to all other neoplasms.

Tobacco products, especially cigarettes, have been suggested frequently since the beginning of the 20th century as an etiologic agent in cancer of the lungs. The present controversy is only a "flare-up" of this problem.

A demonstration of a carcinogen in cigarette tars for the skin of a mouse and a rabbit cannot be accepted scientifically as a carcinogen for the lung of man; such has been known for 25 years or longer. The frequency of the habit of smoking by peoples throughout the world during the past several hundred years and the relative infrequency of cancer of the lung should make us critical of the observations suggesting an association between cigarette smoking and cancer of the lung as "cause and effect." Diseases other than lung cancer occur in individuals who are heavy smokers without anyone suggesting "cause and effect." Furthermore, many individuals have died with cancer of the lung that did not smoke. Scientific studies on this problem should be encouraged because facts may be discovered about the pathogenesis of cancer which may serve as the basis for treatment.

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EXHIBIT 13

London Times June 28, 1957

LUNG CANCER INCREASE "DUE TO SMOKING"

MEDICAL FINDINGS ACCEPTED BY GOVERNMENT

LOCAL AUTHORITIES ASKED TO MAKE FACTS KNOWN

From Our Medical Correspondent

The Government have accepted the Medical Research Council's finding that a major part of the great increase in deaths from lung cancer among men during the past 25 years has been due to smoking tobacco, particularly heavy cigarette smoking.

Announcing this in the House of Commons yesterday, Mr. Vaughan-Morgan, Parliamentary Secretary to the Ministry of Health, said: "The Government feel that it is right to ensure that this latest authoritative opinion is brought effectively to public notice, so that everyone may know the risks involved."

All local health authorities are to be asked by the Ministry to inform the general public of the Medical Research Council's opinion.

The circular to local health authorities comments that the risks should be made known, so that the individual who smokes can make up his or her own mind. It adds that, while health education measures have hitherto been directed primarily to mothers of young children and other special groups, in this case "publicity of a more general character will be required." Material is to be provided by the Central Council of Health Education.

MUCH RESEARCH

The conclusions of the Medical Research Council's statement, which is amplified in the annual report of the council, published to-day, are as follows:—

1. A very great increase has occurred during the past 25 years in the death rate from lung cancer in Great Britain and other countries.
2. A relatively small number of the total cases can be attributed to specific industrial hazards.
3. A proportion of cases, the exact extent of which cannot yet be defined, may be due to atmospheric pollution.
4. Evidence from many investigations in different countries indicates that a major part of the increase is associated with tobacco smoking, particularly in the form of cigarettes. In the opinion of the Council, the most reasonable interpretation of this evidence is that the relationship is one of direct cause and effect.
5. The identification of several carcinogenic substances in tobacco smoke provides a rational basis for such a causal relationship."

RATE DOUBLED IN TEN YEARS

PEAK NOT REACHED

The statement points out that in 1955 the death rate from cancer of the lung reached a level more than double that recorded only 10 years earlier (388 deaths a million of the population in 1955 compared with 188 in 1945). Among males the disease is now responsible for approximately 1 in 18 of all deaths. Although the death rate for females is still comparatively low, it also has shown a considerable increase in recent years and the disease is now responsible for 1 in 103 of all female deaths.

Three comments are made on these figures. In the first place, the trend over the last few years indicates that the incidence has not yet reached its peak. Secondly, the figures are not to be explained as a mere reflection of the introduction and increasing use of improved methods of diagnosis but must be accepted as representing, in the main, a real rise in the incidence of the disease, to an extent which has occurred with no other form of cancer. Thirdly, only a small part of the rise can be attributed to the larger numbers of older persons now living in the population; in the last 10 years the lung cancer death rates among both men and women have risen at all ages from early middle-life on-

wards.

"It is necessary to seek some factor or factors distributed generally throughout the population, and in considering the possibilities it must be borne in mind that a very long period, 20 years or more, may elapse between exposure to a carcinogenic agent and the production of a tumour. From the nature of the disease attention has focused on two main environmental factors: (1) the smoking of tobacco, and (2) atmospheric pollution—whether from homes, factories, or the internal combustion engine.

RESULTS OF 19 INQUIRIES

SMOKING HABITS

The evidence that heavy and prolonged smoking of tobacco, particularly in the form of cigarettes, is associated with an increased risk of lung cancer is derived from two types of special inquiry. In the first, patients with lung cancer have been interviewed and their *previous* histories in relation to smoking and other factors that might be relevant have been compared with those similarly obtained from patients without lung cancer.

The results of 19 such inquiries (in this country, the United States, Finland, Germany, Holland, Norway, and Switzerland) have been published. They agree in showing more smokers and fewer non-smokers among the patients with lung cancer, and a steadily rising mortality as the amount of smoking increases.

In the second type of inquiry information has been obtained about the smoking habits of each member of a defined group in the population and the causes of the death occurring *subsequently* in the group have been ascertained. There have been two such investigations, one in the U.S.A. covering 190,000 men aged 50-69, and the other in this country covering over 40,000 men and women whose names appeared on the Medical Register of 1951. In both the results have been essentially the same.

The investigation in this country, which has now been in progress for more than five years, has shown with regard to lung cancer in men:—

(1) A higher mortality in smokers than in non-smokers.

(2) A higher mortality in heavy smokers than in light smokers.

(3) A higher mortality in cigarette smokers than in pipe smokers.

(4) A higher mortality in those who continued to smoke than in those who gave it up.

It follows that the highest mortalities were found among men who were continuing to smoke cigarettes, heavy smokers in this group having a death rate nearly 40 times the rate among non-smokers. Although no precise calculation can be made of the proportion of life-long heavy cigarette smokers who will die of lung cancer, the evidence suggests that, at current death rates, it is likely to be of the order of 1 in 8, whereas the corresponding figure for non-smokers would be of the order of 1 in 300. The observations on the effect of giving up smoking is particularly important, since it indicates that men who cease to smoke, even in their early forties, may reduce their likelihood of developing the disease by at least one half.

LABORATORY TESTS

EFFECT OF SUBSTANCES ON ANIMALS

It will be apparent from what has been said that the evidence from the many inquiries in the last eight years, both in this country and abroad, has been uniformly in one direction and is now very considerable. It has been further strengthened recently by the observation from several sources that the extent of the relationship with smoking differs for different types of lung tumour which can be distinguished only by microscopic examination.

From the physical and chemical point of view there is nothing inherently improbable in a connexion between smoking and lung cancer. Tobacco smoke consists largely of microscopic oily droplets held in suspension in air, and these droplets are of a suitable size to be taken into the lungs and retained there. Over 100 constituents have so far been identified and, among these, five substances have already been found which are known to be capable, in certain circumstances, of causing cancer in animals.

AIR POLLUTION

It has been known for some years that mortality from lung cancer is greater in urban areas than in the countryside. This fact, together with the identification of carcinogenic substances in coal smoke and in motor vehicle exhausts, has led to the supposition that exposure to atmospheric pollution may be concerned with the increase in lung cancer. The role of atmospheric pollution is particularly difficult to investigate, however, and the evidence is neither so consistent nor so extensive as that relating to tobacco smoking.

On the one hand, no excess mortality from lung cancer has been observed in persons who would be especially exposed by the nature of their work to atmospheric pollution, for example, transport workers, garage hands, and policemen. On the other hand, the results of a number of investigations have suggested that a relationship does exist between atmospheric pollution and lung cancer. Perhaps the best evidence for this relationship comes from studies of the small number of deaths from the disease among *non-smokers* in different types of residential district; in these studies higher death rates have been observed among non-smokers in large towns than among those in rural areas. On balance it seems likely that atmospheric pollution plays some part in causing the disease, but a relatively minor one in comparison with cigarette-smoking.

INDUSTRIAL CAUSES

Knowledge of the causation of lung cancer is still incomplete. Many factors other than tobacco-smoking are undoubtedly capable of producing the disease; for example, at least five industrial causes have been recognized. Nevertheless, the evidence for an association between lung cancer and tobacco-smoking has been steadily mounting throughout the past eight years and it is significant that, during the whole of this period, the most critical examination has failed to invalidate the main conclusions drawn from it. It has indeed been suggested that the fundamental cause may be some common factor underlying both the tendency to tobacco-smoking and to the development of lung cancer some 25 to 50 years later, but no evidence has been produced in support of this hypothesis.

The council is supporting an expanding programme of research in lung cancer, much of which is aided by the "substantial benefaction by the British Tobacco Manufacturers' Association for research into the cause of lung cancer." This work is taking two main directions. The first consists of further investigations of existing suggestions, such as statistical surveys into causative factors other than smoking, and further tests on laboratory animals of the crude products of combustion of tobacco.

TOBACCO SMOKING AND CANCER OF THE LUNG

STATEMENT BY THE MEDICAL RESEARCH COUNCIL

The Increase in Lung Cancer

In its annual report for 1948–50* the Council drew attention to the very great increase that had taken place in the death rate from lung cancer over the previous 25 years. Since that time the death rate has continued to rise, and in 1955 it reached more than double that recorded only 10 years earlier (388 deaths per million of the population in 1955 compared with 188 in 1945). Among males the disease is now responsible for approximately 1 in 18 of all deaths. Although the death rate for females is still comparatively low, it also has shown a considerable increase in recent years, and the disease is now responsible for 1 in 103 of all female deaths.

Three comments may be made on these figures. In the first place, the trend over the last few years indicates that the incidence has not yet reached its peak. Secondly, the figures are not to be explained as a mere reflection of the introduction and increasing use of improved methods of diagnosis but must be accepted as representing, in the main, a real rise in the incidence of the disease to an extent which has occurred with no other form of cancer. Thirdly, only a small part of the rise can be attributed to the larger numbers of older persons now living in the population; in the last ten years the lung cancer death rates among both men and women have risen at all ages from early middle life onwards.

Possible Causes of the Increase

The extent and rapidity of the increase in lung cancer point clearly to some potent environmental influence which has become prevalent in the past half-century and to which different countries, and presumably also men as compared with women, have been unequally exposed. The pattern of incidence of the disease rules out any possibility that the increase can be due, in a substantial degree, to special conditions, such as occupational hazards, affecting only limited groups. It is necessary to seek some factor or factors distributed generally throughout the population, and in considering the possibilities it must be borne in mind that a very long period, 20 years or more, may elapse between exposure to a carcinogenic agent and the production of a tumour. From the nature of the disease attention has focused on two main environmental factors: (1) the smoking of tobacco, and (2) atmospheric pollution—whether from homes, factories, or the internal combustion engine.

Smoking as a Cause of Lung Cancer

(a) Epidemiological Surveys

The evidence that heavy and prolonged smoking of tobacco, particularly in the form of cigarettes, is associated with an increased risk of lung cancer is not based on the observation that the substantial increase in the national mortality followed an increase in the national consumption of cigarettes. It is derived from two types of special inquiry. In the first, patients with lung cancer have been interviewed and their *previous* histories in relation to smoking and other factors that might be relevant have been compared with those similarly obtained from patients without lung cancer. The results of nineteen such inquiries (in this country, the U.S.A., Finland, Germany, Holland, Norway, and Switzerland) have been published. They agree in showing more smokers and fewer non-smokers among the patients with lung cancer, and a steadily rising mortality as the amount of smoking increases. In the second type of inquiry, information has been obtained about the smoking habits of each member of a defined group in the population and the causes of the deaths occurring *subsequently* in the group have been ascertained. There have been two such investigations, one in the U.S.A. covering 190,000 men aged 50–69, and the other in this country covering over 40,000 men and women whose names appeared on the *Medical Register* of 1951. In both the results have been essentially the same. The investigation in this country, which has now been in progress for more than five years, has shown with regard to lung cancer in men:

(1) a higher mortality in smokers than in non-smokers;

(2) a higher mortality in heavy smokers than in light smokers;

(3) a higher mortality in cigarette smokers than in pipe smokers;

(4) a higher mortality in those who continued to smoke than in those who gave it up.

It follows that the highest mortalities were found among men who were continuing to smoke cigarettes, heavy smokers in this group having a death rate nearly 40 times the rate among non-smokers. Although no precise calculation can be made of the proportion of lifelong heavy cigarette smokers who will die of lung cancer, the evidence suggests that, at current death rates, it is likely to be of the order of 1 in 8, whereas the corresponding figure for non-smokers would be of the order of 1 in 300. The observation on the effect of giving up smoking is particularly important, since it indicates that men who cease to smoke, even in their early forties, may reduce their likelihood of developing the disease by at least one-half.

It should be noted that the excess of deaths from lung cancer among smokers was not compensated for by any corresponding reduction in the number of deaths from cancer of other sites in the body; in other words, there was a total incidence of cancer in the smoking groups in excess of the incidence that would have prevailed in the absence of smoking.

It will be apparent from what has been said that the evidence from the many inquiries in the last eight years, both in this country and abroad, has been uniformly in one direction and is now very considerable. It has been further strengthened recently by the observation from several sources that the extent of the relationship with smoking differs for different types of lung tumour which can be distinguished only by microscopical examination.

(b) Laboratory Evidence

From the physical and chemical point of view there is nothing inherently improbable in a connexion between smoking and lung cancer. Tobacco smoke consists largely of microscopic oily droplets held in suspension in air, and these droplets are of a suitable size to be taken into the lungs and retained there. Over a hundred constituents have so far been identified, and, among these, five substances have already been found which are known to be capable, in certain circumstances, of causing cancer in animals. Some workers have succeeded in producing tumours in animals by painting concentrated extracts of tobacco tar on the skin. Known carcinogens are present in tobacco smoke in very small amounts, however, and there is no certainty that such low concentrations could be harmful to human beings. Nevertheless, the finding of carcinogenic agents in tobacco smoke is an important step forward, in that it provides a rational basis for the hypothesis of causation.

Atmospheric Pollution as a Cause of Lung Cancer

It has been known for some years that mortality from lung cancer is greater in urban areas than in the countryside. This fact, together with the identification of carcinogenic substances in coal smoke and in motor vehicle exhausts, has led to the supposition that exposure to atmospheric pollution may be concerned with the increase in lung

*Cmd. 8387. H.M.S.O., 1951.

cancer. The role of atmospheric pollution is particularly difficult to investigate however, and the evidence is neither so consistent nor so extensive as that relating to tobacco smoking. On the one hand, no excess mortality from lung cancer has been observed in persons who would be especially exposed by the nature of their work to atmospheric pollution—for example transport workers, garage hands, and policemen. On the other hand, the results of a number of investigations have suggested that a relationship does exist between atmospheric pollution and lung cancer. Perhaps the best evidence for this relationship comes from studies of the small number of deaths from the disease among *non-smokers* in different types of residential district; in these studies higher death rates have been observed among non-smokers in large towns than among those in rural areas. On balance, it seems likely that atmospheric pollution plays some part in causing the disease, but a relatively minor one in comparison with cigarette smoking.

Assessment of the Evidence Relating to Smoking and Lung Cancer

Knowledge of the causation of lung cancer is still incomplete. Many factors other than tobacco smoking are undoubtedly capable of producing the disease; for example, at least five industrial causes have been recognized. Nevertheless, the evidence for an association between lung cancer and tobacco smoking has been steadily mounting throughout the past eight years, and it is significant that, during the whole of this period, the most critical examination has failed to invalidate the main conclusions drawn from it. It has indeed been suggested that the fundamental cause may be some common factor underlying both the tendency to tobacco smoking and to the development of lung cancer some 25 to 50 years later, but no evidence has been produced in support of this hypothesis.

In scientific work, as in the practical affairs of everyday life, conclusions have often to be founded on the most reasonable and probable explanation of the observed facts, and so far no adequate explanation for the large increase in the incidence of lung cancer has been advanced save that cigarette smoking is indeed the principal factor in the causation of the disease. The epidemiological evidence is now extensive and very detailed, and it follows a classical pattern upon which many advances in preventive medicine have been made in the past. It is clearly impossible to add to the evidence by means of an experiment in man. The Council are, however, supporting a substantial amount of laboratory research which may throw more light on the mechanism by which tobacco smoke and other suspected causative factors exert their effect, and which may thus eventually add to the degree of proof already attained as a result of studies of human populations. It must be emphasized, however, that negative results from work with animals cannot invalidate conclusions drawn from observations on man.

Conclusions

1. A very great increase has occurred during the past 25 years in the death rate from lung cancer in Great Britain and other countries.

2. A relatively small number of the total cases can be attributed to specific industrial hazards.

3. A proportion of cases, the exact extent of which cannot yet be defined, may be due to atmospheric pollution.

4. Evidence from many investigations in different countries indicates that a major part of the increase is associated with tobacco smoking, particularly in the form of cigarettes. In the opinion of the Council, the most reasonable interpretation of this evidence is that the relationship is one of direct cause and effect.

5. The identification of several carcinogenic substances in tobacco smoke provides a rational basis for such a causal relationship.

Medical Notes in Parliament

Smoking and Lung Cancer

Lieutenant-Colonel M. LIPTON (Brixton, Lab.) on November 19 asked the Minister of Health what further evidence he had received about the connexion between smoking and lung cancer, and what action he was taking thereon. Mr. D. CHAPMAN (Birmingham, Northfield, Lab.) asked a similar question. Mr. R. H. TURTON replied that a recent paper by Professor Bradford Hill and Dr. Doll (*British Medical Journal*, November 10, p. 1071) confirmed the statistical association between smoking and lung cancer. He would ensure that the public were kept informed of all relevant information as and when it became available, but he did not consider that a campaign would be at present appropriate.

Lieutenant-Colonel LIPTON suggested that evidence was piling up to show that smoking and lung cancer were connected, and the time had come for the Minister to sponsor a national campaign. Mr. TURTON said he was anxious to see that the important paper by Dr. Doll and Professor Hill got full publicity, but it was only confirming facts previously known. Dr. EDITH SUMMERSKILL (Warrington, Lab.), saying she presumed the Minister had read the paper in the *British Medical Journal* of November 10, asked how he could reconcile that with his previous statements suggesting that the facts had not been confirmed. Was it directly related to the advice he had received from the medical division of his department? Mr. TURTON said it was never the practice to disclose advice given by the medical division. What he had always said was that no scientific proof of the connexion had yet been found. It might be some time before that came. What was happening was that statistical evidence was piling up to show a connexion between lung cancer and heavy cigarette smoking.

Smoking and Lung Cancer
The Minister's Statement in the House of Commons

Squadron Leader Cooper (Ilford, South, C) asked the Minister of Health on February 12, 1954, " whether he has now received the advice of the Standing Advisory Committee on Cancer and Radiotherapy on the question of the relationship between smoking and lung cancer, and whether he will make a statement ".

Mr Macleod replied as follows :

Yes. The Standing Advisory Committee on Cancer and Radiotherapy have had this matter under consideration for three years. As a result of preliminary investigations, a panel under the chairmanship of the Government Actuary was set up in 1953 to enquire and report. I have now been advised by the Committee in the following terms:

Having considered the report of the panel under the chairmanship of the Government Actuary on the statistical evidence of an association between smoking and cancer of the lung, and having reviewed the other evidence available to them, the Committee are of opinion:

(1) It must be regarded as established that there is a relationship between smoking and cancer of the lung.

(2) Though there is a strong presumption that the relationship is causal, there is evidence that the relationship is not a simple one, since:

(a) the evidence in support of the presence in tobacco smoke of a carcinogenic agent causing cancer of the lung is not yet certain;

(b) the statistical evidence indicates that it is unlikely that the increase in the incidence of cancer of the lung is due entirely to increases in smoking;

(c) the difference in incidence between urban and rural areas and between different towns, suggests that other factors may be operating, e.g. atmospheric pollution, occupational risks.

(3) Although no immediate dramatic fall in death rates could be expected if smoking ceased, since the development of lung cancer may be the result of factors operating over many years, and although no reliable quantitative estimates can be made of the effect of smoking on the incidence of cancer of the lung, it is desirable that young people should be warned of the risks apparently attendant on excessive smoking. It would appear that the risk increases with the amount smoked, particularly of cigarettes.

I accept the Committee's view that the statistical evidence points to smoking as a factor in lung cancer, but I would draw attention to the fact that there is so far no firm evidence of the way in which smoking may cause lung cancer or of the extent to which it does so. Research into the causes of lung cancer has been pressed forward by the Government and by other agencies in view of the increase in the incidence of this disease and we must look to the results of its vigorous pursuit to determine future action.

I should also tell the House that before these recommendations were considered by Her Majesty's Government the tobacco companies had offered to give £250,000 for research. They have, on my advice, agreed to offer this money to the Medical Research Council.

The Ministry of Health's Press Statement

The Parliamentary Answer given by the Minister of Health is based on advice given to him by his Standing Advisory Committee on Cancer and Radiotherapy who for three years have been giving close consideration to the problem of the possible relationship between tobacco smoking and cancer of the lung. In view of the public interest and concern over this question it is, in the Minister's opinion, of very great importance that uninformed and alarmist conclusions should not be drawn from the Committee's advice and that the qualifications mentioned by the Committee in their advice should be fully realised.

In the autumn of 1950 an article by Dr Doll and Professor Bradford Hill in the *British Medical Journal* suggested that, on evidence arising from a statistical enquiry, there was a relationship between smoking and lung cancer. The Committee at that time considered that further evidence was needed. Late in 1952 a further article by Dr Doll and Professor Bradford Hill, which confirmed their earlier conclusions, reports from research workers in the USA, and the submission of arguments seeking to demonstrate that the relationship was not proved, led to a panel under the Chairmanship of the Government Actuary being asked in 1953 to enquire and report to the Standing Advisory Committee. The conclusions reached by this panel were considered by the Standing Advisory Committee who advised the Minister as in his Parliamentary Answer.

Although it can be taken as established that a relationship between smoking and lung cancer exists, it is important to realise that this relationship is not a simple matter, that a great deal of information and research is still required and it is not possible to draw final conclusions. The Minister considers that it would be helpful, in order that the matter can be looked at in proper perspective, to set down firstly what facts are known about the relationship, and secondly what must be regarded as speculative and unproved.

Facts Which are Known

The Minister would like to draw attention to the following facts, which are now well established:

(1) There has been an increase in deaths from lung cancer in this country which began about 1919 and has continued ever since. The increase is much greater in males than females. Between 1911 and 1919 the number of deaths from cancer of the lung was about 250 per year. The rise which began about 1919 can be illustrated from the figures of deaths for 1931 as compared with subsequent years. In 1931 the number of deaths attributed to lung cancer in England and Wales was 1,358 for males and 522 for females. These figures represented 5 per cent of all *cancer* deaths and 0.5 per cent of deaths from all causes in males, and 2 per cent of all *cancer* deaths and 0.2 per cent of deaths from all causes in females. In 1951 the number of deaths had risen to 11,166 males and 2,081 females. The latest figures available, for 1952, showed a further

increase in that 11,981 males and 2,237 females died from the disease. These represent 26 per cent of all cancer deaths and nearly 5 per cent of deaths from all causes in males, and 5 per cent of all cancer deaths and 1 per cent of deaths from all causes in females. The figures also show that the highest mortality rate from lung cancer in males occurred in the 65-74 age group, whereas in females the highest rate occurred in the 75 and over age group.

(2) Comparable increases have been reported in all countries from which reliable statistics are available. Factors such as the increasing age of the population and better diagnosis account for some of the rise but not the whole of it.

(3) Tobacco smoking plays some part in this increase. To use the language of statisticians, there is an "association".

(4) It is certain that tobacco smoking cannot be the only factor since the disease occurs in non-smokers. Not one but several factors or a combination of factors must be regarded as responsible.

(5) The disease is more prevalent in urban areas than rural and different parts of the country suffer more than others.

(6) No substance producing cancer of the lung has yet been specifically identified in tobacco smoke. Certain tars derived from tobacco smoke have produced skin cancers in mice but this is not considered as being conclusive evidence of the presence of a substance producing cancer of the lung.

So much is known.

Further Evidence Needed

The following matters must, however, remain speculative until further evidence comes to light:

(i) There is no firm evidence of the way in which smoking may cause lung cancer or of the extent to which it does so. All that can be said at present is that there is a presumption that it does, but the evidence does not permit us to say any more than that.

(ii) The difference in incidence between town and country and between different towns suggests that other factors should be taken into account such as atmospheric pollution or risks from particular occupations, but no evidence is available of the extent to which these factors operate.

(iii) Although the risk of contracting the disease appears to increase with the amount smoked, particularly of cigarettes, no reliable factual estimate can be made of the precise effect of smoking.

In view of this, it is not possible to come to a final and definite conclusion on this matter. A good deal of research and information is needed before anything more firm can be said. Many investigations are taking place both in this country and abroad which bear directly on the problem and also into the related problem of the effect of atmospheric pollution on health. The Ministry are in close touch with the Medical Research Council on this, and there will be no hesitation in launching further research if any particular line shows promise. Opportunity is taken of paying tribute to the valuable pioneer work of Dr Doll and Professor Bradford Hill and other workers who have given us what little information we have.

Ministry of Health,
February 12, 1954 *Savile Row W1*

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MINISTRY OF SOCIAL AFFAIRS AND PUBLIC HEALTH

Information Service
Press Notice No. 1233 The Hague, March 8th 1957

REPORT CONCERNING THE CONNECTION

BETWEEN SMOKING AND LUNG CANCER

At the request of the Director-General of Public Health the President of the National Health Council set up a commission some time ago to inform him on the subject whether there is a possible connection between smoking - particularly the smoking of cigarettes - and lung cancer. This commission issued their report a short time ago. In said report it is stated that the commission have come to the conviction, based on statistical grounds, that there is a connection between the two. The question whether there is a causal connection and if so, how far, can naturally not be answered by statistics, but the other possible factors, which were likewise studied in extenso, all proved to be less probable in this respect than smoking. Basing his opinion on this and other considerations the Minister of Social Affairs and Public Health opines that steps should be taken to restrain young people from smoking heavily and at an early age. In this way he hopes to achieve that with a following generation of adults the increase in the frequency of lung cancer, which is causing so much concern, will not continue.

The National Health Council has now called a new commission into existence, whose task it is to ascertain in what manner the young people can be approached and informed of the dangers connected with habitual smoking and to give directives, which can be followed by the authorities concerned.

(translation from the Netherlands)

J.A.M.A. 162: p. 1075, Nov. 10, 1956.

SWEDEN

Lung Cancer and Government Control of Tobacco.—Tobacco is controlled in Sweden by a government-created monopoly. The Prime Minister was recently asked whether the government was ready to cooperate in an educational campaign concerning the injurious effects of nicotine; whether it would support a scientific investigation of the health risks of tobacco in general and cigarettes in particular; whether the monopoly's advertisements in favor of cigarettes were in the interest of the community and, if not, whether the government would supervise the advertising methods used; and, finally, whether the government would consider any measures calculated to keep cigarettes away from school children and other minors. After consultation with the Ministry of Health, the Prime Minister replied that the nature and effects of tobacco are to be taught in the schools, not only in the teaching of biology and hygiene but also in special courses on temperance. Smoking is forbidden for pupils in schools and their immediate neighborhood, and both school medical officers and nurses are enjoined to promote propaganda against smoking. In 1955, certain bodies, including the School Medical Service and the Tobacco Monopoly, conferred over the steps to be taken to control smoking by school children. In December, 1955, the extent to which school children smoke was investigated. The Tobacco Monopoly has offered a prize of 11,000 crowns for the best essay on the subject, the winning essay to be earmarked for subsequent educational propaganda. The School Medical Service will also shortly cooperate with radio and other mediums in publicizing the findings of this investigation. The Tobacco Monopoly not only has given 300,000 crowns for cancer research but also has discontinued advertising its cigarettes. It is also discouraging the sale of cigarettes in the small quantities that facilitate their consumption by children. Tobacconists are being asked to discourage the sale of cigarettes to minors.

J.A.M.A. vol. 162, No. 10, Nov. 3, 1956

NORWAY

Lung Cancer and Smoking.—Two articles in *Tidsskrift for den norske lægeforening* for Aug. 15 approach lung cancer from somewhat different angles. Dr. Roald Opsahl emphasizes the comparative rarity of primary lung cancer in Norway between 1916 and 1935, during which period, of 2,005 cases of cancer verified by postmortem examinations, only 56 were primary cancer of the lung. These were equally divided between the sexes and included only two patients in whom there was a record of heavy smoking. After pointing out that the death rate from lung cancer has tripled in Norway in the last two decades, Opsahl censures the government on the score of its passive attitude toward cigarette smoking, from which it derives a certain revenue. He also deplores the failure of the National Cancer Association to wage a more vigorous campaign against the inhaling of cigarette smoke. The other article, by Dr. Einar Pedersen, draws attention to the fact that Norway and Denmark are at present the only countries in the world in which a scheme has been adopted for the systematic review of death certificates in order to examine the diagnoses of lung cancer. Thus, in 1953 there were 209 cases originally so diagnosed in Norway, but, on review, 24 of these diagnoses had to be altered. The author further states that in 1930 only 0.9% of all the deaths from cancer in Norway were due to cancer of the respiratory tract, whereas the corresponding figure for 1952 was 3.6%. Up to 1954, the rise seems to have been limited almost exclusively to the urban male population, and for this group the rise has been and still is great. There is also a tendency for the peaks of the age curves for successive years to shift toward older age groups.

EXHIBIT 14A

A Reader's Digest

REPRINT

THE FACTS BEHIND FILTER-TIP CIGARETTES

By Lois Mattox Miller and James Monahan

July 1957

THE READER'S DIGEST • PLEASANTVILLE, NEW YORK

Is one filter better than another? What sort of "protection" do they give? Is it true that low-grade tobaccos are being used more and more widely? Here are the startling findings of a thorough survey

The Facts Behind Filter-Tip Cigarettes

By Lois Mattox Miller and James Monahan

Less than 24 hours before the leaders of the tobacco trade gathered in Chicago for the annual convention of the National Association of Tobacco Distributors last March, newspaper headlines carried what seemed like sad news for cigarette smokers. The Study Group on Smoking and Health, a panel of medical experts organized* to settle the "cigarette controversy," had found that "the sum total of scientific evidence establishes beyond reasonable doubt" that cigarette smoking is a cause of the rapidly increasing incidence of lung cancer.

The tobacco men were undismayed. "This will blow over like other blasts," declared the chief executive of the NATD. Said a Kansas City wholesaler: "People enjoy smoking and aren't going to quit. But they *will* go to filters."

The tobacco men's confidence was based on experience. Since 1953-54,

*At the request of the American Cancer Society, the American Heart Association, the National Cancer Institute and the National Heart Institute.

when the "cancer scare" caused a slump in the cigarette market, sales have bounced back amazingly. This year they seem to be headed for an all-time high. *The filter-tip cigarette has been the salvation of the tobacco industry.*

Prior to 1954 less than ten percent of the cigarettes sold were filter-tips. Filter-tip sales climbed to 30 percent in 1956, and some wholesalers say they passed the 50 percent mark early this year. "By the end of 1957," a major distributor told *The Wall Street Journal,* "I'd say that the filters will command 75 percent of the market."

The reason for this trend was revealed in a survey of smoking habits recently completed by the Sloan-Kettering Institute. More than 70 percent of the smokers who had switched from plain to filter-tip cigarettes said they had done so for "health protection."

This raises some important questions. How much "health protection" do filter-tips provide? Specifically, *how much less tar* (and nicotine) does the smoker get through his present filter-tip than he got from the plain-tip brand he formerly smoked?*

*"The greater the tar reduction in the smoke," says Dr. Ernest L. Wynder of the Sloan-Kettering Institute, "the greater will be the reduction in the risk of lung cancer." As for nicotine, Dr. Dorothy Roth, of the Mayo Foundation for Medical Education and Research, says, "Apparently the content of nicotine in a cigarette must be decreased more than 60 percent before the vascular effects [pulse rate, blood pressure, etc.] produced during smoking will be greatly altered."

The average smoker cannot answer these questions. There is evidence that the public thinks a filter is a filter, that one tip is as good as another. Doctors, who may be asked for advice, are no better off. Whereas foods and drugs can be selected on a basis of known ingredients, no accurate information exists to enable smokers to choose a brand of cigarette on a basis of the amount of tar and nicotine they will get in the smoke.

To meet this need for specific information, The Reader's Digest asked a leading testing laboratory to make scientific determination of the tar and nicotine in the mainstream smoke of popular brands. The laboratory purchased cigarettes on the open market in cities across the country. The cigarettes were smoked in a standard smoking apparatus (originally designed by the American Tobacco Co.) which automatically puffs each cigarette in a manner that approximates human smoking as nearly as possible. Particular care was taken to recover all the tar that a human might inhale in the smoke. After each run, chemical determinations were made of the tar and nicotine content according to approved laboratory methods.

The results of the tests are presented on the opposite page. Let's see how smokers who may have switched from a plain-tip to a filter-tip have fared.

Until a few years ago, the lion's share (between 70 and 80 percent) of the cigarette market belonged to

Here are the scientific laboratory determinations of the amount of tar and nicotine (stated in milligrams per cigarette) contained in the mainstream smoke of the most popular brands of cigarettes. The analyses were made for The Reader's Digest by Foster D. Snell, Inc., consulting chemists and engineers of New York City, selected on the basis of a 37-year record of outstanding service in the fields of food, drug, chemical and industrial research. Technical details concerning the laboratory methods used in these tests are available upon request.

How Much Tar?

PLAIN-TIPS			FILTER-TIPS		
Brand	Size	Tar (mg)	Brand	Size	Tar (mg)
Cavalier	King	42.8	Old Gold	King	39.0
Old Gold	King	41.7	L & M	King	38.5
Chesterfield	King	41.1	Hit Parade	King	36.3
Philip Morris	King	40.8	Marlboro	King	34.4
Raleigh	King	39.3	Parliament	King	34.1
Pall Mall	King	38.6	Salem	King	33.7
Tareyton	King	36.6	Winston	King	32.6
Philip Morris	Regular	35.3	L & M	Regular	31.1
Chesterfield	Regular	32.7	Kool	King	30.4
Lucky Strike	Regular	31.5	Kent	King	30.4
Kool	Regular	31.3	Viceroy	King	30.2
Camel	Regular	31.0	Tareyton	King	27.1
Old Gold	Regular	30.9	Kent	Regular	25.6

How Much Nicotine?

PLAIN-TIPS			FILTER-TIPS		
Brand	Size	Nicotine (mg)	Brand	Size	Nicotine (mg)
Cavalier	King	3.1	Old Gold	King	3.1
Old Gold	King	3.0	L & M	King	3.1
Camel	Regular	2.8	Hit Parade	King	2.8
Philip Morris	King	2.8	Parliament	King	2.7
Chesterfield	King	2.6	Winston	King	2.6
Raleigh	King	2.5	Salem	King	2.5
Chesterfield	Regular	2.4	Marlboro	King	2.4
Old Gold	Regular	2.4	Viceroy	King	2.4
Kool	Regular	2.4	L & M	Regular	2.4
Pall Mall	King	2.4	Kool	King	2.3
Philip Morris	Regular	2.4	Kent	King	2.2
Tareyton	King	2.2	Tareyton	King	1.9
Lucky Strike	Regular	2.1	Kent	Regular	1.8

the regular-size "Big Four"—Camel, Lucky Strike, Chesterfield and Philip Morris. These are also the brands that have lost most customers in the trend toward filters. So, let's first assume that the smokers who abandoned these old favorites switched to one of the four filter-tip brands that the tests show to have the *lowest tar content*—Kent, Tareyton, Kool, Viceroy. Consider these hypothetical shifts, and what the smoker gained:

¶ From Camel to Kent (regular): 17 percent less tar, 36 percent less nicotine.

¶ From Lucky Strike to Tareyton: 14 percent less tar, 10 percent less nicotine.

¶ From Chesterfield (regular) to Kool: seven percent less tar, four percent less nicotine.

¶ From Philip Morris (regular) to Viceroy: 14 percent less tar, same amount of nicotine.

Medical authorities say that these tar reductions (ranging from seven to 17 percent) are too small to be really significant in terms of "health protection." However, the big shift in brands hasn't been along even this mildly favorable line. Of the four filter-tips that give the least tar, only Viceroy has become a big seller.

The other big favorites seem to be those that rank among the *highest* in both tar and nicotine, according to the tests: Winston, L & M, Marlboro and Hit Parade. These brands are produced by the makers of the old "Big Four."

Let's suppose, then, that the plain-tip smoker switched to a filter-tip made by the same company. Only the smoker of Philip Morris (regular size) who went to Marlboro gained even a slight reduction (about 2½ percent) in tar intake. Consider what happened to the others:

¶ From Camel to Winston (both R. J. Reynolds): five percent *more* tar, but seven percent *less* nicotine.

¶ From Lucky Strike to Hit Parade (both American Tobacco): 15 percent *more* tar, 33 percent *more* nicotine.

¶ From Chesterfield regular to L & M king (both Liggett & Myers): 18 percent *more* tar, 29 percent *more* nicotine.

Now some people may argue that these shifts are all from regular size plain-tips to king-size filters—implying that there is more tobacco (hence more tar and nicotine) in the filter kings. Don't be fooled. According to the Department of Agriculture, *there is less tobacco in most king-size filter-tip cigarettes than in the shorter plain-tip cigarette!*

Last year a House-Senate subcommittee investigating the tobacco industry found that in 1955-56 the cigarette companies had used *less* tobacco leaf to produce *more* cigarettes than in the preceding year. "How can you account for this?" the subcommittee asked John C. Whitaker, chairman of R. J. Reynolds Tobacco Co.

"Our regular-size brands are decreasing in sales," Mr. Whitaker explained. "They are being replaced by the filter type, which is a smaller

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cigarette and requires less tobacco. A Camel has eight percent more tobacco than a Winston, for instance. It [Camel] is larger."

Now we begin to see what's wrong with filtration. The old Chesterfield, Lucky Strike and Camel, for example, are all comparatively low (approximately 31 mg.) in tar. Placing a filter-tip on any of these cigarettes could reduce the tar still more. But no! Instead, the tobacco companies offer you a deceptively longer cigarette with *less* tobacco but *more* tar, and—in the case of L & M King or Hit Parade—a whopping 29 or 33 percent more nicotine. How can this be?

To find the answer to this question, the Digest sent investigators to talk to tobacco growers, warehousemen, auctioneers, cigarette manufacturers, government officials. Here are their findings.

Back in 1953, when filter-tip cigarettes were in their infancy, a newcomer called Kent claimed very high tar-filtration. The American Medical Association ran tests on three filter-tip brands, and found that one (designated as B-1) did indeed remove about 55 percent of the tar. Cigarette B-1 was Kent.

At that very moment, however, Kent was having trouble selling at the tobacco counters. The filter-tip was *too* good. It not only filtered out a lot of tar but most of the tobacco taste as well!

Kent quickly modified its tip to give the smoker more "taste satisfaction"—and, of course, more tar and nicotine. The A.M.A. tested the modified filter and found that Kent filtration had dropped from 55 to 44 percent. Later, Kent loosened up its filter even more in an effort to win customers, but the brand (still one of the best filters—see chart) never made the big-sale league.

Other cigarette makers, however, profited from the Kent experience. They went all-out to give the smoker "satisfaction" at any cost. Not only did they adopt filters which, in most cases, are little more than mouthpieces; they also began loading the blends with heavier tobacco.

Cigarette manufacturers used to brag about their high-quality tobacco and superior blends. Even today you can find these proud claims on the Camel and Chesterfield packages, and LS/MFT still reminds us that "Lucky Strike Means Fine Tobacco." But examine the labels on the popular filter-tip packages. On the majority of brands, the word tobacco is never mentioned unless it appears in the firm's name.

Whatever the reason for this peculiar omission, one thing is clear: the tobacco in most popular filter-tip brands today is not high-grade.

During the past few years there has been unrest among the Eastern tobacco growers, particularly in North Carolina, who have always produced the light, flue-cured, premium-priced tobacco for U. S. cigarettes. Sales to cigarette companies have been declining steadily. More and more of the best-quality leaf has

been stored under the government's price-support program. This year acreage of mild, flue-cured tobacco was cut back 20 percent.

Fred Royster, president of the Bright Belt Warehouse Association, Henderson, N. C., told the Congressional subcommittee that the marked change in the tobacco-buying pattern of some cigarette companies became noticeable in 1953. Buyers who formerly demanded the best flue-cured leaf began buying inferior grades. This, said Mr. Royster, "caused a tremendous price increase in the inferior grades."

Mr. Royster was asked, "Does this pattern lead you to believe that lower-quality tobacco is probably being placed in filter-type cigarettes?"

"Yes, sir," he replied. "I think that is generally accepted in the tobacco industry."

Meanwhile, the tobacco growers in the "burley belt" struck a bonanza. Last January *The Wall Street Journal* explained why. "Burley is heavier and stronger flavored than the light-colored, flue-cured leaf," the *Journal* stated. "Cigarette makers have found that the smoke flavor from the mild, flue-cured tobacco could scarcely penetrate the filter in the increasingly popular filter-tip cigarettes. Their solution: mix in more of the stronger leaf to kick a satisfactory taste through the filter."

Tobacco chemists report that, on an average, burley will yield in the mainstream smoke about 16 percent more tar and 40 percent more nicotine than flue-cured tobacco. But that isn't all. The best-grade burley comes from the leaves that grow near the bottom of the plant. There used to be so little demand for the strong, harsh top leaves that they sold for about 20 cents per pound. Today the strong, harsh "tips" are so much in demand that they sell for 62 cents a pound, almost on a par with best-grade burley.

"I can remember when I wouldn't have even brought that stuff to market," one Kentucky burley grower told *The Wall Street Journal*. "I'd have thrown it away."

This trend toward inferior tobacco has even the burley growers confused. "It seems to me that this is poor business," John Berry, vice-president of the Burley Tobacco Growers Coöperative Association, told the subcommittee. "Such practices ultimately will bring into bad repute the manufactured cigarette and the people who manufacture it."

Mr. Berry told the Congressional investigators that burley "scrap," which sold for 18 cents per pound in 1954, had gone to 41 cents in 1955. "All of a sudden that came to be very attractive tobacco," he said.

Where is this scrap going? How are the cigarette makers using it?

At the urging of the Congressional subcommittee, reluctant tobacco men discussed another innovation. It is called "homogenized" or reconstituted tobacco.

The homogenizing process, which is also used to produce cigar binders, is supposed to be a closely guarded trade secret. Most tobacco men re-

fuse to discuss it. But the details may be obtained merely by consulting the disclosures filed in the U. S. Patent Office. Most of the patents are assigned to the American Machine & Foundry Co., which produces equipment for the tobacco industry.

Traditionally, in preparing tobacco for cigarettes, the woody stem and midrib are removed from the leaf and discarded along with the scrap or damaged leaves. In the homogenizing process the *whole* leaves, plus the scrap, are reduced to a fine powder, moistened and rolled into thin, plastic-like "sheet tobacco," which is then shredded for the cigarette blend.

Most of the major tobacco companies deny that they are now using homogenized tobacco, although all admit they are experimenting with it. ("After all," says one tobacco man, "competition may force us to use it some day.") The notable exception is R. J. Reynolds (Camel, Winston, Cavalier), whose chairman, John Whitaker, told the Congressional subcommittee: "We are using it in all cigarettes."

Asked whether a higher percentage of reconstituted tobacco is used in Reynolds' filter-tips than in the company's plain-tips, Mr. Whitaker refused to answer. "You are getting into trade secrets now," he said. Later, Senator W. Kerr Scott of North Carolina remarked: "We have not obtained the information we need. It is apparent that 'trade secrets' are being used as the Fifth Amendment."

From responsible sources the Digest has learned *the* "trade secret" that probably will interest smokers most: *It is entirely possible to manufacture filter tips much more efficient than any now on the market.*

The improved filters would be made from essentially the same materials now being used: that is, cellulose acetate plus, possibly, some form of powdery filler. They would cost no more to produce. They would be as "easy on the draw" as the present filter tips. Yet, unlike present filters (which filter as little as ten percent), the improved tips would filter 40 percent or more of the tar from the mainstream smoke.

Dr. Wynder, of Sloan-Kettering, says: "Such a filter, placed on a regular-size cigarette which normally yields 30 milligrams of tar in its smoke, would reduce the smoker's tar exposure to about 18 milligrams. A reduction to that level, as shown by both animal experiments and human statistical studies, would be a significant reduction in cancer risk."

Why aren't these improved filter tips available? What reasons do cigarette companies give for not adopting them?

Watch for a second article, in the August Reader's Digest, which will answer these and other significant questions.

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EXHIBIT 14B

A Reader's Digest Report to Consumers

WANTED

—*And Available*—

Filter-Tips That Really Filter

The Second of Two Articles

By

Lois Mattox Miller and James Monahan

Based on Laboratory Tests of Filter-Tip Cigarettes

SECRECY is the rule in the cigarette industry. Research is guarded jealously. Developments immediately become highly classified "trade secrets." What goes into, or comes out of a cigarette is considered nobody's business but the manufacturer's. Questioned about such matters, the average tobacco man either freezes up or explodes.

When the Digest began investigating reports that it was possible to manufacture filter-tips more effective than those on the market, inquiries met with polite rebuffs. The cigarette companies professed to be engaged actively in filtration research; but they also declared that, thus far, research had produced nothing better than their present filters.

However, reliable reports came from scientists in the tobacco industry that better filter-tips were prac-

See "The Facts Behind Filter-Tip Cigarettes," The Reader's Digest, July, '57. 43

tical and available. Laboratory men stated confidentially that they had actually smoked and tested standard-brand cigarettes equipped with experimental filter-tips, and that these filters gave much higher tar-filtration as well as satisfactory tobacco flavor and an easy draw.

Then why weren't these improved filters in commercial use?

The most plausible answer was that smokers thought all filters were alike, and chose brands solely on a basis of taste. "The brands that sell best are those with the least effective filters and the strongest tobacco," said one tobacco man. "That seems to be what the public wants, so that is what the public is getting. Improved filtration might even be unprofitable."

While this explanation may be valid, it still leaves the question whether smokers will persist in considering all filter-tips alike *when they know the facts*—facts which have not been available until now.

There is really no mystery about the filtration of cigarette smoke. An average puff of smoke from an unfiltered cigarette contains more than *12 billion* tiny, spherical, semi-solid particles, mainly tar and nicotine. These particles give tobacco smoke its characteristic aroma and flavor. They also represent the stimulants, irritants and the suspected cancer-causing agents responsible for the fundamental tobacco-health problem.

Effective filtration would remove from the smoke enough of these particles to reduce the health risk to a minimum, while passing along to the smoker enough of them to provide a flavorsome smoke. Where to draw the line is still uncertain. But Dr. Ernest L. Wynder of the Sloan-Kettering Institute states that, for practical purposes, a filter-tip capable of filtering out 40 percent of the tar would be a step in the right direction. (Assuming, of course, that the manufacturer does not defeat the purpose by loading the cigarette with stronger tobacco.)

"Such a filter-tip," Dr. Wynder says, "placed on a regular-size cigarette which normally yields 30 milligrams of tar in its smoke, would reduce the smoker's tar exposure to about 18 milligrams. A reduction to that level, as shown both by animal experiments and human statistical studies, would be a significant reduction in cancer risk."

The modern filter-tip is essentially a complex maze of microscopic traps or barriers designed to prevent the passage of a certain percentage of the smoke particles. To do this effectively, however, the filter must contain some elements at least as small as the tiny particles it seeks to stop. Otherwise, the particles will slip through the interstices and defeat the purpose.

One tobacco scientist offers this illustration: "Suppose you had a jumbled pile of telegraph poles. You could drop baseballs into that pile and a large percentage of them might find their way through the intervening spaces to the bottom.

But the spaces in a similar pile of fence posts would probably be small enough to stop baseballs, and yet large enough to let golf balls pass. Similarly, a pile of jackstraws or toothpicks would probably trap small beads. Reduce this principle to the microscopic level and you have the basic problem of smoke-particle filtration."

What material will provide such a maze of microscopic "jackstraws" capable of blocking smoke particles so small that they are measured in fractions of *microns?* After much research, the most suitable substance appears to be a synthetic fiber — *cellulose acetate.*

With the exception of Tareyton (which uses a special type of paper impregnated with activated charcoal), the filter-tips of the popular brands are made from this same basic material. Well over 95 percent of the cellulose acetate comes from the same producer, Tennessee Eastman (a subsidiary of Eastman Kodak), located in Kingsport, Tenn. Tennessee Eastman ships the material to the cigarette factories in a wide, soft ribbon called "tow."

But Tennessee Eastman manufactures the cellulose acetate according to each cigarette company's own specifications. The synthetic filaments can be made in any specified degree of fineness or coarseness. They can be packed tightly or loosely. They can be crimped, twisted, packed parallel or at random. And even the finest hairlike filament can also be shaped or grooved in different ways to increase the particle-trapping efficiency.

In the cigarette factory, the tow is treated with a moist "sizing," shaped to a specified diameter, cut into short plugs and fitted into the mouthpiece by the cigarette-making machinery. But, before this final processing, the cigarette maker can (if he chooses) improve filtration even more by incorporating in the tow certain "fillers" devised by his own laboratory.

The best filter-tips now on the market are ingenious variations or combinations of these unlimited possibilities. The least effective filters are merely haphazard bundles of synthetic fibers.

The laboratory tests reported in The Reader's Digest last month were nearly complete when word came from a reliable source in the tobacco industry that Lorillard had not only developed a new filter-tip for the Kent cigarette but was modifying machinery for immediate production. Lorillard at first denied the report, then admitted that the new Kent would be on the market by midsummer. After some objections, the company finally agreed to provide production-line cigarettes (not laboratory samples) of the new type for testing by an independent laboratory. Lorillard also agreed to tell how the new filter-tip was developed.

The Kent case is important because (1) it spans the modern filter-tip era from 1951 to date; (2) it illustrates the problems and pitfalls

of filter-tip technology; and (3) it demonstrates what a cigarette maker can accomplish if he is sincerely seeking an improved product rather than another advertising gimmick.

In 1951, two years before headlines linked cigarette smoking with lung cancer, Lorillard's general sales manager, Lewis Gruber, sensed a growing public demand for a mild, filtered cigarette. At the time, there were two filter-tips on the market: the original Parliament (since renamed Benson & Hedges), with a cardboard mouthpiece containing a tuft of cotton; and Viceroy, which at that time had a filter-tip made of treated crepe paper.

Dr. Harris B. Parmele, Lorillard's research director, had barely begun the search for a more suitable filter material when he got what seemed like a lucky break. The Atomic Energy Commission declassified a technical report describing an aerosol filter containing crocidolite (a kind of bluish asbestos), which was used to remove radioactive particles from the air in AEC installations. Lorillard developed a filter-tip made of cotton fibers containing crocidolite, and the Kent cigarette made its debut early in 1952.

But Kent paid a penalty for being a pioneer in filter-tip technology. The original filter-tip proved too efficient to satisfy the taste of the average smoker, and had to be modified several times. Meanwhile, the battle of the filter-tips began in earnest. Viceroy switched to cellulose acetate; Liggett & Myers introduced L&M, and Reynolds followed with Winston. Soon the tobacco counters were crowded with filter-tip brands.

Ironically, of the four brands that made the best showing for tar filtration in the Digest tests published last month, only Viceroy has become a substantial seller (over 25 billion cigarettes last year). Tareyton, Kent and Kool all sell well below the four-billion level. By contrast, Winston sold 34 billion cigarettes in 1956.

Until last year, Kent was handicapped by its premium price. But Lorillard claims that (unlike some of its competitors) it steadfastly resisted the temptation to bid for popular appeal by "spiking" the Kent blend with stronger tobacco.

In August 1956, Lewis Gruber, original sponsor of the embattled Kent, became president of Lorillard. He promptly cut Kent's premium price to the popular level, and started production plans for the new Kent filter developed in Lorillard's research laboratory.

Kent's new "Micronite" filter * is made of fine cellulose acetate developed especially for Lorillard by Tennessee Eastman. While the Eastman laboratories in Tennessee are the center of much hush-hush filter-tip research, Lorillard claims that this development is essentially its own.

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* "Micronite" is a Lorillard trademark that has been applied to all Kent filter-tips, regardless of their construction. The cigarettes with the "NEW EXCLUSIVE MICRONITE FILTER" (so designated on the face of the package) went on the retail market early in July.

BY DEVIOUS means proofs of the first Reader's Digest article on filter-tips were obtained by representatives of the tobacco industry well in advance of publication of the July issue. Perhaps as a result, when final inquiries were made prior to the preparation of this second article, there was a sudden and surprising interest among cigarette manufacturers in the production of better filter-tips.

"For competitive reasons, I cannot disclose our plans in detail," wrote Paul M. Hahn, president of The American Tobacco Co. "I can, however, tell you that our company is developing a completely new filter which we are planning to place on one of our brands in the near future."

The president of another company (name omitted by request) stated: "Confidentially, we do hope to have an improved filter-tip on the market by the end of the year."

New filter-tips will be tested and compared when they appear on the retail market, and a report to consumers will appear in the Digest.

The secret of the new tip's higher filtration, the company claims, lies in the tiny natural fibers (so small that, to the naked eye, the stuff looks like fine powder) added to the cellulose-acetate tow after it reaches the Lorillard plant in Greensboro, N. C.

The laboratory of Foster D. Snell, Inc., has made determinations of the tar and nicotine in the mainstream smoke of the new cigarettes and has reported to the Digest the following results: "Kent, king-size, with new filter-tip, 23.4 milligrams of tar, 1.5 milligrams of nicotine; Kent, regular size, with new filter-tip, 19.7 milligrams of tar, 1.1 milligrams of nicotine."

¶ This represents a 23 percent improvement in tar filtration over the Kent cigarettes tested and reported on in the July issue of the Digest.

¶ Compared with some of the filter-kings that ranked *lowest, average,* and *high* in tar (according to the Digest tests), the new Kent king yields 14 percent less tar than Tareyton; 23 percent less than Viceroy; 28 percent less than Winston; 32 percent less than Marlboro; 36 percent less than Hit Parade; 39 percent less than L&M; 40 percent less than Old Gold.

¶ Compared with regular-size unfiltered brands, the new Kent regular-size yields 36 percent less tar than Camel and Old Gold; 37 percent less than Lucky Strike; 40 percent less than Chesterfield; 44 percent less than Philip Morris.

The new Kent may not be the best filter-tip possible. (Lorillard says that research will continue, and that further improvement will be made if developments warrant.) But it is a big step in the right direction. Perhaps competition will compel other cigarette companies to seek filters even better than the new Kent. *(See box above.)*

On June 4, 1957, the final report on the American Cancer Society's 44-month study of the smoking habits of 188,000 men 50 to 70 years old presented evidence of what the American Medical Association called "a spectacular relationship" between cigarette smoking and lung cancer. But the report also showed that *death rates from all causes combined* rise with the number of cigarettes smoked daily. The death rate from coronary artery disease was 70 percent higher among cigarette smokers than among non-smokers. The suspect here is not tar but nicotine.

While the new Kents, according to the tests, give the smoker from 20 to 50 percent less nicotine than the other filter-tip brands tested, this reduction may still fall short of the ideal. Some medical authorities say that the nicotine in the smoke from one cigarette may have to be reduced to about one-half a milligram before nicotine's measurable effects on the cardiovascular system are decreased significantly.

However, there is still much to be learned about the exact role nicotine plays in the tobacco-health problem. The Study Group on Smoking and Health, which declared "scientific evidence establishes beyond reasonable doubt" that cigarette smoking is a causative factor in lung cancer, also stated that "in the few reports available there is no convincing biological or clinical evidence to indicate that smoking *per se* has a causative role in cardiovascular disease."

The Study Group urged an extensive research program to clarify the relationship between smoking and heart and vascular diseases. Until this relationship is clarified, no one can decide to what point the nicotine in tobacco smoke should be reduced.

Meanwhile, we will be hearing a lot more about the filter-tips. In 1955 the Federal Trade Commission quietly informed the tobacco industry that it was opposed to all health claims and comparisons of filter-tip efficiency "when it has not been established by competent scientific proof . . . that the claim is true. . . ." The industry fell into line.

Lately there have been signs that the cigarette advertisers are growing restless. The Federal Trade Commission may soon be facing a rash of new "claims" that it is powerless to deal with—except by prolonged litigation. Because tobacco is not covered specifically by the Food and Drug Act, the Federal Trade Commission Act, or any similar statute, the FTC cannot seek a temporary injunction to suspend allegedly deceptive or fraudulent advertising while the case is still in litigation. Some of the Federal Trade Commission's legal actions against cigarette advertisers have dragged on for as long as five years; by the time a final verdict was handed down the advertising in question had become ancient history.

To deal realistically with this problem, Congress may have to pass legislation that will:

1. Require the cigarette manufacturer to state on the label of each brand: "Laboratory tests have determined that the average cigarette in this package will contain in its mainstream smoke — mg. of tar, and — mg. of nicotine." (The same law should require the industry to standardize laboratory methods of determining the contents of cigarette smoke, and to adhere to those standards.)

2. Require the manufacturer to correct the data on the label whenever any change is made in the brand's tobacco, blending process or filter-tip construction.

3. Require the manufacturer to confine his advertising claims for each brand of cigarettes to statements which he can support with competent scientific proof.

4. Enable the Federal Trade Commission (or other federal agency) to seek a temporary injunction from a federal court in order to stop promptly (and while the issue is in litigation) the dissemination of false or misleading information in connection with the advertising or selling of any brand of cigarettes.

Such practical regulations are now being applied—fairly and effectively—to foods, drugs, cosmetics, alcoholic beverages, flammable fabrics, etc. Tobacco products are directly linked to the public health. And the tobacco industry has a long record of deceptive or irresponsible advertising. Why this industry should remain beyond the reach of effective regulation is a question that probably no one but a tobacco man would undertake to answer.

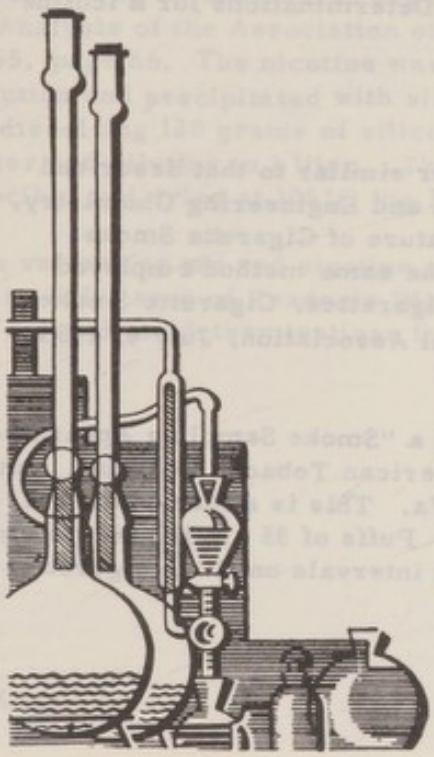
So far as filtration is concerned, proper labeling — backed by realistic enforcement—would draw the line between filter-tips that filter and those that are merely mouthpieces. That, at least, would enable the cigarette smoker to know that he is getting the best protection that a filter-tip can offer at the present time.

EXHIBIT 14C

Report

TO
WHOM IT MAY CONCERN
ON

METHODOLOGY EMPLOYED IN DETERMINATION
OF TAR AND NICOTINE CONTENT OF
CIGARETTE SMOKE FOR READER'S DIGEST



from

FOSTER D. SNELL, INC.

29 WEST 15th STREET, NEW YORK 11, N. Y.

WATKINS 4-8800

CONSULTING CHEMISTS • ENGINEERS

TODAY'S RESEARCH PAYS TOMORROW'S DIVIDENDS

FOSTER D. SNELL, INC.
29 WEST 15TH STREET, NEW YORK 11, N. Y.
CONSULTING CHEMISTS
ENGINEERS

METHODS FOR DETERMINING

TAR AND NICOTINE CONTENT OF CIGARETTE SMOKE

The following are excerpts from our report to Reader's Digest.

The cigarettes were conditioned at 77°F (25°C) and 50 per cent relative humidity. The average weight of the cigarettes was determined and only those cigarettes were selected for test which were fully packed and did not deviate from the average weight by more than 20 milligrams. A mark was made on each cigarette to be smoked 23 millimeters from the mouth-piece end of the cigarettes and the cigarettes were smoked to this butt length.

Five cigarettes were smoked for each single determination. Determinations for tars were carried out in triplicate. Determinations for nicotine were carried out in duplicate.

Smoking

Cigarette smoking was carried out in a manner similar to that described by Bradford, Harlan and Hanmer in Industrial and Engineering Chemistry, vol. 28 (1936) page 836-839 under the title "Nature of Cigarette Smoke. Technic of Experimental Smoking". This is the same method employed by W. Wolman and described in "A Study of Cigarettes, Cigarette Smoke and Filters", Journal of the American Medical Association, July 4, 1953, Pages 917-920.

Smoking of the cigarettes was performed with a "Smoke Sampling Apparatus" designed by the Research Laboratories of American Tobacco Co., Inc., and manufactured by Phipps & Bird, Richmond, Va. This is a four-place solenoid-actuated mechanical smoking machine. Puffs of 35 milliliter volume and 2 seconds duration were taken at 1 minute intervals until the cigarettes were burned to the predetermined butt length.

The smoke passed through an absorption train consisting of a Kjeldahl flask containing 1 ml. of 0.5N hydrochloric acid and 10 ml. of alcohol and then through 2 bubble traps. The first bubbler contained 5 ml. of 0.5N alcoholic hydrochloric acid. The second bubbler contained 5 ml. of 0.5N aqueous hydrochloric acid.

At the conclusion of the run the smoke was allowed to settle for 20 minutes. All portions of the smoke collection train were washed into the flask with a minimum of hot water and alcohol.

Determination of Tar Content

The tar content of the smoke was determined by condensing and collecting it, at ambient room temperature, in the manner described above under Smoking. The apparatus train was washed with alcohol and water to remove tars condensed in the train and all washings placed in the tar condensate flask.

These were transferred to a beaker for evaporation of the solvent and the quantity of tars was determined by complete evaporation of the solvent and weighing of the residue.

Determination of Nicotine Content

Nicotine was determined in accordance with the method described in Methods of Analysis of the Association of Official Agricultural Chemists, Eighth Edition, 1955, page 66. The nicotine was steam distilled into dilute hydrochloric acid solution and precipitated with silicotungstic acid. The reagent was prepared by dissolving 120 grams of silicotungstic acid ($4H_2O.SiO_2.12WO_3.22H_2O$) in water and diluting to 1 liter. The precipitate was filtered through a Gooch crucible and dried at 105°C for 3 hours.

The values for tar and nicotine content of cigarette smoke shown on page 35 of the July issue of Reader's Digest are the averages of nine determinations for tar and six determinations for nicotine.

Respectfully submitted,

FOSTER D. SNELL, INC.

Cyril S. Kimball

Cyril S. Kimball
Executive Vice-President

CSK:bf

EXHIBIT 15A

Exhibit 16A

CIGARETTES

What CU's tests showed . . . the industry and



The thoughtful individual above is an average cigarette smoker. He is contemplating twelve piles of cigarettes, each containing an average month's supply. Although there are no precise statistics on cigarette consumption in the U. S., an average of the estimates indicates that nearly half of the adult population smokes cigarettes, each smoker burning up 24 cigarettes a day, 730 a month, 8760 a year. In the piles above are 27 brands which account for 95% of domestic cigarette sales and represent all of the leading cigarette manufacturers.

its advertising...and: how harmful are they?

The biggest news in the cigarette industry is the fast-growing popularity of king-size and filter-tip brands, both widely promoted as offering the smoker "protection" against nicotine and tar. The biggest news from CU's tests of cigarettes is that a smoker actually gets more nicotine and no less of the tars from most filter-tip and king-size cigarettes than he does from the regular brands. The new filter-tip *Kent* cigarettes, CU's tests showed, are an exception. *Kent* smoke contained about half as much nicotine and less tars than most regular brands. Most filter tips were ineffective, the tests showed, apparently because an equal length of tobacco in the butt filters out more nicotine and no less tar than the usual cotton or paper filter. In king-size brands, the extra tobacco does reduce the amount of nicotine and tar the smoker takes in, but only if he leaves the extra length of the cigarette unsmoked. If he smokes to the same size butt as he does with a regular brand, with most of the king-size brands he inhales more nicotine and tar per cigarette. Again there is an exception; *Embassy* gave no more nicotine than a regular cigarette even when so smoked.

These are among the results from CU's tests of 27 brands of cigarettes accounting for 95% of domestic sales and representing all leading companies.

CU also found that, among the leading popular brands, there were no significant differences in the amount of nicotine and tar in their smoke; and that all of the leading popular brands had more nicotine in their smoke than several other American-made cigarettes, including one brand (*John Aldens*) made from low-nicotine tobacco, the filter-tip *Kents*, several Turkish-type cigarettes, and a low-cost brand (*Yorkshires*, sold by Sears-Roebuck).

CU also tested filter holders for their effectiveness in removing nicotine and tar from cigarette smoke; the brand ranking highest in this respect was the *Zeus*, in which you use a cigarette as a filter.

For a complete report on the results of CU's tests on cigarettes and filter holders, see pages 67 to 70. For a review of the industry which manufactures America's most widely distributed product, turn the page. ▶

Because Americans spend billions of dollars each year on cigarettes, because the number of cigarette smokers in the population is growing fast and has already reached the figure of nearly one out of every two adults in the country (even higher at the younger age levels), because new and serious questions have been raised about the effects on health of cigarette smoking, and because few Americans can avoid the cigarette industry's ubiquitous and often contradictory advertising, CU has given over a major part of this issue to a report on the subject. Sections on the following pages include:

THE INDUSTRY PAGE 60

From the Indians to the trusts
Trust busting and the price story
Advertising: a modern mythology

THE PRODUCT PAGE 67

CU's tests: the findings and the methods
Nicotine and tar in cigarette smoke: how 27 brands compared
Ratings of filter holders

THE HEALTH PROBLEM PAGE 71

What the medical literature really shows
Fact vs. fancy in the advertisements

96946 O—57——41

the industry

Two antitrust suits but still no real price competition . . . millions for ads but a still skeptical public

Smoking, which is a 400-year-old habit of Western civilization, started when the Indians gave tobacco leaves to the Spaniards. Practically from the very first, the use of tobacco was considered by many to be bad for health and to be immoral. King Charles II and King James I of England both issued edicts against smoking; in Russia, at one time, smokers were punished by amputation of the nose. Disapproval is still voiced by many groups in the United States today and is reflected in the paradox that cigarettes, along with alcoholic beverages, are the most heavily taxed consumer products at the same time that they are used by a vast segment of the adult population.

WHAT ARE CIGARETTES MADE OF?



The drawing shows the range of ingredients of domestic cigarettes. Not all are in all brands, but most contain: tobacco and paper, of course, a humectant (moistener), sweeteners, and traces of other flavorings ("casing" in the trade). Popular brands are 45% to 75% flue-cured, 15% to 45% burley, 5% to 13% Turkish tobacco

The background of disapproval and distrust of cigarette smoking is also reflected in many of the slang names for cigarettes, among them: coffin nails, gaspers, weeds, pills, lung dusters, dope sticks, and poison sausages.

Cigarettes are a late entry in the tobacco business; they were invented in South America sometime around the middle of the 18th Century. As late as 1920, cigars and cigarettes were in a close race for the smoker's dollar, with the cigarettes (which were, of course, much cheaper) outselling cigars only about five and a half to one. Today, about 80% of the leaf tobacco used in this country goes into cigarettes, and about 70 or more cigarettes are sold for every cigar. From the bottom of the last depression to date, cigarette sales in the United States have nearly quadrupled, reaching the fantastic record figure of 395 billion in 1952. This represents a retail expenditure of around 4.3 billion dollars. To bring it down to an understandable level, it is estimated by *Business Week* that half a pack of cigarettes is smoked every day for *every* American over the age of 15 and that the average cigarette smoker puts out roughly $90 a year for cigarettes at the present rate of consumption. That rate, incidentally, is still rising very rapidly (see the chart on page 63).

Forty years ago, when cigarettes were still considered somewhat sissyish for men and downright immoral for women, the products were quite different from most modern brands. Turkish or Virginia tobaccos were used practically exclusively and without added flavorings. The change to the present type of cigarette began in 1913.

The break-up of the trust

At the turn of the century, there was much public concern with the dangers from big business, and among the trusts which the government broke up was The American Tobacco Co. When it was dissolved as the result of a government case in 1911, its assets were divided among the present American Tobacco Co., R. J. Reynolds Tobacco Co., Liggett & Myers Tobacco Co., and P. Lorillard Co. R. J. Reynolds did not get any of the cigarette business, but two years later it launched a new brand—*Camels*.

Camels were not only a new cigarette, but a new type; contrary to the current belief that only Virginia- or Turkish-type cigarettes could be sold in quantity, they contained a good proportion of burley tobacco; secondly, they were "cased" or flavored; and finally, they were widely promoted by advertising.

Aside from the taste of the brand and the intensive advertising, a number of social factors may have had a great influence on the acceptance of *Camels;* for one thing, they were introduced at a propitious time for sales, just before the explosive effects of World War I on many social

taboos. Within half a dozen years, R. J. Reynolds had captured 40% of the American cigarette market with *Camels*. Naturally, the other big companies followed suit: American Tobacco launched *Lucky Strike*, a burley mixture cigarette, in 1916, and Liggett & Myers shortly switched *Chesterfield* from a Virginia cigarette to the new type. P. Lorillard didn't get on the band wagon with *Old Golds* until 1926, after the race was well started.

The three biggest companies in the industry today are still the same three which inherited the major share of the assets of the old tobacco trust back in 1911. Among them they divide roughly 77% of the domestic cigarette market (American Tobacco, 32%; R. J. Reynolds, 27%; and Liggett & Myers, 18%). Their major, regular-size brands, however, do not account for all of this; about 18% of total sales comes from their secondary brands, chiefly king-size, but also some others. Tobacco companies, like many other large manufacturers, tend to compete with themselves in order to cover all segments of the very diverse American market.

It is estimated that some 30% or so of the adult women in the country smoke, and that some 60% or more of the adult men are confirmed cigarette smokers. At the older ages, the proportions of both men and women who smoke cigarettes are somewhat less, but as the present generations grow older the ratio of cigarette smokers to non-smokers among the old will almost certainly rise.

Where cigarettes are bought

Cigarettes are today probably the most widely distributed manufactured product in the country. You can get a pack almost anywhere and at almost any time. Cigar stores and cigar stands were superseded years ago as the chief sellers of cigarettes. Along with the great increase in smoking by women has come a boom in cigarette sales at food stores and supermarkets, which do most of their cigarette business by the carton and undersell most other outlets by a few cents per carton. They account now for about one-third of all cigarette sales. Vending machines, which charge more for cigarettes than regular over-the-counter sources, get about one-sixth of the business. Cigar stores and cigar stands get about one-eighth of the business. Drugstores still do a sizable share, about one-seventh; restaurants, bars, and liquor stores do about one-fourteenth. A wide variety of other retailers also get into the act.

The cigarette retailers often compete vigorously for business, and their chief weapon is price. During the 1930s, cigarette price wars were common, and real bargains were often available when stores used cigarettes as loss leaders to attract customers. Today prices are more stable, but some stores cut them and some sellers, such as restaurants and nightclubs, hike them.

So far as the consumer is concerned, the prices generally seem to be climbing. From the depression's two packs for a quarter, the leading brands have risen to a nationwide average of about 21¢ a pack. When the prices rise, they rise together on similar brands. Although you may find one store in your neighborhood selling the "Big Three" brands for a penny or two below another store, you'll look a long time before you'll find any retailer pricing one of the leading brands below another.

A kind of competition

Price competition among the large companies has existed —but only, in the last couple of decades, at the wholesale level, and not there to any serious degree. For two years, from 1946 to 1948, *Camels* were sold at 3¢ less per 1000 (50 packs) than either *Luckies* or *Chesterfields* (or most other popular brands), a saving too small to be practical to pass on to the consumer.

The leaders got a scare during the depression when the cut-rate brands were being pushed. The savings of 3¢ a pack available with such brands as *Wings*, which used to retail for 10¢, looked pretty good to smokers in the Thirties. When the Big Three upped their prices in 1931—as one put it, to show their confidence—the sales of 10¢ brands skyrocketed. In one year total consumption of the 10¢ brands was increased almost 30 times. Shortly after that, the leaders started lowering prices again and took other action to prevent the 10¢ smokes from becoming a national institution.

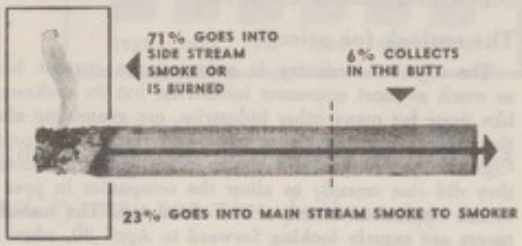
The story was made public in the government's second antitrust suit against the cigarette manufacturers, started in 1940 and completed with a Supreme Court decision in 1946. When the 10¢ brands were garnering a good piece of the market, the large companies began buying up the type of tobacco used in the 10¢ cigarettes (but not in their own). They stored it in warehouses, withholding it from the market to keep it out of the hands of their competitors.

This was only one of the methods used to prevent real price competition in the industry. Despite variations in their budgets, the Big Three generally priced their cigarettes to wholesalers at precisely the same figures, down to the penny. And when one changed, the others changed in consort. But when R. J. Reynolds raised its prices in 1931 and American followed shortly, Liggett & Myers went along only reluctantly. The company announced that it considered the price rise a mistake but that, if it failed to play along, it would have less money available for advertising and would therefore be at a competitive disadvantage. The

Continued on next page

WHERE THE NICOTINE GOES

(Percentage figures are approximate)



cigarettes continued

statement made clear what the industry leaders considered proper and improper competition.

What the government accomplished

As a result of the second antitrust case, the three major companies stood convicted not only of manipulating the tobacco markets to prevent competition, but also of collusion to prevent price competition by keeping their own prices the same and by bringing pressure on dealers to prevent any but the few recognized cheap brands from being sold at a lower price than the regular brands. Some six and a half years after the decision, the major brands and many others, including some of the king-size cigarettes, are still being sold at identical retail prices within stores throughout the country. Even *Philip Morris* and *Old Gold*, which are fighting for position with the leaders (last year *Philip Morris* lost ground when *Pall Mall* moved past it to fourth place in sales standing), have not made any noticeable use of retail price competition.

Since World War II, the cheap cigarettes, the leader of which was *Wings*, have done very poorly. Their percentage of sales has declined to a figure substantially below 1%, compared to more than 22% for one peak month in 1932 before the Big Three really gave them the works. *Wings* is still cheaper than the leading brands, by the same 3¢ per pack savings it offered in 1931. A late comer to the cut-price field is *Yorkshire*, a regular-sized brand which is sold exclusively by Sears Roebuck. *Yorkshire* averages about 4¢ per pack less than the leading brands, but it is considerably less convenient for most consumers to buy, because of its limited availability.

In its survey, CU found many cigarettes priced higher than the popular brands, which ranged from 18¢ to 26¢ a pack, from store to store and place to place. The king-sized *Chesterfields* usually got a 1¢ per pack premium. Filter-tip brands purchased ranged from 1¢ higher than average per pack for *Viceroys* to a premium of 8¢ and more on *Kents*, *Parliaments*, and *du Mauriers*. The highest priced brands CU bought for testing were the Turkish: *Murad* and *Melachrino* at from 32¢ to 46¢ a pack. *Helmar*, another Turkish brand, was found at prices from 24¢ to 36¢, and low-nicotine *John Aldens* at 23¢ to 31¢, and the denicotinized *Sano*, at 26¢ to 34¢.

The outlook for prices

The cigarette industry is enjoying the current boom as much as most consumer industries, but its spokesmen, like those for many other industries, are grumbling about the squeeze between rising costs and fairly rigid prices. Cigarette prices are still under price control, although they did rise recently to allow the companies to pass on to the smoker an increase in Federal tax. The manufacturers are eagerly looking forward to April 30, when the present price control law is expected to die, and price rises seem almost sure to be on the way. Even with record sales during the last few years, the companies did not make record profits. In 1951, for example, when total cigarette consumption was up more than 3% from 1950, the profits of the manufacturers fell off an average of 15%. (The Big Three still made a total of 87 million dollars net profit that year.)

There are all kinds of trade guesses, but one of the most widely repeated is that most cigarettes will go up 1¢ a pack as soon as price controls permit. Some of the king-size brands—those now frozen at the same prices as the popular brands—are expected to rise too, ending up possibly 2¢ a pack higher than at present.

What the smoker pays for

Taxes take the biggest slice of the smoker's dollar—8¢ a pack going into the Federal treasury, and from 2¢ 6¢ a pack into state coffers (1¢ in the District of Columbia). On top of that, some cities have local sales or other taxes which add a penny or so to the total. In New York City, 13¢ out of the regular 23¢ price of a pack of ordinary cigarettes goes for taxes. The return to the manufacturer runs around 7¢, which leaves a margin of 3¢ or so for the distributor and retailer.

Tobacco leaves

It takes a huge crop of tobacco to make the raw material for 400 billion cigarettes and still leave some for other uses. The total crop in this country in 1951 was in excess of a million tons, grown on more than one and a half million acres. With total demand always rising, the manufacturers of cigarettes have to hold large inventories of tobacco leaves, particularly since some kinds must be cured for 18 months to two years before use. The market in which the manufacturers buy is far from a free one. The Federal government supports tobacco prices and this method of support has the effect of strictly limiting acreage. That helps to keep cigarette costs up. Right now leaf tobacco prices are off slightly, but the consumer won't benefit, at least for a few years, until the tobacco now being bought comes on the market, and he probably won't benefit then unless the manufacturers are forced by economic circumstances to pass on the savings.

Tobacco is under one of the tightest controls the Department of Agriculture has over any cash crop. Acreage allotments are closely held and highly prized, and the total allotment is usually increased very little from year to year. If you grow tobacco without an allotment, you end up by paying a penalty tax when you market it, and the tax may be a high percent of the value of the crop.

The cost of cured leaf tobacco makes up more than half of the cost of making and marketing a cigarette. The cost of manufacturing and shipping together, according to published figures for the standard brands for 1940-1941, approximately equaled the cost of advertising, which was about one-seventh of the manufacturers' costs of doing business. Selling costs other than advertising are an appreciable item, also, although less than half of advertising costs.

Although they spend more on tobacco than on advertising, the cigarette companies give the impression of being more concerned with their advertising campaigns than with the kind of tobacco they buy. Considering the low value

of the dollar today compared to the early 1930s, their present campaigns are actually conservative in size. In 1930, the Big Three alone spent roughly 46 million dollars on advertising; the entire industry spent only a little more than 60 million dollars in 1952. Five of the companies, however—American Tobacco, Reynolds, Liggett & Myers, Philip Morris, P. Lorillard—ranked among the only 20 firms in the nation to spend 4 million dollars or more on ads during the first six months of last year. Those five companies account for 92.7% of all the cigarettes sold in this country, and a very sizable portion of other tobacco products.

Just what these huge outlays have bought, not even the advertisers seem sure. For one thing, they have bought a fair amount of trouble with the Federal Trade Commission, which has objected at one time or another to claims made by all of the big companies and many of the smaller ones.

Perhaps the most famous cigarette advertiser was the late George Washington Hill, president of American Tobacco Co. and an autocratic ruler of its advertising and other policies. (It is generally assumed in the trade that he was the model for the character of the sponsor in *The Huckster*.) Under Hill, American Tobacco's advertising was loud and often pioneering. The slogan "Reach for a *Lucky* instead of a sweet," which was used during Hill's regime, got *Lucky Strike* a lot of attention as well as denunciation from candy companies. Hill was the first cigarette manufacturer to bid for the women's market by running pictures of a good-looking girl smoking a cigarette. When these ads were bitterly attacked, Hill replied: "Why should I justify [my advertising]? It has paid."

Advertising themes

Three main themes have run through cigarette advertising: 1) pleasure, 2) snob appeal, and 3) health appeal. The snob appeal works in several ways: the idea is built up that Brand X is smoked by famous, rich, successful people or those otherwise to be envied or admired; the name of Brand X is repeated until it is an accepted thing socially; and Brand X is associated so far as possible with certain traits generally considered to be desirable (such as masculinity, or, for women, good looks).

The pleasure from smoking a particular brand is not, of course, subject to precise scientific measurement, and is pretty much a personal matter, anyway.

The health theme in advertising is the one that has caused most of the run-ins with the FTC and has brought the industry much criticism. One recent survey indicated that smokers are often aware that cigarette smoking may not be good for them and they don't like to be reminded of it by ads. Some members of the industry also insist that ads claiming health advantages for a particular brand merely underscore the possible dangers from smoking, to the detriment of the whole industry and without helping the advertised cigarette. But health themes have been and are still widely used.

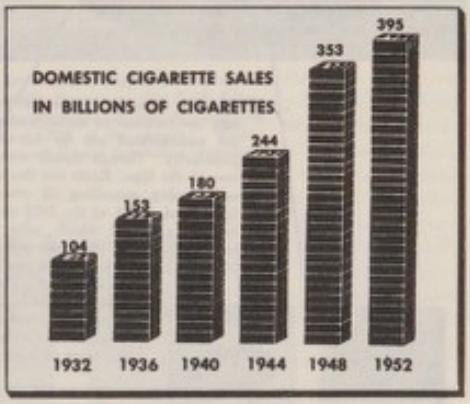
The claims have often been pretty bald: for example, R. J. Reynolds' former statement to the effect that *Camels* were helpful for athletes who had to keep fit, or the assertion that smoking *Kools* would give extra protection against colds. These are among the claims which were dropped as a result of FTC action.

In general, the government has held that among the leading brands there is no meaningful difference in nicotine content or in the other important chemicals in the smoke, and no significant difference in the physiological effect of smoking them. It has held that tobacco cannot be smoked without irritation to throat tissue. It has also objected to specific statements which it found to be untrue (such as *Lucky's* former claim that, among independent tobacco dealers, it was "*Luckies* two to one"). One of the most frequently used cliches in the trade is the reference to the "independent laboratory." The laboratory and the "medical specialist" may be independent in the sense that they are not part of the advertiser's organization, but, as has been pointed out by the government, they are usually paid by the company for their opinions or tests and may therefore be something less than completely free from bias.

The FTC began its campaign to clean up the health aspects of cigarette advertising in 1942, and by 1950 had proceeded against all of the five largest companies except Liggett & Myers. The proceedings in completed cases have not served to inhibit seriously the advertising of other companies. No copywriter worth his Brooks Brothers suit would let an FTC order stop him from using health appeals if he felt they would be effective.

Advertising today

The *sine qua non* of cigarette advertising is mildness. Rare is the copywriter who can restrain himself from



For a breakdown of sales by brands and types, see the following two pages. Text continues on page 66

what's happening in cigarettes

The status of the continuing changes in consumer preferences for types and brands of smokes is shown at each year's end by sales figures. This is the picture for the end of 1952. Since expenditures for cigarettes last year were 4.3 billion dollars, by conferring even a tiny bit of their business on a company the country's cigarette smokers can make it a multi-million dollar concern. Shown here are the major types of cigarettes and all of the 27 brands (95% of the market) tested by CU. For a picture of how cigarette smoking has grown in the last two decades, see the preceding page.



Sales of king-size brands rose 54% to a new popularity record (18% of the market)

Filter-tips jumped 65% but to only 1½% of total sales



Regular cigarettes fell off 3% but still dominate the field with 80% of sales

Of these three groups—mentholated, Turkish, and low-nicotine or denicotinized cigarettes—the mentholated are by far the highest in popularity. Though *Spuds* were the originators of the type, *Kools* are the only really big seller today, outselling all other minor types for a total of 3% of the 1952 market. Turkish brands, some of which were comparatively important before the trust was broken up in 1911, and the low-nicotine smokes, are today unimportant. The low-nicotine tobacco used in *John Aldens* is a modern hybrid type. The nicotine in *Sanos* is reduced by washing some of it out of the tobacco















King-size cigarettes cut sharply into sales of the regular brands last year. *Pall Mall* was the first king to squeeze into the top four in sales, got 10.5% of the country's total. *Chesterfield's* king-size smokes, introduced in June, got a 3% share in a half year's time. It is rumored that other major companies may follow the lead of Liggett & Myers and soon bring out kings under major brand names. The other brands here ranged in sales from slightly under 1% (*Fatima*) to about 0.1% (*Dunhill*)



Filter-tips make up one-third or more of sales in South America and other places. Though still a minor part of the American industry, many in the trade say that they, along with kings, are the coming thing. One influence on the popularity of both types is said to be the increase in smoking by women. *Viceroys* are the leading filter-tip today, but they got only 0.7% of last year's sales



Camels have been the hit of the parade since *Luckies* slipped back to second place in 1949. *Chesterfields*, *Pall Malls*, *Philip Morris*, and *Old Golds* follow. Current estimates are that slightly less than half of the 104,000,000 population aged 18 and over smoke (60% of the men, 30% of the women). Women in the Middle West are expected to be target of new promotion campaigns



These English-style brands, all made in America, have a high prestige-appeal, cost more than the popular cigarettes, and sell comparatively little. The U.S. market has many minor brands—CU counted 30 odd last fall, not including roll-your-own tobacco

cigarettes continued from page 63

claiming that the cigarette whose praises he sings is, for example, "mild" (*Camel* and *Luckies*); "much milder" (*Chesterfields*); "far milder" (*Dunhills*); or endowed with "tasty mildness" (*Philip Morris*); or with "mildness you can measure" (*Pall Mall*).

The filter-tip and king-size cigarettes today are usually advertised as offering protection from nicotine, tar, or sometimes simply "irritants." For *Viceroys* the largely irrelevant and self-evident statement is made that "The nicotine and tars trapped by the *Viceroy* filter cannot reach your throat or lungs!" *Kent* ads read, "First time ever! Sensitive smokers get real health protection with new *Kent*. . . . If you're sensitive to the nicotine and tars in tobacco . . . (published medical reports show about 1 out of 3 smokers is) . . . *Kent's* exclusive 'micronite' filter removes up to 7 times more nicotine and tars than other leading filter cigarettes!" For *Pall Mall*, the biggest selling king-size smoke, the copywriters say you can "guard against throat-scratch with *Pall Malls*." For *Dunhill*: "*Dunhill*—because of its king size, its finer tobaccos and its natural filtering qualities—screens out irritants. . . ." The list of similar quotes is not quite endless, but it's long.

Health and the popular brands

But the king-size and filter-tip brands are not alone in stressing health. *Lucky Strike* emphasizes that its tobaccos are "clean!" *Old Gold*, still smarting from an FTC order not to claim less nicotine or less irritation than other leading brands, turned the FTC case to its own advantage and currently works both sides of the street. On the one hand, *Old Gold* ads carry the head, pooh-poohing health claims, "A treat instead of a treatment." On the other hand, the same ads say, "No other leading cigarette is less irritating, or easier on the throat, or contains less nicotine than *Old Gold*. This conclusion was established on evidence by the United States Government." What the government established, and the *Old Gold* ads don't say, is that all leading brands are substantially alike in these respects, and no other leading brand is more irritating, either.

Camel ads are still full of health hints, despite a long list of "don'ts" from the FTC. Though prohibited from saying directly that *Camels* are efficacious where a sport demands unfailing energy or good condition, the ads often feature famous athletes smoking *Camels* and saying things such as, "My choice for steady smoking is mild, flavorful *Camels!*" In addition, *Camel* ads announce, "More doctors smoke *Camels* than any other cigarette," which may be true without being significant, because more people in general smoke *Camels*, which are at present and have been for some time the most popular brand.

Two other leading regular brands, *Chesterfield* and *Philip Morris*, both used to make strong statements about freedom from irritation of one sort or another, but they may change their copy. Both are presently involved with the FTC. During the first week of January, as the culmination of a long case against *Philip Morris*, the Commission ordered the company to discontinue its claims that: irritation caused by smoking other leading brands is of longer duration than that caused by smoking *Philip Morris;* the humectant used in *Philip Morris* (diethylene glycol) helps to make the smoke from the cigarettes significantly less irritating to the upper respiratory tract than the smoke from cigarettes containing glycerine as a humectant; smoking *Philip Morris* will protect the smoker from smoker's cough or the effects of inhaling.

Liggett & Myers, now in the midst of a court battle with the Commission, uses some advertising claims for *Chesterfield* very similar to those the FTC has forbidden other companies to use. Some recent *Chesterfield* ads say, "Nose, throat, and accessory organs not adversely affected by smoking *Chesterfields*," and add that this is the report of a "continuing study by a competent medical specialist and his staff. . . ." For the first time in a cigarette case, the FTC asked for a Federal court injunction to prevent Liggett & Myers from continuing the ads pending an FTC hearing. The Commission is empowered to use court injunctions in cases involving drugs; in its plea to the court, the Commission said that tobacco is a drug and pointed out that to allow the company to continue with advertising claims forbidden to other firms while the case was being heard and probably appealed would place the others at a competitive disadvantage. The District Court, however, refused the injunction on the grounds that it did not consider tobacco a drug within the meaning of the law. The government is appealing the District Court's decision.

What about the public?

It is probably fairly obvious to most consumers that the FTC's efforts to control cigarette advertising haven't been very successful. Also, no matter what solemn and pious statements they may make about health-appeal ads, advertising men and cigarette merchandisers obviously think they are very effective because they continue to use them. Whether the health appeals are believed by many smokers is another question. When Dr. Gallup conducted a poll on cigarette smoking in 1949, more than half of the cigarette smokers questioned said they thought cigarette smoking harmful and had tried at one time or another to give it up.

One secondary effect of the great volume of advertising is that it seems to protect the cigarette companies, to a very large degree, from unfavorable press comment. Although some of the more responsible newspapers run news stories on FTC actions against cigarette companies and on medical research into the effects of smoking, many newspapers completely ignore these stories or bury them. The *Reader's Digest*, probably the only mass-circulation publication in the country which doesn't have to worry about losing cigarette advertising, carries on what is practically a war against cigarette smoking. Some of the publications connected with religious organizations (such as *The Christian Science Monitor* and the *Christian Herald*) speak out on the evils of smoking and the excesses of cigarette advertising from time to time, but aside from this the public doesn't read much about the controversial aspects of smoking and the cigarette industry.

the product *Substantial differences among types of cigarettes in nicotine, and among filter holders in effectiveness*

Sixteen cigarettes of each brand, each cigarette from a different pack, were "smoked" in CU's laboratory tests for nicotine and tar in cigarette smoke. Twenty-seven brands, purchased in 19 cities throughout the United States, were included in the tests. All kinds of cigarettes were represented: regular-size and king-size; regular-price, low-price, and premium; the common American blend and Turkish and Virginia blends; low-nicotine, denicotinized, and untreated; flavored and mentholated; and untipped, cork-tipped, ivory-tipped, and filter-tipped. For so great a variety, most of the differences found were surprisingly small. But some of the differences were, nevertheless, significant. They are summarized in the table on page 68. Several positive conclusions can be drawn from the data.

The kind of tobacco

The test results indicate that the nicotine content of the smoke from a cigarette is influenced, first of all, by the type of tobacco used. The cigarette with the least nicotine in its smoke was one made with tobacco especially developed for its low nicotine content. This brand, *John Alden*, had an average of only 0.4 milligrams of nicotine per cigarette in the smoke puffed in by the laboratory smoking apparatus. (A milligram is about 1/28,000th of an ounce.) Other cigarettes with less nicotine in their smoke than the big-selling brands were in general those made largely of Turkish tobacco. The low-priced *Yorkshire* cigarette was as low in nicotine as any of the Turkish types.

The "popular" brands

The most widely advertised and widely sold regular-length cigarettes—*Camels, Lucky Strikes, Chesterfields, Philip Morris,* and *Old Golds*—were practically identical in the nicotine content of their smoke, which averaged about five times as high in nicotine per cigarette as the smoke from *John Alden*.

The smoke of king-size cigarettes (about 3⅓ inches in length) generally contained about a fifth more nicotine than the smoke of regular-length American brands (about 2¾ inches in length), when both were smoked down to a butt of a little under an inch. When, however, the extra length of the king-size cigarette is left unsmoked—that is, when only as much of the longer cigarette is consumed as of a shorter one—the total nicotine from the king-size cigarette is lower. The single exception found—*Embassy*—gave only about as much nicotine as a regular cigarette, even when smoked down to a butt just under an inch.

Most filter-tip cigarettes tested—contrary to the statements in the ads for them—actually had about 20% more nicotine in their smoke than regular brands—as much as that of the typical king-size brands smoked down to about a one-inch butt. Among the filter-tip brands tested, there was the single exception already noted: *Kents* were second only to *John Aldens* in freedom from nicotine in their smoke, averaging only about one milligram of nicotine per cigarette.

As the smoke passes through it, the tobacco in the cigarette itself acts as a fairly effective filter; CU's tests included a determination of the filtering effect of the length of tobacco which is replaced by other materials in filter-tip cigarettes. Two-thirds of an inch of a regular-size cigarette were cut off, and the remainder smoked to the usual extent; 16% more nicotine was found in the smoke than in the smoke of uncut cigarettes.

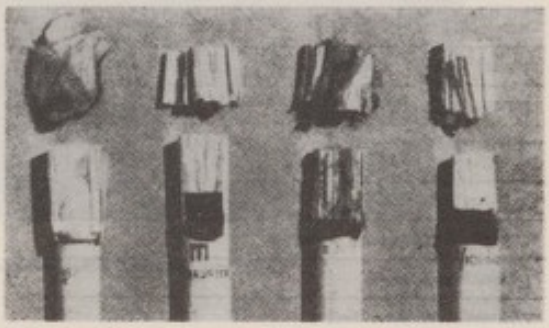
Some special cases

Slightly above *Kent* in nicotine content—but appreciably below the big-selling regular brands—were *Sano*, a "denicotinized" cigarette, *Murad*, a Turkish brand, and *Yorkshire*, the Sears brand, which appears similar to the more widely sold brands. Two other Turkish cigarettes, *Helmar* and *Melachrino*, also ranked between *Kent* and the big sellers in nicotine content of their smoke, but they were only a little below the popular regular-size American blends.

Some brands—*Virginia Rounds* and *Craven A*—had a higher nicotine content than the popular brands, probably because of their particular blends of tobacco.

Kools, the largest selling mentholated cigarette, are, like so many other brands, promoted as being "mild," but in

Continued on next page



What's in the filter tips (left to right): cotton in *Parliaments;* creped paper in *du Maurier's;* paper and other fibrous material in *Kent's* (relatively effective); creped paper in *Viceroy's*

RESULTS OF TESTS FOR NICOTINE AND TAR IN THE SMOKE FROM 27 BRANDS OF CIGARETTES

Brands are listed in order of increasing nicotine content of their smoke. Because of the variability between different cigarettes of the same brand and inherent shortcomings of the test method, the differences in tar content between brands can be considered only approximate; a difference of a few milligrams is of no significance.

BRAND AND MANUFACTURER	AVERAGE NICOTINE IN SMOKE PER CIGARETTE (milligrams)	AVERAGE TAR IN SMOKE PER CIGARETTE (milligrams)
JOHN ALDEN John Alden Tobacco Low-nicotine tobacco	0.4	12
KENT P. Lorillard	1.0	7
MURAD P. Lorillard	1.1	17
SANO United States Tobacco Denicotinized	1.2	10
YORKSHIRE Sears, Roebuck	1.2	11
MELACHRINO American Tobacco	1.6	13
HELMAR P. Lorillard	1.7	16
CAMEL R. J. Reynolds	1.9	15
PHILIP MORRIS Philip Morris	1.9	14
OLD GOLD P. Lorillard	2.0	12
CHESTERFIELD Liggett & Myers Regular-size	2.0	11
LUCKY STRIKE American Tobacco	2.1	16
EMBASSY P. Lorillard	2.1	14
MARLBORO Philip Morris	2.2	13
RALEIGH Brown & Williamson	2.2	13
KOOL Brown & Williamson Mentholated	2.2	16
du MAURIER Columbia Tobacco	2.3	14
WINGS Brown & Williamson	2.3	12
VICEROY Brown & Williamson	2.4	13
FATIMA Liggett & Myers	2.4	14
CHESTERFIELD Liggett & Myers King-size	2.5	13
PALL MALL American Tobacco	2.6	15
VIRGINIA ROUNDS Benson & Hedges	2.6	15
DUNHILL Philip Morris	2.7	17
PARLIAMENT Benson & Hedges	2.7	12
CRAVEN A Cerreras Limited	2.8	15
CAVALIER R. J. Reynolds	2.8	19

cigarettes continued

nicotine content of their smoke *Kools* were about the same as the popular regular brands.

Tars are not found in unsmoked cigarettes, but are produced as a result of incomplete combustion. Because of the variability between different cigarettes of the same brand, and because of inherent shortcomings in available test methods, the differences in tar content between brands can be considered only approximate; differences of a few milligrams are of no significance. The tar content of the smoke from *Kent* cigarettes was found, however, to be consistently low.

Comparison with previous tests

The tests on which this report is based showed a somewhat higher nicotine content for many brands than was found in the limited tests of these brands reported on in June, 1952. CU pointed out at the time that the very small number of cigarettes of each brand tested made those results only approximate. The present results reflect the greater accuracy afforded by the greater number of cigarettes tested. The large difference found in the two tests with respect to *Kent* cigarettes appears to be mainly the result of a change in the filter during the interval. CU noted last summer that *Kents* required "considerably more effort to draw on than do other cigarettes." This characteristic appears now to have been largely corrected

Continued on page 70

HOW CU TESTS CIGARETTES

The testing of a cigarette begins with the collection of its smoke. The cigarette is first fitted into a glass holder **1** and then "smoked" in the laboratory apparatus **2**. The smoke drawn from the cigarette is collected in acidified alcohol in the flask (any smoke carried into the glass "train" is trapped in the two vials.) Lowering the level of the water in the vertical glass tube at the end of the train by means of the bulb at the extreme right of this picture draws air through the cigarette. Each puff is made to last for two seconds and one puff is taken each minute. Slightly more than two cubic inches (35 cubic centimeters) is taken in with each puff, and each cigarette is smoked down to a butt of just under an inch (23 mm.). In CU's tests, 16 samples of each brand were "smoked" in two runs of eight cigarettes each; the results of each run were determined separately and then averaged. After each run, the washings from the apparatus are added to the smoke solution in the collecting flask. One portion is steam distilled **3** to isolate the nicotine; amounts are measured with an ultra-violet spectrophotometer **4**. Chloroform is added to another portion of the smoke solution diluted with water. The tar is then extracted by vigorous shaking **5**, and the chloroform is then drained from the separatory funnels. After this, the chloroform is boiled off **6** and the tar residue weighed on a chemical balance **7**.



1



2



3



5



4



6



7

cigarettes continued

with a looser and, therefore, a more permeable filter.

CU's current tests were for nicotine and tar content. Blindfold tests, which have been performed many times by many groups, including CU, and which have almost uniformly shown that smokers cannot with any regularity distinguish among the popular brands by taste, were not a part of this survey. In the course of its investigation, CU did have members of its staff who were habitual smokers try out various brands, but no attempt was made to conceal the names of the cigarettes being smoked. Some of the comments were interesting. With almost no exceptions the smokers showed the same reluctance to shift from their favorite brands that some advertising surveys have shown to exist widely. Many of the smokers said they would not shift from the brand they smoked to some of the brands even if these cost appreciably less.

Filter holders

The filter holders CU tested were the *DeNicotea*, in which silica is used as the filtering agent; the *Medica*, in which a roll of paper is used; the *Pura-Smoke*, in which alumina is used; the *Weber*, in which charcoal is used; and the *Zeus*, in which a cigarette in the tube acts as the filter. Two of each brand were tested.

Cigarettes held in the filter holders were "smoked" as for the cigarette tests, and the smoke was collected and analyzed by the same methods. A typical regular-size cigarette was used. Each holder was "smoked" on a run of five cigarettes. The two brands which proved most effective on the initial run—the *Zeus* and the *DeNicotea*—were further tested for their effectiveness when the same filters were used without change for longer runs. The filtering action of the *DeNicotea* was determined for the last five of 15 cigarettes, and the effectiveness of the *Zeus* for the last five of 20. Both were found effective even after such long use. (The *DeNicotea* effectiveness had decreased and the *Zeus* effectiveness had increased slightly.)

In length the holders ranged from 3½ to 4¾ inches; the long metal tube of the *Zeus* holders was found to be more effective by itself in removing nicotine than some of the holders with the filters in them. When used without a cigarette inside as a filter, the *Zeus* tube condensed nearly 20% of the nicotine in the smoke from the first five cigarettes smoked.

Most of the members of a panel of smokers who normally did not use cigarette filter holders found the odor of the filters after a few cigarettes extremely unpleasant. One may have to suffer through a period of building up a tolerance to the odor in order to be able to use one of these filters regularly. Some users found the extra effort of drawing through them objectionable; others disliked the weight of the holders. One found the smoke relatively tasteless, and noted that he smoked more cigarettes when using the filter. While cigarette holders have the advantage of keeping tobacco grains out of the smoker's mouth (an advantage shared by filter-tip cigarettes), they cause the smoke to impinge on a small area of the tongue, which can cause the same "bite" that pipes cause.

One unhappy tendency of smokers who use a filter holder is to smoke cigarettes down to the very end, thus negating at least a part of the nicotine-removing effectiveness of the filter. Another tendency, which has the same effect, is to smoke deeper puffs with a holder than without.



Zeus (at top) uses a cigarette as a filter; the stained cigarette was taken from a Zeus after a pack of cigarettes was smoked through it. Pura-Smoke uses a cartridge of alumina granules. It differs from the other holders tested mainly in the cartridge material

RATINGS OF FILTER HOLDERS: *Listed in order of their effectiveness in removing nicotine from smoke.*

ZEUS (L. & H. Stern, Inc., NYC), $1.50 for regular size which uses an ordinary cigarette as the filter; $2 for the "deluxe" which can use a king-size cigarette as a filter. Removed 44% of the nicotine and 33% of the tar from the smoke of the first five cigarettes smoked, when used with a regular-sized cigarette as a filter; it would do slightly better with a king-size cigarette as a filter. It removed 50% of the nicotine and 33% of the tar from the smoke of the fourth set of five cigarettes smoked. Has a butt ejector.

DENICOTEA (Alfred Dunhill of London, Inc., NYC). $2.50 for men's holder with 10 refill cartridges; $3.50 for longer women's holder with 12 refill cartridges. Silica filter. Removed 35% of the nicotine and 33% of the tar from the smoke of the first five cigarettes smoked; 29% of the nicotine and 17% of the tar from the smoke of the third set of five cigarettes. Refill filters, 10 for 35¢. Has a butt ejector.

PURA-SMOKE (Continental Briar Pipe Co., NYC). $1 with 5 refill cartridges. Alumina filter. Removed 21% of the nicotine and 33% of the tar from the smoke of the first five cigarettes smoked. Refill filters, 10 for 25¢. Has a butt ejector.

MEDICO (S. M. Frank & Co., NYC). $1. Paper roll filter. Removed 13% of the nicotine and 17% of the tar from the smoke of the first five cigarettes smoked. Refill filters, 10 for 10¢.

WEBER (no manufacturer shown on package). $1 with 5 refill cartridges. Charcoal filter. Removed 10% of the nicotine and 11% of the tar from the smoke of the first five cigarettes smoked. Refill filters, 10 for 35¢.

are they harmful? *Perhaps—each smoker will have to decide for himself whether the satisfaction is worth the risk*

So intimately is tobacco bound up with the life of America —from the early Virginia settlements and the plantation economy of the South to the growth of the big tobacco companies and the daily advertising barrage—that any discussion of the health aspects of smoking is bound to excite the hostility of some groups and individuals.

Before the first World War, when cigarettes were much less important in the tobacco business, they were damned principally as a foppish affectation and, on moral grounds, as a kind of symbol of loose living. The indictment now is that cigarettes, healthwise, are the most pernicious member of the tobacco family. The catalog of evils generally associated with smoking them includes irritation of the respiratory tract, eyes, and tongue; interference with appetite; bad breath; pollution of the atmosphere; and many other such. In addition, some doctors insist that smoking, particularly cigarette smoking, leads to addiction, and that addiction impairs the intellect, interferes with judgment, depresses the emotions, inhibits sexual potency, injures the heart and blood vessels, and causes cancer of the lung.

On the other side of the controversy, we find the tobacco industry and its hucksters claiming that smoking will steady the nerves, relieve irritated throats, banish coughs caused by any rival company's tobacco, and promote social ease.

Against this background, it might be a good idea first to clear away some of the misconceptions concerning the effects of tobacco and tobacco ingredients on human beings.

Some misconceptions and some facts

Thomas Edison, like many others distinguished in the arts or sciences, had strong opinions on this subject; he believed that cigarette paper was responsible for many of the toxic symptoms caused by smoking. Subsequent study showed that the part played by paper in the reaction to smoking is insignificant.

Proper moisture content of the tobacco is important for a satisfactory smoke; a moistening agent, such as glycerine or diethylene glycol, is usually employed in the processing of the tobacco. According to Professor H. B. Haag, of the Medical College of Virginia, Richmond:

> Discussion, at times somewhat acrimoniously tinctured, has waxed concerning the relative merits of these two agents as they affect the irritating properties of cigarette smoke. The more recent studies all are inclined to the view that neither agent shows superiority over the other in this respect.

Tobacco smoke is invariably irritating, irrespective of the type of moistening agent applied.

In addition to nicotine, tobacco smoke contains carbon monoxide, ammonia and other volatile alkaline materials, acids, phenols, aldehydes, and other materials comprising tar. The heavy smoker inhales a considerable amount of carbon monoxide from the incomplete combustion of tobacco in his cigarette, pipe, or cigar. The carbon monoxide combines readily with the hemoglobin of the red cells and the smoking of two packs could result in saturation of as much as 7% of the hemoglobin with carbon monoxide. But it has not been established that this degree of saturation is responsible for any of the toxic effects of heavy smoking. Nor is there any knowledge about possible long-time effects of this amount of carbon monoxide. Obviously, more research on this problem is needed.

Nicotine: pure and in smoke

Of all the components of tobacco, nicotine has been the focus of greatest interest. The use of denicotinized tobacco, and of special tobaccos cultivated for their low nicotine content, are reflections of this special concern with the properties of nicotine. While the toxic effects of pure nicotine in specific doses are well known, there is a considerable difference of opinion concerning the extent to which nicotine of tobacco *smoke* is harmful to health.

In a limited study recently made in the Laboratory of Applied Physiology of Yale University, Drs. Greenburg, Lester, and Haggard found the average nicotine content of the cigarettes they tested to be 17.2 milligrams per gram of dry tobacco. (The average nicotine content of the cigars tested was found to be 17.4 milligrams per gram of dry tobacco.) Since nicotine must be absorbed by the body in order to exercise any effect, the Yale doctors investigated, among other things, the effect of inhaling on the amount of nicotine absorption. They found that a very small amount is absorbed when a cigarette or cigar is smoked without inhaling (other studies have shown that if the smoker does not inhale, but does hold the smoke in his mouth even for a few seconds before expelling it, the nicotine absorption is much higher). But with inhalation, virtually all of the nicotine present in the smoke is retained by the body. In sum, they found that when cigarette smoke is inhaled, about two and a half milligrams of nicotine is retained in the body (see page 68 for CU's findings on the amount of nicotine in cigarette smoke). Inhaling is much more common among cigarette smokers than among cigar and pipe smokers. Whatever the reason, it is apparent that, in general, for the same quantities of tobacco, inhaling cigarette smokers absorb considerably more nicotine than non-inhaling cigar and pipe smokers—or non-inhaling cigarette smokers.

According to one investigator, there is no relation between the so-called "strength" of tobacco and its nicotine content. The strength of a tobacco depends primarily on

Continued on next page

cigarettes continued

aromatic substances which are volatile and which, for the most part, are formed during the process of fermentation before the tobacco is dried.

Modern knowledge of the properties of nicotine began with the famous experiments of the English investigator, Dr. J. N. Langley, who painted solutions of nicotine on the ganglia of the autonomic nervous system. It soon became apparent both from these and later studies that the effects of nicotine on human beings are exceedingly complex. The drug acts on three main areas of the nervous system: 1) on all the ganglia of the autonomic nervous system; 2) on the junction between the nerves and the muscles; and 3) on the brain itself. At each of these sites, it first stimulates and then depresses function. Consequently, six possible types of reaction may go on at different rates and at different times, producing involved, and at times unpredictable, effects.

Opponents of smoking are fond of pointing out that a few milligrams of nicotine applied to the skin can cause death. Fortunately, this drastic result is impossible except in chemical plants or laboratories where the concentrated drug is handled.

One's first smoke, whether of a cigarette, a cigar, or a pipe, is almost always associated with acute toxic symptoms—notably, nausea, vomiting, headache, and dizziness—and it is probable that most of these symptoms are caused by the nicotine. With continued smoking, habituation to the drug occurs; the chronic heavy smoker can absorb and handle amounts of nicotine that would seriously poison a non-smoker. Since acute intoxication by nicotine is a rare accident, or a passing phase in the development of the smoking habit, it is proper to turn to the effects of nicotine on the habitual smoker.

Heart and blood vessels

The chronic effect of nicotine in smoke on the heart and blood vessels has been the subject of considerable experimental research and clinical observation. But even if one excludes research sponsored and financed by tobacco companies with special interests, it is still difficult to draw incontestable conclusions from much of the experimental data. At one time the opinion was expressed that smoking caused arteriosclerosis, or hardening of the arteries. Persons with "angina pectoris" were forbidden to use tobacco, particularly when tests showed that injections of pure nicotine caused a transient rise in blood pressure and heart rate. More critical studies have since shown that neither nicotine nor any other component of tobacco causes arteriosclerosis, a disease whose essential cause or causes are still unknown.

Smoking sometimes does cause palpitations, extra heart beats, or even paroxysms of rapid beating, but such symptoms occur without evidence of interference with the blood supply to the heart and probably reflect the influence of nicotine on the nervous and muscular irritability of the heart itself. Certainly, it is simple common sense for anyone sensitive to nicotine either to refrain from smoking, to reduce the number of cigarettes smoked daily, or to try cigarettes of low nicotine content. Switching to a pipe might also be helpful.

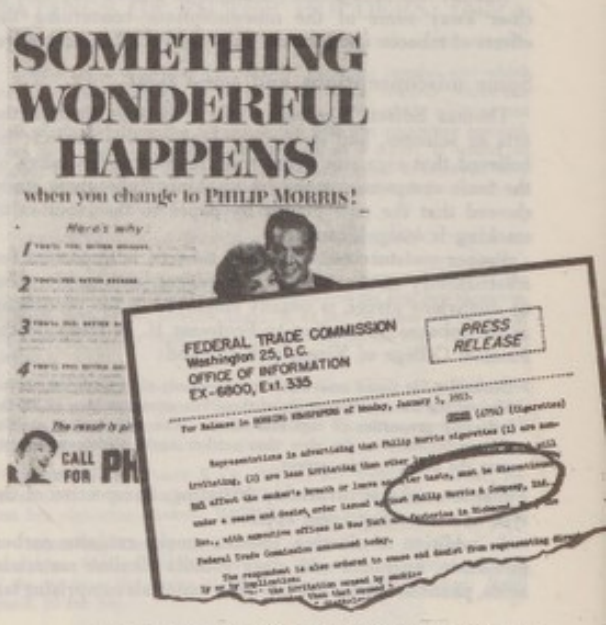
True "tobacco angina," a condition in which pain in the chest occurs after smoking, is quite uncommon; even when it does occur, it does not necessarily mean that the pain is associated with changes in the coronary artery circulation of the heart. It does mean special sensitivity in the smoker, however, and again simple common sense should tell him what to do. As for persons with some form of organic heart disease (valvular disorders, coronary disease, etc.) the comments of Dr. Robert L. Levy of Columbia University, College of Physicians and Surgeons, probably represent the point of view of most heart specialists:

> Patients with any form of heart disease usually are advised to abstain from the use of tobacco; yet it has been our experience that over a period of years most of these individuals have smoked moderately without apparent harm. If one may judge by the amount of tobacco consumed, smoking affords a good deal of pleasure to a large number of persons; for many it provides emotional stability.

In this connection, it must be pointed out that "moderate" means different things to different persons. To the heavy smoker who consumes two to three packs of cigarettes a day, moderate smoking may be one pack; to others, moderate may mean five to ten cigarettes a day. Each individual must determine for himself, or with the aid of his physician, just what his tolerance for tobacco is.

Dr. Levy also points out that, on the basis of new techniques for estimating heart function, tobacco smoke causes relatively little change in the activity of the heart.

> There is [says Dr. Levy] considerable variability of effect in both normal persons and patients with heart disease, but this depends to



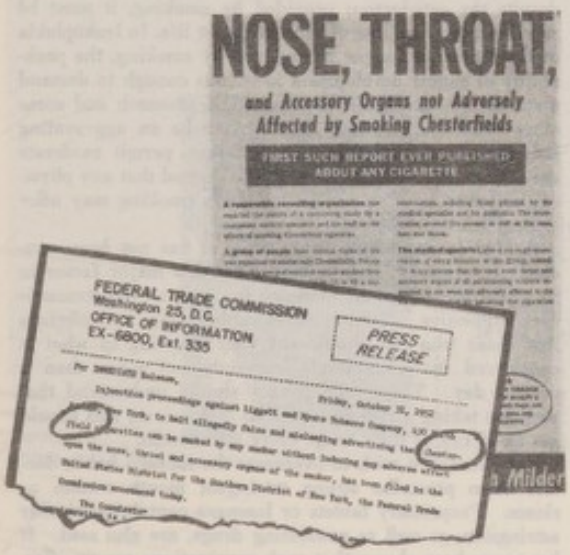
Here are two ads, both with health themes, which were part of campaigns resulting in the FTC's latest actions against cigarette companies. All other important brands have been on the receiving end of FTC actions against health themes, but health themes still appear (see page 63). Other big brands which are currently using such appeals include Camels, Pall Malls, and Old Golds

a greater extent on individual susceptibility than on the presence of a cardiac disorder. . . . Because of the enjoyment afforded and the feeling of satisfaction obtained, it is our opinion that the majority of patients with inactive forms of heart disease may be permitted to smoke in moderation. Those who are sensitive to nicotine will fare better by abstinence. There are certain conditions in which smoking should be forbidden, because any increase in the work of the heart, however slight, is to be avoided; among these are congestive heart failure, the acute stages of cardiac infarction [coronary thrombosis] and active rheumatic fever. No patient with peripheral vascular disease should use tobacco because of its constricting action on the peripheral vessels.

The last sentence states an incontestable fact; that nicotine, because of its action in reducing blood flow through the vessels of the extremities, is harmful to patients with Buerger's Disease (thrombo-angitis obliterans) or similar disorders of the blood vessels of the extremities. Because of the unusual sensitivity of such patients to nicotine, complete abstinence from smoking is the single, most important requirement for successful treatment. The benefits of complete abstinence have been proved so conclusively that it is doubtful that any physician could justify the use of even one cigarette daily, even one with the lowest available nicotine content, for a patient with Buerger's Disease or peripheral vascular disease of the extremities.

The respiratory tract

That all cigarette smoke is irritating, particularly to the mucous membrane of the respiratory tract, from the nose and throat down to the bronchi, is also another established and incontestable fact about smoking. Naturally, those who inhale deeply will have a greater tendency to irritation than those who simply puff without inhaling. A chronic cough, usually most apparent in the morning on arising, is perhaps the commonest symptom of irritation of the respiratory tract from smoking. Even those who smoke only a few cigarettes a day may be subject to an annoying tickle or cough. However, it is dangerous to assume that a cough is caused by smoking until at least three steps have been taken: 1) an X-ray examination of the lungs; 2) a history and physical examination by a physician to exclude the presence of other disorders that can cause chronic cough; and 3) abstinence from smoking for a while, to find out whether this will lead to complete relief from the cough.



Varying degrees of hoarseness and voice fatigue are symptoms of "smokers' larynx." Swelling, redness, and even thickening of the vocal chords are consequences of prolonged, heavy smoking in some persons.

Cancer of the lung

In the past few years the focus has shifted from the influence of smoking on vascular disease to its possible relation to cancer of the lung. Vital statistics show few trends more dramatic than the rise during the past 30 years in the death rate from cancer of the lung. Only a part of this increase can be attributed to aging of our population, or to improvements in diagnosis by physicians. The rising incidence of cancer of the lung is assumed by some experts to result from several environmental factors, among them surface dust from tarred roads; exhaust fumes from motors; fumes, dust, and smoke from gas works and industrial plants; and, lastly and closer to many hearts, tobacco. The most vigorously advocated hypothesis today is that heavy smoking (more than 20 cigarettes a day) over a period of many years is one of the most important factors, if not the most important of all. This hypothesis seems to be particularly plausible since there has been a progressive rise in the use of tobacco, particularly of cigarettes, parallel with the rising incidence in lung cancer.

Experimental work with animals has not conclusively implicated tobacco as a cause of cancer of the lung, despite the fact that tars collected from tobacco smoke can produce cancer in some animals. Arsenic is also a well known carcinogenic agent, capable of causing cancer both in humans and experimental animals, and in the United States tobacco leaves are sprayed heavily with lead arsenate to control insect infestation. Studies of the arsenic content of whole tobacco and of tobacco smoke, both in England and in this country, show that appreciable amounts of arsenic are present in cigar, pipe, and cigarette tobacco; that from about 8% to 18% of the arsenic in the tobacco may be found in the smoke; and that "main stream" smoke, when inhaled, can introduce into the respiratory tract an appreciable amount of arsenic over a period of years.

That arsenic is inhaled in tobacco smoke does not prove that arsenic causes cancer of the lung, nor, even more important, that smoking causes cancer of the lung. More conclusive experimental and clinical data are needed. To help settle this point, the American Cancer Society has undertaken a special project of real importance. From 200,000 to 300,000 men, between the ages of 50 and 69 (the period in which cancer of the lung has its highest incidence), will be questioned and kept under observation for several years

Continued on next page

cigarettes continued

by volunteer workers of the Society. Volunteers in selected counties of ten different states will record the smoking and medical histories of ten men each.

The follow-up method of study of the American Cancer Society should overcome some of the deficiencies in previous methods of investigation. But it will be several years before answers will be forthcoming. If it should be proved that heavy tobacco smoking (particularly of cigarettes) over many years is a factor in the cause of cancer of the lung, it will then be up to the smoker to determine if he wishes to take his chances in acquiring cancer of the lung in exchange for the comforts, pleasures, and stimulant or sedative effects that smoking provides. Car-driving on American roads is the leading cause of accidental death today, yet most of us take the risk.

There is also inconclusive evidence about the effects of tobacco on the body in physical performance and endurance in athletic contests. About all that has ever been shown is that susceptibility to the effects of tobacco varies widely among individuals. While some men can smoke in moderation without impairment of athletic proficiency, others have exhibited poor performance after smoking for some time.

Heavy smoking of pipes, cigars or cigarettes is known to cause iritation of the mouth and tongue, often of such intensity as to lead to the development of localized areas of thickening or "leukoplakia" of the mouth. This thickening is considered a potential cancerous or pre-cancerous condition. When the condition is detected in the pre-cancerous stage, it will usually disappear if smoking is stopped.

With respect to tobacco or its ingredients as a factor in causing disease of the stomach or intestines, a recent, critical study was done by Drs. Batterman and Ehrenfeld of New York University, College of Medicine. A careful review of published work has convinced them that "tobacco smoking is not . . . a factor in the causation of peptic ulcer," either of the stomach or of the small intestine. But there is evidence that "smoking may result in functional disturbances which may simulate organic disease or aggravate . . . functional or organic disease." It is particularly the person with an active peptic ulcer who is likely to be harmed by smoke. His response to active treatment will be much less satisfactory than that of the patient who gives up smoking entirely.

Rarely, tobacco smoking may cause a disorder of the eye described as "retrobulbar neuritis," associated with varying degrees of impairment of vision.

In view of the incontestable toxic and irritating effects of tobacco smoking, and in view of the possible connection between heavy cigarette smoking and cancer of the lung, it may properly be asked whether the pleasures and comforts of smoking are worth the candle. To denounce the habitual smoker as a weak-willed, degenerate addict digging his own grave is hardly a reasonable answer to a problem involving millions of normal, well-adjusted persons who are habitual smokers. Although craving is exhibited by heavy smokers when tobacco is removed, there is no evidence that true physical addiction occurs, as with opium drugs. Nor is there evidence that profound psychological disturbances occur when tobacco is withdrawn. According to Professor William T. Salter of the Yale University School of Medicine, "It would seem . . . that tobacco is as habit forming as breakfast coffee, chewing gum, lollipops and several other forms of habitual indulgence."

The issue is not whether tobacco is habit forming—since everyone is agreed that it is. The issue is whether the tobacco habit is so harmful as to require a serious reduction in smoking or even complete abstinence. This issue is not resolved by pointing to Raymond Pearl's famous report of 15 years ago, which showed that beyond the age of 30 only 63% of habitual heavy smokers could expect to survive to the age of 50, as against 81% of abstainers, since it was not at all made clear in that report whether the shortened life expectancy was due to tobacco or to some nervous condition which predisposed toward the use of tobacco.

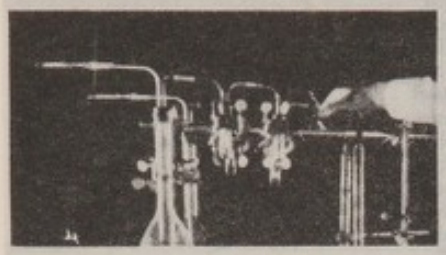
However the habit is acquired, smoking is an activity that serves to reduce the inner nervous tensions and strains resulting from man's exposure to the stresses and responsibilities imposed by society. It helps him to perform more effectively in his work and personal relationships. It would seem to be, at the least, a less harmful method of adaptation than alcohol addiction or even, in many cases, overeating. Many doctors have noted that when a patient successfully gives up tobacco, his appetite increases and he puts on weight. But it is questionable that overeating (and its consequence, overweight) is a more desirable habit than smoking; certainly it is not for some persons with heart or vascular disorders.

To summarize

There are instances, as in Buerger's Disease and similar affections, where the risks of smoking are so serious that despite the satisfaction provided by smoking, it must be sacrificed to avoid loss of limb, sight, or life. In leukoplakia of the mouth or tongue brought on by smoking, the probability of cancer development is serious enough to demand a similar prohibition. In ulcer of the stomach and some other ailments, although nicotine can be an aggravating influence, some physicians, nevertheless, permit moderate use of low-nicotine cigarettes on the ground that any physical irritation is offset by the analgesia smoking may offer against life's problems.

As for cancer of the lung, while it has not been conclusively proved that heavy smoking is a major factor in its rising incidence, the evidence for such an indictment is very suggestive. Until proof is forthcoming, it is obvious that those who can should cut their smoking to what is considered moderate levels—certainly not more than a pack a day. Those who cannot should understand that they are taking a risk of unknown dimensions, and should get an X ray of their lungs every six months.

For those who wish to overcome the tobacco habit, doctors often prescribe strong astringent mouth washes or rinses. Proprietary tablets or lozenges containing similar astringents, as well as nauseating drugs, are also sold. It has never been shown that such preparations are an effective long-range curb on the smoking habit.



Laboratory Tests

37 brands of cigarettes, regular and king size, with both plain and filter tips, were analyzed for the tar and nicotine content of their smoke

The Industry



Under the impetus of the "cancer scare," the tobacco business is going through its second revolution



Medical Aspects

Smoking is often a health hazard, but evidence that it is a major cause of lung cancer is still inconclusive







Here are the results of CU's recent laboratory tests

Will filter-tip cigarettes give you less nicotine and tar than plain cigarettes? What about the king-size cigarette which "travels the smoke further," or king size *plus* a filter? What about the cigarettes which use a low-nicotine tobacco, or a denicotinized tobacco—is there really less nicotine in their smoke? And the "popular" brands—how do they compare with the other brands on the market with regard to their nicotine and tar content?

These are some of the questions to which smokers would like to have answers. But no answers, at least no reliable ones, are to be found in the hazy, happy, healthy world of cigarette advertising. The purpose of this report is to provide some of the answers. In February 1953, CU reported on laboratory tests of 27 widely sold brands of cigarettes, and readers were informed of brand-to-brand, and type-to-type differences in terms of nicotine and tar content. Since that time, however, many new king-size and filter-tip cigarettes have appeared on the market, and some of the older brands have changed their lengths and their filters. CU's tests for nicotine and tar embrace the newcomers as well as the old standbys.

The following report covers tests of 37 brands of cigarettes, purchased in 28 cities throughout the country. They are of four broad types: regular size, filtered; regular size, unfiltered; king-size, filtered, and king-size, unfiltered. Within these types are cigarettes made of American blends, and Turkish cigarettes; low nicotine, denicotinized, and mentholated smokes; regular-price, low-price, and premium-price; plain, and filter-tipped.

Sixteen cigarettes of each brand (each from a different pack) were mechanically "smoked" in a laboratory apparatus in tests for nicotine and tar. The test results are summarized in the table on page 59. Apart from the obvious determinations of how the brands rank in nicotine and tar content, there are a number of interesting conclusions which can be drawn from the figures.

First of all, the test results indicate that the use of tobacco which is low in nicotine results in lower nicotine content in the smoke than regular tobacco even with a filter. *John Alden*, a regular-size cigarette without a filter, had far less nicotine in its smoke than any other cigarette; this brand is made from a special low-nicotine tobacco. Additional information was provided by some check tests: for the five largest selling brands, not only the smoke but the tobacco itself was analyzed for nicotine content. There was a direct correlation between the nicotine content of the smoke and of the tobacco in each case. Because crops of any given variety of tobacco vary in nicotine content from year to year, and from region to region, depending on such things as climatic and soil conditions, the nicotine in the smoke of the cigarettes made from these tobaccos can also be expected to vary. And in most cases it does.

Of the 20 brands included in both CU's previous tests and the current tests, only *John Alden* and *Sano* regular were found to be unchanged in the nicotine content of their smoke. *John Alden* was lowest in nicotine content in both series of tests; it averaged only 0.4 milligram of nicotine per cigarette in the 16 test samples "smoked" by the laboratory apparatus. *Sano* regular also remained at its previously found low nicotine level, although it was not quite as low as the recently introduced *Sano* king size with filter tip. *Sanos* are made of a denicotinized tobacco: that is, tobacco from which a large part of the nicotine has been extracted.

Continued on next page

cigarettes LABORATORY TESTS

Nearly all the other cigarettes tested for both this and the 1953 report were higher in nicotine this time. The nicotine content in the smoke from *Camels* increased from 1.9 milligrams to 3.3 milligrams per cigarette; the regular-size *Old Golds, Lucky Strikes, Philip Morris,* and *Chesterfields* also rose in nicotine content, although not as sharply as *Camels*. The range in nicotine content, except for *John Alden* and *Sano*, was from about 1.5 milligrams (for *L&M* regulars) to 4 milligrams (for *Old Gold* king size), and the range among the five most popular regular-size brands was even less, running from 2.3 milligrams to 3.3 milligrams.

There is no generally accepted definition of the "tars" so much talked about in connection with smoking, and consequently no generally accepted method for their measurement. In its new tests, CU utilized a modification of its previous test method to obtain what is believed to be a more precise determination of the tar content of the smoke; the results, therefore, are not strictly comparable with those obtained in the previous tests. The range in tar content among the 37 brands tested was not as great as the range in nicotine content. By smoking *John Alden*, you can cut your nicotine intake down to 10% of that of the brand highest in nicotine. But the cigarette lowest in tar content (*L&M* regular) will still give you 40% of the tar content of the smoke of the cigarettes highest in tar content.

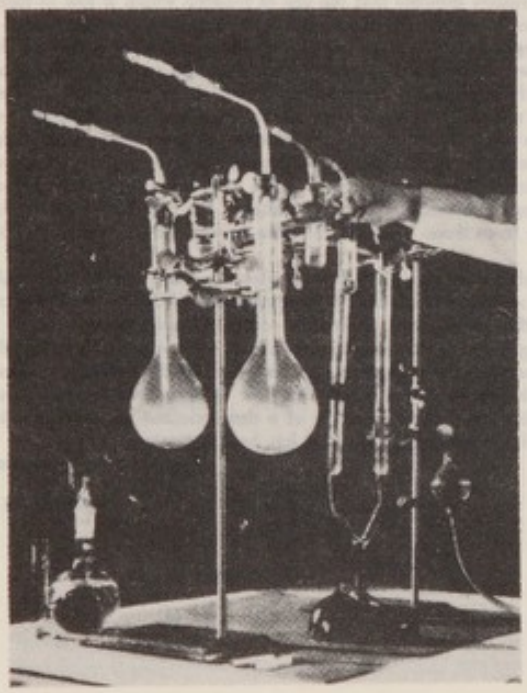
THE FILTER-TIP BRANDS

Open up the filter tip of different cigarettes and you will find a variety of things—absorbent cotton, paper containing activated carbon, cellulose acetate fibers, creped and uncreped paper, asbestos fibers, or combinations o. several of these materials. No laboratory tests were run to determine whether it was the filters, or other factors, that were responsible for high or low nicotine content in each case. The main concern was with the final product—the amount of nicotine and tar in the smoke.

The test results indicate, however, that for a cigarette of given size there was in most cases less tar in the smoke of the filter-tip brands—on the average, about a third less. Within a given size too, the filter-tip cigarettes were also generally lower in nicotine content, although there were several exceptions. The brands whose smoke yielded the

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HOW CU TESTS CIGARETTES





The testing of a cigarette begins with the collection of its smoke. The cigarette is first fitted into a glass holder and then "smoked" in the laboratory apparatus (left). The smoke drawn from the cigarette is collected in acidified alcohol in the flask (any smoke carried into the glass "train" is trapped in the two vials). Lowering the level of the water in the vertical glass tube at the end of the train by means of the bulb at the extreme right of this picture draws air through the cigarette. Each puff is made to last for two seconds and one puff is taken each minute. Slightly more than two cubic inches (35 cubic centimeters) is taken in with each puff, and each cigarette is smoked down to a butt of just under an inch (23 mm.). In CU's

least tar were all filter-tipped. Three of them—*L&M* regular, *Sano* king size, and the premium-priced *du Maurier* regular—were also below the 2 to 4 milligram range of nicotine content into which 80% of all brands tested fell.

Kent, while relatively low in tar, did not fare so well in holding down the nicotine in its smoke. In the 1953 survey, *Kent* was noted as having less nicotine than any other brand with the single exception of *John Alden*. The *Kents* which CU tested for this project had little edge in this respect over most of the other brands tested.

Regardless of the effectiveness of filter tips in keeping nicotine and tars out of your lungs, they have some value in keeping bits of tobacco from getting into your mouth and they do afford a firmer butt.

KING-SIZE CIGARETTES

The smoke of king-size cigarettes contained, on the average, more nicotine and tar than the smoke of regular-size brands when both were smoked down to a butt of 23 millimeters (just under an inch), though here also there were several exceptions. King-size brands with filter

Continued on next page



tests, 16 samples of each brand were "smoked" in two runs of eight cigarettes each; the results of each run were determined separately and then averaged. After each run, the washings from the apparatus are added to the smoke solution in the collecting flask. One portion is steam distilled to isolate the nicotine; amounts are measured with an ultraviolet spectrophotometer (center). Chloroform is added to another portion of the smoke solution, which is then diluted with water. The tar is then extracted by vigorous shaking, first from acid solution, then from alkaline solution, and the chloroform is then drained from separatory funnels. After this, the chloroform is boiled off and the tar residue weighed on a chemical balance (above).

RESULTS OF TESTS FOR NICOTINE AND TAR IN THE SMOKE FROM 37 BRANDS OF CIGARETTES

Brands are listed in order of increasing nicotine content of their smoke. A difference of a few milligrams in tar content is of no significance.

BRAND AND TYPE	AVERAGE NICOTINE IN SMOKE PER CIGARETTE (MILLIGRAMS)	AVERAGE TAR IN SMOKE PER CIGARETTE (MILLIGRAMS)
JOHN ALDEN Low nicotine tobacco John Alden Tobacco	0.4	16
SANO Denicotinized (KF) United States Tobacco	1.0	12
SANO Denicotinized	1.2	15
L&M (F) Liggett & Myers	1.5	11
DU MAURIER (F) Columbia Tobacco	1.7	12
YORKSHIRE Larus & Bro.	1.7	15
MURAD P. Lorillard	1.8	21
KENT (F) P. Lorillard	2.0	12
YORKSHIRE (K)	2.0	18
CHESTERFIELD Liggett & Myers	2.3	17
DU MAURIER (KF)	2.3	16
REGENT (KF) Riggio Tobacco	2.4	19
PARLIAMENT (F) Philip Morris	2.4	14
L&M (KF)	2.5	18
HERBERT TAREYTON (KF) American Tobacco	2.5	19
PARLIAMENT (KF)	2.6	15
CHESTERFIELD (K)	2.7	22
PHILIP MORRIS Philip Morris	2.7	21
LUCKY STRIKE American Tobacco	2.7	21
KENT (KF)	2.8	15
OLD GOLD (KF) P. Lorillard	2.9	14
WINGS (K) Brown & Williamson	2.9	20
OLD GOLD	2.9	18
FATIMA (K) Liggett & Myers	2.9	24
VICEROY (KF) Brown & Williamson	3.0	18
DUNHILL (K) Philip Morris	3.0	23
WINSTON (KF) R. J. Reynolds	3.1	20
HERBERT TAREYTON (K)	3.1	22
CAMEL R. J. Reynolds	3.3	20
KOOL Brown & Williamson	3.4	20
PHILIP MORRIS (K)	3.4	26
PALL MALL (K) American Tobacco	3.5	26
EMBASSY (K) P. Lorillard	3.7	24
KOOL (K)	3.7	23
RALEIGH (K) Brown & Williamson	3.7	25
CAVALIER (K) R. J. Reynolds	3.8	26
OLD GOLD (K)	4.0	25

K = king size; F = filter tip; KF = king size with filter tip. Where no letter follows name, brand is regular size.

cigarettes LABORATORY TESTS

tips were also higher in nicotine and tar than the regular-size filtered brands. King-size cigarettes measure about 85 millimeters compared with about 70 millimeters for regular-length cigarettes. However, if you smoke the same length of a king-size cigarette as you do of a cigarette of regular size (that is, you leave a longer butt), the extra tobacco serves as an effective filter, and the total nicotine from the king-size cigarette will, under such conditions, generally be lower than from a regular-size cigarette.

Not all king-size cigarettes tested were high in nicotine and tar when smoked to usual butt size, however. The relatively low-priced *Yorkshire* king size (available only at Sears-Roebuck retail stores in larger cities) yielded only about as much nicotine as *Kent* regular, and less than any of the regular-size "popular" brands.

Also lower in nicotine than the popular brands was *Murad*, a premium-priced, regular-size Turkish cigarette. *Murad*, however, had an above-average tar content.

A NOTE ON FLAVOR

CU made no attempt to evaluate flavor in any of the cigarettes tested, but a note on this hotly disputed subject may be of some interest. It is now pretty generally agreed —and CU has in the past verified this with mass tests— that in blindfold tests most smokers can't tell the difference between one "popular" brand and another. The situation is a little different when it comes to Turkish blends, special tobaccos, or specially treated or flavored tobaccos. It is widely conceded that a reasonably sensitive smoker can tell one from the other, and can distinguish all of them from the "popular" brands. The preference for one or another of the flavor types seems to be a mixture of personal taste and habit. A number of smokers who tried the low-nicotine *John Alden* commented adversely on its flavor; however, those bent on reducing their nicotine intake without reducing their cigarette consumption might be willing to bear with *John Alden's* initially less-pleasing flavor in the hope that they would get to like it.

CIGARETTE PRICES

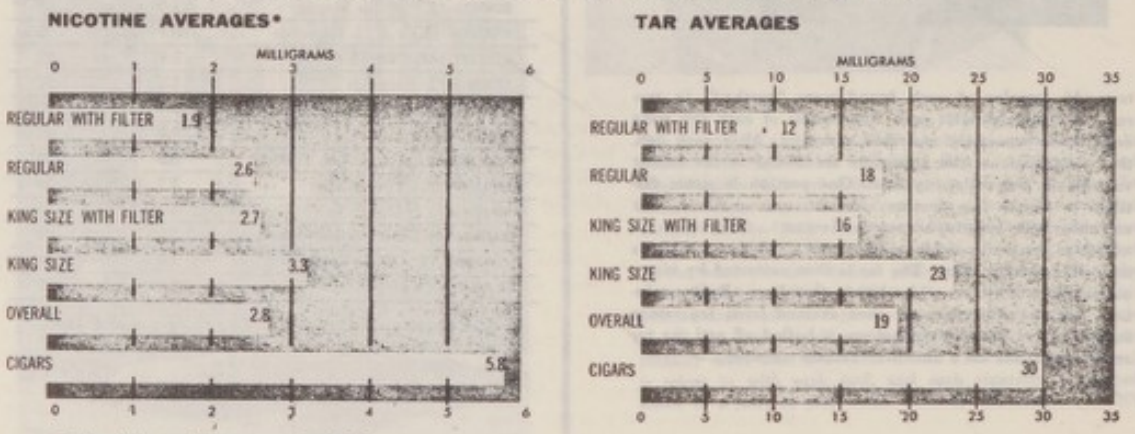
Cigarette prices often vary from store to store. The following prices are representative of those paid by CU's shoppers in New York City. *Murad*, at 37¢ was the most expensive brand tested, with *Parliament* and *du Maurier* (both regular- and king-sizes) close behind at 33¢. Other premium-priced brands included *Kent*, in both sizes, at 31¢. and *Sano* king-size, 30¢. At the low end of the scale were *Yorkshires* (they averaged 17¢ for the regular and 18¢ for the king-size in Sears retail stores throughout the country), and *Wings* at 21¢. Nearly all other brands were priced at 24¢ or 25¢, with some filter-tip and king-size cigarettes at a penny or two more. *John Alden*, the brand lowest in nicotine, was 26¢ a pack.

WHAT ABOUT CIGARS?

Recent medical literature has dealt largely with the hazards of cigarette smoking, but little has been said about cigars. CU ran limited tests, to give readers some information on nicotine and tar in cigar smoke. A random selection of 24 cigars—thin ones, fat ones, long ones. short ones—were "smoked" in the same laboratory apparatus used in the cigarette tests, and the collected smoke of each cigar was analyzed in the same manner.

On the average these cigars, which gave about six times as many puffs as cigarettes, had in their smoke only about twice as much nicotine and one and a half times as much tar. On a weight-for-weight basis, the cigars had on the average only one-third as much nicotine and one-fourth as much tar in their smoke as cigarettes. The relationship of cigars to cigarettes in this respect is shown below.

NICOTINE AND TAR CONTENT OF SMOKE PER CIGARETTE



*John Alden and Sano are not included.

The Industry

Medical reports have upset the cigarette business; regular brands decline as 19 new versions enter the retail arena

The tobacco industry today is undergoing one of the greatest periods of change and upheaval since Americans first started smoking cigarettes before the Civil War." This was the sweeping judgment, as the year 1954 ended, of Lewis Gruber, vice-president in charge of sales for P. Lorillard, one of the "Big Six" tobacco companies which together manufacture more than 98% of the cigarettes sold in America. "Upheaval" is a startling word in the tobacco industry; two years ago it would have been almost unthinkable.

The tobacco industry had its revolution 40 years ago, the revolution which turned it into the *cigarette* industry. The revolution consisted of two fundamental changes in American habits, which began around the time of the first World War: first, a great increase in smoking, especially among women, as the social pressures against it were relaxed; and second, a switch to cigarettes from the older forms of tobacco—pipes, cigars, snuff, and chewing tobacco. This expanding consumer demand, shaped and standardized by a few companies, hoisted cigarettes to a retail position which was simple, secure and gigantic.

Even by the massive standards of the American economy, cigarettes are big business. In 1952—to date, the year of greatest total sales—some 395 billion cigarettes were bought in the United States, an average of 187 packs a year for every American over the age of 15. (Almost 43 billion more cigarettes were sold tax-free, largely to the armed forces.) In this one year, American consumers spent $4,300,000,000 for cigarettes alone.

THE BIG SIX AND THE BIG THREE

Since the 1920s, the cigarette market has been increasingly dominated by a few big companies. In the order of their total sales, the "Big Five" have been: the American Tobacco Company (makers of *Lucky Strike, Pall Mall, Herbert Tareyton*), R. J. Reynolds (*Camel, Cavalier, Winston*), Liggett & Myers (*Chesterfield, L&M, Fatima*), Philip Morris (which, besides its namesake, also makes *Dunhill* and *Parliament*), and P. Lorillard (*Old Gold, Kent, Embassy*). In 1954, Brown & Williamson (*Viceroy, Kool, Raleigh, Wings*) rose to fourth position to make the Big Five the Big Six. The first three—American, Reynolds, and Liggett & Myers—are much the biggest; they account for a whopping 75% of all cigarettes sold in 1954, and are usually grouped in a super-king-size class as the "Big Three."

Each of the big companies won its commanding share of the market with one leader—*Camels, Lucky Strikes, Chesterfields, Philip Morris,* or *Old Golds.* Each of these brands was picked as a "leader" by its manufacturer and the leader got top priority, especially in the company's advertising budget. Each company made a relentless effort to imprint one brand name on the consumer's mind, to make "*Luckies*" or "*Camels*" his sole, automatic response to the idea of "cigarettes." These insistent, "Johnny One-Note" advertising campaigns have been going on for 40 years, and on a very large scale. The cigarette industry spends upwards of 65 million dollars a year on advertising. Among the 20 largest American advertisers have been four tobacco companies, along with such giants as Procter & Gamble, General Motors, General Foods, and Ford. And their advertising paid off; by 1947, the five leading cigarettes held more than 92% of the market. In this "win-or-lose-with one" race, the Big Three entries—*Luckies, Camel,* and *Chesterfields*—came in first, with a combined total of 80% of the market.

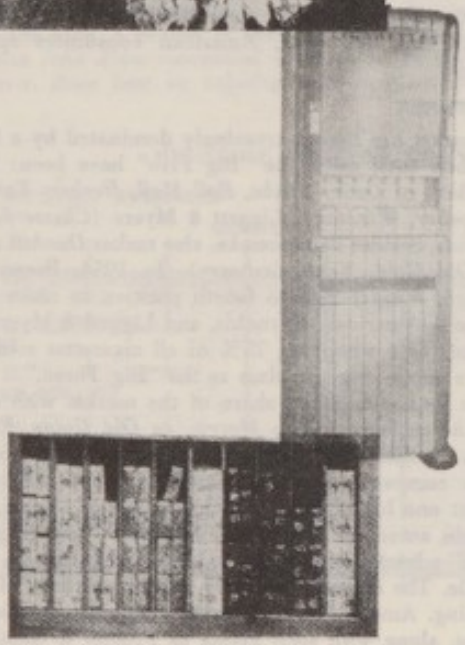
In effect, each of the five big companies was selling the American smoker one

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cigarettes THE INDUSTRY





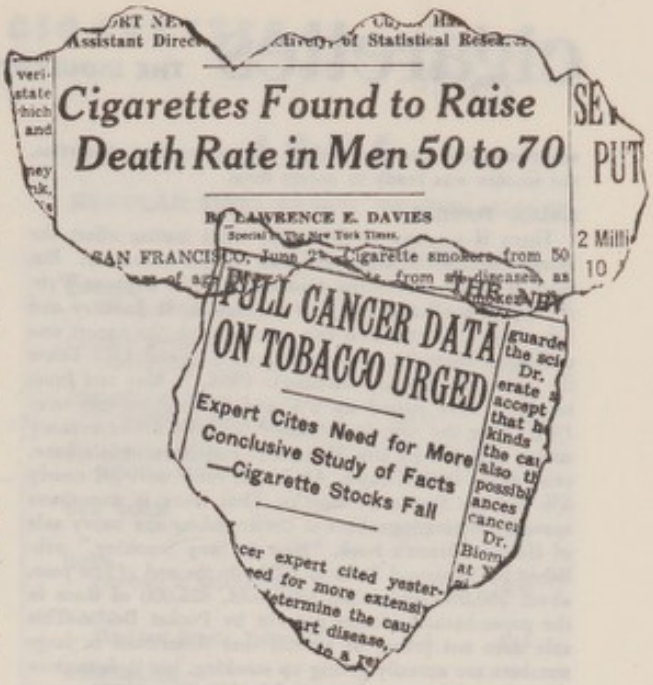


product—and, basically, their five products were the same. In simplifying the cigarette market, the big companies had also standardized it: whatever its name, the "American cigarette" was a plain cylinder of thin paper, 70 millimeters long, filled with a mixture of burley and flue-cured tobacco. Among the five brands there were, of course, differences in the proportion of burley to flue-cured, in the small amounts of Turkish tobacco mixed with this basic filler, in added flavorings, and in "processing." The resulting differences in taste and quality of smoke were supposed to be the basis for the consumer's brand loyalty. It is true that many smokers believe in these "differences," and can describe them, usually in vague terms, though they may frequently contradict each other as to which brand is "mild," which is "strong," which "hurts my throat," etc. Blindfold tests indicate, however, that most smokers cannot tell, by taste alone, whether they are smoking their favorite big-name brand, or another. Nevertheless, the "differences" have been stressed vociferously in big-brand advertising. So simple and standardized a product did not permit any other kind of competition—except, perhaps, in price.

NO PRICE COMPETITION

Despite their almost frantic appeals for public acceptance, however, the big brands have never tried to compete in price. When one of the Big Three has moved its price up or down, the other companies have promptly done the same. In the depths of the depression, smaller tobacco companies launched cut-rate brands (such as *Wings, Avalons* and *Marvels*) which sold at 10¢ a pack—3¢ under the regular brands—and for a brief time in 1932 captured 22% of the market. The big companies, which had raised their prices in 1931, answered this challenge by a uniform price drop. (According to testimony in a successful federal antitrust suit against them, they also bought up the kind of tobacco used in the cut-rate cigarettes and hoarded it.) This solidarity in pricing among the dominant Big Three was condemned by the courts in 1946 as collusion. But prices remained identical. Competition in price was left to the retailers. By reducing their own margin of profit, the small retailers can reduce the price of cigarettes a cent or two per pack. Thus, they can compete—but only against each other. When supermarkets or drugstores cut the prevailing local price, it is almost always by the same amount on all regular brands.

What reason was there, after all, for the big companies to lower their price? There was certainly no resistance by the consumer. The market for cigarettes kept on expanding, despite a series of price hikes, which, in the last 20 years, have raised the price per pack of the big selling brands from 13¢ to 25¢. In fact, it was during this same period of inflation and higher federal cigarette taxes that the industry scored its most spectacular gains. Between 1932 and 1952, the number of cigarettes sold almost quadrupled, and industry spokesmen were confidently predicting sales rises of 4% to 6% a year.


Assistant Direc… …ivery of Statistical Rese…

Cigarettes Found to Raise Death Rate in Men 50 to 70

By LAWRENCE E. DAVIES

Special to The New York Times.

SAN FRANCISCO, June 2… Cigarette smokers from 50 … of age … from all diseases, as …

FULL CANCER DATA ON TOBACCO URGED

Expert Cites Need for More Conclusive Study of Facts —Cigarette Stocks Fall

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SE… PUT

2 Milli… 10 …

At the beginning of 1953, then, the industry seemed to be safe against any upset: 1952 had been the peak year for sales; cigarettes were an "essential" to a vast, and apparently growing, sector of American life; the market was dominated by a few prosperous companies, selling their product at a standard price, and with half-a-dozen brands which bore a comforting resemblance to one another.

What has happened, in only two years, to cause so much concern in the industry? Briefly, three developments of major importance to the consumer:

1 A series of reports which, according to the scientists and statisticians who prepared them, imply that there is a significant association between the rising incidence of lung cancer and the smoking of cigarettes.

2 For two years running, a significant drop in the number of cigarettes sold—the first since 1932; in 1953, cigarette consumption fell by 2.9%, and another drop of about 4.6% is expected to be shown for 1954.

3 A big change-over by smokers from "regular" brands to king-size cigarettes (85 millimeters long), and a marked shift to those with some kind of filter tip.

Since these three developments all took place during roughly the same period of time, the big question is: are they related? The recent "health" attacks on cigarette smoking are evaluated in a special report on pages 67 to 73. The point here is: what effect have they had on the industry —and the consumer?

THE CANCER SCARE

The industry's first reaction came after the publication in December 1953 of the report by Dr. Ernest L. Wynder and Dr. Evarts A. Graham, "Experimental Production of Carcinoma [cancer] with Cigarette Tars." This medical report was quickly "translated" for the public by an outburst of stories in newspapers and such mass-circulation magazines as *Life* and *Time*. On January 4, 1954, a full-page advertisement in 448 newspapers announced the formation of the Tobacco Industry Research Committee to investigate the relation of tobacco to health. The Committee's members were 14 tobacco companies and associations—growers, warehousers, and manufacturers—and included all the Big Six, with the exception of Liggett & Myers. The Committee followed up the ad by naming a scientific advisory council headed by Dr. Clarence Cook Little, founder and director of the Roscoe B. Jackson Memorial Laboratory at Bar Harbor, Maine.

Since then, the TIRC has not been publicly active. Dr. Little's occasional statements have followed the "wait-and-see" line taken in the first ad, namely, that no single cause of lung cancer has been established, and that there is no proof of a cause-and-effect relationship between smoking and lung cancer. In November of last year, the TIRC made its first six grants (totaling $82,000) for research projects involving the composition of tobacco, the physiology of smokers, and other pertinent subjects.

Industry spokesmen have followed the "not proven" line. They imply that the current medical controversy is only the latest in a long series of "health scares" which have blown over in the past. But business and trade journals generally agree that during the last year and a half the cigarette business has received the most damaging publicity in its history. And last November, this was virtually confirmed by one of the Big Three, when E. A. Darr, president of Reynolds, accused the American Cancer Society of attempting "to destroy the tobacco industry."

INDUSTRY HEALTH CLAIMS

The industry does not admit in public that "health" publicity may have caused the unprecedented drop in cigarette sales. But critics close to the industry—for example, in the advertising trade journals—have said flatly that it is fear which is causing Americans to turn away from cigarettes. They also say that the companies themselves are largely to blame. Long before the current medical attacks, the companies were building up suspicion in the consumer by the discredited "health claims" in their ads. For a generation, the companies have been insisting, in big red letters, that their particular brand had "less nicotine," was "less irritating," "guarded against throat scratch," was "safe in your T-zone," and so on, to the outer limits of the copywriter's imagination and the Federal Trade Commission's patience. Such medicine-show claims may have given the smoker temporary confidence in one brand, but they also implied that cigarettes in general were distasteful, probably harmful, and certainly a "problem." When the

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scientists came along with their charges against cigarettes, the smoker was ready to accept them.

SALES TUMBLE

There is no way to determine what lasting effect the recent "health" attacks have had on the consumer. But there is pretty convincing evidence that the Graham-Wynder report made a strong first impression. In January and February, 1954, the first two months after the report was published, cigarette sales went down 9% and 17% below the figures for the same months in 1953. In May and June, however, sales moved up 2% and 8% respectively over 1953. After the American Cancer Society's life-expectancy survey reported by Drs. Hammond and Horn in late June, sales again turned down. All in all, sales were off nearly 4% for the first nine months. That there is uneasiness among American smokers is confirmed by the heavy sale of Herbert Brean's book, "How to Stop Smoking," published by Vanguard Press in 1951. By the end of last year, about 500,000 copies had been sold, 425,000 of them in the paper-backed edition put out by Pocket Books. This sale does not prove, of course, that Americans in large numbers are actually giving up smoking, but it does show that a lot of them are interested in doing so.

THE NEW LOOK IN CIGARETTES

The industry may appear sluggish, or just cautious, in its reaction to the medical attacks, but it has a lot of merchandising fight left, as shown by its quick response to the new consumer demand for king sizes and filter tips. Last year, at least 19 new brands, or new versions of old brands, came on the market. All of these new cigarettes were either king size or filter tip, and 11 of them were both. This is "fast service" indeed for the restless or discontented smoker. It is also good business for the tobacco companies, since the "new look" cigarettes are the growing sector of an otherwise shrinking market.

For the last seven years, the tobacco industry has watched the demand for the Big Three brands—*Camels*, *Luckies*, and *Chesterfields*—steadily contract. In 1947, over 80% of all the cigarettes sold bore these familiar names. By 1953, the Big Three regulars' share of the market had gone down to 58%. This 22% decline meant a loss in retail sales of about a billion dollars.

The decline also made more striking the rise of *Pall Mall*, a king-size cigarette which American Tobacco brought out in 1939 at a "popular" price, and promoted heavily. By 1947, *Pall Malls* had attracted a modest 3.4% of the market. By 1953, the *Pall Mall* share had quadrupled to 12.4%, and this king-size entry had passed both *Old Golds* and *Philip Morris* to become the nation's fourth largest seller. In 1954, *Pall Mall* passed regular-size *Chesterfields* to become the third largest seller.

Although American Tobacco was losing business with its old leader, *Lucky Strike*, it was more than regaining it on *Pall Mall* and another king size, the cork-tipped *Herbert Tareyton*. This new way to balance sales did not escape the notice of rival companies, all of whom put out longer smokes of their own—*Cavalier* (Reynolds), *Fatima* (Liggett & Myers), *Dunhill* (Philip Morris), and *Embassy* (Lorillard).

MORE KINGS APPEAR

Three years ago, Liggett & Myers made another concession to the king-size demand. It offered *Chesterfield* smokers a choice between king size and regular size, under the same brand name. The king-size *Chesterfields* were marketed as merely a longer version of the old cigarette, in the familiar white and gold package. In 1953, three other old favorites also appeared in the "new look"— *Philip Morris*, *Old Gold* and *Raleigh*. *Raleigh* smokers were given no choice about going king size; the regular-length *Raleigh* was taken off the market.

Clearly, the number of smokers who want king size is growing—whether it is because they feel they are getting more health protection, a more "distinguished" look, or just a longer smoke for their money. In 1953, the year when total cigarette sales first fell, extra-length brands had grown to almost 26% of the market, as against 18.5% in 1952. Last year, the figure jumped again, to an estimated 30%.

The king-size conversion has been gathering momentum for some time, but with the impact of cancer fears, the change to filter tips was abrupt, a real "rush." In 1952, only 1.4% of the cigarettes sold were filter-tipped; in 1953, the number more than doubled, to 3.4%; when the returns for 1954 are in, experts estimate that the filter brands will have 10%, and possibly 12%, of the total market. Lorillard's Lewis Gruber flatly predicts that within two more years, 40 of every 100 smokers will be smoking filtered cigarettes, most of them king size.

WHAT IS A FILTER TIP?

What is this "revolutionary" product? It is simply a cigarette in which about one-half inch of the butt has been filled with a filtering substance instead of tobacco. Most of the filters are made of finely creped (crinkled) paper, absorbent cotton, and cellulose-acetate fibers, either alone or in combination. Some have added "purifying agents," such as *Herbert Tareyton's* activated charcoal, and *Kent's* patented Micronite, an asbestos-like substance.

Filter cigarettes are not new; they have been on the American market for some time in *Parliament* and *Du Maurier*, which sold at a premium price (32¢ or more

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CHANGES IN THE CIGARETTE MARKET

The tables on the facing page show how the cigarette market has changed in two years. The halftone areas backing the tables show the trend, not exact percentages: the figures for 1954 include estimates for the last months of the year and do not distinguish between regular- and king-size filter-tip brands

SALES OF CIGARETTES

1952

REGULAR SIZE: 81.56% OF THE MARKET

	BILLIONS OF CIGARETTES	% OF MARKET
CAMEL (R. J. Reynolds)	113.7	26.0
LUCKY STRIKE (American Tobacco)	82.2	18.8
CHESTERFIELD (Liggett & Myers)	65.8	15.1
PHILIP MORRIS (Philip Morris)	40.4	9.2
OLD GOLD (P. Lorillard)	23.6	5.4
KOOL (Brown & Williamson)	11.5	2.6
RALEIGH (Brown & Williamson)	8.3	1.9
DOMINO, YORKSHIRE (Larus & Bro.)	2.6	0.6
MARLBORO (Philip Morris)	0.5	0.1
PIEDMONT, SPUR (Liggett & Myers)	0.2	0.05
VIRGINIA ROUNDS (Benson & Hedges)	0.06	0.01
EXPORT BRANDS (Brown & Williamson)	5.3	1.2
ALL OTHERS	0.9	0.2

KING SIZE: 17% OF THE MARKET

PALL MALL (American Tobacco)	45.5	10.4
HERBERT TAREYTON (American Tobacco)	12.8	2.9
CHESTERFIELD (Liggett & Myers)	8.5	1.9
FATIMA (Liggett & Myers)	3.0	0.7
CAVALIER (R. J. Reynolds)	1.3	0.3
DUNHILL (Philip Morris)	1.0	0.2
EMBASSY (P. Lorillard)	0.8	0.2
WINGS (Brown & Williamson)	0.7	0.2
REGENT (Riggio Tobacco)	0.55	0.1
HOLIDAY, LORDS (Larus & Bro.)	0.5	0.1

FILTER TIP: 1.3% OF THE MARKET

VICEROY (Brown & Williamson)	2.7	0.6
PARLIAMENT (Benson & Hedges)	1.66	0.4
ENCORE (United States Tobacco)	0.7	0.2
KENT (P. Lorillard)	0.5	0.1

1954

REGULAR SIZE: 63.22% OF THE MARKET

	BILLIONS OF CIGARETTES	% OF MARKET
CAMEL (R. J. Reynolds)	89.8	22.3
LUCKY STRIKE (American Tobacco)	65.2	16.2
CHESTERFIELD (Liggett & Myers)	42.0	10.4
PHILIP MORRIS (Philip Morris)	24.8	6.1
OLD GOLD (P. Lorillard)	15.6	3.9
KOOL (Brown & Williamson)	12.1	3.0
SANO (United State Tobacco)	0.4	0.1
MARLBORO (Philip Morris)	0.3	0.07
EXPORT BRANDS (Brown & Williamson)	4.5	1.1
ALL OTHERS	0.2	0.05

KING SIZE: 27.06% OF THE MARKET

PALL MALL (American Tobacco)	54.5	13.5
CHESTERFIELD (Liggett & Myers)	15.5	3.8
HERBERT TAREYTON (American Tobacco)	11.3	2.8
PHILIP MORRIS (Philip Morris)	8.2	2.0
RALEIGH (Brown & Williamson)	6.5	1.6
OLD GOLD (P. Lorillard)	5.0	1.2
CAVALIER (R. J. Reynolds)	4.1	1.0
DOMINO, YORKSHIRE (Larus & Bro.)	2.3	0.6
FATIMA (Liggett & Myers)	1.0	0.25
DUNHILL (Philip Morris)	0.4	0.1
EMBASSY (P. Lorillard)	0.3	0.07
REGENT (Riggio Tobacco)	0.3	0.07
HOLIDAY, LORDS (Larus & Bro.)	0.2	0.05
WINGS (Brown & Williamson)	0.1	0.02

FILTER TIP: 9.64% OF THE MARKET

VICEROY (Brown & Williamson)	14.9	3.7
L&M (Liggett & Myers)	7.7	1.9
WINSTON (R. J. Reynolds)	7.5	1.9
KENT (P. Lorillard)	2.5	0.6
PARLIAMENT (Philip Morris)	2.3	0.6
HERBERT TAREYTON (American Tobacco)	1.5	0.4
MARVEL (Stephano Bros.)	1.5	0.4
OLD GOLD (P. Lorillard)	0.5	0.1
REGENT (Riggio Tobacco)	0.1	0.02
ENCORE (United States Tobacco)	0.1	0.02

Statistics reprinted from Printers' Ink, December 31, 1954. *Copyright 1954 by Printers' Ink Publishing Co., Inc., N. Y.*

cigarettes THE INDUSTRY

a pack in New York City, where regular brands have been selling at 24¢ to 25¢), and which took a distinctly ritzy tone in their ads. As long ago as 1936, Brown & Williamson introduced the regular-size filter-tip *Viceroys*, at only a cent or two above the price of regular brands. *Viceroys* and *Parliaments* divided most of the insignificant filter-tip market until 1952. Then Lorillard offered *Kents*, a premium-priced filter-tip which was promoted and distributed with big-brand energy. The Big Three made its first entry into the filter market in 1953, when Liggett & Myers launched *L&Ms*, also at a premium price.

THE FILTER BAND WAGON

The other big companies, which had stood aloof from this "small-time" market, suddenly joined in the scramble last year. In March, Philip Morris bought out Benson & Hedges—and its successful filter-tip brand, *Parliaments*. A month later, Reynolds started off with an exclusive king-size filter-tip *Winston*, which was described in ads as "made by the makers of *Camels*," an appeal to old brand loyalties. Finally, in August, American Tobacco took the field with a filter version of its king-size *Tareyton*, in the same white package, but distinguished by thin red and blue stripes across the lower left corner. Both these new filter tips sold at 26¢, the "popular" price for filter-tips.

Filters have restored price-competition to the industry, if only temporarily. In March, the price of *L&M* was cut by 4¢, dropping both the regular and new king-size style out of the premium class and into the popular arena. *Kent*, which Lorillard says can never compete on the lower price level because of the high cost of its Micronite filter, went to king size. Some observers thought this was indirect price competition, as well as an invitation to smokers of king-size brands. *Parliaments*, another premium product, showed its sensitivity to the price factor in September by cutting the price on its king size by 2¢.

THE PRICE OF FILTERS

Price competition thus far has not brought filter-tips down to the price level of regulars and some king sizes. "Popular price" for filter-tip brands still means around 26¢, a cent or two more than the "popular price" for plain cigarettes. Leaf tobacco accounts for about half the cost of a cigarette, and it is doubtful that the material in the average filter costs any more than the tobacco it replaces. Putting the two elements—filter and tobacco—together in one cigarette, however, is a more complicated process. (The trade magazine *Sales Management* reports that Lorillard had to import machinery from Europe to produce the first *Kents*.) Also, the industry claims the production machinery for filter cigarettes is slower and more subject to mishaps. One big-company executive says that a regular machine can turn out around 1200 cigarettes a minute, but that he has yet to hear of a filter-tip machine doing better than 750.

Though the cigarette companies have subdued the scare-head health advertising of the regular brands (the ads no longer proclaim that "more doctors smoke *Camels*" but rather that "*Camels* agree with more people than any other cigarette"), in their filter-tip advertising, they seem to be working both sides of the street lest the smoker feel that in protecting his health, he will not enjoy smoking. Thus *Tareyton's* filter "holds back elements that can detract from the pleasure of smoking," and *Old Gold Filter Kings* proffer "a treat you can trust." *Winston* "the filter cigarette with flavor . . . tastes good—like a cigarette should."

The Federal Trade Commission has been trying for years to temper the more blatant assertions of the cigarette advertisers, but if there has been any recent amelioration, it has been the result of the industry "upheaval" rather than of legislative or judicial action. In late 1952 and early 1953, the courts rejected FTC's claim that cigarettes should come under the Federal Trade Commission Act. In September 1954, the FTC tried a different tack and submitted to the cigarette companies a draft code of advertising standards. This code would be entirely voluntary and without the force of law. But though meetings have been held to discuss it, no cigarette manufacturer has yet indicated his willingness to accept even this dilute regulation.

HOW TO SELL CIGARETTES

Such, then, is the "upheaval"; where does it leave the huge cigarette industry at the beginning of 1955?

Cigarettes are still the major product of the industry. Some of the medical reports which condemn cigarettes also tend to exonerate pipes and cigars, but thus far there has been only a minor rise in the sale of these two forms of tobacco. In the near future, the companies must make up their losses on regular brands with increased sales of filter-tipped and king-size brands.

The manufacturers must get their new products in front of as many consumers as possible, as quickly as possible. Distribution of a number of brands can be costly, and difficult. Retailers complain at the extra space for new brands which must be found on their crowded shelves and counters, at the extra bookkeeping, at the extra dollars tied up in their inventories of slow-selling items. Space is also a major problem for the estimated 500,000 cigarette vending machines in the United States. Heretofore, most vending machines had ten columns, and could sell, at most, ten different brands. To offer their customers even a minimum sampling of the present array, the vendors have to install two machines in one location, or else invest in the new 20-column type. Differences in price are also a problem for the machine distributors, since the machines can be paid only in standard coins.

The consumer's taste is changing fast, and no manufacturer can be sure what direction it will take. In 1953, 21 brands were listed as "significant" (that is, as selling over a billion cigarettes a year); by July 1954, the number had gone up to 30. Each company will have to make hard decisions on which of its brands to push, which to modify, and which, perhaps, to discard. The industry seems to feel that already there are too many varieties on the market to survive, and that a "weeding out" is inevitable.

Medical Aspects

A survey of current medical knowledge on the relationship between the smoking of cigarettes and cancer of the lung

Every year for about 30 years the frequency of cancer of the lung as a cause of death has progressively increased. Today many serious medical investigators are convinced that the increase in frequency of lung cancer is closely related to the smoking of cigarettes. The concern with cigarettes as a medical problem has called forth other research to determine the relation between smoking and heart trouble; smoking and peptic ulcer; the particular ingredients in tobacco which may be harmful; the extent to which cutting down on tobacco intake will diminish the risk of disease; and the psychological and physiological effects of the tobacco habit. But it is the relation between cancer of the lung and smoking which has stirred up the most controversy.

Regular cigarette smoking is believed by such leading American surgeons as Dr. Evarts Graham and Dr. Alton Ochsner, by investigators and physicians in medical schools and health departments, and by the American Cancer Society, to be an important or major factor contributing to the development of cancer of the lung. Many physicians in private practice are apparently convinced that the relation between regular cigarette smoking and cancer of the lung is now proved. They have discontinued their own cigarette smoking and also advise their patients to cut out or cut down their smoking.

The tobacco industry has met the challenge in part by setting up a medical research program of its own, headed by Dr. Clarence Cook Little of the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine. This project is still too new to have yielded any significant data, and its formation has been criticized as resembling burglar-financed research toward the perfection of a crackproof safe. However, several independent criticisms of the cigarette theory of lung cancer have been advanced by outstanding investigators, the foremost being Dr. W. C. Hueper, Chief of the Environmental Cancer Section of the National Cancer Institute in Bethesda, Maryland. Dr. Hueper and some other experts regard the evidence linking lung cancer and cigarette smoking as insufficient or contradictory, and the theory generally as not proven.

No one has claimed that regular cigarette smoking is *the* cause of lung cancer. Many factors or "causes" can operate to produce cancer. Many "carcinogens"—cancer-producing agents—pervade our environment. Those who consider smoking an important or major factor contributing to the development of cancer of the lung say merely that tobacco smoke inhaled into the lungs is one of the carcinogens and possibly the most important.

Since World War I thoracic surgeons and physicians specializing in diseases of the lung have been impressed by the frequency with which they have encountered cancer of the lung among patients who gave a history of heavy cigarette smoking. When this association between smoking and lung cancer became so common that it could no longer be ignored, and when statistics indicated that cancer of the lung was becoming a leading cause of death in men, groups of investigators in the United States, England and other countries began to study 1) whether the increase in the frequency of lung cancer was real or apparent, and 2) whether its association with regular cigarette smoking was more than coincidence.

Prior to 1900, cancer of the lung was infrequently recorded in post-mortem studies and even more rarely diagnosed during life. After 1900, with the increasing use of chest X rays and the bronchoscope, cancer of the lung began to be diagnosed

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cigarettes MEDICAL ASPECTS

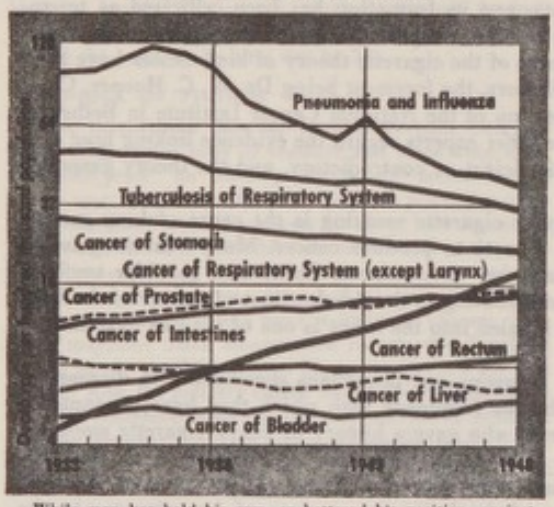
more frequently and to be observed more regularly in post-mortems. Although the progressive increase in the incidence of cancer of the lung has not been the same in different countries, the disease is now recognized as a leading cause of death on all continents and in every country, or at least wherever there are adequate diagnostic and pathological facilities and where vital statistics are gathered.

Some doctors argued at first that the increase in reported cases of cancer of the lung merely reflected an improvement in diagnostic techniques. But the recorded increase in cancer of the lung is greater in males than in females (from two men for every woman in 1933 to 1936 to an estimated ten for one today), and it is hard to believe that physicians are better able to diagnose lung cancer in men than in women.

THE INCREASE IN LUNG CANCER

During 1951, nearly 20,000 deaths in the United States were attributed to cancer of the lung—1.3% of all deaths during that year, and 9.2% of all deaths charged to cancer. According to Dr. Charles Cameron, medical director of the American Cancer Society, "Cancer of the lung is showing the most rapid increase ever ascribed to any non-infectious disease in medical history." Since cancer of the lung usually starts in the surface lining of the bronchial tubes of the lung, the carcinogens must be those that are inhaled into the bronchial tubes and lungs. Among suspect inhaled materials are industrial fumes; industrial and domestic soot derived from coal and fuel oil furnaces; exhausts from internal combustion machines (gasoline and diesel); asphalt or bituminous road surface dust; and tobacco smoke. All these materials are believed to have cancer-inducing properties.

DEATH RATES FOR SELECTED DISEASES AMONG WHITE U. S. MALES



While man has held his own, or bettered his position, against many fatal diseases, lung cancer deaths have risen sharply

THE SEARCH FOR CAUSES

Up to this point there is no disagreement among the health experts. The controversy starts when doctors estimate the relative importance of these environmental materials in causing cancer of the lung. The conflicting reports so far released have been based on animal experiments, on medical studies in human beings, and on statistical analyses.

The cancer-inducing effects of industrial and other air pollutants have been studied experimentally in laboratory animals. These experiments have yielded conflicting data, particularly because of the difficulty of finding animals which develop lung tumors comparable to those in man. Experiments with the smoke of tobacco and cigarette paper have also yielded inconclusive data. One group of experiments showed that benzpyrene, a chemical long known for its carcinogenic effects, is not only present in the smoke from cigarette paper, but can actually cause cancer of the lung in certain strains of mice. This report has been highly publicized especially by those who would like to see the cancer onus removed from tobacco itself.

In general, animal experiments have been of doubtful value to investigators of human lung cancer. Dr. Cameron says: "The problems of carcinogenesis [origin of cancer] . . . are extremely complex and far from understood as yet. What will cause cancer in one species will not necessarily cause it in another. What will cause cancer in one tissue of a given animal will not necessarily cause it in another tissue of the same animal. Thus it is conceivable that tobacco smoke does contain an agent which is carcinogenic for the lungs of human beings. It may not be so for the lungs or any other tissue of a mouse, or a guinea pig, or a dog. Here it is appropriate to point out that no one has ever succeeded in producing cancer in an experimental animal with chromium or any chromium-containing compounds, yet the statistical evidence that chromates can cause cancer of the lung in workers occupationally exposed to them is conceded by everyone."

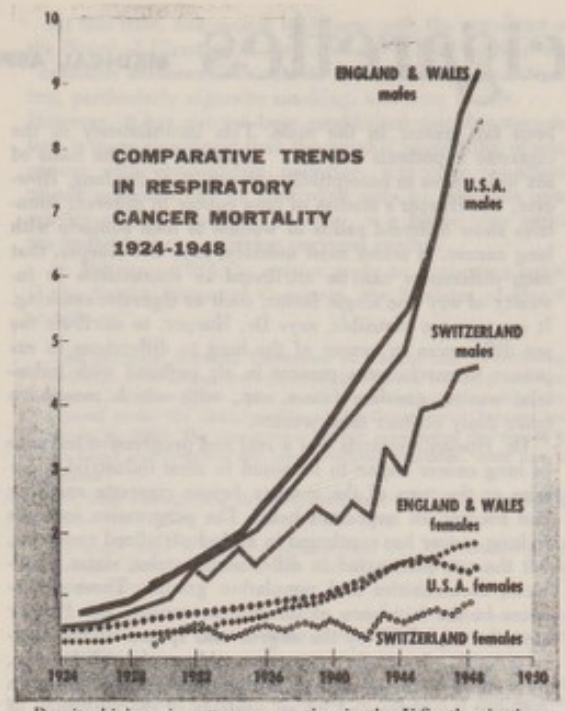
STATISTICAL EVIDENCE

Since actual experimentation on living human beings is ruled out by the nature of the disease, researchers have fallen back on a technique known in public health as "epidemiology." Broadly speaking, epidemiology deals with the mass behavior of all diseases, not just acute epidemic infectious diseases. Statistical evidence is always a very important part of the study of the epidemiology of a disease. According to Dr. Leonid S. Snegireff, Associate Professor of Cancer Control of the Harvard School of Public Health: "The epidemiologist often finds puzzling situations which he can solve by applying epidemiological principles without first knowing all the detailed pieces of the puzzle. For example, experience with many diseases shows that exact knowledge of the specific agent which causes a given disease process is not essential for the achievement

of effective control over morbidity and mortality in a particular disease." Thus, to cite a few examples, the last major cholera epidemic in Great Britain was brought under control in 1866 by appropriate measures of sanitation and purification of water supply—17 years before Dr. Koch isolated the cholera vibrio germ. The precise cause of rheumatic fever is not known, yet there are effective measures for controlling the disease. The precise cause of dental caries is not known, yet there is no doubt that fluoridation of the water supply can help control the disorder. It is not necessary to await the isolation and identification of all the carcinogenic agents in our environment, to point the way to halt the rapid increase in the mortality from cancer of the lung. Of all possible environmental carcinogens, tobacco smoke is one of the few that the individual can control. According to Dr. Morton Levin, Assistant Commissioner for Medical Services of New York State and a leading student of the epidemiology of cancer of the lung, "There is already more than enough evidence incriminating cigarette smoking to justify advising the public to stop smoking cigarettes as a means of lowering the incidence of lung cancer" (Problems in the Study of Occupation and Smoking in Relation to Lung Cancer, Drs. Morton L. Levin et al. October 11, 1954). This point of view is shared by others, here and abroad.

What is the statistical evidence of a causal relationship between smoking and cancer of the lung? Since 1950 more than a dozen independent studies have reported a disproportionately high percentage of heavy cigarette smokers among lung cancer patients. Most of these studies are what is known as "case-history" or "retrospective" studies. The tobacco intake of patients in whom cancer of the lung has already been diagnosed is compared with that of control groups (non-cancer patients) representative of the population from which the cancer patients were drawn. All these studies have been interpreted as demonstrating a greater liability to cancer of the lung in regular cigarette smokers, to cancer of the lip in regular pipe smokers, and to cancer of the mouth in regular cigar smokers—a liability that increases with the quantities smoked.

Because of certain weaknesses in the design of retrospective or case-history studies, the American Cancer Society, the British Research Council and individual investigators have also undertaken statistical studies by the "prospective" or "population" method. Preliminary reports of the studies of the American Cancer Society and the British Medical Research Council are available and they confirm in their broad features the results of the case-history studies. From all these studies, says Dr. Alexander G. Gilliam of the National Cancer Institute, "It may now . . . be regarded as an established fact that white, male cigarette smokers in England and the United States suffer a substantially greater risk to cancer of the lung than non-smokers. The evidence from other countries is less convincing and complete and no substantial data bearing directly on this question are yet available for non-white population or for female population." However, according to Dr. Hueper, a world-renowned authority on environmental carcinogens, "the cigarette theory is almost entirely based on statistical data having at best circumstantial value and being in part of questionable origin." (Address before American Pharmaceutical Manufacturers Association, December 10, 1954.)



Despite higher cigarette consumption in the U.S., the death rate from cancer of the lung, larynx, and sinuses is higher in England

In support of the cigarette smoke thesis it is stated that both in England and in the United States an increase of per capita cigarette consumption, particularly by men, has generally paralleled the trend of mortality in cancer of the lung. This evidence is, however, disputed by Dr. Hueper and others. Dr. Hueper points out that the age-adjusted rate of increase in the frequency of lung cancer deaths was greater between 1914 and 1930 than between 1931 and 1944. He believes that the rapid increase in cigarette smoking during the Thirties and Forties should have given the later period a greater increase in lung cancer fatalities if cigarette smoking were a significant factor.

Women as a group did not begin to smoke cigarettes regularly until well after World War I. Since then there has been a relatively greater increase in smoking by women than by men, but the relative increase in lung cancer has

Continued on next page

cigarettes MEDICAL ASPECTS

been far greater in the male. This inconsistency in the cigarette hypothesis has been "explained" on the basis of sex differences in susceptibility to cancer of the lung. However, Dr. Hueper's studies of lung cancer in different countries show different ratios of women to men subjects with lung cancer. It seems most unlikely, says Dr. Hueper, that such differences can be attributed to fluctuations in intensity of any one single factor, such as cigarette smoking. It seems more plausible, says Dr. Hueper, to attribute the sex differences in cancer of the lung to differences in exposure to carcinogens present in air polluted with industrial wastes, gasoline fumes, etc., with which men have more daily contact than women.

Dr. Hueper contends that a real and progressive increase in lung cancer began to be noted in most industrial countries at the turn of the century *before* cigarette smoking had become an important habit. The progressive increase in lung cancer has continued in all industrialized countries, but the rate has varied in different countries, states, provinces, communities and population groups. These differences in the incidence of the disease cannot, Dr. Hueper says, be explained by the degree and spread of the cigarette habit. It *can* be more reasonably explained, he says, by a study of the growth of industry and transportation in different countries and by an analysis of the multitude of carcinogenic agents released into the air. Carcinogenic materials are believed to be present in effuents of domestic and industrial furnaces, exhaust of gasoline and diesel engines, dust from rubber tires and asphalted and oiled roads —in other words, in the air breathed by residents and workers in urban-industrial regions. Thus the marked and growing predominance of males among lung cancer victims could be traced to the following factors:

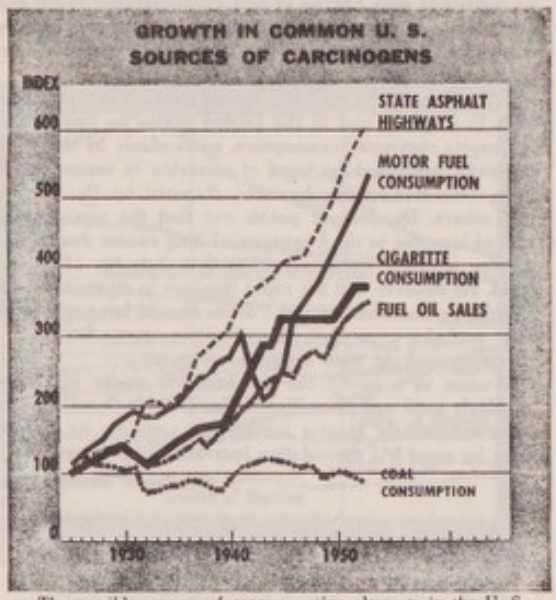
1 Males are more extensively employed than females in occupations where known or suspected atmospheric carcinogens are produced and used, and they work in such jobs more consistently over longer periods of their lives.

2 Males predominate in outdoor occupations, especially in urban areas, where they are exposed to carcinogenic pollutants contained in the general atmosphere.

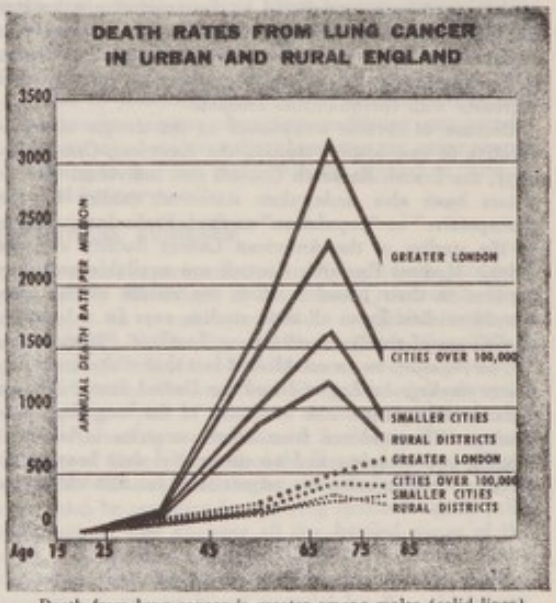
3 Males far more often than females perform heavy physical labor requiring deep and rapid breathing, and consequent greater penetration of air pollutants into the lungs.

4 Males more often than females work through the entire span of their occupational lives within urban areas with proven carcinogenic atmospheric pollution, while females tend to remain in the cleaner residential areas.

All studies have shown a remarkable drop in lung cancer incidence when the researchers turn from urban-industrialized areas to rural districts. English studies as well as those of Drs. Hammond and Horn of the American Cancer Society explain these differences on the ground that cigarette smoking is more common among urban than among rural residents. Dr. Hueper and others, however, believe



The possible sources of cancer-causing elements in the U. S. show the same sort of increase as lung cancer mortality



Death from lung cancer is greater among males (solid lines) than females (dots) and in the city than in the country (1950)

that urban air—polluted with carcinogens derived from industry, transportation, etc.—is the greater villain.

LUNG CANCER IN INDUSTRIAL WORKERS

Dr. Hueper has shown that the incidence of lung cancer is highest among certain industrial workers, especially those in non-ferrous metals, transportation, rubber and plastics, iron and steel. Many industrial workers are exposed to fumes and vapors of chromium, nickel, arsenic, coal tars, petroleum oils, furnace soot and mineral pigments; all of which have known carcinogenic properties.

The English lung cancer rate is considerably higher than of the United States, although the English smoke 30% fewer cigarettes per capita than Americans. This observation, which appears to undermine the cigarette theory, has been argued away on the assumption that Americans do not smoke cigarettes to the very end, while the English, for economic reasons, do. A group of Austrian doctors has pointed out that lung cancer mortality is at different rates in Upper Austria, Kaernten, Steirmark, and Tirol, although the four provinces show approximately the same per capita consumption of cigarettes. The doctors suggest that the high lung cancer mortality among the people living along the northern slope of the Alps may be related to climatic-atmospheric conditions such as winds bringing the industrially polluted air of Vienna into the valleys of the northern alpine regions. Similar observations have been reported on the possible role of prevailing winds in altering the relative frequency of lung cancer in different areas of the English Midlands. The Austrian doctors, moreover, found that lung cancer mortality rates in communities located along main highways, where the atmosphere was polluted with exhaust fumes and dust from asphalted roads, were double those of villages and towns far away from heavy traffic.

Few proponents of the cigarette smoke theory will claim that all the differences between town and country can or need be explained on the basis of differences in cigarette smoking. They agree that urban residence imposes a more sustained and intense exposure to atmospheric pollutants, and that some of these pollutants might of themselves induce the disease. Should the present population studies in England and America demonstrate that the relative risk of cancer of the lung is substantially greater in rural smokers than in rural non-smokers, and that the absolute risk in rural non-smokers of the two countries is the same, then the cigarette hypothesis would be very greatly strengthened.

According to Dr. Gilliam, another question that must be answered by current studies is why there is so much variation in the incidence of cancer of the lung in different large cities of 100,000 or more population. "It is difficult to believe," says Dr. Gilliam, "that smoking habits in our large cities are sufficiently different to account for variations of this magnitude."

It is hoped that these and other questions will be answered, at least partly, by population and other studies presently in progress.

At this time, one cannot take issue with the statement of the Board of Directors of the American Cancer Society that "available evidence indicates an association between smoking, particularly cigarette smoking, and lung cancer. . . ." However, it has not yet been established that this association is more significant than the parallel association of lung cancer with atmospheric pollution. Exposure to atmospheric pollution can be controlled only by vigorous community action. Smoking, however, is a factor over which the individual can exercise personal control.

In February 1954, in written reply to a question asked in the British House of Commons, the Minister of Health stated:

> "The Standing Advisory Committee on Cancer and Radiotherapy had been considering the relationship between smoking and lung cancer for three years. As a result of preliminary investigations, a panel under the chairmanship of the Government Actuary was set up in 1953. The committee, having considered the report of the panel and reviewed the other evidence available, were now of opinion:
>
> 1. It must be regarded as established that there is a relationship between smoking and cancer of the lung.
>
> 2. Though there is a strong presumption that the relationship is causal, there is evidence that the relationship is not a simple one, since:
>
> (a) the evidence in support of the presence in tobacco-smoke of a carcinogenic agent causing cancer of the lung is not yet certain;
>
> (b) the statistical evidence indicates that it is unlikely that the increase in the incidence of cancer of the lung is due entirely to increases in smoking;
>
> (c) the difference in incidence between urban and rural areas and between different towns, suggests that other factors may be operating—e.g., atmospheric pollution, occupational risks.
>
> 3. Although no immediate dramatic fall in death rates could be expected if smoking ceased, since the development of lung cancer may be the result of factors operating over many years, and though no reliable quantitative estimates can be made of the effect of smoking on the incidence of cancer of the lung, it is desirable that young people should be warned of the risks apparently attendant on excessive smoking. It would appear that the risk increases with the amount smoked, particularly of cigarettes."

Although "excessive" smoking is usually defined as more than one pack a day, susceptibility to the effects of tobacco varies widely. A half pack may be excessive for one person and moderate for another.

THE BRITISH ANSWER

It is interesting to note in passing how the British tobacco companies have met the challenge of lung cancer. Instead of setting up their own study, they have offered about $700,000 to the British Medical Research Council, the leading medical body in the country, to develop an objective research program on the relation between lung cancer and environmental carcinogens.

If there is doubt as to whether regular smoking is a major contributory cause of cancer of the lung, there is none about the causal relation between smoking and "chronic

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bronchitis" or "cigarette cough." This is a cough which tends to be worst when one rises in the morning and often produces a considerable amount of thick sputum. Such a cough, of course, is not necessarily due to smoking. Persons with a chronic cough should have a physical examination and chest X ray. *Every* adult should have a chest X ray at least once a year; heavy smokers should have chest X rays twice a year. Other tests, such as bronchoscopy and cytological smear studies of the sputum, are often recommended by physicians when the patient reports spitting blood; suffers repeated attacks of "pneumonia"; or experiences delayed healing of "pneumonia."

EFFECTS ON OTHER ORGANS

At a conference at Cornell Medical Center (December 1953), Dr. Graydon Boyd, specialist in diseases of the nose and throat and Associate Professor of Surgery at New York University Medical School said, apropos the effects of smoking on the nose and throat: "I think as far as prevalence of symptoms is concerned there are more in the nose and throat than from any other effect of tobacco. Of 18 leading otolaryngologists interviewed prior to this meeting, 100% believed tobacco smoking to be irritating to nose and throat." Smoking is also believed to have a close relation to chronic laryngitis and larynx tumors.

Smoke not only irritates the respiratory tract; it contains chemicals that can affect other organs by toxic and possibly by allergic effects. The most important of these chemicals is nicotine, which acts on the nervous system in a variety of ways. One effect appears to be a tranquilizing action on the higher nervous centers. Nicotine also has important effects on the blood vessels and the heart. Patients with Buerger's disease (thrombo-angiitis obliterans) must give up smoking of any kind—cigarette, cigar, or pipe—if they are to avoid gangrene. Even a single cigarette can produce a recurrence or an aggravation of the disease. Even if smoke is not inhaled and the nicotine therefore not absorbed from the lungs, much of it is absorbed from the mouth. The effect of smoking on Buerger's disease may be due to the capacity of nicotine, absorbed from smoke, to cause spasm or constriction of the walls of the blood vessels; or to an allergic sensitivity of the person to some ingredient of tobacco. Many doctors also prohibit smoking of any kind in ordinary arteriosclerosis of the vessels of the extremities; in eye disorders associated with spasm of the blood vessels of the retina, and in Raynaud's disease, a disorder in which there is intermittent impairment of circulation in the fingers or toes. Smoking tends to cause a constriction of the blood vessels in many people but it is not known how much of this effect is due to the toxic action of nicotine; how much to allergy to tobacco products; and how much to nerve reflexes induced by smoke in the mouth and the lungs.

Smoking has also been known to cause extra heart beats and rapid heart beat. How harmful these toxic side effects are, can be determined only by the physician.

Some physicians believe that smoking has a harmful effect on the coronary arteries of the heart and can aggravate coronary artery disease—today the greatest killer of Americans. Their view received unexpected support from the population studies of the American Cancer Society. Drs. Hammond and Horn of the Society, reported that in their study of 187,000 men between the ages of 50 and 69, heavy smokers had a death rate from coronary artery disease twice as high as men who had never smoked. "Even light cigarette smokers were somewhat affected. On the other hand, cigar and pipe smoking seemed to have little or no influence on death rates from the disease." These findings, if confirmed, may mean that cigarette smoking imposes an added burden on the coronary artery circulation and the work of the heart. An alternative explanation is that nervous or psychological factors which predispose to the use of tobacco aggravate existing coronary artery disease.

Some physicians believe that *moderate* smoking has no adverse effect on the normal heart or on coronary artery disease. In December, 1954 the Board of Directors of the American Heart Association, cognizant of the importance of the problem and the paucity of reliable data, approved the following statement relating to this subject: "The American Heart Association is seriously interested in any possible relationship between smoking and heart disease. A committee is currently studying the problem. As soon as clear evidence is available a statement will be forthcoming."

DO FILTERS PROTECT?

Manufacturers of filter-type cigarettes have implied in their advertising that such cigarettes add a safety factor, protecting smokers from the effects of nicotine, tar and other materials in smoke. According to Dr. Irving S. Wright, past president of the American Heart Association and a leading authority on vascular—heart and blood vessels—diseases, "There is absolutely no evidence that there is any protection in terms of vascular disease from these brands; on the other hand there is quite strong evidence that such protection is completely lacking." According to Dr. Wright, this statement holds true for *all* types of filter cigarettes.

Filters do reduce the inhalation of tars and other materials in smoke, and conceivably may reduce the intake of carcinogenic materials. However, Dr. Ernest L. Wynder of the Memorial Hospital, New York's leading cancer hospital, points out that, "As long as we do not know what the carcinogenic material is we do not know whether the filter will remove any or all of this material."

The effect of smoking on gastro-intestinal function and on patients with ulcer of the stomach or duodenum has not received the attention it deserves. Drs. Batterman and Ehrenfeld of New York University School of Medicine, reported in 1949 that while tobacco smoking is not a factor in the *cause* of peptic ulcer, smoking "may result in functional disturbances which may simulate organic disease or

aggravate . . . functional or organic disease. It is particularly the person with an active peptic ulcer who is likely to be harmed by smoke. His response to active treatment will be much less satisfactory than that of the patient who gives up smoking entirely." These views are also shared by leading specialists in gastro-intestinal diseases. It has not been shown that low-nicotine or filter-type cigarettes reduce the damage of smoking in persons with active stomach or duodenal ulcer.

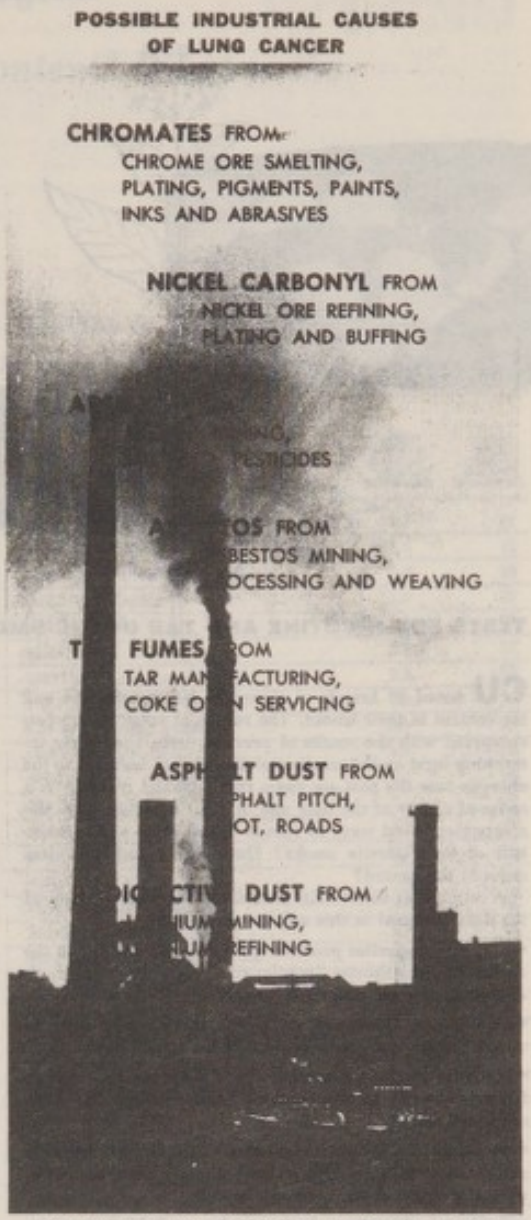
SUMMING UP

In summing up the evidence, it seems established that smoking is harmful in vascular disorders of the extremities and the eyes; and in active peptic ulcer. Inhaling smoke is also definitely irritating to the nose, throat and larynx. The evidence is inconclusive with respect to the effects of smoking on athletic ability, and on heart disorders.

As for cancer of the lung, the most reasonable view at this time relates its phenomenal and alarming rise in frequency in industrial countries partly to the diffusion into the air of carcinogens derived from industry and transportation, partly to unusual exposures to carcinogens in many occupations, and partly to excessive cigarette smoking. The control of respiratory or lung cancer would appear to require a three-pronged effort by individuals, communities, industries and voluntary health agencies (such as the American Cancer Society) against 1) air pollution, 2) occupational exposure and 3) excessive cigarette smoking. How significant a factor regular cigarette smoking is in contributing to lung cancer remains to be determined. Until more conclusive evidence is reported, it would seem prudent to reduce cigarette smoking to less than a half pack a day or to smoke a pipe or cigars in moderation. There is no conclusive evidence that smoking low-nicotine, or filter, or king-size cigarettes offers substantial protection to those with disorders which are known to be aggravated by smoking.

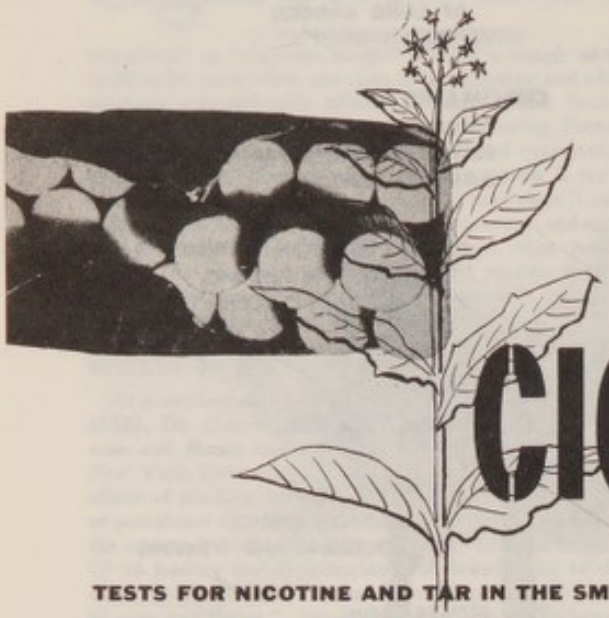
What can be said *for* the smoking habit?

All living organisms are characterized by the property of "irritability." When an organism receives a stimulus, it reacts by a discharge of energy. Human beings are exposed to an enormous number of stimuli in their environment. Among the most important of these are the stresses and strains of modern society—of family, occupation and community tensions. To contend with these tension stimuli, to find relief from them, man has adopted a number of habits, including smoking. Reaching for a cigarette, lighting it, holding it in the mouth, inhaling and blowing out clouds of smoke are motor activities that relieve tensions. Tobacco also provides at least one chemical, nicotine, which appears to have a tranquillizing effect on the nervous system. These effects may be so valuable to some individuals that they will willingly risk whatever harm may be associated with the habit. On the other hand, there are many for whom these risks are not worthwhile and who should therefore cut out or cut down on their smoking.



The tremendous growth of industry, no less than the increases in cigarette smoking, has paralleled the rise in lung cancer mortality. Many industrial waste products contain known carcinogens capable of polluting the air

EXHIBIT 15C



The cancer scare has

Have the new filters and new

present medical view of

CIGARETTES

TESTS FOR NICOTINE AND TAR IN THE SMOKES OF 33 BRANDS

CU tested 33 brands of cigarettes for the nicotine and tar content in their smoke. The results of these tests, when compared with the results of previous tests, shed some interesting light on the reaction of the tobacco industry to the charges over the past six years that cigarette smoking is a cause of cancer of the lung and of other diseases. Have the companies found ways to reduce the nicotine and tar content of the cigarette smoke? Have filters and new sizes reduced the hazard?

A number of interesting facts stand out from a study of the data obtained in this and previous tests:

▪ King-size cigarettes produce more nicotine and tar in the smoke than the shorter "regular-size" cigarettes do, if both are smoked to the same butt length.

▪ As between filters and no filters, there's very little to choose so far as nicotine content of the smoke goes.

▪ While the nicotine content is about the same, the average filtered-cigarette smoke contains somewhat less tar than unfiltered smoke.

▪ So-called low nicotine cigarettes do show low nicotine content as compared with others—about a third as much, on the average, as the ordinary brands.

▪ Unfiltered cigarettes, both regular and king-size, are remarkably similar in nicotine and tar levels to their levels of two years ago. In contrast, the average nicotine and tar levels of filter-tip cigarettes have risen.

The brand-by-brand comparison (see Table on page 101) reveals some facts worthy of special mention:

▪ Among popular regular-size, non-filter cigarettes, *Chesterfield* was lowest in both nicotine and tar levels.

▪ Among popular king-size, filter-tip cigarettes, *Tareyton* was lowest in nicotine level and about average in tar; *L&M* was lowest in this class in tar level and second only to *Tareyton* in nicotine.

▪ *John Alden*, a cigarette made of tobacco especially bred for low nicotine content had, indeed, the lowest nicotine level, but it was among the highest in tar.

▪ *Sano*, made of a denicotinized tobacco, was lowest of all the cigarettes in tar level and higher only than *John Alden* in nicotine.

▪ The lowest in both nicotine and tar levels among cigarettes sold with no special claim to low nicotine content was *Yorkshire*, a brand distributed only by Sears-Roebuck retail stores.

▪ Reynolds Tobacco Co. held the dubious but indisputable distinction of supplying more nicotine for the money than any of its competitors. Its *Winston* ("tastes good like a cigarette should") and *Cavalier* brands edged out its mentholated *Salem* by an insignificant fraction; the fourth Reynolds product, *Camel*, exceeded all other regular-size cigarettes in nicotine level. *Cavalier* was also the tarriest of all cigarettes tested.

The case history of *Kent* cigarettes provides a rather illuminating sidelight on the cigarette industry's reaction to the health problem. First put on the market in 1952 as P. Lorillard's answer to the raging cancer-of-the-lung scare, the *Kent* cigarette did a creditable job of filtering out a high percentage of both nicotine and tar from tobacco smoke. But *Kent's* "Micronite" filter ("developed by researchers in atomic energy plants") was, it would appear, a shade too effective. Smokers complained that it was hard to draw smoke through the filter and that all they got for their effort was a mouthful of tasteless warm air. CU's 1952 tests showed that the original *Kent* contained, in the smoke of one cigarette, ½ milli-

brought many changes to the industry.

sizes affected the nicotine and tar content? What is the

the effect of smoking on health?

gram of nicotine and 2 milligrams of tar. By 1955 the filter had been loosened up and made easier-drawing, and *Kent's* nicotine level had about quadrupled while its tar level had increased about six-fold. As of CU's current investigation, *Kent* had, without fuss or furor, abandoned the original Micronite completely and substituted a filter tip which appears similar to the almost universally used cellulose filter tip. And its nicotine and tar levels had risen so that it had about five times as much nicotine and about eight times as much tar as the original 1952 *Kent*.

CU shoppers in 18 cities from coast to coast bought 16 packs of each brand, and, as in the earlier tests, one cigarette from each pack was mechanically "smoked" with the apparatus shown on page 105. Since neither the true medical significance of tars nor the permissible level, if any, of nicotine in cigarette smoke is known, CU does not rate the brands, but simply presents the results of its tests.

No attempt was made to gauge the effectiveness of filters as such nor to measure the quantity of nicotine and tars in the tobacco of the cigarettes. The tests were confined to the *smoke*, because this obviously is the smoker's only real concern.

Significantly, brands which have appeared in all three of CU's projects have ranked, within type, in roughly the same order of nicotine content in all three. As before, *John Alden*, made with low-nicotine tobacco, produced the least nicotine of any brand in the latest tests. Next in line this time came denicotinized *Sano* (also low in the earlier projects) and *Lords* (included for the first time), both made with tobacco from which a large part of the nicotine had been extracted by processing.

L&M filter regulars took the biggest jump in nicotine from 1955 to 1957. Of the ten filter brands covered in both 1955 and 1957, six—led by *Parliament* and *Old Gold*—were somewhat higher in tar content in the latest tests.

In contrast to the variety of filter materials—absorbent cotton, paper with activated carbon, cellulose-acetate fiber, creped and uncreped paper, asbestos fiber, and combinations of these—that turned up in the 1955 tests, most of the filters encountered this time were made of a cellulose derivative. Regardless of their effectiveness as filters, all appeared to offer two advantages: they help to keep bits of tobacco out of the smoker's mouth, and they provide a firmer butt for his lips to grip.

Continued on next page

RESULTS OF TESTS FOR NICOTINE AND TAR

The listing is in order of increasing nicotine content of the smoke. Small differences in nicotine and tar content are of no significance.

BRAND AND TYPE	AVERAGE NICOTINE IN SMOKE PER CIGARETTE (MILLIGRAMS)	AVERAGE TAR IN SMOKE PER CIGARETTE (MILLIGRAMS)
JOHN ALDEN Low nicotine tobacco, regular	0.7	21
SANO Denicotinized, king-size, filter-tip	1.0	11
LORDS Denicotinized, regular	1.2	16
YORKSHIRE Regular	1.8	15
MURAD Regular	1.8	17
CHESTERFIELD Regular	2.1	17
TAREYTON King-size, filter-tip	2.3	19
L & M Regular size, filter-tip	2.6	15
L & M King-size, filter-tip	2.6	15
LUCKY STRIKE Regular	2.6	19
KENT Regular size, filter-tip	2.7	16
OLD GOLD Regular	2.7	18
VICEROY King-size, filter-tip	2.8	18
KOOL Mentholated, regular	2.8	20
MARLBORO Long, filter-tip	2.9	18
PHILIP MORRIS Regular	2.9	18
HERBERT TAREYTON King-size	2.9	22
HIT PARADE King-size, filter-tip	3.0	20
CHESTERFIELD King-size	3.0	22
PALL MALL King-size	3.0	24
KOOL Mentholated, king-size, filter-tip	3.1	16
SPUD Mentholated, long, filter-tip	3.1	17
OLD GOLD King-size, filter-tip	3.1	19
PHILIP MORRIS Long	3.1	21
REGENT King-size, filter-tip	3.1	21
FATIMA King-size	3.1	24
CAMEL Regular	3.2	17
OLD GOLD King-size	3.2	19
PARLIAMENT Long, filter-tip	3.4	20
SALEM Mentholated, king-size, filter-tip	3.7	19
RALEIGH King-size	3.7	23
WINSTON King-size, filter-tip	3.8	22
CAVALIER King-size	3.8	26

CIGARETTES continued

Although the king-size brands, on the average, had more nicotine and tars in their smoke than the regular-size cigarettes did, these results should be viewed in the light of the fact that all samples, regardless of size, were smoked to the same butt length (approximately one inch). Consequently, more tobacco was burned in smoking the king-size cigarettes. Whether or not "fine tobacco filters best," as the *Pall Mall* ads claim, it is a fact that if you leave a proportionately longer butt when you smoke a king-size cigarette (in other words, smoke the same length of tobacco as you would with a regular-size cigarette), the extra tobacco left in the butt will act as a filter and reduce both the nicotine content and tar content of the smoke.

NICOTINE AND TAR LEVELS BY CIGARETTE TYPE

	AVERAGE NICOTINE IN SMOKE OF ONE CIGARETTE (MILLIGRAMS)			AVERAGE TAR IN SMOKE OF ONE CIGARETTE (MILLIGRAMS)		
	1957	1955	1953	1957	1955	1953*
REGULAR SIZE NO FILTER	2.5	2.6	2.0	18	18	17
REGULAR SIZE WITH FILTER	2.7	1.9	2.1	16	12	14
KING SIZE NO FILTER	3.2	3.3	2.5	22	23	18
KING SIZE WITH FILTER	3.1	2.7	**	18	16	**
SPECIAL LOW NICOTINE	1.0	0.7	0.8	16	14	13

** Different methods were used for tar extraction in the 1953 tests than in the 1955 and current tests. The 1953 figures have been increased by 20% in an attempt to make them directly comparable.*
*** This type of cigarette was not among the popular brands on the market at the time of the test.*

THE INDUSTRY: NEW BRANDS AND NEW TYPES HIT THE GROWING MARKET

In a recent issue of a tobacco trade publication, the editors, speaking of the research that has indicated a relationship between cigarettes and lung cancer, tossed off this seemingly lighthearted comment: "The only certainty that has emerged is that mice shouldn't smoke!"

The editors' flippancy may have been inspired by the apparently complete recovery of the cigarette industry from the staggering punches medical investigators landed on it a few years ago.

Last year, according to authoritative estimates, Americans broke all records by spending nearly five billion dollars for 395 billion cigarettes. This was 170 million more dollars and 13 billion more cigarettes than in 1955 and 570 million more dollars and about a billion more cigarettes than in the previous record year: 1952, the year before the full impact of cigarette-cancer publicity hit the cigarette business.

Obviously, 1956 was a banner year for most big cigarette companies and their stockholders. So it is not surprising that industry spokesmen now appear to be taking the health problem considerably less seriously than they did a year or two back. In those days, president Edward A. Darr of R. J. Reynolds (*Camel, Winston, Cavalier, Salem*) accused the American Cancer Society of "trying to destroy the tobacco industry." Last December in *Printers' Ink*, an advertising trade magazine, tobacco-business consultant Harry M. Wootten wrote that "the smoking-and-health controversy . . . may be collapsing for lack of nourishment." Even the Department of Agriculture concedes that many people who had reduced their smoking or cut it out entirely in 1953 and 1954 "probably have resumed or increased their consumption, mainly using filter-tip cigarettes."

At least a million and a half of this country's 38 million smokers (54% of all men and 24% of all women over 18) gave up the habit between the fall of 1953 and early 1955, a Census Bureau survey for the National Cancer Institute indicates. How many more simply cut down, the published findings do not reveal, but it must have been a sizable number, because in 1953, after 21 years of uninterrupted growth, cigarette sales dropped by 7.3 billion cigarettes. The slide continued through the next year, to bring the total drop for the two-year period to 25.4 billion cigarettes.

Sales turned upward again in 1955, however. That year's total, although still well below 1952's, bettered the 1954 mark by some 13 billion cigarettes. Then came 1956 —and joy was complete again in the hearts of the men who know tobacco profits best.

Last year's cigarette sales and profits records (and 1955's partial recovery) were largely the result of the soaring popularity of filter-tip cigarettes, and the upsurge

of this once-minor cigarette breed has wrought some fairly startling changes in the cigarette business. The major ones are: a spate of new brands, new versions of old brands, new packages (in the face of a traditional, virtually industry-wide reluctance to make any change in the products for fear of spoiling a good thing); an upheaval in the tobacco market (resulting from the fact that filter cigarettes not only require less tobacco than nonfilters, but are being made with lower-grade leaf and, in some cases, with parts of the tobacco plant that used to be thrown away); the highest profits in years (because most filter cigarettes cost less to make than non-filter cigarettes, but have sold at premium prices); and, finally, a marked weakening of the time-honored sales dominance of three or four top brands.

Filter-tip cigarettes are far from being a new product. Handmade ones were on sale in Europe at least as far back as 1900, and such brands as *Obak* and *Imperiale* turned up in the United States a few years later. Benson & Hedges' *Parliament*, introduced in 1931, and Brown & Williamson's *Viceroy*, which appeared five years later, were the first major domestic brands, but they made no dent worth mentioning in the American market. As a matter of fact, a mass market was the last thing their manufacturers had in mind at the time: the advertising pitch was aimed at snobs only.

In 1952, P. Lorillard Co. trumpeted its new filter-tip *Kents* onto the stage with full-page newspaper ads describing the virtues of the "Micronite" filter ("developed by researchers in atomic-energy plants").

Since 1952, sales of filter-tip cigarettes have shot up from 5.2 billion a year to a 1956 record of over 119 billion, from a relatively insignificant 1.4% of total cigarette sales to nearly 30%. Not only were they the only cigarette type to sell better in 1956 than in 1955: their gain amounted to nearly 60%. In achieving it, they passed the king-size group for the first time and captured second place among the three major cigarette types. By the end of this year, they are expected to have 40% of the market.

The chief loser in the shifting cigarette market has been the erstwhile mainstay of the business, the regular-size nonfilter cigarette. As smokers turned to filters, sales of regulars have fallen off—from 319 billion cigarettes in 1952 to 182 billion in 1956, a drop of nearly 43%. Regulars still hold the top spot, but in contrast to 1952, when they were in command of four-fifths of the entire cigarette market, their share last year was under 46%. Kings have slipped too, but their decline last year was only about 6%, compared with the regulars' 10%.

Within the general pattern of a rising filter-tip market and a shrinking market for regulars and kings, individual brands have been having their ups and downs.

Camel, the best-selling brand since 1950, is still on top, despite a sales drop of 34% since 1952. *Pall Mall*, top seller among kings and third-ranking among all brands, was the only king to buck the filter tide successfully last year. Hardest hit by the filter competition were *Chesterfield* and *Old Gold* regulars, both down about 20% in 1956 sales from 1955.

Winston's 1956 sales of 34 billion not only made it the top filter for the second straight year, but lifted it to fourth place among all brands, well ahead of such oldtimers as regular-size *Chesterfield*, *Philip Morris*, and *Old Gold*. *Viceroy*, the filter leader until 1954, came in second again, with *L&M* hot on its heels and coming up fast. *Marlboro's* 1956 gain (120%) was the biggest of all, and by the end of the yea. it had shot past seven older brands to become the fourth-ranking filter.

New brands appear

Not since the emergence of 10¢-a-pack cigarettes during the Great Depression has there been such a rash of new brands and changes in old brands as the last few years have seen. As a result, the industry's take is being split more ways, and three or four brands no longer dominate sales as they once did. While the four leading brands of six years ago, for example, accounted for 80% of total cigarette sales, last year's top four did only 57% of the overall business.

Every one of the industry's giants sent a new entry into the filter race last year—including American Tobacco, the last of the Big Three (the other two being R. J. Reynolds and Liggett & Myers) to bring out a high-powered contender. Perhaps because its *Pall Malls* and its *Luckies* had held their combined sales volume against the filters, American didn't get around to launching its king-size filter-tip *Hit Parade* until last October. *Hit Parade* was not expected to achieve national distribution until early this year, but even without this it had scored sales of 2.8 billion by the end of 1956.

The nation got its first mentholated filter brand when Reynolds trotted out *Salem* last May. A few weeks later Philip Morris reintroduced *Spud* (a mentholated brand it had acquired in the liquidation of the Axton-Fisher Tobacco Co. some years back) as a filter-tip, mentholated, king-size cigarette. Then, in August, a new filter-mentholated-king (Brown & Williamson) *Kool* hit the market, replacing nonfilter king-size *Kool*.

So-called brand splitting (using the same brand name

Continued on next page



CIGARETTES continued

for two or more cigarette types) has grown apace. Liggett & Myers was the first of the brand splitters (with king-size *Chesterfield* in 1952). Later, P. Lorillard took the process a step further, splitting *Old Gold* three ways (regular, king, and filter), and last year both Philip Morris and Liggett & Myers went three-way too (regular, long, and king).

Other recent cigarette-business debuts included the advent of the "long"-size cigarettes (*Marlboro, Philip Morris, Parliament, L&M, Regent, Spud*), 80 mm in length, as compared with 70-mm regulars and 85-mm kings, and Philip Morris' introduction of formerly premium-price filter-tip *Parliament* in a flip-top box, to sell for only 1¢ to 2¢ above "popular" filter prices.

Although the tobacco crop—at least four-fifths of which goes into the making of cigarettes—ranks near the bottom of the list of United States crops in acreage (1,366,000 in 1956) it stands near the top in cash value. In 1955, it brought its growers about one and a quarter billion dollars (about 9% of total U. S. farm income for that year).

Tobacco is grown from New Hampshire to Florida and as far west as Minnesota, but as a result of differences in soil, climate, and plant varieties, nearly every region produces a different kind and quality of leaf. Some cigarette tobaccos are flue-cured; others are air-dried or fire-cured. Flue-curing, in which the leaves dry quickly in a heated barn, results in a light brown, almost yellow, leaf, while fire-cured leaves, dried in the smoke of wood fires, are very dark. Maryland tobacco, and Kentucky and Tennessee burley usually are air-dried in unheated barns.

For many years, flue-cured tobacco, which comes mainly from North Carolina, was used straight in American cigarettes, but now nearly all the leading brands are made with "blended" tobaccos. The mixture varies, but "popular" nonfilter cigarettes are said to contain 45% to 75% flue-cured leaf, 15% to 45% burley, 5% to 13% Turkish, and 5%, more or less, Maryland. Other ingredients include a "humectant," or moistener, and one or more of such flavoring materials as rum, wine, brandy, honey, oil of cloves, oil of cinnamon, oil of peppermint, and licorice.

What kinds of tobacco are going into filter cigarettes these days seems to be largely each company's secret. Manufacturers are reported to be switching from light-colored, light-bodied, flue-cured tobacco to darker, heavier-bodied leaf in an effort to compensate for a loss of flavor that occurs as the smoke passes through a filter. In making this switch, they are turning the tobacco market upside down. The price of "low-grade" darker leaves, which used to be much lower than that of flue-cured leaf, is climbing rapidly, while "high-grade" flue-cured tobacco is bringing bids below government support prices—and going begging at that. At the end of last year, the supply of flue-cured leaf on hand had mounted to a record 3.5 billion pounds, enough to last almost three years—even at the old rate of consumption. As a result, the government, which, in connection with the price-support program exercises strict control over tobacco acreage, has ordered a 20% cut in flue-cured growing for 1957.

TOTAL CIGARETTE SALES*

	1952	1953	1954	1955	1956
BILLIONS OF CIGARETTES	394	387	369	382	395

CIGARETTE SALES BY TYPE*

REGULAR	80.3%	69.8%	61.8%	53.2%	45.8%
KING SIZE	18.3%	26.9%	28.1%	27.2%	24.3%
FILTER	1.4%	3.3%	10.1%	19.6%	29.9%

**Figures are from Printers' Ink magazine*

Growers not happy

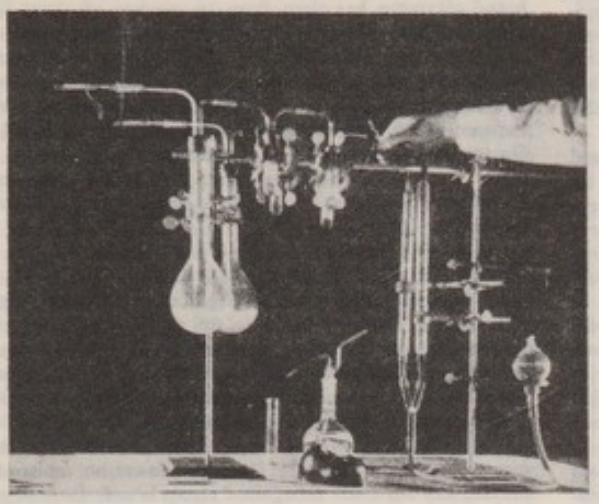
For the growers, the "tremendous switch," as *Business Week* magazine calls it, is only one of three setbacks they have suffered at the hands of the cigarette and cigar makers during the last few years. In the first place, the growers are somewhat less enthusiastic than the manufacturers about the filter-cigarette boom, for the simple reason that filter cigarettes take less tobacco than plain ones (7% to 20% less).

Not only that, but some manufacturers have taken to buying and using parts of tobacco plants that used to be thrown away: stems, broken and inferior leaves, and fragments. These are ground into small particles, mixed with a binder to form a paste, rolled out into a sheet, dried, shredded, and then blended with higher-grade tobaccos and made into cigarettes. The use of such "homogenized" (or "reconstituted" or "synthetic" or "processed," as it is variously called) tobacco could cut tobacco costs by up to 50%, according to some estimates. The companies are said to be ready, after eight to ten years of experimenting, to begin using the homogenized product in a big way.

When a Senate-House agriculture subcommittee tried to look into the matter last May, it found the manufacturers pretty close-mouthed. The reason for this reticence, *Business Week* guessed, were the companies' uncertainty about the public's reaction and fear on the part of manufacturers who are using or planning to use homogenized leaf that the competition might begin boasting of its "100% top-grade" tobaccos. However, a spokesman for R. J. Reynolds, which is said to have been using homogenized tobacco for several years, insists that its man-made leaf, being a controlled blend, is milder and more flavorful than natural leaf.

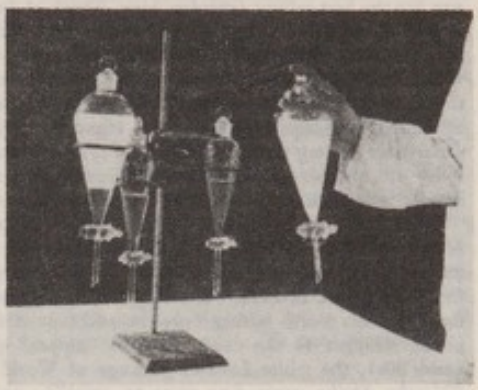
Even though homogenized tobacco does not appear to have come into general use yet, filter cigarettes are proving considerably more profitable than plain ones. Value Line Investment Survey credits them with an important role in the 6% rise in the tobacco companies' net income last year. The reason is clear: Three-quarters of the cost of making a cigarette goes for the tobacco, according to *Forbes*, a business magazine, hence filter cigarettes, which take less tobacco and can be made with lower-grade leaf, can mean an appreciable saving to the manufacturer. At the same time, the filters have been selling for up to 5¢ more

A



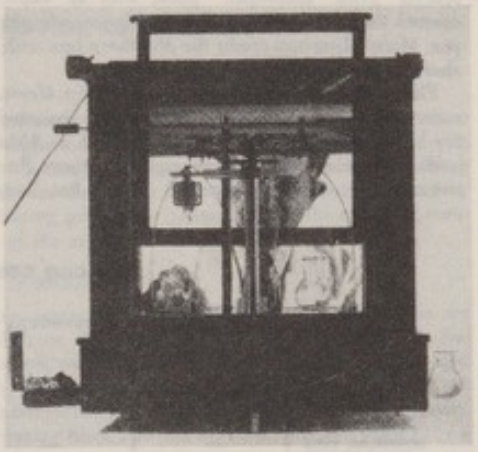


B



C

D



HOW CU TESTED CIGARETTES

Two cigarettes at a time were fitted into glass holders and then "smoked" by the laboratory apparatus (A). Raising and lowering the leveling bottle at the right raises and lowers the level of the water in the vertical glass tubes (below technician's hand) at the end of the "train." When the water level in the tubes falls, air is drawn through the cigarettes. The smoke is collected in acidified alcohol in the flasks (left); any smoke that escapes the flasks is trapped in the vials between the flasks and the end of the train. The apparatus took one puff a minute, each puff lasting for two seconds and containing about two cubic inches (35 cubic centimeters) of smoke. The cigarettes were smoked down to a butt of just under an inch (23 mm.) Sixteen samples of each brand were smoked in two runs of eight cigarettes each, and the results of each run were determined separately and averaged. After each run, the washings from the vials were added to the smoke solution in the collecting flasks. One portion of the solution was steam-distilled to isolate the nicotine, which was measured with an ultra-violet spectrophotometer (B). Chloroform and water were added to another portion of the smoke solution (C) and then the tars were separated out in a series of steps and weighed on a chemical balance (D).

per pack than the nonfilters. Small wonder, then, that, as Mr. Wootten has said, filter cigarettes, once "reasonable unit volume" is attained, "are, in most instances, the most profitable end of the tobacco business."

Cigarettes probably are sold in more places—and more kinds of places—than any other manufactured product: cigar stores and stands, drug stores, grocery stores, restaurants, bars, liquor stores, newsstands, and just about any other place where they can pay, in cash or good will, for the space and trouble they take. However, such traditional retailers as tobacco shops, cigar stores, and drug stores are gradually yielding ground to supermarkets, which now have become the principal outlets. Self-service merchandising (via supermarket and vending machine) is supplanting over-the-counter sales. The latest available statistics, released in 1952 by the National Association of Tobacco Distributors, show food stores making 32% of total sales, followed by vending machines with 16%, drug stores with 13%, and cigar stores and stands with 12%. Along with the shift of business to supermarkets has gone a shift, on the consumer's part, from buying packs to

Continued on next page

CIGARETTES continued

buying cartons. At least half of all cigarette sales today are said to be by the carton.

Fifty per cent of the 490,000 vending machines in operation in 1955, according to a *Redbook* magazine report were run by wholesale tobacco distributors. The same source quotes trade informants as saying that the big cigarette companies pay annual subsidies of $3 to $12.50 per column per machine to have their brands stocked.

The recent outpouring of new cigarette brands and types has created more than its share of stocking, inventorying, and pricing problems for the retailer. Some supermarkets, report *Printer's Ink*, are refusing to carry any but the best-selling (regular, king, filter) of any brand, and a "brand survival battle, with weak entries forced out" is predicted. Operators of vending machines, most of which have only ten columns, now are confronted with the fact that it takes at least 20 columns to offer even minimum coverage of the current cigarette crop. Price differences among the many cigarette types on the market today are another headache, because vending machins can handle money in only two or three denominations.

Until January, 1955, when Philip Morris brought out *Marlboro* in a flip-top box, cigarette packaging had remained practically unchanged since *Camel* appeared in the first "cup" pack in 1913. The only intervening packaging developments worth noting were the addition of the cellophane wrapper to the cup pack, the "zipper" pull tabs (mid-'30s), the white *Luckies* package of World War II ("*Lucky Strike* green has gone to war!"), and Philip Morris' 1954 "snap-open" pack, with an extra tab that opened the package itself along with the cellophane wrapper. Many observers credit the *Marlboro* box with a sizable share in the brand's rapid sales growth.

Philip Morris (which now boxes *Philip Morris*, *Parliament*, and *Spud* as well as *Marlboro*) imported its flip-top-box-making machines from England at $30,000 each and had exclusive American rights to them through last summer. Since then Liggett & Myers has bought some machines and adopted the box as an alternative package for *L&M* filters ("Pick the pack that suits you best . . ."). Other companies are reported to be experimenting with box-packaging.

In the past 20 years, the retail price of a pack of major-brand cigarettes has risen from an average of about 13¢ (two for 25¢) to about 25¢. Tax hikes—cigarettes are one of the most heavily taxed of all products—have accounted for part of this increase. The Federal excise tax now amounts to 8¢ a pack, while the average of the taxes imposed by 42 states and the District of Columbia is 3¾¢. On top of these, there is a local tax of a penny or so in many areas. For the fiscal year that ended last June Federal and state taxes alone were expected to total about 2.1 billion dollars.

Movement in unison

As they always have, the retail prices of "popular" cigarette brands continue to move up or down virtually in unison—despite a 1946 Supreme Court anti-trust case decision convicting the three largest companies (American Tobacco, R. J. Reynolds, and Liggett & Myers) of collusion to prevent retail price competition. The companies accomplished their end, the court found, by keeping their own price the same and by putting pressure on dealers to prevent any but a few recognized cheap brands from being sold for less. At the same time, the companies were judged guilty of manipulating the tobacco market to hold down the prices paid to growers.

According to testimony in the case, which was begun in 1940, the Big Three dealt ruthlessly with the manufacturers of the 10¢ brands (*Wings*, *Avalons*, *Marvels*, etc.) that burst on the scene during the Depression. When these upstarts captured 22% of the market for a brief period in 1932, the big companies went into action, dropping their own prices and buying up the kind of tobacco used in the 10¢ brands. To date, the only apparent price competition in the cigarette business is at the retail level, generally between small dealers and supermarkets and drug chains.

It is a popular notion that most of the price of a pack of cigarettes goes to pay for the advertising. Actually,

TOBACCO COMPANIES AND THEIR OFFSPRING

	AMERICAN TOBACCO	REYNOLDS	LIGGETT & MEYERS	PHILIP MORRIS	LORILLARD	BROWN & WILLIAMSON
REGULAR SIZE NO FILTER	Lucky Strike	Camel	Chesterfield	Philip Morris	Old Gold, Murad	—
REGULAR SIZE WITH FILTER	—	—	L & M	—	Kent	—
KING SIZE NO FILTER	Tareyton, Pall Mall	Cavalier	Chesterfield, Fatima	Philip Morris	Old Gold	Raleigh
KING SIZE WITH FILTER	Hit Parade, Tareyton	Winston	L & M	Marlboro, Parliament	Old Gold	Viceroy
MENTHOLATED	—	Salem	—	Spud	—	Kool

figures published several years ago indicate that the manufacturers of the major brands spend only about a fourth as much for their advertising as they do for their tobacco, that advertising accounts for a little over 14% of their total cost of doing business. Even so, six cigarette companies made a list of the 100 leading advertisers of 1955 published in the trade weekly *Advertising Age* last December.

Through the years and into the present, the three main *themes* of cigarette advertising have been pleasure, health, and snob appeal, sometimes used separately, sometimes in combination. In the '40s and early '50s, the emphasis was on health ("Just what the doctor ordered," "Guard against throat scratch," "Safe for your T-zone," "More doctors smoke *Camels* than any other cigarette," etc.)

Most of the industry's run-ins with the Federal Trade Commission in the past 15 years or so were brought on by health claims. For example, in 1942, *Old Gold* ads claimed that impartial tests reported in the *Reader's Digest* had shown that *Old Gold* had less nicotine than any other cigarette tested. The FTC objected to this claim, pointing out that the actual difference in average nicotine content per cigarette between *Old Gold* and the other two brands tested was 1/177,187 ounce.

At other times, the FTC has ordered an end to claims, all of which it found to be untrue, that "*Camels* will never harm or irritate the throat," that *Philip Morris* "protects against smoker's cough," and that among "independent tobacco experts, it's *Luckies* two to one!"

In September, 1955, the FTC released an eight-point guide for its staff's use in judging cigarette advertising. According to the guide, the following claims are improper:

- References to the presence or absence of any physical effects of smoking in general or in any brand in particular.
- Unproved or insignificant claims concerning nicotine content.
- References to the effects of cigarette smoking on the nose, throat, nerves, or other parts of the body, or on energy.
- Claims of medical approval of cigarette smoking in general or any brand in particular.
- Comparisons of the sales volume of competing brands without substantiating data.
- Unprovable claims to the use of particular types or qualities of tobacco.
- Testimonials that are not genuine and the current opinion of the testifier.
- False or misleading disparagement of other cigarette companies and their products.

As *Business Week* observed, the guide, if it were enforceable would eliminate "nearly every selling point that the tobacco companies have used in the past generation."

But 1954 brought a new look in cigarette advertising. *Business Week* summed it up this way: "The FTC couldn't do it. The tobacco growers couldn't do it. Warnings from marketing experts went unheeded. But economics did the trick. Faced with dropping sales, cigarette manufacturers have pulled an abrupt about-face in advertising tactics. . . . Today there is no word of fear, no talk of throat scratch—just comfortable, reassuring phrases about how good a cigarette tastes. Smoking, it seems, is no longer a health cure; it's pure pleasure."

MEDICAL ASPECTS: THE CANCER LINKAGE IS STILL NOT CLEAR

Although smoking has been a human activity for hundreds of years, it is only in the last decade or so that it has become the subject of intensive scientific study. Occasional reports of the effects of smoking on both body and mind have appeared since the middle of the last century, when a famous Irish physician, Dr. Robert J. Graves, found heart disturbances in heavy smokers. However, most of the studies on living subjects were made without adequate statistical controls, diagnostic precision, or the benefits of chemical and pharmacological analysis. Often the emotional attitude of the observer toward smoking would result in either exaggeration or underestimation of the effects of tobacco. The development of improved medical, biological and statistical techniques in recent decades has made possible more reliable studies of the properties and effects of tobacco smoke.

According to present knowledge, tobacco smoke contains tars, nicotine, carbon monoxide, arsenic and a miscellaneous group of acids, phenols, aldehydes, and other chemicals. The relative amounts of these chemicals vary in different tobacco leaves. The ingredients which are introduced by the burning of the cigarette paper are not fully known, but current research efforts should provide useful information in this neglected field. Physical factors associated with smoking—holding a hot pipe stem in the mouth, or holding hot smoke in the mouth and the respiratory tract—may have irritating properties independent of the chemical composition of the smoke itself.

Smoke is absorbed

Because many of the chemicals in tobacco smoke are absorbed from the mouth and respiratory tract into the blood stream, smoking probably affects all the organs and tissues of the body. The most important known effects are on the gastrointestinal tract, the heart and blood vessels, the respiratory tract, and the nervous system. People vary a great deal, however, in their reaction to tobacco. For one person, 15 cigarettes a day constitutes heavy smoking; for another, heavy smoking means 40 cigarettes a day. British investigators usually term more than 25 cigarettes a day as "heavy smoking." In American research the term "heavy smoking" means from 20 to 40 or more cigarettes daily.

Notwithstanding the testimonials of movie stars and fa-

Continued on next page

CIGARETTES continued

mous sports personalities regarding the mildness or non-irritating properties of the cigarettes they are paid to advertise, tobacco smoke does irritate the mucous membrane. Dentists are familiar with the stains and tar deposits on the teeth and gums of habitual smokers, and some believe that heavy smoking aggravates gum diseases. "Leukoplakias"—pre-cancerous, localized surface thickenings of the tongue, cheek and other parts of the mouth—are seen predominantly in heavy smokers, and they usually disappear if smoking is discontinued. "Birdcage mouth," the flat, morning taste well known to heavy smokers, is in part due to the local irritative effects of tobacco smoke.

The nausea-producing action of nicotine is well known to almost everyone who remembers his first attempt at smoking. Fine discrimination in taste sensation and smell are known to be impaired by smoking, and gourmets make a point of never smoking before dining and wining. Even in some habitual smokers, hunger contractions of the stomach are suppressed by smoking, and it is a common complaint that there is a great increase in appetite after the smoking habit is broken.

While smoking does not of itself cause stomach or duodenal ulcers, most doctors prohibit smoking in the presence of active ulcer symptoms. The apparent effect of the after-breakfast cigarette in promoting a bowel movement is believed to be due to the stimulating action of nicotine on the autonomic ganglia supplying the colon. Because of this effect, persons with diarrhea or with acute or chronic colitis are sometimes advised to avoid tobacco.

The effect of smoking on the heart and blood vessels was perhaps the first aspect to engage the attention of physicians. Smoking accelerates the heart rate, raises the blood pressure, increases the work of the heart and constricts the blood vessels of the extremities. The degree to which such changes occur varies a great deal in different persons. Persons sensitive to tobacco often complain of "extrasystoles" or other irregularities in the heart beat.

Nicotine tolerance

Although the habitual smoker acquires a certain tolerance to nicotine, and can absorb amounts of the drug that would kill a non-smoker, he never gets over the vascular effects of smoking. Elevation of the blood pressure and the heart rate, and constriction of the peripheral arteries, does occur after one or two cigarettes even in the heaviest smokers. The studies of Dr. Grace Roth of the Mayo Clinic, among others, have made it evident that nicotine is the agent chiefly responsible for the vascular changes induced by smoking. About 2½ milligrams of nicotine are contained in the puffed smoke of an average, regular-length cigarette, somewhat more in the smoke of a king-size cigarette. About 90% of nicotine in inhaled smoke is absorbed, compared to about 25% to 60% absorption when the smoke is simply drawn into the mouth and then expelled, as is usual in pipe and cigar smoking.

Some investigators believe that the vascular disturbances occurring after smoking are little influenced by the nicotine content of the tobacco or the extent of inhalation of the smoke; others consider that individuals sensitive to the nicotine effects of ordinary cigarettes may be able to tolerate a low nicotine tobacco (such as *John Alden*) or a denicotinized tobacco (such as *Sano*) with fewer harmful vascular effects.

Appreciable amounts of carbon monoxide are present in tobacco smoke, and in the heavy smoker as much as 5% or more of the circulating hemoglobin may be converted to "carboxyhemoglobin." Whether this concentration of carbon monoxide in the blood impairs physical and mental efficiency under ordinary circumstances is not known, but it has been speculated that heavy smoking may be a contributing factor in impairing judgment and nervous reflexes in auto driving and other activities requiring physical and mental alertness. At high altitudes, where there is a tendency to oxygen deficiency, a concentration of 5% carbon monoxide in the blood may have serious effects.

Smoking and the heart

Smoking tends to cause a significant rise in blood pressure in persons with a tendency to hypertension, and some doctors caution against smoking for those who have severe high blood pressure or hypertensive heart disease. Other doctors believe that the tranquilizing effects of moderate smoking on the nervous system offset the blood pressure elevating effects.

In the course of their studies on the association between heavy cigarette smoking and lung cancer, Drs. Hammond and Horn of the American Cancer Society reported that among 187,000 men aged 50 to 69, the heavy smokers had a death rate from coronary artery disease (hardening of the coronary arteries, "coronary sclerosis" or "thrombosis") twice as high as men who had never smoked. Even light cigarette smokers were somewhat affected, according to the report, though cigar and pipe smoking seemed to have little or no influence on death rates.

The thesis that heavy cigarette smoking imposes a serious burden on the coronary artery circulation and on the heart is not accepted by most physicians, however. An alternative explanation may be that nervous or psychological factors which dispose a person to indulge in heavy smoking also dispose to coronary artery disease. A scientific committee of the American Heart Association, appointed to appraise all available scientific evidence relating to smoking and heart disease, concluded (March 14, 1956):

> 1. There is evidence supported by clinical observations in a large number of cases that tobacco smoking is harmful in certain diseases of the peripheral blood vessels of the arms and legs. This harmful effect was demonstrated most clearly in the condition known as thromboangiitis obliterans (Buerger's disease). It is known that this disease will usually . . . become stationary or even improve if the patient stops smoking.
>
> 2. It is recognized that a small percentage of persons with known disease of the coronary arteries will develop symptoms and will display signs detectable by laboratory tests when they smoke. Such people may be harmed by smoking.
>
> 3. The committee believes that the available evidence is not sufficient to define the effect of tobacco smoking upon the coronary arteries or upon the heart itself, except in the small group mentioned above who already have coronary artery disease. It is believed that if smoking plays any part in the causation of heart disease, it is only one of many factors.

4. It is the belief of the committee that much greater knowledge is needed before any conclusion can be drawn concerning relationship between smoking and increased death rates from coronary heart disease.

It has been suggested that some of the vascular and other effects of tobacco are the result of allergy to some component of tobacco. Many persons with abnormal responses to tobacco show a positive reaction when tobacco extracts are injected into the skin. More studies are required, however, to determine the significance of allergy in relation to some of the effects of smoking.

Tobacco "amblyopia" is a relatively rare but serious eye disorder associated with smoking. There is a gradual or sudden decrease of visual acuity, especially for colored objects. If the connection with smoking is not realized, the condition may progress to optic nerve atrophy and permanent injury to vision.

While nicotine is chiefly responsible for tobacco's effects on the heart and blood vessels, the elements chiefly responsible for the effects on the respiratory tract—the nose, throat, bronchi, and lungs—are the tars, and possibly arsenic and other combustion products. The respiratory effects are of two types—irritating and carcinogenic (cancer-inciting).

Irritating effects

The irritating effects of tobacco smoke on the respiratory tract seem indisputable. Many otolaryngologists believe that chronic inflammation of the larynx and warty growths on the vocal cords can be caused by heavy smoking. Many studies indicate that heavy smoking can result in chronic bronchitis. So-called "cigarette cough" is believed to be caused by the chronic irritation of the nose, throat, larynx, trachea, and bronchi. Although tobacco smoke has not been demonstrated to cause bronchial asthma, in most cases asthma is made worse by inhalation of tobacco smoke.

Recent studies indicate—though final proof is still lacking—that inhaled tobacco smoke may reach far down into the smaller air passages of the lung, causing spasm of the muscles surrounding the passages as well as inflammation of the mucous membranes. This may lead to partial or intermittent obstruction of air from the alveoli of the lungs and eventually to "pulmonary emphysema"—a disease of the lungs marked by breathlessness, and almost always associated with coughing. The lungs become over-inflated, the tissues are stretched, the elastic recoil of the lung is reduced, and the diaphragm becomes low and fixed. The disease, which usually begins after the age of 40, tends to progress and to lead eventually to severe respiratory and even circulatory failure. Since pulmonary emphysema is much more common than lung cancer, if the relation between heavy smoking and pulmonary emphysema is confirmed, its public health importance may be even greater than the relation between smoking and cancer of the lung.

So far as public interest goes, the most important question is the extent to which smoking is responsible for the progressive rise in cancer of the lung in recent decades. Last year about 24,000 men and 4000 women died of cancer of the lung—about eight times as many as 20 years ago. Mortality from no other type of cancer is increasing at a comparable rate.

News on cancer and tars

As we go to press, investigators at the Radium Institute of the University of Paris report the discovery of a chemical in cigarette smoke—3-4-9-10 dibenzpyrene—"which invariably causes cancer when injected into mice." Although it has long been known that benzpyrene chemicals are capable of inducing cancer in mice, the amounts present in tobacco tars have not been found sufficient to produce cancer consistently in a high percentage of experimental animals. The newspaper report does not indicate the relationship between the amount of dibenzpyrene which caused cancer in the mice and the amount of the chemical present in cigarette smoke.

Without a great deal more information than is yet available, it is impossible to know whether the Paris studies make a significant contribution to previous knowledge based on animal experiments.

There is no dispute among medical authorities that many materials in the air we breathe have cancer-inducing effects and probably play an important part in the rising incidence of cancer of the lung. There is dispute, however, as to the comparative importance of air pollutants as against heavy smoking as a causative factor in cancer of the lung. Heavy cigarette smoking is believed by many investigators—and by the American Cancer Society—to be an important, or even the major, factor contributing to the development of lung cancer. A different opinion is held by Dr. Clarence C. Little, Chairman of the Scientific Advisory Board of the Tobacco Industry Research Committee. "Any possible role of smoking in the etiology [cause] of lung cancer," says Dr. Little, "remains an unresolved question. It cannot be said that smoking has been absolved from suspicion; neither have the charges . . . been proven."

The point of view of the American Cancer Society, derived from several types of statistical studies on thousands of men and women with and without lung cancer, is summarized in a recent (1956) pamphlet "Where We Stand Today on Cigarettes and Lung Cancer," published by the American Cancer Society. According to this pamphlet, lung cancer is about 27 times as frequent among those who smoke two packages of cigarettes a day as among those who have never smoked. However, not everyone who smokes heavily develops lung cancer. The man who for many years has smoked heavily, two packages or so a day, has about one chance in ten of developing lung cancer eventually. A man who smokes less than a pack a day has about one chance in 36 of developing the disease. The chances of a non-smoker developing lung cancer are about one in 270.

Two important, large-scale studies on population groups in the United States and England, one by the American Cancer Society and the other by the British Medical Research Council, though not yet fully completed, appear to support the conclusion that heavy cigarette smoking is one

Continued on next page

CIGARETTES continued

of the most important causes of cancer of the lung. In both studies, lung cancer seems to occur less frequently in those who have given up cigarettes than in those who have smoked them continuously, though moderately. The British report considers significant a study of the smoking habits of doctors living in rural and urban districts. The high and closely similar incidence of cancer of the lung in the two groups of smokers could not, according to this study, have been affected by varying exposure to such atmospheric pollutants as industrial wastes and automobile exhausts.

Environmental factors

A different point of view about the relationship between environmental air carcinogens and cancer of the lung is contained in a recent Public Health Monograph by Dr. W. C. Hueper, head of the Environmental Cancer Section of the National Cancer Institute, U. S. Public Health Service. According to Dr. Hueper any final conclusion about the relative importance of cigarette smoking as a cause of lung cancer should be kept in abeyance until more valid and conclusive evidence becomes available. Dr. Hueper believes that tobacco smoking may play a role in causing cancer of respiratory organs, but that it is neither the only cause nor the main cause, nor is it predominantly responsible for the rising frequency of lung cancer observed during recent decades. He believes that exposure to many industrial fumes (chromates, nickel, arsenic and others) gasoline fumes, tire and asphalt dust, and radioactive dust is of great importance in producing cancer of the lung. Although only a small proportion of the 28,000 Americans who died of lung cancer last year were occupationally exposed to cancer-inciting environmental substances, it is Dr. Hueper's belief that in view of the far-flung pollution of the air, these carcinogenic substances are inhaled by the general population as well as by those occupationally exposed.

With the expansion of industry in the past 50 years, there has been a corresponding increase in air pollution. Fuel oil consumption is now 3½ times as great as it was 30 years ago. Exhausts from motor vehicles contribute to air pollution; motor fuel consumption is five times what it was in 1933. Roads surfaced with asphalt and oils are a source of fine dust which adds to air pollution.

Experiments with different animals to learn whether suspected substances in tobacco or tobacco smoke or in cigarette paper can actually produce lung cancer have so far provided inconclusive clues. Not only must one be cautious in relating to humans the results of experiments performed on mice, rats and other species, but these experiments have yielded contradictory results. However, recent experimental work by Dr. Ernest Wynder of the Sloan-Kettering Institute is of interest since it appears to show that carcinogenic effects depend upon the dose of the tar. Tars and hydrocarbons such as benzpyrene are at present among the suspected carcinogenic materials in tobacco smoke. As CU's tests show, all cigarettes contained appreciable amounts of tars. If Dr. Wynder's work is confirmed, low tar cigarettes should be less likely to induce lung cancer than cigarettes with normal tar level.

It would be gratifying, says the American Cancer Society, if it could "cut through the smoking turbulance and produce a neat final answer" to the controversy about the relation between various environmental agents, including smoking, and cancer of the lung. However, it cannot. The Society's policy is this: "It intends to make widely available facts that bear on the question of whether to smoke or not to smoke. . . . The American Cancer Society has no plans for a campaign against cigarette smoking. Smoking is a habit which gives comfort and pleasure to millions, supports a great industry, and brings millions to the government in taxes. . . . The final decision on whether to smoke cigarettes rests with the individual . . ."

A medical and statistical study of several hundred thousand veterans of World War I is being conducted by the U.S. Public Health Service to determine the relation between smoking and cancer of the lung and other disorders. It is hoped that the findings of this survey will help to clear up important aspects of the smoking controversy.

The most reasonable view of cancer of the lung at this time would relate its phenomenal and alarming rise partly to the diffusion into the air of carcinogens derived from industry and transportation, partly to unusual exposures to carcinogens in many occupations, and partly to excessive cigarette smoking. The control of lung cancer would, therefore, appear to require a three-pronged effort against 1) air pollution, 2) occupational exposure to specific carcinogenic agents, and 3) excessive cigarette smoking. Until more conclusive evidence is reported, it would seem prudent to reduce cigarette smoking to less than a pack a day, or to smoke a pipe or cigars in moderation.

Stop smoking?

For anyone to argue that everyone should stop smoking because of its hazards would be highly unrealistic. Relatively few smokers adopt the tobacco habit because of a sensuous appreciation of the fragrance of the cured tobacco leaf. In adolescence, smoking is frequently a symbol of emancipation from the fetters of childhood. Heightened nervous tension is the usual explanation given for the tobacco habit, and there is much evidence to support it.

During times of national crisis, as in a war, tobacco consumption rises sharply. At all times, man is exposed to a multitude of stresses and tensions. Smoking is doubtless one of the habits man has adopted to contend with these tensions. Reaching for a cigarette, cigar or pipe, holding it in the mouth, sucking on it, lighting it, inhaling it, and blowing out the smoke all tend to relieve tensions.

The stimulating or comforting effects of tobacco may be so valuable to some persons that they are willing to risk whatever physical harm may be associated with the habit. On the other hand, there are many for whom these risks are not worthwhile and who will, without too great a sacrifice, cut out or cut down their cigarette smoking.

It would be gratifying to conclude this article with a successful formula for giving up the tobacco habit. Mark Twain said that to stop smoking was easy. "I ought to know," he asserted, "because I've done it a thousand times." Modern-day medicine has no better answer.

EXHIBIT 16

CHEMICAL LABORATORY

Following is the first of several reports on cigarettes, cigarette smoke, and filters by the Chemical Laboratory of the American Medical Association. The work was done by the Laboratory at the request of the Advertising Committee of the American Medical Association. Many of the technical details of the work, which was carried out by Mr. Robert R. Stark of the laboratory staff, have been omitted in this presentation and can be obtained upon request from the Chemical Laboratory.

WALTER WOLMAN, PH.D., *Director.*

A STUDY OF CIGARETTES, CIGARETTE SMOKE, AND FILTERS

1. Filter-Tip Cigarettes

Considerable interest has been expressed concerning the effectiveness of filter-tip cigarettes and the so-called denicotinizing process in reducing the amount of nicotine and tars in cigarette smoke. Because of the scarcity of published data on the subject, the Chemical Laboratory of the American Medical Association has undertaken a study of nicotine and tars in the smoke of filter cigarettes, special low-nicotine cigarettes, and "regular" cigarettes and the effectiveness of filter holders in removing nicotine and tars from cigarette smoke. This first report will deal only with the effect of the filters of the three largest selling filter-tip cigarettes. They represent the three types of filter-tips on the market, namely, paper, asbestos, and cotton. During the course of this investigation changes were made in the filters of each of the three brands, either to improve the filtering efficiency or to change the smoking characteristics of the cigarette. The cigarettes with the original filters are designated as A-1, B-1, and C-1, and the cigarettes with modified filters are designated as A-2, B-2, and C-2 in table 1. A "regular" all-tobacco cigarette, brand D, was also tested. These experiments are not designed to interpret such qualities of cigarettes as flavor, aroma, or smoking characteristics.

As stated by Bradford, Harlan, and Hanmer,[1] a laboratory smoking process should be sufficiently like human smoking to allow acceptance of the data collected, and it must be reproducible. Puffing the cigarette by mouth with an absorption apparatus placed between the cigarette and mouth meets the first requirement, but is not reproducible. Continuous suction applied to the cigarette as employed by Bogen,[2] Koperina,[3] and others is far from meeting the first requirement as the intensified heat produced from the long incandescent coal changes the composition of the smoke. Constant suction interrupted at timed intervals by a stopcock or valve has been employed by Baumberger,[4] Bogen,[2] Jensen and Haley,[5] and others. This method is open to criticism because the volume of puff varies between cigarettes, depending on differences in tightness of packing. Moreover, the volume increases significantly in each cigarette as the smoking proceeds and the air resistance decreases as the cigarette becomes shorter.

1. Bradford, J. A.; Harlan, W. R., and Hanmer, H. R.: Nature of Cigaret Smoke: Technic of Experimental Smoking, J. Indust. Engin. Chem. **28**: 836-839, 1936.

2. Bogen, E.: The Composition of Cigarets and Cigaret Smoke, J. A. M. A. **93**: 1110-1114 (Oct. 12) 1929.

3. Koperina, A.: Untersuchung der Stickstoffhaltigen Verbindungen des Tabakrauches, Biochem. Ztschr. **219**: 258-276, 1930.

4. Baumberger, J. P.: (a) The Carbon Monoxide Content of Tobacco Smoke and Its Absorption on Inhalation; (b) The Nicotine Content of Tobacco Smoke; (c) The Amount of Smoke Produced from Tobacco and Its Absorption in Smoking as Determined by Electrical Precipitation, J. Pharmacol. & Exper. Therap. **21**: 23-57, 1923.

5. Jensen, C. O., and Haley, D. E.: Studies on the Nicotine Content of Cigarette Smoke, J. Agric. Res. **51**: 267-275, 1935.

6. Pfyl, B.: Zur Bestimmung des Nicotins im Tabakrauch: II: Normung des künstlichen Verrauchens der Tabakerzeugnisse, Ztschr. f. Untersuch. d. Lebensmitt. **66**: 501-510, 1933.

7. Pyriki, C.: Über die Verteilung des Nicotins beim Rauchen von Zigaretten, Chem.-Ztg. **58**: 279, 1934.

These objections are overcome by a constant volume smoking device, such as that described by Pfyl,[6] in which the cigarette was puffed by air displacing mercury in a tube as the mercury level was lowered by means of a leveling bulb. Pyriki,[7] using Pfyl's apparatus to smoke oriental cigarettes to a 15 mm. butt, taking 40 ml. puffs every 30 seconds, found 19 to 27% of the nicotine in the mainstream smoke (smoke emerging from the puffed end of the cigarette), 37 to 47% in the sidestream (smoke rising from the burning tip), and 13 to 17% in the butts, with a total recovery of 74 to 85% of the nicotine. The remainder of the nicotine is apparently decomposed by the heat of the coal.

The cigarettes in the present tests were smoked with the automatic smoking machine described by Bradford, Harlan, and Hanmer.[1] This is a constant volume smoking device in which the mercury is replaced by water as the operating fluid. The smoke is passed through a glass tube and collected by gravity deposition in a 300 ml. flask to which only two supplementary bubblers are attached. These are specially designed to reduce the back pressure to about two inches of water. The smoking machine itself consists of a constant-level reservoir that supplies water to a buret equipped with a glass float. Four cigarettes are smoked simultaneously by taking a puff from each in succession. This is accomplished by motor-driven valves timed by a clock motor. During the puff, suction created by the emptying buret draws air through the cigarette. The puff is terminated when the falling water level seats the glass float in a ground glass constriction at the bottom of the buret.

In smoking, a large number of variables are encountered. Six of these listed by Pfyl are rapidity of the puff, time of the puff, volume of the puff, number of puffs, duration of smoking, and the length of the butt. The effect of the length of the butt on the nicotine content of the smoke is confirmed by Jensen and Haley,[5] who have shown that as a 70 mm. cigarette is smoked from four-sevenths to six-sevenths of its length, that is from a 30 mm. to a 10 mm. butt, the nicotine appearing in the smoke of each cigarette increases from 1.3 mg. to 3.8 mg. These variables may be eliminated by assigning fixed values to each of them. The results of the smoking experiments will depend on the values selected, but under any given set of conditions comparisons between cigarettes and comparisons in the effectiveness of filter-tips are made possible.

The smoking conditions adopted by the A. M. A. Laboratory are those of Bradford and co-workers.[1] In this process 47 mm. of each cigarette is smoked (two-thirds of a "standard" 70 mm. cigarette) using 35 ml. puffs of two seconds' duration taken once a minute. The size of the puff is considered reasonable in view of Pfyl's observation that puffs varied among individuals from 27 to 61 ml. The time between puffs is sufficiently long to allow the cigarette to return to a free burning state and to prevent the formation of an unnaturally long and hot coal.

In addition to the variables encountered in the smoking process certain variables are inherent in the cigarettes themselves within a given brand, and the results of the experiments will depend to a certain degree upon them. Variations in type, weight, cut,[8] and tightness of packing of the tobacco may be expected. By careful selection of cigarettes based on their weight, some of these variations can be minimized but they cannot be eliminated completely.

Five cigarettes were smoked for each single determination. The smoking of a larger number of cigarettes (up to 50) did not produce more reliable or consistent results, and the sheer volume of smoke was difficult to collect without losses. The average weight of each lot of cigarettes was determined and only those cigarettes weighing within 20 mg. of this average were smoked to eliminate gross differences in amount of tobacco and tightness of packing. The cigarettes were smoked

8. Wenusch, A.: Influence of the Width of the Cuttings on the Passage of the Nicotine into the Main Smoke Stream, Ztschr. f. Untersuch. d. Lebensmitt. **75**: 182-184, 1938.

9. Bradford, J. A.; Harlow, E. S.; Harlan, W. R., and Hanmer, H. R.: Nature of Cigaret Smoke: Volatile Bases and Acids, J. Indust. Engin. Chem. **29**: 45-50, 1937.

in a room maintained at 77° F (25° C) and 45% relative humidity.

The mainstream smoke was collected and the nicotine in the smoke determined by the method of Bradford, Harlow, Harlan, and Hanmer.[9] The smoke was collected in a 300 ml. Kjeldahl flask containing 5 ml. of alcohol and 5 ml. of alcoholic 0.5 *N* sulfuric acid. The smoke was not bubbled through the solution, but the large free space above the solution allowed the smoke to settle undisturbed by subsequent puffs. The effectiveness of gravity deposition of smoke obviated the need for a long absorption train. The two supplementary bubblers filled with glass beads and containing 5 ml. of alcoholic 0.5 *N* acid and 5 ml. of aqueous 0.5 *N* acid, respectively, trapped any nicotine not collected in the flask.

The smoke solution was rendered alkaline and the nicotine steam distilled into a dilute acid. The nicotine was then precipitated as the silicotungstate.

The nicotine content of the tobaccos of each of the brands of cigarettes smoked was also determined. The method used was that of the Association of Official Agricultural Chemists.[10]

In separate runs, tars were determined by again collecting the smoke by gravity deposition over 25 ml. of 0.1 *N* sulfuric acid in the 300 ml. Kjeldahl flask. The two bubbler tubes, loosely packed with asbestos held in place with cotton plugs, were placed between the flask and the smoking machine. Upon completion of a run, the smoke was allowed to settle 20 minutes and then the smoke tube and bubbler tubes were washed into the flask with chloroform. The material in the flask was extracted with chloroform. The chloroform was evaporated, and the tars were dried for three hours at 100° C.

The results of the experiments are given in tables 1 and 2 and in the graph. Table 1 lists the type of filters tested, the physical characteristics of the cigarettes and filters, and the percentage of moisture and nicotine found in the tobacco. Table 2 and the graph give the results obtained from the smoking of the cigarettes. It should be noted that the weights of nicotine and tars found in cigarettes and their smoke will vary in different lots of the same brand of tobacco. The amount of nicotine in the smoke is a function of the amount of nicotine originally present in the tobacco. The nicotine content of tobacco is subject to great variations, depending on the position of the leaf on the plant, weather conditions during the growing season, type of tobacco, region of growth, and cultural practices. The nicotine content of different brands of cigarettes will vary, depending on the blend of tobaccos, and cigarettes of the same brand may vary considerably if the manufacturer's controls are poor or nonexistent.

In table 2, column 2, the figures are obtained by multiplying the average number of puffs required to smoke the cigarette to the 47 mm. mark by the volume of each puff (35 ml.) and represent the average volume of smoke sucked into the absorption train. This mainstream smoke represents the smoke that presumably would reach the smoker's mouth. Columns 3 and 4 of table 2 show the weight of nicotine in this smoke with the filters removed and intact, respectively. The percentage reduction in nicotine in the mainstream effected by the filter is listed in column 5 and is based on the difference between columns 3 and 4.

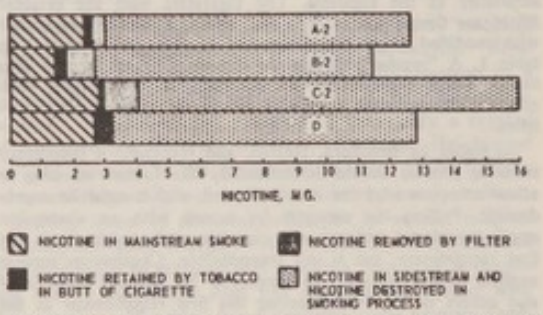
In columns 8 and 9 of table 2, the weights of the tars appearing in the mainstream smoke per cigarette are given without and with the filters attached, respectively. Column 10 lists the reduction in tars in the mainstream effected by the filter. These figures are based on the difference between columns 8 and 9 and are in close agreement with the figures in column 5.

The figures on reduction in nicotine and tars in columns 5 and 10 favor the filters and are deceptive because the figures for nicotine and tars in the mainstream smoke listed in columns 3 and 8 were obtained from the short cigarettes resulting from the removal of the filters. Tobacco itself is a rather good filter, and, if the filter-tips were to be replaced by an equal length of tobacco, the mainstream smoke from the resulting all-tobacco cigarettes of brands A, B, and C would contain less nicotine and tars than are listed for the shortened cigarettes in column 5 and 10, because the tobacco replacing the filter-tips would remove a certain amount of these substances.

This laboratory has found that, if the paper filter of brand A-1 is replaced by 11 mm. of tobacco, the additional tobacco will remove 8% of the nicotine and tars from the mainstream smoke. This is comparable with the 9% reduction in nicotine and 5% reduction in tars effected by the paper filter of brand A-1. Similar reductions might be expected to occur in columns 5 and 10, table 2, for brands B and C if the removed filter-tips were replaced by equal lengths of tobacco.

In the graph, the distribution of the nicotine from the smoked portion of the cigarette is shown for the brand 2 series of filter-tips and the all-tobacco cigarette, brand D. Comparisons between brand D and other members of the series are difficult to make when the initial nicotine contents of the smoked portions of the cigarettes are different as shown by differences in the total lengths of the bars. A comparison between brand D and brand A-2 can be made because the initial nicotine contents of the smoked portions of the two cigarettes are very nearly equal. It can readily be seen from the graph that the amount of nicotine in the mainstream smoke from the two cigarettes is almost identical. It is also apparent that the amount of nicotine filtered by the tobacco of the butt of cigarette brand D is almost as great as the amount of nicotine filtered by the tobacco remaining in the butt plus the filter of brand A-2. It is interesting to note from the graph that although the total amount of nicotine trapped by the filter of brand C-2 is greater than that trapped by the filter of brand B-2, the percentage efficiency is lower (column 5 of table 2). This is a direct result of the fact that the tobacco of brand C-2 has a larger nicotine content in the portion of the cigarette smoked, and consequently the concentration of nicotine in the smoke passing through the C-2 filter is greater than that passing through the B-2 filter.

Since the amount of nicotine that reaches the smoker's mouth is of primary importance, the practical ineffectiveness



Distribution of nicotine from smoked portion of four brands of cigarettes.

of the filter of brand C-2 is demonstrated by the length of the bar that measures the nicotine in the mainstream smoke. Brand C-2 contains more nicotine in this portion of the smoke than any other brand shown in the graph. Brand C-1, an earlier product of the same brand name, showed an even larger amount of nicotine in the mainstream smoke.

Columns 6 and 7, table 2, show the percentage, expressed as the nearest whole number of the total nicotine in the smoked portion of the tobacco that is transferred to the mainstream smoke without and with the filters. In column 6 the figures are again shown for the shorter cigarette resulting from the removal of the filter. To compensate for the shorter cigarette, corrections may again be used here that are based on the substitution of a length of tobacco equal to the length of the filter. They are proportional to the corrections that may be applied to columns 5 and 10 and would reduce the figures in column 6 by about 2%.

The figures in column 6 show that the proportion of nicotine transferred from the smoked portion of the cigarette to the mainstream smoke is surprisingly constant. If a 2% correction were applied to compensate for the shorter cigarettes, the values would range from 19 to 23%. These corrected figures would be in close agreement with the work of Bradford and co-workers.[9] The values in column 7 for brands A-1, A-2, C-1, C-2, and D agree closely with one another and with the corrected values for the cigarettes without filters. These values and the bars of the graph indicate that the filters are responsible for removing very little of the total nicotine in the smoked portion of the cigarette. The bulk of this nicotine, about 75% in the case of "regular" cigarettes, is lost in the sidestream or is destroyed by the burning coal.

On the basis of results obtained in the smoking experiments, it can be seen that the over-all effectiveness of the filters of brands A and C is not significant. Indeed, it can be seen from column 4 of table 2 and from the graph that the nicotine content of the mainstream smoke of brand C is exceptionally high—higher than that of "regular" cigarettes. The filter of brand B-1 is the most effective in removing nicotine and tars from the mainstream smoke. However, the sample of later manufacture, brand B-2, is much less effective, as the filter is more loosely packed, apparently in an effort to improve the smoking qualities.

In all cases the fraction of nicotine removed by the filter from the smoked portion of the cigarette is small. Even in a "regular" cigarette with no filter-tip only 21% of the total nicotine reaches the smoker in the mainstream smoke. In recent advertisements calculations have been contrived to advance claims for very low percentages of nicotine in the mainstream smoke. Widely divergent values can be obtained from the same analytical data choosing different bases for the calculations. For example, an extremely low value can be obtained by basing the calculation on the total weight of the cigarette. Using this stratagem in calculating, the figure 0.25% nicotine in the smoke of the "regular" cigarette, brand D, is obtained. Thus an apparently low figure is obtained from a normal nicotine content.

10. Association of Official Agricultural Chemists, Official Methods of Analysis, ed. 7, Menasha, Wis., The Collegiate Press, George Banta Publishing Company, 1950, p. 69.

TABLE 1.—*Moisture, Nicotine Content, and Physical Characteristics of Four Brands of Cigarettes*

Column	1	2	3	4	5	6	7	8	9
	Type of Filter	Date Sample Obtained	Average Weight of Cigarette, Gm.	Average Weight of Filter-Tip, Gm.	Total Length of Cigarette, Mm.	Average Length of Filter-Tip, Mm.	Average Circumference of Cigarette, Mm.	Moisture in Tobacco, %	Nicotine in Tobacco (Moisture-Free Basis), %
Brand A-1	Paper	May, 1952	1.005	0.105	70.0	11.0	25.0	8.06	1.93
Brand A-2	Fiber	December, 1952	1.133	0.183	69.5	11.8	25.3	9.76	1.92
Brand B-1	Asbestos laminated with paper	April, 1952	1.059	0.159	69.5	10.9	25.7	8.61	1.88
Brand B-2	Asbestos laminated with paper	October, 1952	1.011	0.110	69.5	10.9	25.7	9.84	1.94
Brand C-1	Cotton	May, 1952	1.047	0.168	69.0	10.0	25.3	9.27	2.54
Brand C-2	Cotton	August, 1952	1.053	0.173	69.0	10.0	25.3	10.77	2.44
Brand D	No filter	February, 1953	1.004	.....	70.0	....	25.5	10.71	2.02

TABLE 2.—*Analysis of Smoke from Four Brands of Cigarettes*

Column	1	2	3	4	5	6	7	8	9	10
	Weight of Tobacco Actually Smoked (Dry Weight), Gm.	Average Volume of Mainstream Smoke per Cigarette, Ml.	Weight of Nicotine Found in Mainstream Smoke per Cigarette, Mg.		Reduction of Nicotine in Mainstream Smoke, %	Nicotine in Tobacco Actually Smoked Which is Transferred to Mainstream Smoke, %		Weight of Tars Found in Mainstream Smoke per Cigarette, Mg.		Reduction of Tars in Mainstream Smoke, %
			With Filter-Tip Removed	With Filter-Tip		With Filter-Tip Removed	With Filter-Tip	With Filter-Tip Removed	With Filter-Tip	
Brand A-1	0.979	340	2.85	2.60	9	24	21	16.9	15.1	6
Brand A-2	0.980	351	2.82	2.43	14	22	19	16.8	13.9	17
Brand B-1	0.635	328	2.52	1.00	60	21	8	15.0	6.8	55
Brand B-2	0.591	350	2.49	1.47	41	22	13	16.7	9.4	44
Brand C-1	0.681	461	3.01	2.38	14	23	20	18.5	15.6	16
Brand C-2	0.655	415	4.00	2.93	27	25	18	19.6	15.1	23
Brand D	0.635	356	....	2.74 *	..	..	21 *	....	17.8 *	..

* Placed in this column for comparison with full-length cigarettes.

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CHEMICAL LABORATORY

This is the second of several reports on cigarettes, cigarette smoke, and filters by the Chemical Laboratory of the American Medical Association. The first report appeared in THE JOURNAL, *July 4, 1953, page 917.*

WALTER WOLMAN, PH.D., *Director.*

A STUDY OF CIGARETTES, CIGARETTE SMOKE, AND FILTERS

2. Special Low-Nicotine Cigarettes

There is considerable difference in the nicotine content of the various types of tobacco used in cigarettes. Bailey and Petre [1] and Bogen [2] reported the following approximate values for nicotine content of several types of cigarettes:

Type of Cigarette	Approximate Content, % Nicotine
Blended	1.0-2.7
Virginia (flue-cured)	2.0-3.1
Levantine	1.0-1.5
Denicotinized	0.8-1.4
Puerto Rican	0.8-1.2

A typical modern blended cigarette contains approximately the following proportions of tobacco [1]:

Flue-cured	60%
Burley	15%
Maryland	10%
Levantine types	15%

Some of the more inexpensive brands omit the Levantine types.

The nicotine content of flue-cured tobaccos was found to range from an average of 2.36% for the low-nicotine South Carolina tobacco (U. S. type 13) to 3.36% for the higher nicotine Danville tobacco (U. S. type 11)[3] for the five years 1928 through 1932. Burley tobaccos, on the other hand, contain more nicotine than flue-cured tobaccos. A nicotine content of about 3.5% is probably representative for this type. Maryland tobacco and Levantine types have a relatively low nicotine content. Bailey and Petre [1] found 2.12 and 2.26% nicotine in the leaf web of a sample of Maryland tobacco from the 1930 season.

These nicotine values are subject to considerable variation, depending on the position of the leaf on the plant, weather conditions during the growing season, type of tobacco, region of growth, and cultural practices. The values reported by Bailey and Petre [1] for the Maryland tobacco grown in 1930 were high because hot, dry weather had prevailed during the growing season. A sample of the same grade and type from the 1932 crop gave values of 0.86 and 0.83% nicotine.

Many investigators have reported that the nicotine content of the leaf increases from the bottom to the top of the plant. Darkis and his collaborators [4] reported that the nicotine in flue-cured tobacco from a good growing season ranged from 1.94% in the lower leaves (secured by the first priming—a method of harvest in which leaves are pulled from the plant as they mature) to 3.76% in the upper leaves (the sixth priming), with a composite sample containing 3.04% nicotine. This entire range will not appear in tobacco destined for cigarettes, however, since generally the leaves from near the middle of the plant are employed for this purpose. Most of the remainder goes into other smoking mixtures and chewing tobacco.

Tobaccos for the so-called denicotinized cigarettes are prepared from regular tobaccos. According to Bailey,[5] they are usually prepared by a resweating process. This is accomplished by treatment of the tobacco with superheated steam or by heating in vacuum chambers. Numerous other processes are based on the extraction of the nicotine by organic solvents or on the treatment of the tobacco with the vapors of an aqueous ammonia solution. These vapors decompose the naturally occurring salts of nicotine (oxalate, acetate, citrate, and malate), and the free nicotine is entrained in the vapors.

Certainly the term "denicotinized" is a misnomer, as it would connote "nicotine-free" to the uninitiated, and the presently available brands contain about one-half the nicotine found in "regular" cigarettes. Pyriki [6] has proposed the terms "nicotine-poor" for tobacco containing 0.5 to 0.6% nicotine and "nicotine-free" for tobacco containing 0.1 to 0.15% nicotine. None of the tobaccos examined are nicotine-free in this sense.

The tobacco used in the cigarette designated as brand G in this report was developed by the Kentucky Agricultural Experiment Station [7] from three strains of low-nicotine Cuba cigar tobacco obtained in 1933 from the Kaiser Wilhelm Institute. This tobacco was crossed with burley in order that the new variety "should carry the recessive color factors of burley because burley is a light-colored tobacco when cured and light color is commonly associated in the mind of the public with mildness."[7] The varieties resulting from the crosses and back crosses of the low-nicotine cigar tobacco with burley are like burley but have a low nicotine content. They have been designated a subtype of burley and have been established as class 3, type 31-V, air-cured tobacco by the United States Department of Agriculture.

This report gives the results of smoking experiments on two brands of so-called denicotinized cigarettes, brands E and F, and one brand of cigarette containing the tobacco especially bred for its low nicotine content, brand G. Brand D, the "regular" cigarette from the first report,[8] is again included for comparison.

The methods of analysis used and the conditions employed have been described in the first report.[8] Briefly, 47 mm. of each cigarette was smoked (two-thirds of a "standard" 70 mm. cigarette) using 35 ml. puffs of two seconds' duration taken once a minute.

Brand G is the only cigarette examined thus far that has contained significant quantities of nornicotine. Nornicotine differs chemically from nicotine in that the nitrogen atom of the pyrrolidin ring is not methylated. The figures in column 6 of the table were obtained by the method of the Association of Official Agricultural Chemists [9] with the exception of the figure for brand G. These figures for brand G were obtained by the method of Bowen and Barthel,[10] which quantitatively measures the amounts of both nornicotine (0.45%) and nicotine (0.31%). The value found by the Association of Official Agricultural Chemists method for the total steam volatile alkaloids including nornicotine was 0.79%.

As in the first report, the figures in column 8 of the average volume of mainstream smoke (smoke emerging from the puffed end of the cigarette) per cigarette were obtained by multiplying the average number of puffs required to smoke 47 mm. of a

1. Bailey, C. F., and Petre, A. W.: The Modern Cigaret Industry, J. Indust. Engin. Chem. **20**: 11-19, 1937.
2. Bogen, E.: The Composition of Cigarets and Cigaret Smoke, J. A. M. A. **93**: 1110-1114 (Oct. 12) 1929.
3. Darkis, F. R.; Dixon, L. F., and Gross, P. M.: Flue-Cured Tobaccos Factors Determining Type and Seasonal Differences, J. Indust. Engin. Chem. **27**: 1152-1157, 1935.
4. Darkis, F. R.; Dixon, L. F.; Wolf, F. A., and Gross, P. M.: Flue-Cured Tobacco: Correlation between Chemical Composition and Stalk Position of Tobaccos Produced under Varying Weather Conditions, J. Indust. Engin. Chem. **28**: 1214-1223, 1936.
5. Bailey, E. M.; Nolan, O. L., and Mathis, W. T.: "Denicotinized" Tobacco, Conn. Agr. Expt. Sta. Bull. **295**: 338-351, 1928.
6. Pyriki, C.: Über das Auftreten von Nicotin in Zigarettenrauch, Ztschr. f. Untersuch. d. Lebensmitt. **62**: 95-98, 1931.
7. Valleau, W. D.: Breeding Low-Nicotine Tobacco, J. Agric. Res. **78**: 171-181, 1949.
8. A Study of Cigarettes, Cigarette Smoke, and Filters: I. Filter-Tip Cigarettes, report of the Chemical Laboratory, J. A. M. A. **152**: 917-920 (July 4) 1953.
9. Association of Official Agricultural Chemists, Official Methods of Analysis, ed. 7, Menasha, Wis., The Collegiate Press, George Banta Publishing Company, 1950, p. 69.
10. Bowen, C. V., and Barthel, W. F.: Determination of Nicotine and Nornicotine in Tobacco, J. Indust. Engin. Chem., Anal. Ed. **15**: 740-741, 1943.

cigarette by the volume of each puff (35 ml.). The figures in column 10 showing the proportion of nicotine transferred from the smoked portion of the cigarette to the mainstream smoke are again relatively constant as in the first report, with the exception of brand G.

The weight of nicotine found in the mainstream smoke as shown in column 9 shows that the smoke of the "denicotinized" brands E and F contains about one-half the nicotine found in the smoke of a "regular" cigarette, brand D. The amount of tars in the smoke of these cigarettes does not differ materially from the tars found in the smoke of brand D as shown in column 11. The smoke of brand G contains significantly less nicotine than that of other brands but it contains a greater amount of tars.

Physical Characteristics and Analysis of Four Brands of Cigarettes

	1	2	3	4	5	6	7	8	9	10	11
	Date Sample Obtained	Average Weight of Cigarette, Gm.	Length of Cigarette, Mm.	Average Circumference of Cigarette Mm.	Moisture in Tobacco, %	Nicotine in Tobacco (Moisture-Free Basis), %	Weight of Tobacco Actually Smoked (Dry Weight), Gm.	Average Volume of Mainstream Smoke per Cigarette, Ml.	Weight of Nicotine Found in Mainstream Smoke per Cigarette, Mg.	Nicotine in Tobacco Actually Smoked which is Transferred to Mainstream Smoke, %	Weight of Tars Found in Mainstream Smoke per Cigarette, Mg.
Brand E	October, 1952	1.003	69.5	24.4	10.01	0.99	0.625	344	1.37	22	15.4
Brand F	April, 1953	1.039	70.0	24.0	9.66	0.93	0.667	321	1.41	25	16.4
Brand G	April, 1953	1.028	69.7	25.3	8.99	0.31 0.45*	0.664	331	0.67 0.35*	36 0*	28.2
Brand D	February, 1953	1.001	70.0	25.5	10.71	2.02	0.635	356	2.74	27	17.8

* Nornicotine.

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CHEMICAL LABORATORY

This is the third of several reports on cigarettes, cigarette smoke, and filters by the Chemical Laboratory of the American Medical Association. The first report appeared in THE JOURNAL, *July 4, 1953, page 917. It concerned filter tip cigarettes. The second report appeared in* THE JOURNAL, *July 11, 1953, page 1035. It concerned cigarettes in which the tobacco is claimed to have a low nicotine content.*

WALTER WOLMAN, PH.D., *Director.*

A STUDY OF CIGARETTES, CIGARETTE SMOKE, AND FILTERS

3. Cigarette Holders

The Chemical Laboratory has examined five brands of cigarette holders that contain filters to determine their efficiency in removing nicotine and tars from cigarette smoke. The methods of analysis and experimental conditions for smoking have been described in a previous report.[1] Briefly, 47 mm. of each cigarette was smoked (two-thirds of a "standard" 70 mm. cigarette) using 35 ml. puffs of two seconds' duration taken once a minute.

A "standard" 70 mm. cigarette, one of the best-selling cigarettes, was used in the smoking experiments. These cigarettes weighed an average of 1.096 gm., were 69.9 mm. long, and had a circumference of 26.5 mm. The moisture content of the tobacco was 11.52% and the tobacco contained 2.08% nicotine on a moisture-free basis. The dry weight of the tobacco actually smoked was 0.631 gm. per cigarette.

Of the cigarette holders examined, both numbers 1 and 2 employ a metal trap like those found in many pipes. Number 3 uses a cylindrical paper filter measuring 56 mm. in length and 6 mm. in diameter and containing a number of small paper baffles. Number 4 uses a plastic cylinder measuring 36 mm. in length and 9 mm. in diameter and filled with granules of silica as a filter. Number 5 uses a cigarette inserted in the holder as the filter.

All cigarette holders tested changed the smoking characteristics of the cigarettes in two ways: First, air leaked in between the cigarette and the holder in all cases and around the ejectors in holders number 4 and 5. This leakage amounted to more than 5% in some individual holders. Second, all the holders used a compression fit to reduce this air leakage. The specially machined Bakelite holders used in this experiment for smoking the comparison cigarettes provided a rather snug fit with an internal diameter of 8.3 mm. (The cigarettes were sealed in these holders with a concentrated dextrose syrup that prevented any air from leaking between the holder and the cigarette and insured that the entire 35 ml. puff was drawn through the cigarette.) Holder number 1, however, compressed the butt end of the cigarettes to 7.4 and 7.2 mm. and holder number 2 to 7.6 and 7.2 mm.; holders 3, 4 and 5 all compressed the butts to 7.8 mm. diameter.

The results of the experiments are given in the table. The figures in column 1 are obtained by multiplying the average number of puffs required to smoke the cigarette to the 47 mm. mark by the volume of each puff (35 ml.) and represent the average volume of smoke drawn into the absorption train. This mainstream smoke represents the smoke that presumably would reach the smoker's mouth. Column 2 shows the weight of nicotine in this smoke. Column 3 shows the weight of nicotine recovered from the holder and its trap or filter, and represents the nicotine removed from the mainstream smoke as it passes through the holder. Column 4 is the total of columns 2 and 3. The percentage reduction in nicotine in the mainstream effected by the holder is listed in column 5 and is based on the difference between columns 4 and 2.

In columns 6 and 7, the weight of tars appearing in the mainstream smoke per cigarette and the weight of tars recovered from the holder and its trap or filter are given. The totals of these tars are listed in column 8. The percentage reduction in tars in the mainstream effected by the holder is listed in column 9 and is based on the difference between columns 8 and 6.

Removal of Nicotine and Tars from Smoke by Cigarette Holders

Column	1 Average Volume of Mainstream Smoke per Cigarette, Ml.	2 Weight of Nicotine Found in Mainstream Smoke per Cigarette, Mg.	3 Weight of Nicotine Recovered from Holder per Cigarette, Mg.	4 Total Nicotine, Mg.	5 Reduction of Nicotine in Mainstream Smoke by Holder, %	6 Weight of Tars Found in Mainstream Smoke per Cigarette, Mg.	7 Weight of Tars Recovered from Holder per Cigarette, Mg.	8 Total Tars, Mg.	9 Reduction of Tars in Mainstream Smoke by Holder, %
"Standard" Cigarette	375	2.65	....	2.65	..	18.2	...	18.2	..
Holder 1	380	2.50	0.13	2.63	5	15.5	1.4	16.9	8
Holder 2	393	2.45	0.10	2.55	4	15.8	1.9	17.5	11
Holder 3	367	2.57	0.20	2.77	7	15.1	1.5	16.6	9
Holder 4	373	2.28	0.37	2.65	14	14.7	3.8	18.5	21
Holder 5	378	1.53	1.06	2.59	41	10.8	...	....	41

The tars absorbed by the tobacco in the cigarette used as the filter in holder number 5 could not be recovered. An approximate value for the weight of tars absorbed by holder number 5 may be calculated by assuming that the value for total tars found for the "standard" cigarette may be substituted for the total tars of holder number 5. The weight of the tars that were recovered from holder number 5 may then be calculated by subtracting the weight of tars found in the mainstream smoke (10.8 mg.) from the substituted value (18.2 mg.).

The figures in the table are the averages per single cigarette for runs of the first five cigarettes smoked through a clean holder and filter. The efficiency of most of the holders dropped as additional cigarettes were smoked. When 20 cigarettes were smoked through the holders, number 1 lost 27% of its original effectiveness, number 2 lost 42%, number 3 lost 14%, number 4 lost 34%, and number 5 lost none of its effectiveness.

Holder 5 was found to be the most effective of the filters tested. Holders 1, 2, and 3 were ineffective. The performance of holder 4 was intermediate between that of number 5 and the ineffective group.

1. A Study of Cigarettes, Cigarette Smoke, and Filters: I. Filter-Tip Cigarettes, a report of the Chemical Laboratory, J. A. M. A. 152:917-920 (July 4) 1953.

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CHEMICAL LABORATORY

This is the fourth report on cigarettes, cigarette smoke, and filters by the Chemical Laboratory of the American Medical Association. The first report, concerning filter-tip cigarettes, appeared in THE JOURNAL, *July 4, 1953, page 917. The second report, concerning cigarettes claiming to contain tobacco of low nicotine content, appeared in* THE JOURNAL, *July 11, 1953, page 1035. The third report, on cigarette holders, appeared in* THE JOURNAL, *Feb. 20, 1954, page 678. The laboratory work reported in this series was done by Mr. Robert R. Stark of the Chemical Laboratory.*

WALTER WOLMAN, Ph.D., *Director.*

A STUDY OF CIGARETTES, CIGARETTE SMOKE, AND FILTERS

4. Regular Cigarettes, King-Size Cigarettes, and Additional Filter-Tip Cigarettes

The Chemical Laboratory of the American Medical Association has examined a number of the largest selling brands among the regular and king-size cigarettes as well as a number of brands of filter-tip cigarettes that have appeared on the market since the Laboratory's original report [1] on filter-tip cigarettes. Reports on the effects of cigarette smoke on the health of habitual smokers have prompted the introduction of many new brands of filter-tip cigarettes during the past several months.

The methods of analysis and experimental conditions for smoking have been described in a previous report.[1] Briefly, 47 mm. of both the "standard" and "king-size" cigarette were smoked (67% of a standard 70 mm. cigarette and about 55% of a king-size cigarette) using 35 ml. puffs of two seconds' duration taken once a minute. In addition, king-size cigarettes were smoked 62 mm. to a 23 mm. butt (the same length butt as a 70 mm. cigarette smoked 47 mm.).

The results of the experiments are given in tables 1 and 2. Table 1 lists the type of cigarettes tested, the type of filter used, if any, the physical characteristics of the cigarettes and filters, and the percentage of moisture and nicotine found in the tobacco. Letters assigned to brands reported in previous papers have been retained as the first letter for the same brands in this report. The addition of the letters k and f to the letter symbolizing the brand name designates king-size and filter-tip respectively. Table 2 gives the results obtained from the smoking of the cigarettes.

In table 2, column 2, the figures are obtained by multiplying the average number of puffs required to consume the designated length of cigarette smoked by the volume of each puff (35 ml.) and represent the average volume of smoke drawn into the absorption train. This mainstream smoke represents the smoke that would reach the smoker's mouth. Column 4 of table 2 shows the weight of nicotine in the smoke from intact cigarettes and column 3 gives the weight of nicotine in the smoke obtained from the cigarettes in which the filter-tip was removed. The percentage reduction in nicotine in the mainstream smoke effected by the filter is listed in column 5 and is based on the difference between columns 3 and 4. In column 9 of table 2, the weights of the tars appearing in the mainstream smoke per cigarette are given for the intact cigarettes and column 8 gives the weights of tars in the smoke obtained from the cigarettes in which the filter-tip was removed. Column 10 lists the percentage reduction in tars effected by the filter, based on the difference between columns 8 and 9.

The figures for nicotine and tars in the mainstream smoke listed in columns 3 and 8 were obtained from the short cigarettes resulting from the removal of the filters, and are higher than would be obtained from intact all-tobacco cigarettes of regular or king-size length, since tobacco itself is a rather good filter. If the filters of the filter-tip cigarettes were to be replaced by an equal length of tobacco, this tobacco would filter out a certain amount of the nicotine and tars passing through it. This filtering effect of tobacco is illustrated in table 2 by the king-size cigarettes from which 15 mm. of tobacco was cut and the resulting 70 mm. cigarettes were smoked 47 mm. The amount of nicotine and tars appearing in the smoke was considerably greater than the amount obtained from the intact 85 mm. king-size cigarettes smoked 47 mm.

If a king-size cigarette is smoked 62 mm. instead of 47 mm. (to the same 23 mm. butt length as a regular 70 mm. cigarette smoked 47 mm.), 31.9% more tobacco is consumed; however, the nicotine and tars found in the mainstream smoke are higher than can be accounted for by the extra weight of tobacco smoked. As the cigarette is puffed and the smoke travels through the cigarette, the tobacco filters out a portion of the nicotine and tars. At each succeeding puff, therefore, the remaining tobacco becomes richer in nicotine and tarry material. As this enriched tobacco is burned during subsequent puffs a portion of the deposited nicotine and tars is transferred to the mainstream smoke. It is plain that one cannot have the added protection of the extra length of tobacco and at the same time have a 21% longer smoke as some ads for king-size cigarettes have claimed.

Columns 6 and 7, table 2, show the percentage of the total nicotine in the smoked portion of the tobacco that is transferred to the mainstream smoke without the filters and with the intact cigarette respectively. Under comparable conditions, the proportion of nicotine transferred from the tobacco of the smoked portion of the cigarette to the mainstream smoke is rather constant.

A factor contributing to differences in the smoking of the filter-tip cigarettes was the variation in the filters of the individual cigarettes. There were differences in porosity of the filters, but the greatest variation was in the length of the separate filters. The filters of brand M cigarettes ranged from 10.4 to 17.1 mm. long, with an average of 13.1 mm.; brand A filters ranged from 10.7 to 16.0 mm. long, with an average of 13.0 mm.; brand N filters ranged from 12.3 to 17.8 mm. long, with an average of 15.0 mm.; brand B filters ranged from 8.8 to 14.5 mm. long, with an average of 11.5 mm.; and brand O filters ranged from 9.5 to 17.0 mm., with an average of 13.2 mm. These differences are of little significance when the efficiency of the filter is about the same as the tobacco it replaces, but as the efficiency of the filter increases beyond this point, the variations become increasingly evident. Chemical examination showed that the brand M filter was not α-cellulose as claimed by the carton insert. Additional tests indicated that it was cellulose acetate.

1. A Study of Cigarettes, Cigarette Smoke, and Filters: 1. Filter-Tip Cigarettes, a report of the Chemical Laboratory, J. A. M. A. 152:917-920 (July 4) 1953.

At the present state of filter-tip development, filtering efficiencies in the range of those found in brands A, B, M, and O, seem to be the optimum for a commercially successful product, striking a sort of balance between effectiveness and a smoke too thinned to please many habitual smokers. The early history of the brand B filter seems to indicate this. These early brand B filters removed about 60% of the nicotine and tars from the mainstream smoke, but smokers apparently found it too difficult to draw through the tight filters and the smoke too thin to be satisfying. Consequently, the brand B filters were modified to their present state of effectiveness.

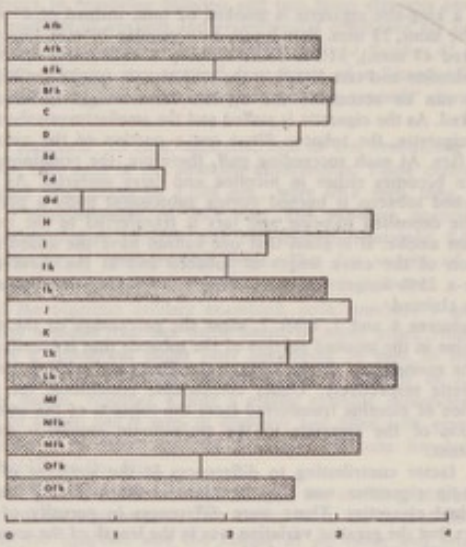
From the series entitled "A Study of Cigarettes, Cigarette Smoke, and Filters," data of greatest pertinence to the physician have been gathered together in the following résumé and are presented in graph form for ease of reference.

NICOTINE AND TARS IN MAINSTREAM SMOKE

The recent rise in the popularity of king-size and filter-tip cigarettes may be caused partly by the feeling that these types of cigarettes are giving a measure of health protection to the smoker. This feeling has been encouraged by advertisers who have dwelt on the real or fancied importance of such features as the efficiency of the filter, the type of filter material, and the length of the cigarette. Seldom has the advertiser given the information that is of prime importance to the smoker, namely, the amounts of smoke constituents that reach the smoker's mouth. The importance of this information lies in the fact that the amounts of these constituents that reach a smoker's mouth are proportional to the amounts assimilated by the smoker. The determinations of the amounts assimilated cannot easily be made because of the biological variations among individuals and the differences in their smoking habits.

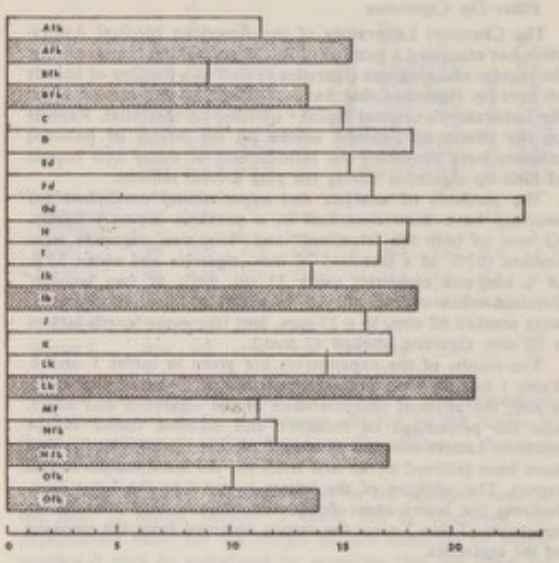
The amounts of nicotine and tars that reach a smoker's mouth can be approximated in the laboratory by the use of a smoking machine that simulates human smoking under carefully controlled conditions. The nicotine and tars in the mainstream smoke (the smoke that reaches the smoker's mouth) have been determined by this laboratory[1] for the best-selling cigarettes and brands for which special claims have been made. For ease of comparison and reference these data accumulated in the previous studies are brought together in figures 1, 2, 3, and 4.

Figures 1 and 2 show the amounts of nicotine and tars, respectively, that were found in the mainstream smoke of the cigarettes tested. Using standard cigarettes, brand D, figures 3 and 4 show the average nicotine and tar contents in the mainstream smoke based on the first five cigarettes smoked through various cigarette holders. The capital letters used to identify the brand names of the cigarettes correspond to the letters used in the earlier papers.



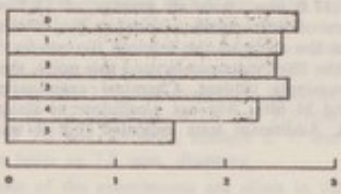
Nicotine, Mg.

Fig. 1.—Nicotine in mainstream smoke of cigarettes. Gray bars represent cigarettes smoked 47 mm. Cross hatched bars represent king-size cigarettes smoked 62 mm.



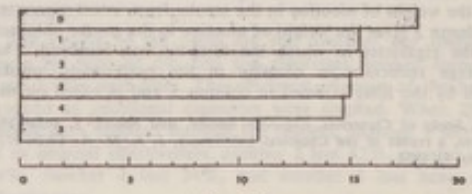
Tars, Mg.

Fig. 2.—Tars in mainstream smoke of cigarettes. Gray bars represent cigarettes smoked 47 mm. Cross hatched bars represent king-size cigarettes smoked 62 mm.



Nicotine, Mg.

Fig. 3.—Nicotine in smoke drawn through cigarette holders.



Tars, Mg.

Fig. 4.—Tars in smoke drawn through cigarette holders.

1. A Study of Cigarettes, Cigarette Smoke, and Filters: 1. Filter-Tip Cigarettes, a report of the Chemical Laboratory, J. A. M. A. **152**: 917-920 (July 4) 1953. 2. Special Low-Nicotine Cigarettes, ibid. **152**: 1035-1036 (July 11) 1953. 3. Cigarette Holders, ibid. **154**: 678 (Feb. 20) 1954. 4. Regular Cigarettes, King-Size Cigarettes, and Additional Filter-Tip Cigarettes, ibid. **157**: 1309 (April 9) 1955.

TABLE 1.—*Moisture, Nicotine Content, and Physical Characteristics of Cigarettes*

Column	1	2	3	4	5	6	7	8	9
Brand	Type of Filter	Date Sample Obtained	Average Weight of Cigarette, Gm.	Average Weight of Filter-Tip, Gm.	Total Length of Cigarette, Mm.	Average Length of Filter-Tip, Mm.	Average Circumference of Cigarette, Mm.	Moisture in Tobacco, %	Nicotine in Tobacco (Moisture-Free Basis), %
H	.....	Feb., 1954	1.038	.....	70.0	....	20.4	11.95	2.51
D	.....	Aug., 1953	1.096	.....	69.9	....	20.5	11.52	2.68
I	.....	Feb., 1954	1.088	.....	70.0	..	20.1	11.22	1.98
J	.....	Aug., 1954	1.053	.....	70.0	....	20.2	10.63	2.60
K	.....	May, 1954	1.072	.....	69.8	....	20.2	10.54	2.11
Lk	.....	June, 1954	1.235	.....	84.8	....	25.7	11.89	2.29
Ik	.....	Sept., 1954	1.297	.....	84.8	....	26.0	11.57	1.86
Mf	Cellulose acetate fibers.....	April, 1954	1.102	0.178	69.9	13.1	20.1	11.10	1.78
Afk	Cellulose acetate fibers.....	Jan., 1954	1.288	0.181	85.0	13.0	25.3	10.89	2.00
Nfk	Cellulose acetate fibers.....	July, 1954	1.205	0.156	85.0	15.0	25.8	11.78	2.12
Bfk	Asbestos laminated with paper.....	Oct., 1954	1.102	0.130	85.0	11.5	25.8	10.41	2.53
Ofk	Activated charcoal with paper.....	Nov., 1954	1.274	0.141	85.0	13.2	25.0	11.68	2.36

TABLE 2.—*Analysis of Smoke from Cigarettes*

Column		1	2	3	4	5	6	7	8	9	10
				Weight of Nicotine Found in Mainstream Smoke per Cigarette, Mg.			Nicotine in Tobacco Actually Smoked That Is Transferred to Mainstream Smoke, %		Weight of Tars Found in Mainstream Smoke per Cigarette, Mg.		
Brand		Weight of Tobacco Actually Smoked (Dry Weight), Gm.	Average Volume of Mainstream Smoke per Cigarette, Ml.	With Filter-Tip Removed	Intact	Reduction of Nicotine in Mainstream Smoke, %	With Filter-Tip Removed	Intact	With Filter-Tip Removed	Intact	Reduction of Tars in Mainstream Smoke, %
H	47 mm. smoked.....	0.588	350	....	2.31	..	..	22	....	18.0	..
D	47 mm. smoked.....	0.631	375	....	2.05	..	..	20	....	18.2	..
I	47 mm. smoked.....	0.622	368	....	2.34	..	..	20	....	16.8	..
J	47 mm. smoked.....	0.595	371	....	2.13	..	..	20	....	16.0	..
K	47 mm. smoked.....	0.621	345	....	2.77	..	..	21	....	17.2	..
Lk	47 mm. smoked.....	0.577	336	....	2.05	..	..	19	....	14.4	..
	62 mm. smoked.....	0.702	452	....	3.54	..	..	20	....	21.0	..
	Cigarette cut to 70 mm. 47 mm. smoked.....	0.577	338	....	2.91	..	..	22	....	15.6	..
Ik	47 mm. smoked.....	0.609	357	....	1.98	..	..	17	....	13.6	..
	62 mm. smoked.....	0.804	457	....	2.90	..	..	19	....	18.4	..
	Cigarette cut to 70 mm. 47 mm. smoked.....	0.609	350	....	2.39	..	..	21	....	15.4	..
Mf	47 mm. smoked.....	0.633	356	2.43	1.60	34	22	14	20.9	11.3	33
Afk	47 mm. smoked.....	0.654	372	2.44	1.97	23	19	14	14.1	11.3	20
	62 mm. smoked.....	0.903	481	....	2.85	..	..	17	....	15.4	..
Nfk	47 mm. smoked.....	0.614	400	2.70	2.33	14	21	16	14.9	12.1	19
	62 mm. smoked.....	0.839	521	....	3.29	..	..	19	....	17.1	..
	Cigarette cut to 70 mm. 47 mm. smoked.....	0.614	394	....	2.57	..	..	20	....	12.7	..
Bfk	47 mm. smoked.....	0.565	345	3.13	1.88	40	22	13	15.3	9.0	41
	62 mm. smoked.....	0.745	441	....	2.96	..	..	16	....	13.4	..
	Cigarette cut to 70 mm. 47 mm. smoked.....	0.565	348	....	2.12	..	..	15	....	10.3	..
Ofk	47 mm. smoked.....	0.611	371	2.55	1.76	31	18	12	14.7	10.1	31
	62 mm. smoked.....	0.807	494	....	2.61	..	..	14	....	14.0	..
	Cigarette cut to 70 mm. 47 mm. smoked.....	0.611	375	....	2.02	..	..	14	....	12.1	..

EXHIBIT 17

STATEMENT BY IRVING MICHELSON, HEAD OF THE SPECIAL PROJECTS DIVISION OF CONSUMERS UNION, BEFORE THE SUBCOMMITTEE ON LEGAL AND MONETARY AFFAIRS OF THE HOUSE COMMITTEE ON GOVERNMENT OPERATIONS, WEDNESDAY, JULY 24, 1957

ATTACHMENT A.—*Results of Consumers Union tests on Kent cigarettes over a 4-year period*

[Milligrams]

	1953	1955	1957 [1]	1957 [2]	1957, [2] total smoke method [3]	Current Kent ads
Tars:						
Regular	8.5	12.0	16.0	11.0	17.0	17.0
King	----------	15.0	----------	12.0	22.0	21.0
Long	----------	----------	----------	10.0	----------	----------
Nicotine:						
Regular	1.0	2.0	2.7	1.3	----------	1.4
King	----------	2.8	----------	1.7	----------	1.7
Long	----------	----------	----------	1.6	----------	----------

[1] From the March 1957 issue of Consumer Reports.
[2] Results of tests on Kents with new filter, July 1957, unpublished as yet.
[3] See explanation in the covering statement.

EXHIBIT 18

TECHNICAL DETAILS OF ANALYSIS FOR NICOTINE AND TARS IN CIGARETTE SMOKE

The smoking machine and absorption train used in these experiments have been described by Bradford, J. A.; Harlan, W. R., and Hanmer, H. R.: Nature of Cigaret Smoke: Technic of Experimental Smoking, J. Indust. Engin. Chem., 28: 836–839, 1936. The authors state that machine drawings and details of construction are available to interested persons.

The conditions of smoking have been described in J. A. M. A., 152: 917–920, 1953 (July 4).

DETERMINATION OF NICOTINE

The smoke was collected in a tared 300-milliliter Kjeldahl flask containing 5 milliliters of alcohol and 5 milliliters of alcoholic 0.5 *N* sulfuric acid. The smoke tube did not dip below the level of the liquid, i. e., the smoke was not bubbled through the solution. The bubblers contained glass beads. Five milliliters of alcohol 0.5 *N* sulfuric acid were placed in the first bubbler and 5 milliliters of aqueous 0.5 *N* sulfuric acid were placed in the second bubbler.

After the 5 cigarettes had been smoked [1] to the mark, the smoke was allowed to settle 20 minutes. Then the bubblers were blown out into the flask and the bubblers washed two times with boiling hot water. The cigarette holders and the smoke tube (glass tubing 7 millimeters, outside dimension) were washed into the flask with boiling hot water, alcohol, and hot water again; 2.5 *N* sodium hydroxide was added to neutralization of the sulfuric acid, and the contents of the flask made to 80 grams. Exactly 15 milliliters of 0.025 *N* sulfuric acid were apportioned between flasks D and E (made from 50-milliliter distilling flasks), about 12 milliliters in E (Bradford, J. A.; Harlow, E. S.; Harlan, W. R.; and Hanmer, H. R., J. Indust. Engin. Chem., 29, 46, 1937). Fifteen milliliters of trisodium phosphate solution (ibid., p. 47) were added to the contents of the Kjeldahl flask, and the distillation apparatus connected (used gum-rubber tubing and clean gray stoppers). Concentration of the contents of the Kjeldahl flask was begun when the bath under the flasks D and E was hot (this laboratory used a silicone bath and found that about 125° C. was sufficient to keep the con-

[1] This laboratory followed the suggestion of Bradford and coworkers, ibid., p. 838, and used dextrose sirup (Karo) concentrated in vacuo to seal the cigarettes in a bakelite holder. The concentrated sirup was put into collapsible ointment tubes for convenience in handling.

tents of flasks D and E constant). Seventy milliliters of distillate was collected in 23 minutes. Steam was admitted to the flask, but concentration of the flask's contents continued until 20 milliliters remained, and then this volume maintained for the remainder of the distillation. An additional 130 milliliters of distillate was collected at the rate of 2.5 milliliters per minute.

The contents of the flasks D and E were washed into a 250 milliliter beaker, cooled to room temperature and titrated with 0.025 N sodium hydroxide using methyl red as indicator. A blank was run on the reagents. The titration gives total volatile bases.

The contents of the beaker were made to 100 milliliter and 0.5 milliliter of hydrochloric acid added giving a solution of about 0.05 N hydrochloric acid. One milliliter of silicotungstic acid (12 grams of $4H_2O.SiO_2.12WO_3.22H_2O$ dissolved in water and diluted to 100 milliliter) was added for each 10 milligrams of nicotine supposed to be present. The contents of the beaker were stirred and allowed to stand overnight. The precipitate was filtered and washed with 0.005 N hydrochloric acid. The percipitate was ignited for 30 minutes at 650° C. A correction of 0.001 milligram per milliliter was applied to compensate for the solubility of nicotine silicotungstate in 0.05 N hydrochloric acid (see Spies, J. R., Determination of Small Quantities of Nicotine by a silicotungstic Acid Micromethod, J. Indust. Engin. Chem., Anal. Ed. 9, 46–47, 1937.).

DETERMINATION OF TARS

The smoke was collected in a 300 milliliter Kjeldahl flask containing 25 milliliters of 0.1 N sulfuric acid. The smoke tube did not dip below the level of the liquid, i. e., the smoke was not bubbled through the solution. The bubblers were loosely packed with asbestos held in place, top and bottom, with cotton plugs.

After the five cigarettes had been smoked to the mark, the smoke was allowed to settle twenty minutes. Then the bubblers were washed into the flask with chloroform. The cigarette holders and the smoke tube were also washed into the flask with chloroform. The contents of the flask were transferred to a separatory funnel and the aqueous phase extracted with 25 milliliter portions of chloroform. This laboratory has found that 6–25 milliliter portions of chloroform are sufficient to extract all the tars. These 25 milliliter portions of chloroform can be passed through the asbestos in the bubblers to insure complete removal of tars from them. The pooled chloroform washings were extracted with a 10 milliliter portion of water. The chloroform was transferred to a tared 50 milliliter beaker and the chloroform evaporated on a steam bath in a current of air. The beakers were dried 3 hours at 100° C., cooled, and weighed.

EXHIBIT 19

SELECTED LIST OF REFERENCES

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with the av. amt. present in the tobacco consumed. The amts. of I retained from the smoke were 22.04, 30.36, 45.25 and 16.35% respectively. The efficiency of the stubs for removing $CHCl_3$-sol. tars was in the same order. The data are discussed in regard to government regulation stipulating that denicotization is defined as reducing original I content at least 50%.

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EXHIBIT 20

UNITED STATES DEPARTMENT OF AGRICULTURE

AGRICULTURAL MARKETING SERVICE

TOBACCO DIVISION

TOBACCO INSPECTION, MARKET NEWS, AND DEMONSTRATION SERVICES

(Type 31—Burley)

Inspection of tobacco according to standard grades as an aid to growers in marketing their crop was inaugurated in 1929 by the United States Department of Agriculture. In August 1935, the Tobacco Inspection Act was made law. This act offered growers free and mandatory inspection on various tobacco markets where it was desired. That is, the growers could request a referendum to be held on a market or group of markets, and if two-thirds of the votes cast favored inspection, those markets would then be designated by the Secretary of Agriculture for free and mandatory inspection.

In 1936 several markets in the Burley Belt took advantage of this service, and other growers, recognizing the aid it was to marketing their tobacco, requested it on their markets. By 1940 the service had spread to take in almost half of the markets in the burley area. In October 1941, a general referendum was held covering the remainder of the burley markets. Since the marketing season of 1941–42 all burley markets in Kentucky, Tennessee, Virginia, North Carolina, West Virginia, Ohio, Indiana, and Missouri have had free inspection service.

Reasons for the inspection and market news service are readily understood by every farmer who has sold a split lot of tobacco for two widely different prices, or has rejected a bid and resold the same tobacco, on the same day, on the same warehouse floor, possibly for twice or three times the first price offered. Every tobacco grower knows there is much uncertainty about the price any basket of tobacco will bring, and there is a wide range in the price paid for lots of tobacco of the same quality.

Inspection and market news service, based on Government standard grades, has been developed to provide a measuring stick and price guide to the grower. Prior to the sale, a trained inspector examines each basket of tobacco and certifies it as to grade. The market price reports, furnished daily by the market news service, cooperating in some instances with State agencies, give the grower the current

average price paid for each specific grade. With this information he can determine whether his tobacco is selling in line with current prices, and is enabled, when necessary, to make intelligent rejections.

Since January 1940, the Government through its lending agencies, has made nonrecourse loans available to growers. These are administered through cooperative associations or other responsible organizations, that place a support price on each grade of growers tobacco that is in sound and merchantable condition.

The inspection service inspects and certifies the grade of tobacco, before sale, at all auction markets. Packed and processed tobacco is also inspected and grade certified upon application by interested parties.

The Tobacco Inspection Act provides for three distinct services: inspection, market news, and demonstration.

Inspection service.—The inspection is done by highly trained specialists in tobacco judging, who have taken training courses and passed tests to qualify them for this type of work. They precede the auction sale and certify each basket as to grade, according to Federal standards. This grade is, in reality, a description of each basket of tobacco as to group, quality, and color. These grades can be readily understood by the grower, if given a little application.

Grades for tobacco are determined by the simple process of division and subdivision, until a point is reached at which further subdivision is neither essential nor desirable. Each final subdivision is called a grade.

Class: The first division is made on the basis of distinct characteristics of tobacco caused by varieties of seeds, soils, climate, and methods of cultivation, harvesting, and curing; this division is called class. (See Brief classification of leaf tobacco, p. 3).

Type: Each class is subdivided into types. A type is a division of a class of tobacco having certain common characteristics which permit its being divided into a number of closely related grades. Tobacco which has the same characteristics and corresponding qualities, colors, and lengths is treated as one type.

Grade: The next subdivision breaks down each type into grade. A grade of burley tobacco is made of three factors: Group, quality, and color.

(1) *Group.*—The first factor of a grade, is a division of a type, covering severly closely related grades, based on the general quality of the tobacco, including body, the percentage of injury, shape of leaf, and other characteristics. Different groups are Flyings, Lugs, Leaf, Mixed, Nondescript, and Scrap with a subgroup of short leaves called Tips.

Flyings (X) normally consist of the leaves at the lower part of the plant, show a material amount of injury characteristic of leaves grown near the ground, tissuey to very thin in body, generally ripe to thoroughly ripe, low tensile strength and solidity.

Lugs (C) usually longest and widest leaves on the plant, thin to medium body, have a very light color shade, and have sufficient strength and solidity to be stemable into whole strips, and have very little injury.

Leaf (B) compared with general characteristics of the type, is relatively medium to heavy body, has higher percentage of oil, is narrower in width with larger stems (midribs) compared with lug group, and varies in maturity from ripe to fairly ripe, according to quality.

Tips (T) a subgroup of leaf of which 20 percent or more of its leaves are under 16 inches in length. Normally consist of relatively narrow sharp pointed leaves under 16 inches long which grow on top or upper part of the plant, but may consist of any short leaves having the characteristics of tips.

BRIEF CLASSIFICATION OF LEAF TOBACCO

(Covering classes, types, and groups)

Class 1. Flue-cured types

Type 11: Old Belt flue-cured; produced in the Piedmont sections of Virginia and North Carolina.

Type 12: Eastern North Carolina flue-cured; produced in the coastal sections of North Carolina, north of the South River.

Type 13: South Carolina flue-cured; produced in the coastal sections of South Carolina and the southeastern counties of North Carolina, south of the South River.

Type 14: Georgia and Florida flue-cured; produced in southern Georgia and northern Florida.

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Class 2. Fire-cured types

Type 21: Virginia fire-cured; produced in the Piedmont and mountain sections of Virginia.

Type 22: Tennessee-Kentucky fire-cured, eastern district, Clarksville, Springfield, and Hopkinsville; produced in a section east of the Tennessee River, in southern Kentucky and northern Tennessee.

Type 23: Kentucky-Tennessee fire-cured, western district, Mayfield and Murray; produced in a section between the Tennessee, Ohio, and Mississippi Rivers in western Kentucky and northwestern Tennessee.

Class 3. Air-cured types

(Light Air-Cured (Class 3a)

Type 31: Burley; produced in Kentucky, Tennessee, Ohio, Indiana, West Virginia, Virginia, North Carolina, and Missouri.

Type 31–V: Low nicotine burley; produced in Kentucky.

Type 32: Southern Maryland; produced in five counties of southern Maryland—Prince Georges, Anne Arundel, Calvert, Charles, and St. Marys.

Dark Air-Cured (Class 3b)

Type 35: One Sucker; produced in north-central Tennessee and south-central Kentucky.

Type 36: Green River; produced in the northern part of Kentucky in the territory adjacent to Owensboro and Henderson.

Type 37: Virginia sun-cured; produced in central Virginia, north of the James River.

Class 4. Cigar-filler types

Type 41: Pennsylvania Seedleaf; produced in Lancaster County, Pa., and the adjoining counties.

Types 42–44: Ohio filler:

Type 42: Gebhardt; produced in the Miami Valley section of Ohio and extending into Indiana.

Type 43: Zimmer; produced in the Miami Valley section of Ohio and extending into Indiana.

Type 44: Dutch; produced in the Miami Valley section of Ohio.

Type 46: Puerto Rican sun-grown including primed (Deshojado) and stalk-cut (Mata); produced on the island of Puerto Rico.

Class 5. Cigar-binder types

Type 51: Connecticut Broadleaf; produced in the Connecticut Valley sections of Connecticut and Massachusetts.

Type 52: Connecticut Havana seed; produced in the Connecticut and Housatonic Valley sections of Connecticut and Massachusetts.

Type 53: New York and Pennsylvania Havana seed; produced in the Big Flats and Onondaga sections of New York, and extending into Pennsylvania.

Type 54: Southern Wisconsin; produced in Wisconsin, south and east of the Wisconsin River.

Type 55: Northern Wisconsin; produced in Wisconsin, north and west of the Wisconsin River, and in eastern Minnesota.

Class 6, Cigar wrapper types

Type 61: Connecticut Shade; produced in the Connecticut Valley sections of Connecticut and Massachusetts.

Type 62: Georgia and Florida Shade; produced in southwestern Georgia and north-central Florida.

Miscellaneous types of domestic tobacco

Type 72: Louisiana Perique.

Type 73: All domestic types of tobacco not otherwise classified.

Type 77: Domestic Aromatic.

Foreign types

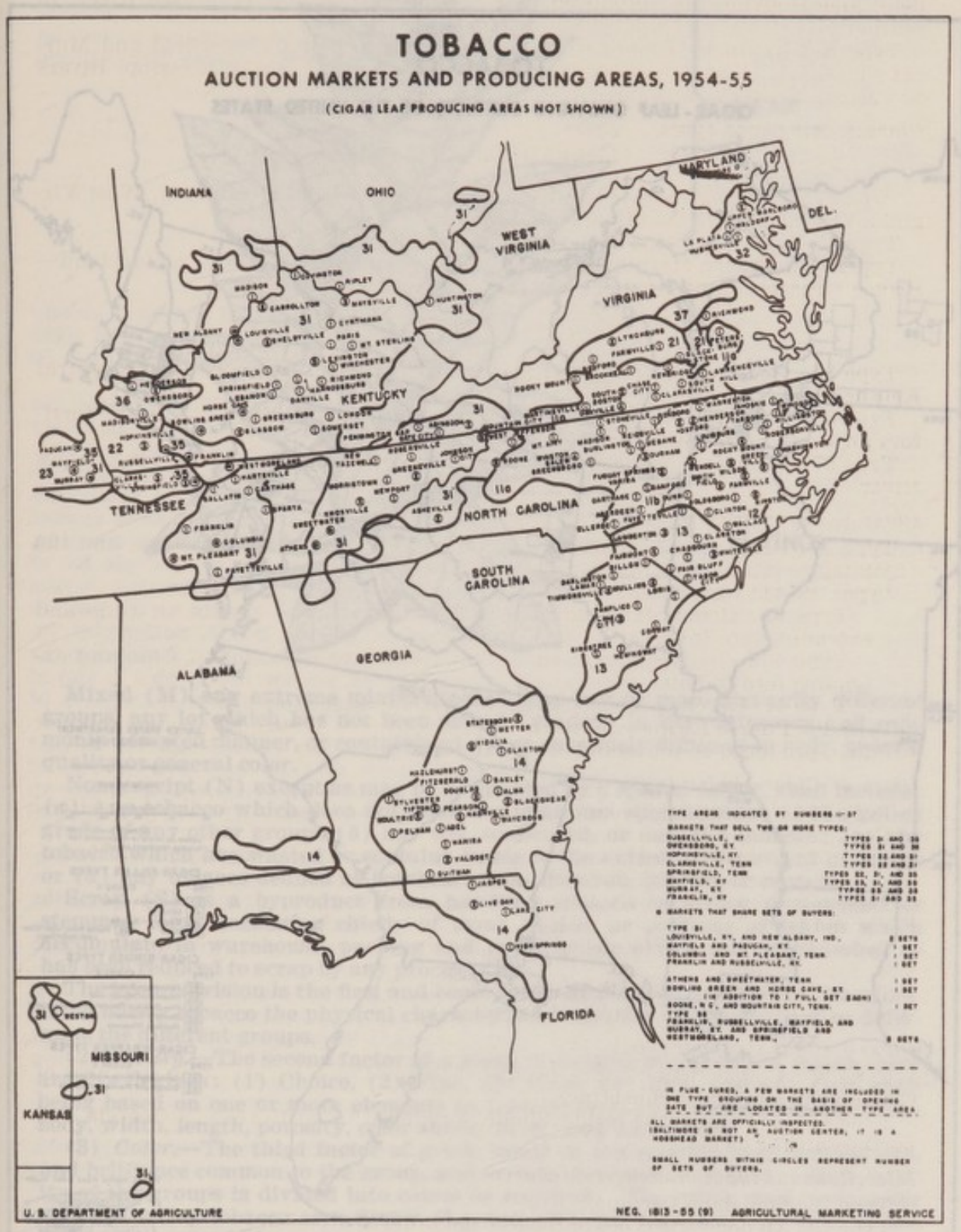
Type 81: Cuba (Havana).

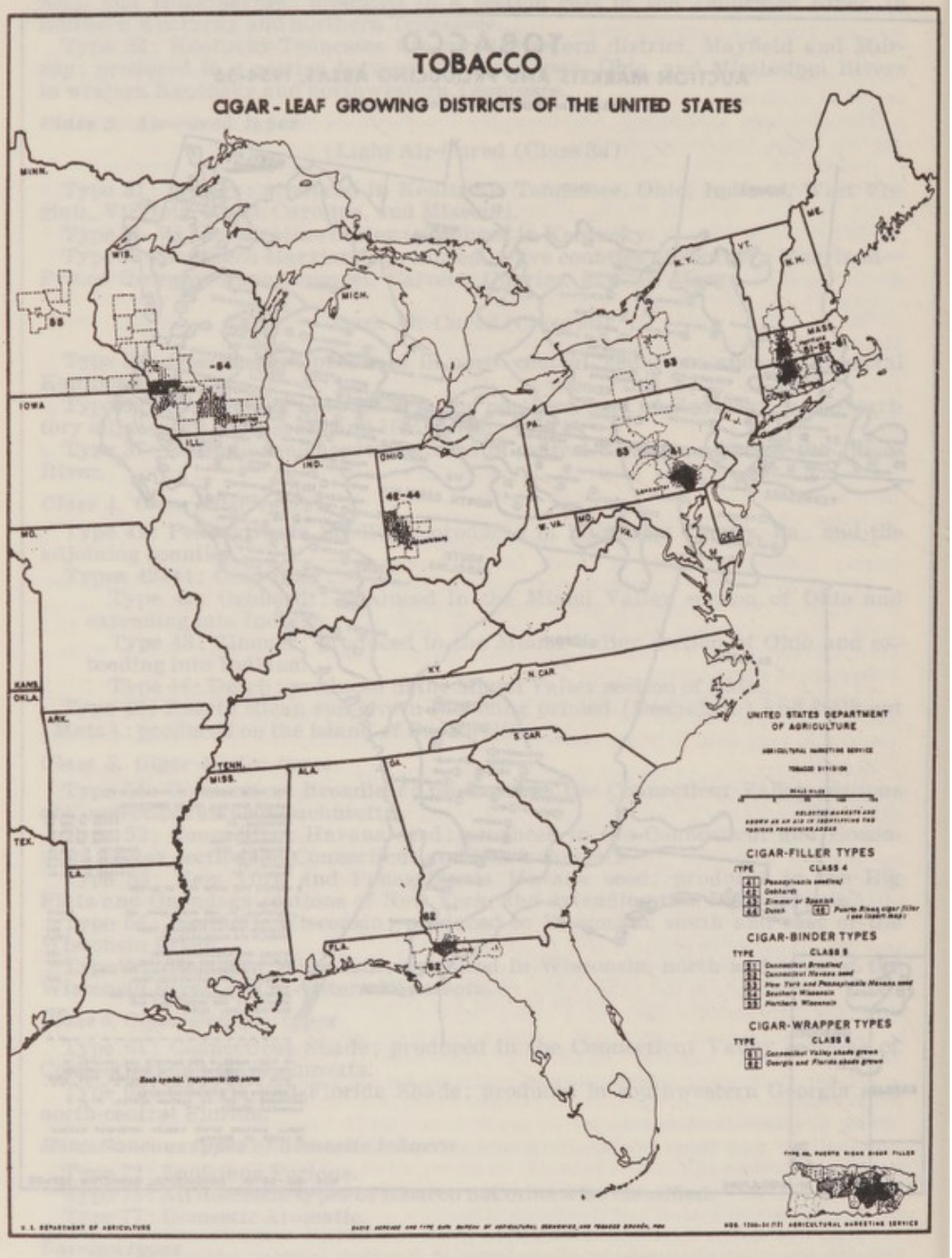
Type 82: Sumatra and Java.

Type 83: Philippine Islands (Manila).

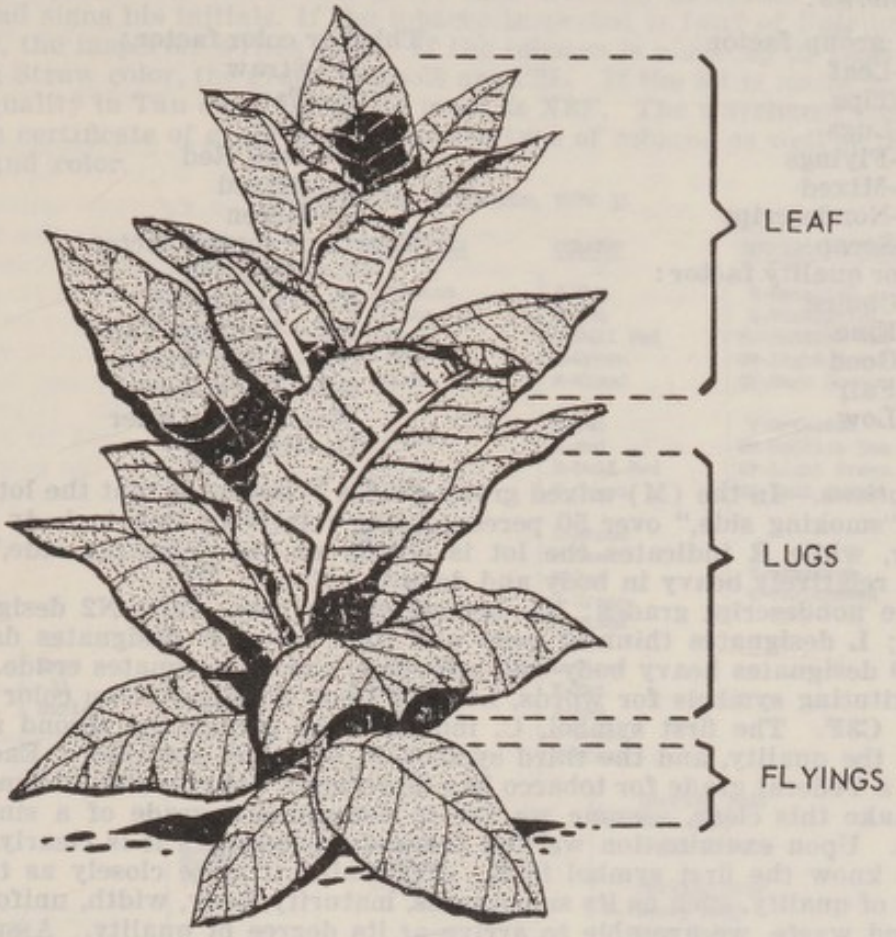
Type 84: All foreign-grown cigar-leaf not otherwise classified.

Type 90: Foreign-grown cigarette and smoking tobacco (Turkish and other).


TOBACCO
AUCTION MARKETS AND PRODUCING AREAS, 1954-55
(CIGAR LEAF PRODUCING AREAS NOT SHOWN)
INDIANA
OHIO
WEST VIRGINIA
MARYLAND
DEL.
VIRGINIA
KENTUCKY
TENNESSEE
NORTH CAROLINA
SOUTH CAROLINA
GEORGIA
ALABAMA
FLORIDA
MISSOURI
KANSAS
U. S. DEPARTMENT OF AGRICULTURE
NEG. 1813-55 (9) AGRICULTURAL MARKETING SERVICE


TOBACCO
CIGAR-LEAF GROWING DISTRICTS OF THE UNITED STATES
MINN.
WIS.
MICH.
N.Y.
VT.
N.H.
ME.
MASS.
IOWA
ILL.
IND.
OHIO
PA.
N.J.
W.VA.
MD.
DEL.
KY.
N.CAR.
S.CAR.
GA.
ALA.
MISS.
TENN.
ARK.
OKLA.
KANS.
MO.
TEX.
LA.
FLA.
UNITED STATES DEPARTMENT OF AGRICULTURE
CIGAR-FILLER TYPES
TYPE
CLASS 4
CIGAR-BINDER TYPES
TYPE
CLASS 5
CIGAR-WRAPPER TYPES
TYPE
CLASS 6

BURLEY TOBACCO PLANT



Mixed (M) any extreme mixture, containing two or more distinctly different groups, any lot which has not been sorted (graded) in the customary and commonly accepted manner, or containing tobacco extremely different in body, general quality or general color.

Nondescript (N) except as may be designated by a special factor, shall include: (*a*) Any tobacco which does not meet the minimum specifications of the lowest grade of any other group; (*b*) any wet, semicured, or unsound tobacco; (*c*) any tobacco which has wasted or contains waste to the extent of 40 percent or more; or (*d*) any tobacco defined as botched, crude, decayed, offtype, or nested.

Scrap (S) is a byproduct from handling tobacco in either unstemmed or stemmed form, consisting chiefly of loose, untied, or portions of leaves which accumulate in warehouse, packing and conditioning plants and stemmeries, or has been reduced to scrap by any process.

The group division is the first and basic factor of grades for tobacco. In examining burley tobacco the physical characteristics detailed above are used to determine the different groups.

(2) *Quality.*—The second factor of a grade, a division of a group, of which there are five degrees: (1) Choice, (2) Fine, (3) Good, (4) Fair, and (5) Low, each being based on one or more elements in tobacco such as: smoothness, maturity, body, width, length, porosity, color shade, finish, and uniformity.

(3) *Color.*—The third factor of grade based on the relative hues, saturations, and brilliance common to the group, and certain elements of quality. Each quality of the groups is divided into colors as required. The terms used to describe color in burley tobacco are: Straw (L), tan (F), red (R), dull red (D), mixed color (M), green (G).

In addition to aforementioned three main factors of a grade, when a lot of tobacco has a peculiar side or characteristic, varying from or not covered by the specifications, the department uses a special factor to help describe these tobaccos which could be a greenish tinge (V), variegated (K), reddish tan (FR), light green (GF), dark green (GR), unsafe order (W), unsound (U).

Any combination of group, quality and color, or special factor when needed, can be made to form a grade. To simplify the use of groups, qualities, colors and

special factors, letters and numerals are used to represent the words they stand for as follows:

First or group factor:
- B—Leaf
- T—Tips
- C—Lugs
- X—Flyings
- M—Mixed
- N—Nondescript
- S—Scrap

Second or quality factor:
- 1—Choice
- 2—Fine
- 3—Good
- 4—Fair
- 5—Low

Third or color factor:
- L—Straw
- F—Tan
- R—Red
- D—Dull Red
- M—Mixed
- G—Green

Fourth or special factor:
- V—Greenish
- K—Variegated
- FR—Reddish Tan
- GF—Light Green
- GR—Dark Green
- W—Unsafe Order
- U—Unsound

Exceptions.—In the (M) mixed group grades F indicates that the lot is chiefly on the "smoking side," over 50 percent being relatively thin in body and light in color, while R indicates the lot is chiefly on the "chewing side," over 50 percent relatively heavy in body and dark in color.

In the nondescript grades: N1 designates the best, while N2 designates the poorest; L designates thinnest body and light color, F designates dark dingy color, D designates heavy body dull red color, and G designates crude.

Substituting symbols for words, Lugs of Good quality in Tan color would be written C3F. The first symbol, C, indicates the group, the second symbol, 3, denotes the quality, and the third symbol, F, describes the color. Each symbol used in a Federal grade for tobacco has therefore a definite and known meaning.

To make this clear, assume we are to determine a grade of a single lot of tobacco. Upon examination we find from characteristics it is clearly a Flying and we know the first symbol is X. Examining it more closely as to relative degrees of quality, such as its smoothness, maturity, body, width, uniformity, injury, and waste, we are able to arrive at its degree of quality. Assuming the flyings are a major portion of straw-colored leaves, we use the symbol L and of good or third quality, making it X3L. Now we have a complete description of the tobacco.

The Federal system of grades for tobacco differs from private systems in that they must be more comprehensive, since it must describe any and all lots of tobacco offered for sale, each symbol has a definite meaning which is known to the general public, and symbols are a complete description of lot applied thereto.

The details of the groups, qualities, colors, and special factors used in connection with the grades for burley tobacco are shown on page 691.

Farmers are sometimes confused by the fact that the several groups, qualities, colors, and special factors can be combined to form a large number of grades. They say that too many grades are recognized because the total number cannot be applied to their particular crops. It should be remembered that grades are used only as required, and only a limited number are necessary to describe all tobacco in an individual crop. However, crops from different farms and from different areas show such a wide variation that a wide range in grades is necessary to describe all burley tobacco offered for sale.

OPERATION OF INSPECTION SERVICE

On markets where the inspection service operates, the procedure is as follows:

(1) Growers deliver their tobacco to the market of their choice and to any warehouse they may select.

(2) The tobacco is arranged for sale on flat baskets.

(3) Each lot, or basket, is then weighed and a warehouse ticket is placed on the lot. The ticket shows the name of the seller and the number of pounds of tobacco in the lot, and may give other information for the purpose of identification. Space is provided on the ticket for the name of the buyer, the grade symbol of the buyer, and the price at which the tobacco is sold. It also has a space in the upper right corner for the Federal grade (see p. 692).

(4) The lots, or baskets, are placed in line on the warehouse floor.

(5) As soon as there is good light, the official inspectors start at the beginning of the "break" ahead of the sale, and make a proper examination of each basket of tobacco.

(6) Having made a careful examination, the inspector writes on the ticket, in the space provided, the Federal grade that correctly describes the tobacco in the lot, and signs his initials. If the tobacco inspected is Leaf of Fair quality in Red color, the inspector writes B4R. If the tobacco is made up of Lugs of Fine quality in Straw color, the grade symbols are C2L. If the lot is made up Flyings of Good quality in Tan color the grade mark is X3F. The warehouse ticket then becomes a certificate of grade and shows the type of tobacco as well as its group, quality, and color.

CLASSIFICATION OF TOBACCO, TYPE 31

		GROUPS	QUALITIES	COLORS	SPECIAL FACTORS*
Class 3 Air-cured	Type 31 Burley	Leaf (B)	1-Choice 2-Fine 3-Good 4-Fair 5-Low	F-Tan R-Red D-Dull Red G-Green M-Mixed	V-Greenish K-Variegated FR-Reddish Tan GF-Light Green GR-Dark Green
		Tips (T)	3-Good 4-Fair 5-Low	F-Tan R-Red D-Dull Red G-Green	V-Greenish FR-Reddish Tan GF-Light Green GR-Dark Green
		Lugs (C)	1-Choice 2-Fine 3-Good 4-Fair 5-Low	L-Straw F-Tan R-Red G-Green M-Mixed	V-Greenish K-Variegated
		Flyings (X)	1-Choice 2-Fine 3-Good 4-Fair 5-Low	L-Straw F-Tan R-Red G-Green M-Mixed	
		Mixed (M)	3-Good 4-Fair 5-Low	Smoking Side F- Thin Body Light Color Chewing Side R- Heavy Body Dark Color	
		Nondescript (N)	1-Best 2-Poorest	L-Thin body light color F-Thin body dingy color R-Heavy body dark color G-Crude	Botched Nested Off-type Decayed
		Scrap	As defined		

*Special factor U and W can be used as the fourth factor of any grade

Market news service.—The average farmer cannot spend enough time on the warehouse floor to keep posted on the approximate value of the different grades of tobacco, but this knowledge is essential to the equitable sale of his tobacco. In order to provide for this need, the tobacco market news service was developed, and operates as a companion activity to the inspection service.

Studies made by the Department of Agriculture show that the greater part of the tobacco sold at auction is sold at prices within the normal range of each grade and therefore are in line with equitable prices as established by sales. The studies also show that some lots sell considerably higher than the normal price range for the grade, and about the same number of lots sell in the price range that is considerably lower than the normal range. In both cases these prices are entirely out of line with equitable sales. In the one instance the seller receives too much for his tobacco and in the other he receives far too little. It is therefore evident that whereas one grower, for some unexplained reason, is paid a premium for his tobacco, another grower is likely to be penalized. It is this situation which causes so much dissatisfaction and which could be eliminated by proper and consistent use of the inspection and market news services.

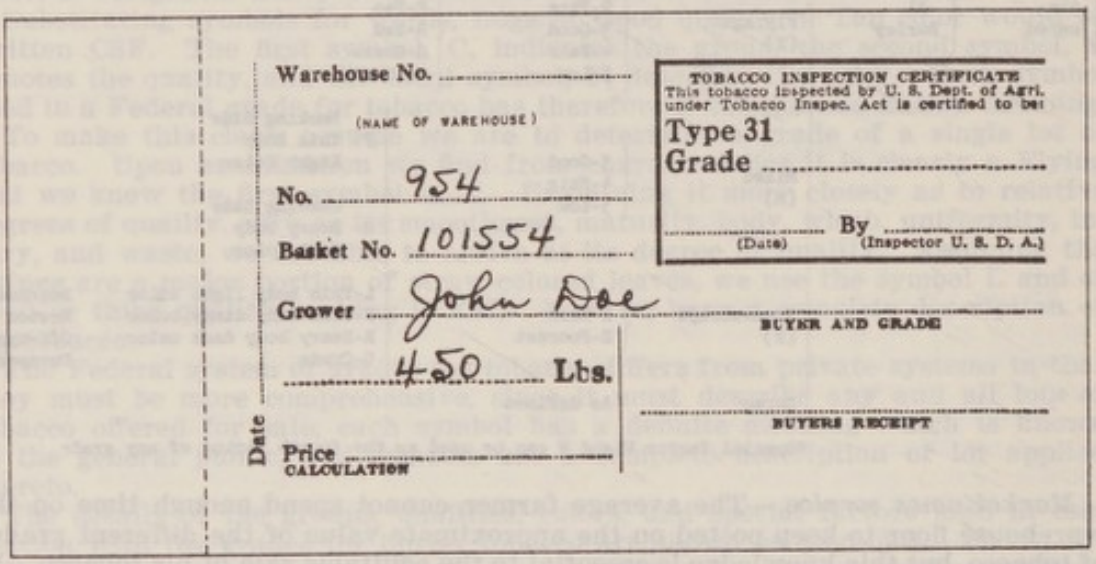
The value of the inspection and market news services lies in the fact that the certificate of grade on the warehouse ticket provides tobacco growers with unbiased information regarding the grade or quality of each lot of tobacco offered for sale, and the price reports furnish a definite basis for making an intelligent decision on whether or not to accept a bid.

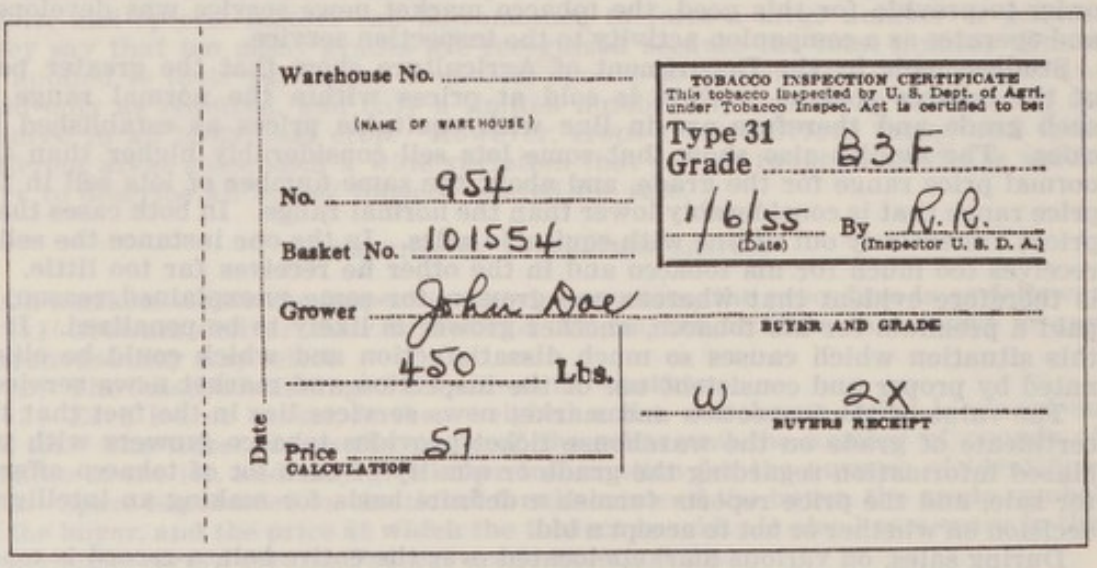
During sales, on various markets located over the entire belt, a record is made of grade, pounds, and price for each lot sold. These data are transmitted to a central market news office, where averages are calculated for representative

grades and issued in the form of a daily press release. This report carries quotations on grades which represent around three-fourths of the total volume of offerings. The release reviews the highlights of the day's sale covering such items as quality, price, trend, high and low price for individual baskets, etc. Sales figures for the eight-State belt are also included. Newspapers and radio stations are furnished with the release daily.

Weekly price reports are sent to every market showing the average price at which each grade sold during the preceding week along with the advance price for the grade. This information is made available to growers selling on the market. In addition a weekly news report is issued which reviews the marketing activities for the week. This report is sent by mail upon request to the Market News Office and may be obtained throughout the season without cost.

Market price reports furnished growers give current average prices by grade and also support price of each grade. If a certain grade is averaging well above support price it would be well for grower to think before accepting loan price: For example, B5FR average was $40 while support price was $33. If grower was not offered bid above support price, it would be worth his time to reject this lot and offer it at next sale. He is assured of loan price being the same all season and in most cases his lot would bring near average sale price on second sale thus giving him a nice profit over first sale.





TOBACCO WAREHOUSE TICKET - BEFORE AND AFTER INSPECTION AND SALE

SAMPLE PRICE REPORT

United States Department of Agriculture, Tobacco Division, Agricultural Marketing Service, Kentucky, North Carolina, Tennessee, Virginia, and West Virginia Departments of Agriculture, cooperating

No. 8
(1954 Crop)

Weekly Tobacco Market Price Report, Type 31, Burley

Gross sales in the 8-State area for the period January 4 through 6 totaled 81,749,841 pounds and averaged $49.56 per hundred. Season gross sales through the same date were 541,342,903 pounds averaging $50.71.

Auction averages and advance prices per hundred pounds by United States grades:

Week ending Thursday, Jan. 6, 1955

United States grade	Auction average	Advance price	United States grade	Auction average	Advance price
Leaf:			Lugs—Continued		
B1F	$61.00	$61	C2L	[1] $67.00	$67
B1FR		50	C2F	67.00	66
B1R		38	C3L	67.00	66
B2F	60.00	59	C3F	66.00	65
B2FR	[1] 50.00	48	C3FV		60
B2R	[1] 45.00	36	C3FK	[1] 59.00	54
B3F	57.00	55	C3R	58.00	57
B3FV	50.00	49	C3RV		50
B3FK		44	C4L	66.00	65
B3FR	45.00	43	C4F	64.00	63
B3R	37.00	32	C4FV	[1] 60.00	58
B3RV		25	C4FK	[1] 55.00	52
B3M		41	C4R	56.00	54
B3GF		31	C4RV		47
B3GR		21	C4M	[1] 58.00	50
B4F	52.00	50	C4G		34
B4FV	45.00	44	C5L	63.00	60
B4FK		39	C5F	59.00	58
B4FR	43.00	39	C5R	50.00	46
B4R	34.00	28	C5M	53.00	44
B4RV	28.50	21	C5G	[1] 38.00	27
B4D	[1] 29.00	19	Flyings:		
B4M	[1] 48.00	37	X1L	[1] 68.00	68
B4GF	33.00	28	X1F		67
B4GR	23.50	18	X2L	[1] 68.00	67
B5F	45.00	42	X2F	[1] 67.00	66
B5FR	40.00	33	X3L	67.00	66
B5R	32.00	25	X3F	66.00	65
B5D	23.50	16	X3R	56.00	55
B5M	[1] 41.00	27	X4L	64.00	62
B5GF	28.00	23	X4F	62.00	61
B5GR	22.00	14	X4R	52.00	50
Tips:			X4M	[1] 55.00	47
T3F	45.00	43	X4G		37
T3FV		36	X5L	59.00	53
T3FR	40.00	36	X5F	55.00	52
T3R	$35.00	$25	X5R	44.00	40
T3RV	[1] 26.50	22	X5M	[1] 48.00	37
T4F	42.00	38	X5G		27
T4FV	35.00	30	Mixed:		
T4FR	37.00	31	M3F	50.00	47
T4R	30.00	22	M3R		35
T4RV	25.50	19	M4F	49.00	42
T4D	21.50	16	M4R	[1] 39.00	31
T4GF	26.00	20	M5F	42.00	37
T4GR	19.00	14	M5R	33.00	26
T5F	37.00	31	Nondescript:		
T5FR	32.00	25	N1L	46.00	38
T5R	23.50	18	N1F	38.00	26
T5D	18.00	13	N1D	19.50	12
T5GF	19.50	16	N1G	14.50	10
T5GR	16.50	12	N2L	29.50	[2]
Lugs:			N2D	13.75	[2]
C1L	[1] 68.00	68	N2G	13.50	[2]
C1F	[1] 67.00	67			

[1] Latest average established. Averages based on auction prices inclusive of association's advance Tobacco graded "W" (Unsafe Order), "U" (Unsound), Nested, Botched, Off-type, or Decayed will not be accepted by the associations.

[2] No advance.

"NOTICE TO GROWERS"

The information furnished in this report is intended to help you in obtaining the market price for your tobacco.

USE PRICE REPORTS REGULARLY

Note both auction average and advance price shown. You have 1 of the following 3 choices:

1. Accept auction price offered.
2. Reject auction price and reoffer.
3. Deliver to the association at advance.

Be present when your tobacco is sold. Consult your warehouseman about delivery of any tobacco to the association.

CAREFUL PREPARATION OF TOBACCO FOR MARKET PAYS A PROFIT

1. Keep green and ripe tobacco separated.
2. Be sure and remove all rubber bands before tying leaves into bundles. Failure to do so may result in a lower price to the grower.
3. Tie heads of bundles uniformly, about size of silver dollar.
4. Handle and market your tobacco in proper keeping order. Wet tobacco is always sold at a loss to the grower.

Consult your tobacco grader. Ask him any questions or advice about marketing your tobacco. He is on the market to assist you.

KEY TO STANDARD GRADE MARKS FOR BURLEY TOBACCO

Group:
- B—Leaf
- T—Tips
- C—Lugs or Cutters
- X—Flyings
- M—Mixed Group
- N—Nondescript

Quality:
- 1—Choice
- 2—Fine
- 3—Good
- 4—Fair
- 5—Low

Color:
- L—Buff
- F—Tan
- R—Red
- D—Dull Red
- M—Mixed Color
- G—Green

Special Factor:
- V—Greenish
- K—Variegated
- FR—Reddish Tan
- GF—Light Green
- GR—Dark Green
- W—Unsafe Order
- U—Unsound, damaged under 20 percent

For example: B3F designates leaf, good quality, tan color.

Information like this will enable every farmer to know after his tobacco is sold whether the prices, lot by lot, are in line with those already established on a grade basis by the buyers. This information will enable farmers to market their tobacco on a basis of fair competition.

Records of offerings and sales, compiled by the Department of Agriculture, show that when bids are materially below the price range per grade farmers make money by rejecting the bids and putting their tobacco up for sale again. The data also show that when bids are within the price range per grade or above the average for the grade, farmers seldom profit by rejection but usually lose money. The point is that if farmers are to benefit from the operations of the inspection and market news services they must apply the information obtained. No one can refute the statement that information which enables farmers to reject low bids and to obtain for their tobacco substantially higher prices at the second offering is of great value to producers in marketing their tobacco.

Demonstration service.—The demonstration service is educational. It acquaints farmers with the objectives of inspection and market news and how these services can benefit them, and instructs them in the better preparation of their tobacco for market so that it can be sold at the highest price consistent with quality.

Through cooperation with county agents and agricultural teachers, tobacco inspectors hold meetings directly at tobacco barns, where actual stripping demonstrations are given. These demonstrations show the farmer the best way to prepare his tobacco for market to enable him to obtain the best possible dollar value

for his crop. Inspectors are also made available to classes of agricultural students to explain the advantage of knowing how to use the inspection and market news services.

Agricultural colleges, county agents, vocational teachers of agriculture, chambers of commerce, and other civic and farmer organizations are given opportunities by the Department to further the service.

WHY THE GOVERNMENT ACTS

It may be asked, "Why cannot warehouse starters and buyers determine the grade of tobacco as well as Government inspectors?" Studies show that in the majority of instances they can, and do, since the bulk of sales are made at prices within the normal range for the several grades. But there are the sales at abnormally low prices to be explained.

One explanation may be found in the rate at which tobacco is sold at auction. The normal rate at which burley tobacco is auctioned is 360 lots an hour, or 1 lot each 10 seconds. Under normal conditions the warehouse starters and buyers must determine the group, quality, and color of a lot of tobacco every 10 seconds. It is extremely doubtful whether these determinations can be accurately and consistently made at this rate of speed during the whole period of sales. To a large extent the inequality in prices for the same grade of tobacco may be explained by errors in judgment on the part of starters and buyers, because of the speed at which tobacco is sold.

Another important factor is the light under which tobacco is sold. Some tobacco may be placed on the warehouse floor where the light is unsatisfactory. It may be in a dark corner, or under a skylight through which the sun shines directly on the tobacco. Both conditions render accurate determinations very difficult, and often adversely affect the sales price of the tobacco. Neither of these unfavorable conditions—rate of sales or unfavorable light—can be directly attributed to either the starters or the buyers, but the ill effects of both can be reduced materially by inspection service.

Under Federal inspection, to eliminate errors in judgment caused by speed and unfavorable light, sufficient inspectors are provided for each sale. Furthermore, as they begin inspecting tobacco sometime before the sale starts, they are not rushed and are able to make thorough examinations, and consequently more accurate and consistent determinations of grade. In addition, lots of tobacco that are under unsatisfactory light can be carried to proper light for better inspection and determination of grade.

Farmers should realize that both services are informational. The inspection service does not promote sales; it supplies information regarding the quality of tobacco offered for sale. The market news service does not establish prices; it only records current average prices established by sales of tobacco. If the service is rightly used it will go far toward preventing the losses now suffered by growers when tobacco is knocked down at less than its current market value. If the information is not applied it will have no influence on prices.

The object of inspection, market news, and demonstration is to supply information regarding the quality of tobacco offered for sale. These services (1) to certify to the seller the grade of his tobacco, (2) furnish him with current average price, and support or loan price for each grade, (3) keep him informed as to best way to prepare his crop for marketing and (4) to aid him in all ways to get the utmost dollar value for his year's work.

In addition, the Department issues a market review each year which covers such information as opening and closing dates of all markets, numbers of warehouses and buyers on each market, warehouse charges, summary of sales by crop years, summary of sales by States and months, producers' sales and resales by markets, price charts showing fluctuations over a period of years, average price per grade each week of marketing season, percentages of each group, quality, and color in each crop, and other valuable data.

All or any of these reports will be sent upon request to Market News Service, Tobacco Division, AMS, 620 South Broadway, Lexington, Ky.

EXHIBIT 21

Reprinted from
The British Journal of Cancer,
1956, Vol. X, p. 485.

3,4-BENZPYRENE IN THE SMOKE OF CIGARETTE PAPER, TOBACCO, AND CIGARETTES*

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RECENT statistical studies suggest a relationship between the increasing incidence of lung cancer and smoking. This implies carcinogenic activity by the smoke (Doll and Hill, 1950 ; Hammond and Horn, 1954 ; Wynder and Graham, 1950). Accordingly, for several years this laboratory has been investigating the nonvolatile fractions of the smokes of cigarettes, cigarette paper, and tobacco for possible known carcinogens. The statistical studies (Hammond and Horn, 1954) on the relation of lung cancer and smoking pointed especially at cigarette smoking and indicated little or no relation to cigar and pipe smoking. One major difference between these types of smoking is, of course, the cigarette paper. Initially, it was thought the carcinogenic activity might be wholly due to the paper, and we were thus stimulated to start with the paper alone.

Indications of fluorescence, characteristic of the benzanthracene derivatives, was first noted by Carroll and Rand (unpublished observations) in the tars from cigarette paper smoke. The fluorescence was found by us to be due to 3,4-benzpyrene.

The technique for burning the paper was not designed to simulate the conditions of actual cigarette smoking. The paper was smouldered in a stream of air (Fig. 2), the highest temperature reached at the burning front (650–950° C.) being in the same range as that attained in a cigarette during inhalation (Wynder, Graham and Croninger, 1953). It was felt that the combusion products would vary with temperature of burning but that qualitatively the same products would be formed. Work subsequently published by Cooper and Lindsey (Cooper and Lindsey, 1954 ; Cooper, Lindsey and Waller, 1954) on cigarettes made wholly of cigarette paper without tobacco and smoked in an apparatus designed to simulate actual smoking conditions supports this assumption ; 3,4-benzpyrene was obtained by these workers although in smaller yields than obtained by us. Additional substantiation was provided by the work on the whole cigarette ; the quantity of 3,4-benzpyrene produced was in line with that expected from the results on the paper and tobacco burned separately.

Subsequently we found 3,4 benzpyrene in the smokes of tobacco, cigarettes, and cigars. The cigarettes and cigars were smoked in a smoke sampling apparatus designed to approximate average conditions of actual smoking.

Much of the work reported here is a duplication with some extension of similar work by Lindsey and Cooper and is further confirmation of their results.

* Material from this paper has been presented at Regional Meeting of South-Eastern Section of Amer. Chem. Soc.. Birmingham, Ala., Nov., 1954; Tobacco Chemists Meeting, Raleigh, N. Car. Oct., 1955 ; and Annual Meeting of AAAS, Atlanta, Ga., Dec., 1955.

EXPERIMENTAL

Apparatus and Chemicals

Benzene.—Thiophene free.

Cyclohexane.—Practical. This solvent was purified by freezing 4/5 of it and discarding the supernatant liquid.

Alumina.—Harshaw chromatographic grade, heated to 130° C. in an oven for several hours before use. For the iodination experiment, Alcoa F-20 alumina, heated to 130° C. for 1 hour, was used.

Tobacco.—Popular brand of pipe and cigarette tobacco.

Cigarettes.—Popular brands.

Cigars.—2 small size and 2 regular size ; popular brand.

Silica gel.—28–200 mesh. Fisher Scientific.

Glass wool.—Pittsburgh Plate Glass Co. SE 3 × 5—621 glass unbonded B.

Cigarette paper.—Commercial brand.

3,4 benzpyrene.—Eastman.

Methylcholanthrene.—Eastman.

Dibenz(ah)anthracene.—Eastman.

1,12 benzpyerylene.—Aldrich Chemical Co.

Initially, a Beckmann DU ultraviolet spectrophotometer was used, then a Beckmann DK-1 recording spectrophotometer for fluorescence and ultraviolet absorption work. For fluorescence (Burdett and Jones, 1947) the light housing backplate was modified to make the fluorescence sample the light source (Fig. 1). A small platform with a holder for a vial 1 cm. in diameter extended from the backplate, placing the vial at the same position normally occupied by the filament of the tungsten lamp. The ultraviolet exciting light was provided by a GE mercury vapor lamp (CH3) with appropriate transformer. This light passed through a hole in the backplate covered by a glass light filter (Corning 5860) which permitted light under 390mμ to enter the lamp housing. The mercury vapor lamp and transformer were mounted in a metal box provided with a hole adjacent to the lamp. A strip of aluminium foil was fitted to the back half of the lamp to serve as a crude reflector. The " energy " setting of the spectrophotometer and high sensitivity were used for fluorescence measurements.

Smoke Sampling

Cigarettes and cigars were smoked in a smoke sampling apparatus designed by the research laboratory of The American Tobacco Company, Inc. and manufactured by Phipps and Bird, Inc. of Richmond, Virginia. In this apparatus each cigarette was smoked individually, one 3-second inhalation of 60 c.c. of air each minute. The smoke was drawn through acetone. Four cigarettes were smoked simulataneously.

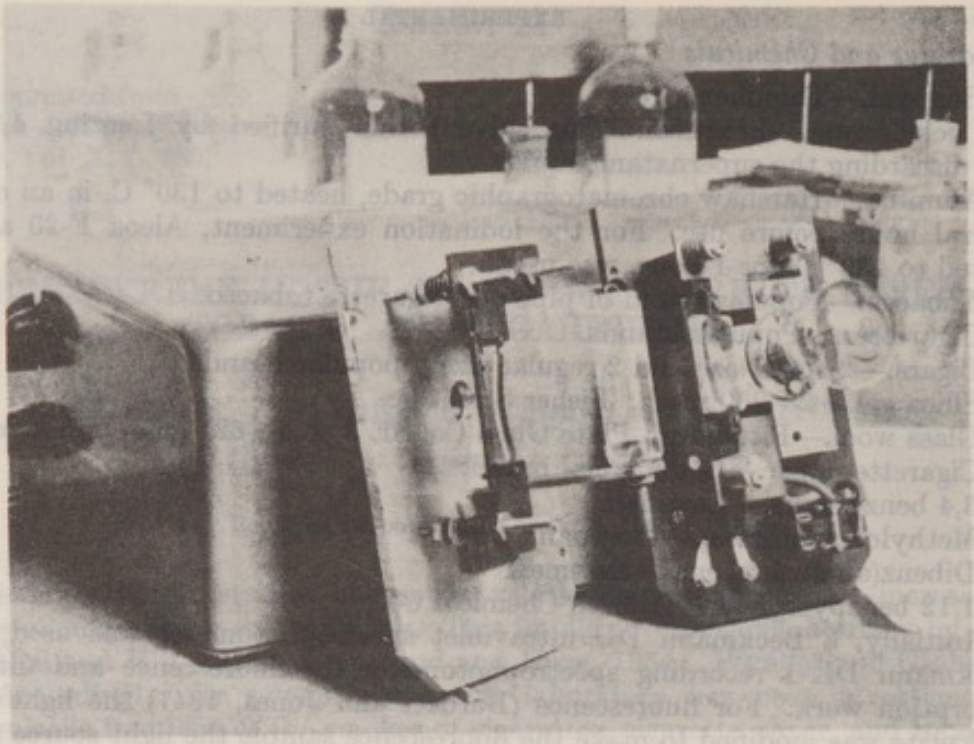
Cigarette paper and tobacco were smouldered in a glass tube 1¼ inches in diameter and 12 inches long (Fig. 2). This tube was connected by means of a

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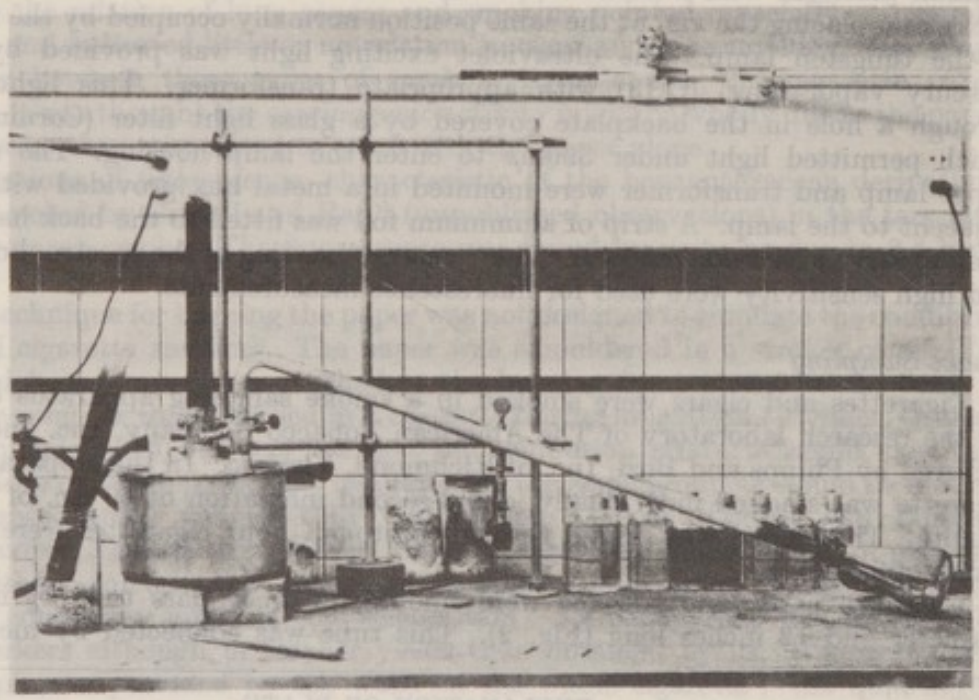
EXPLANATION OF PLATE.

FIG. 1.—Beckmann spectrophotometer modified to make the fluorescence sample the light source.

FIG. 2.—Vertical glass tube 1¼ inches diameter and 12 inches long in which cigarette paper and tobacco were smouldered.



1



2

Cardon, Alvord, Rand and Hitchcock.

34/45 joint to the center neck of a 3-neck flask set in a water bath at room temperature. A piece of wire gauze was placed toward the bottom of the burning tube to prevent the ashes from falling into the flask. To the flask was fitted an adaptor leading to a glass tube tapered at the lower end containing a wad of glass wool 1 to 2 inches long. The tapered end of the condenser tube passed through a rubber stopper into a 200 c.c. filter flask. The filter flask was connected by vacuum tubing to a trap and then to a water aspirator.

Chromatography

Glass tubes 1½ inches to 1 cm. in diameter and 2 to 3 feet long were used. Eluents were collected in narrow-mouth glass bottles 500 to 15 c.c. capacity.

Procedure

I. Cigarette Paper

Cigarette paper was unwound from a commercial roll in 5 to 6 feet lengths. Thirty such lengths (about 30 g.) were wound together and torn into approximately 12 inch lengths. The burning tube was loaded with these wads. With the aspirator on full, the paper was lit and allowed to smoulder down to ashes. The burning front was periodically measured with a bare thermocouple junction and the temperature found to vary between 650 and 900° C. Temperatures to 850° C. were measured with an optical pyrometer. The air flow measured with a flowmeter averaged 5 l. per minute. Thirty minutes was the average time required for the complete burning of the 30 g. samples of paper and about 150 l. of air was passed. This corresponds to 30 l. of oxygen or 1·3 mols. or 7 mols. 0_2/162 g. of cellulose. Theoretically, 6 mols of oxygen are required for the complete combustion of each 162 g. unit of cellulose so that an excess of oxygen was always used in these experiments.

Ten batches (300 g.) of paper were burned in an average day's experiment. Reddish tarry condensate accumulated on the sides of the flask, connecting tube, condenser, and especially in the glass wool plug. Liquid condensed in the glass wool and dropped into the filter flask. When 300 g. of paper had been burned, the apparatus was dismantled and rinsed down with 300 to 500 ml. of acetone (except for the burning tubes), which dissolved all of the condensed tars. The nonvolatile tars produced by the smouldering paper were determined by evaporating a sample of the acetone solution to dryness on a steam bath. Fifteen to 18 g. (5 to 6 per cent) of tars were obtained.

To the acetone solution in a separatory funnel, 500 ml. of cyclohexane was added, followed by 500 ml. of water. The aqueous-acetone water layer was drawn off and discarded. The cyclohexane solution was washed with two 500 ml. portions of water and dried over amhydrous calcium chloride. The dried cyclohexane solution was a sharp smelling, irritating reddish-brown liquid.

A 1 × 6 inch column of Harshaw chromatographic alumina, activated by heating in an oven at 130° C. for several hours, was prepared. The cyclohexane solution was added to the column and it was followed by pure benzene. The cyclohexane passed through the alumina, leaving all the colored material in a narrow band within 1 inch of the top of the column. If the alumina was insufficiently activated, it could be noted by the fact that the colored material spread through the column and came through in the cyclohexane eluents.

The addition of benzene started a yellow material down the column. When irradiated with ultraviolet light, this yellow material fluoresced a bright blue. Fifty milliliter eluents were collected and the fluorescence spectra of all fractions determined. The first benzene eluents were yellowish-green and fluoresced in one wide bright band with a peak at 440–450 mμ (Fig. 3). At the fourth or fifth benzene eluent, two peaks could be discerned at 410 to 412 and 432 to 435 (Fig. 4). The fluorescence spectrum of pure 3,4-benzpyrene in benzene solution has three major peaks at 410, 432, and 455 mμ, with a smaller peak at 413 on the shoulder

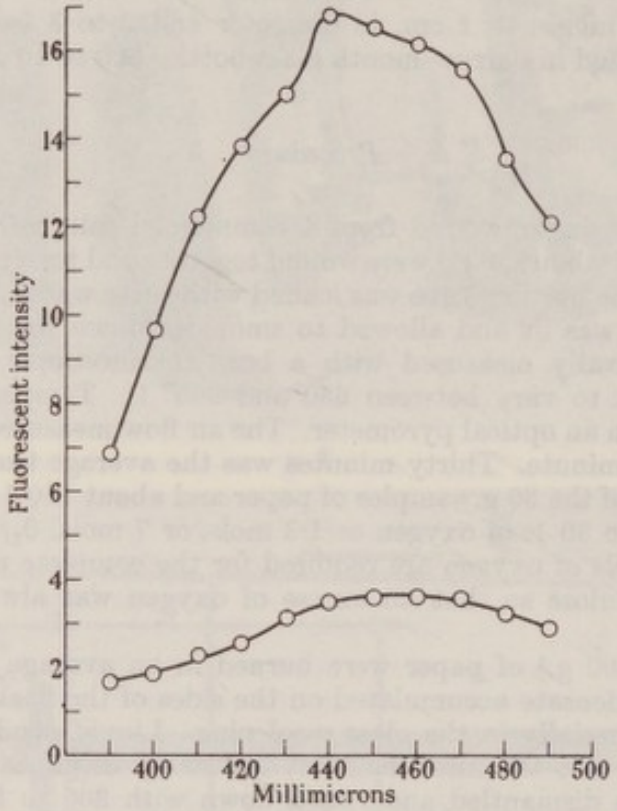


FIG. 3.—Fluorescent spectra of eluents from the cigarette paper tar separation which do not contain 3,4-benzpyrene.

of the 410 peak. The 2 peak fluorescence noted in the benzene eluents of the chromatographic column was the first indication of the presence of 3,4-benzpyrene or other benzanthracene derivatives in the tar. Five to 6 eluents (about 300 ml.) had indications of the 2 peak fluorescence. Subsequent eluents fluoresced with a wide band background fluorescence. Much dark brown material remained on the column.

The eluents showing the 2 peak fluorescence were combined and evaporated to dryness in vacuo at 50 to 60° C. and the residue dissolved in 50 ml. of cyclohexane. This solution was added to a column, $\frac{3}{4} \times 4$ inches, of silica gel. A colored band remained within $\frac{1}{2}$ inch of the top of the absorbent layer. The column was developed with a solution of 10 per cent benzene in cyclohexane. Twenty-milliliter eluents were taken. The first benzene containing eluents fluoresced in bands with two prominent peaks at 410 and 432 mμ and a flat spot

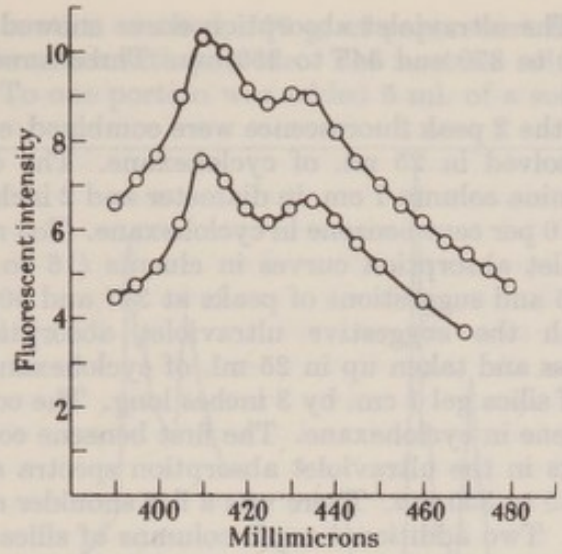


FIG. 4.—Fluorescent spectra of eluents from the cigarette paper tar separation which contains 3,4-benzpyrene.

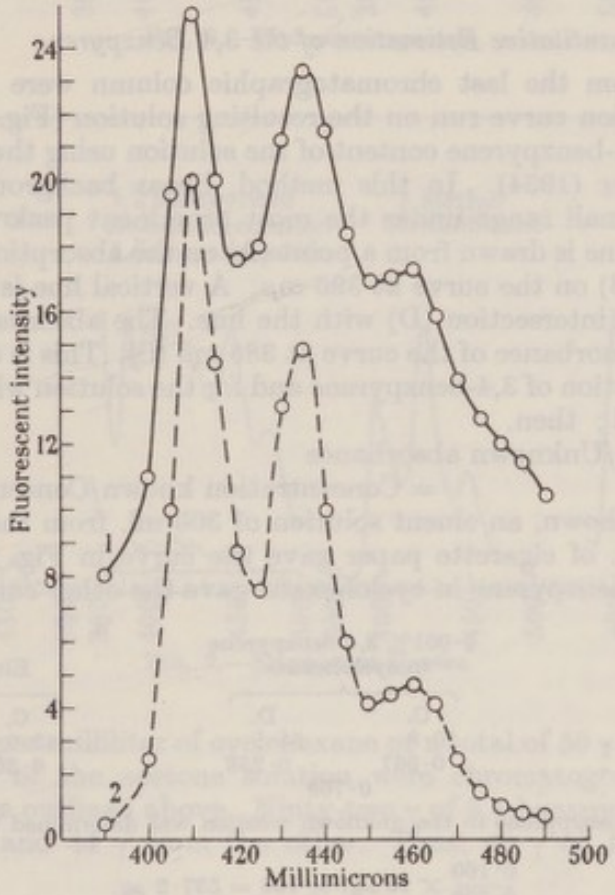


FIG. 5.—Fluorescent spectra. 1. Purified fraction of cigarette paper tar rich in 3,4-benzpyrene. 2. 3,4-Benzpyrene.

at 450 to 455 mμ. The ultraviolet absorption curve showed a small peak at 385 and flat spots at 360 to 370 and 345 to 350 mμ. Three hundred to 400 ml. were obtained.

The eluents with the 2 peak fluorescence were combined, evaporated to dryness and the residue dissolved in 25 ml. of cyclohexane. The cyclohexane solution was added to an alumina column 1 cm. in diameter and 2 inches long. The column was developed with 10 per cent benzene in cyclohexane. Ten milliliter eluents were taken. The ultraviolet absorption curves in eluents (15 to 25) showed definite peaks at 385 and 365 and suggestions of peaks at 347 and 405 mμ.

The eluents with the suggestive ultraviolet absorption were combined, evaporated to dryness and taken up in 25 ml. of cyclohexane. This solution was added to a column of silica gel 1 cm. by 3 inches long. The column was developed with 5 per cent benzene in cyclohexane. The first benzene containing eluents had sharply defined peaks in the ultraviolet absorption spectra at 365, 385, and 405 with a flat spot at 345 to 350 mμ. There was a flat shoulder at 380 mμ just below the peak at 385 mμ. Two additional small columns of silica gel, developed with 3 and 1 per cent benzene in cyclohexane solutions, gave eluents with more clearly defined 3,4-benzpyrene absorption. Peaks at 333 and 317 suggest 1,2-benzpyrene is also present in these samples (Fig. 12). The fluorescence spectrum was very close to that of 3,4-benzpyrene (Fig. 5 and Fig. 6).

Quantitative Estimation of the 3,4 *Benzpyrene*

The eluents from the last chromatographic column were combined and an ultraviolet absorption curve run on the resulting solution (Fig. 12). An estimate was made of the 3,4-benzpyrene content of the solution using the method described in detail by Cooper (1954). In this method, linear background absorbance is asssumed over a small range under the most prominent peak of 3,4-benzpyrene (385 mμ). A base line is drawn from a point (A) on the absorption curve at 375 mμ to another point (B) on the curve at 395 mμ. A vertical line is dropped from the 385 peak (C) to its intersection (D) with the line. The absorbance at (D) is subtracted from the absorbance of the curve at 385 mμ (C). This is done for a solution of known concentration of 3,4-benzpyrene and for the solution whose concentration is to be determined ; then,

Known absorbance/Unknown absorbance
= Concentration known/Concentration unknown.

In the sample shown, an eluent solution of 300 ml. from the chromatography of tars from 550 g. of cigarette paper gave the curve in Fig. 12. A solution of 0·001 per cent 3,4-benzpyrene in cyclohexane gave the other curve in Fig. 12.

	0·001% 3,4-benzpyrene in cyclohexane.			Eluent solution.	
	C.	D.		C.	D.
% transmission . .	10·8	55·2	.	43·0	62·1
Absorbance . .	0·967	0·258	.	0·367	0·207
$A_C—A_D$ . . .	0·709		.	0·160	

The percentage of 3,4-benzpyrene in the unknown solution was determined by substituting in the equation

$$\frac{0{\cdot}160}{0{\cdot}709} \times 10\ \mu g. \times 150 = 337{\cdot}2\ \mu g.$$

$$\frac{550}{0{\cdot}000337} = 1 \text{ part benzpyrene per } 1{,}610{,}000 \text{ part of paper burned.}$$

To check the estimation method, 150 g. of paper were smouldered as above. The apparatus was washed with acetone. The acetone solution was divided in two equal portions. To one portion was added 5 ml. of a solution containing 10γ

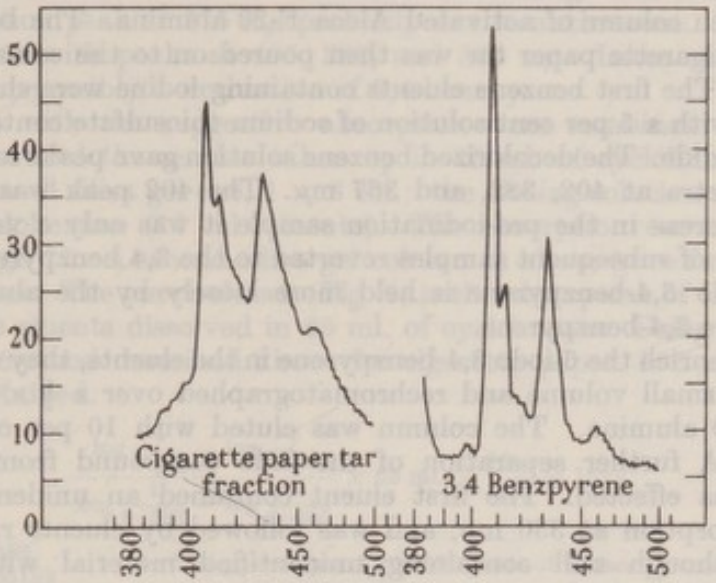


FIG. 6.—Fluorescent spectra.

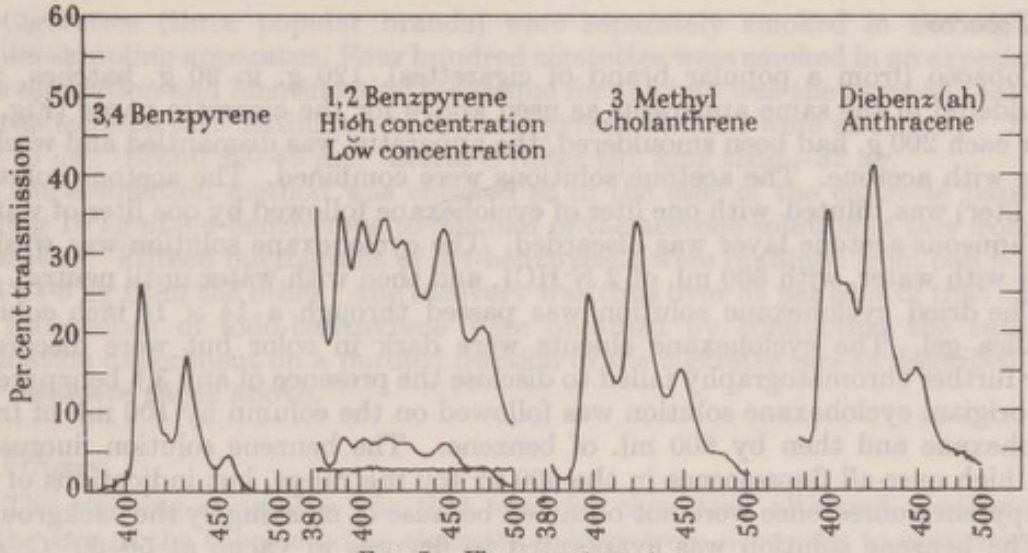


FIG. 7.—Fluorescent spectra.

of 3,4-benzpyrene per milliliter of cyclohexane or a total of 50 γ of 3,4-benzpyrene. The two portions of the acetone solution were chromatographically analyzed using the procedure outlined above. Ninty-two γ of 3,4-benzpyrene were obtained from one portion and 44 γ from the other. Thus, 48 γ of the added 50 were recovered.

*6-Iodo 3,4-Benzpyrene**

* See Tye, Graf and Horton (1955).

A solution obtained by chromatography of tars from cigarette paper smoke containing 97 γ of 3,4-benzpyrene was evaporated to dryness. To a solution of the residue in 10 ml. of benzene was added half of a solution prepared by dissolving 1 g. of iodine in 10 ml. of benzene. The other half of the iodine solution was added to a ¾ × 1½ inch column of activated Alcoa F-20 alumina. The benzene solution of iodine and cigarette paper tar was then poured on to the column followed by pure benzene. The first benzene eluents containing iodine were shaken in a separatory funnel with a 5 per cent solution of sodium thiosulfate containing a crystal of potassium iodide. The decolorized benzene solution gave peaks in the ultraviolet absorption spectra at 402, 382, and 367 mμ. The 402 peak was now the most prominent, whereas in the pre-iodination sample it was only a very small peak. The absorption of subsequent samples reverted to the 3,4 benzpyrene absorption; evidently 6-iodo 3,4-benzpyrene is held more loosely by the alumina and thus comes off before 3,4-benzpyrene.

In order to enrich the 6-iodo 3,4-benzpyrene in the eluents, they were combined, evaporated to small volume and rechromatographed over a ½ × 3 inch column of Alocoa F-20 alumina. The column was eluted with 10 per cent benzene in cyclohexane. A further separation of the iodo compound from unreacted 3,4 benzpyrene was effected. The first eluent contained an unidentified material with peak absorption at 330 mμ, and was followed by eluents rich in the iodo compound, although still containing unidentified material with background absorption and some 3,4-benzpyrene (Fig. 8).

II. Tobacco

Tobacco (from a popular brand of cigarettes), 120 g. in 30 g. batches, was smouldered in the same apparatus as used above for the cigarette paper (Fig. 2). After each 200 g. had been smouldered, the apparatus was dismantled and washed down with acetone. The acetone solutions were combined. The acetone solution (one liter) was diluted with one liter of cyclohexane followed by one liter of water. The aqueous-acetone layer was discarded. The cyclohexane solution was washed twice with water, with 600 ml. of 2 N HCl, and then with water until neutral.

The dried cyclohexane solution was passed through a 1½ × 10 inch column of silica gel. The cyclohexane eluents were dark in color but were discarded after further chromatography failed to disclose the presence of any 3,4 benzpyrene. The origianl cyclohexane solution was followed on the column by 100 ml. of fresh cyclohexane and then by 800 ml. of benzene. The benzene solution fluoresced with high over-all fluorescence in the 400 to 450 mμ range, but indications of 3,4 benzpyrene fluorescence were not obtained because of masking by the background.

The benzene solution was evaporated to dryness in vacuo at 50–60° C. and the residue dissolved in 100 ml. of cyclohexane. This solution was added to a ¾ × 4 inch column of alumina and followed by pure benzene; 20 ml. eluents were collected. Two hundred milliliters were obtained with 2 peak fluorescence at 410 to 420 and 435 to 445 mμ superimposed on a high background fluorescence.

These eluents were combined and evaporated to dryness as above. The residue was taken up in 50 ml. of cyclohexane and this solution added to a ¾ × 5 inch column of silica gel. The column was eluted by a solution of 10 per cent benzene in cyclohexane. The 2 peak fluorescence was improved but there were only slight breaks in the absorption curves at 385 mμ.

The eluents showing 2 peak fluorescence were again combined, evaporated to dryness and the residue dissolved in 20 ml. of cyclohexane. This solution was chromatographed on alumina, 1 × 8 cm., eluting with 10 per cent benzene in cyclohexane. The first 200 ml. of eluents showed nothing. The next 50 to 100 ml. gave an absorption peak at 375 mμ probably due to anthracene. These were followed by eluents with peaks at 405, 385, and 365 mμ. Later eluents showed a peak at 383 mμ, possibly a derivative of 3,4-benzpyrene.

The eluents with the suggestive absorption were combined, evaporated to dryness in vacuo and the residue taken up in 25 ml. of cyclohexane. The next column was a small silica gel one, 1 × 8 cm.; the eluting solution was 10 per cent benzene in cyclohexane (10 ml. eluents). The absorption curves improved in eluents 5 through 12. A second silica gel column as above gave further improved absorption curves. The curve shown (Fig. 9) is the absorption of the residue from the appropriate eluents dissolved in 65 ml. of cyclohexane. Using this curve and the analytical method described above for cigarette paper, 152 μg. of 3,4-benzpyrene were obtained.

$$\frac{\log \frac{1}{0{\cdot}30} - \log \frac{1}{0{\cdot}44}}{\log \frac{1}{0{\cdot}108} - \log \frac{1}{0{\cdot}565}} \times 65 \text{ ml.} \times \frac{10\ \gamma}{\text{ml.}} = 152\gamma$$

$$\frac{1250}{0{\cdot}000152} = 1 \text{ part benzpyrene per 8,200,000 parts of tobacco burned.}$$

III. Cigarettes

Cigarettes (three popular brands) were separately smoked in the cigarette smoke-sampling apparatus. Four hundred cigarettes were smoked in an experiment. The separation and identification technique for the tars was the same as that used on the tobacco tar. Estimates of 3,4-benzpyrene content were as follows : Brand A—40 μg.; Brand B—49 μg.; Brand C (filter tip, king size)—32 μg.

The estimation method was checked as in the case of the cigarette paper by adding 10 μg of 3,4-benzpyrene to one half of the acetone solution of tars from 380 cigarettes. Fifteen micrograms of 3,4-benzpyrene were obtained from one portion and 23·5 μg from the other ; the recovery was thus over 85 per cent of that added.

Indications of iodo-benzpyrene were obtained by conducting the catalytic iodination experiment on a purified fraction of the cigarette tars as described with the cigarette paper above.

IV. Cigars

The smoking apparatus was that used for cigarettes, although modifications in the size of the tubes used as cigarette holders were necessary to accommodate the larger size cigars. Regular size and small size cigars were separately smoked. The separation and identification was that used on the tobacco described in the foregoing. The results were :

Brand A (regular size)	82·5 γ per 20 cigars (wt. of cigar 8 g.)
Brand A (small size)	94·0 γ per 49 cigars (wt. of cigar 2·75 g.)
Brand B (small size)	10 γ per 24 cigars (wt. of cigar 2·75 g.)
Brand C (regular size)	25 γ per 24 cigars (wt. of cigar 8·5 g.)

In the eluents from Brand A containing 3,4-benzpyrene, a prominent peak is present at 340 mμ. This may be due to coronene.

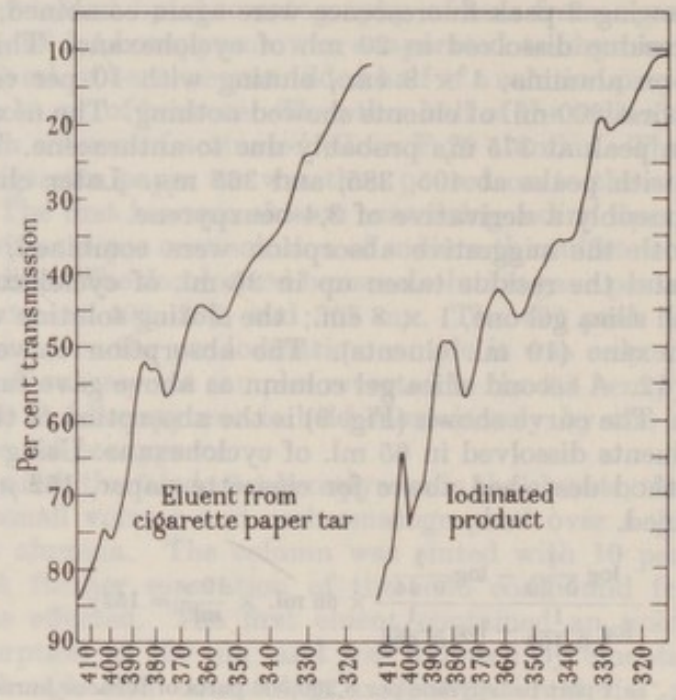


FIG. 8.—UV absorption.

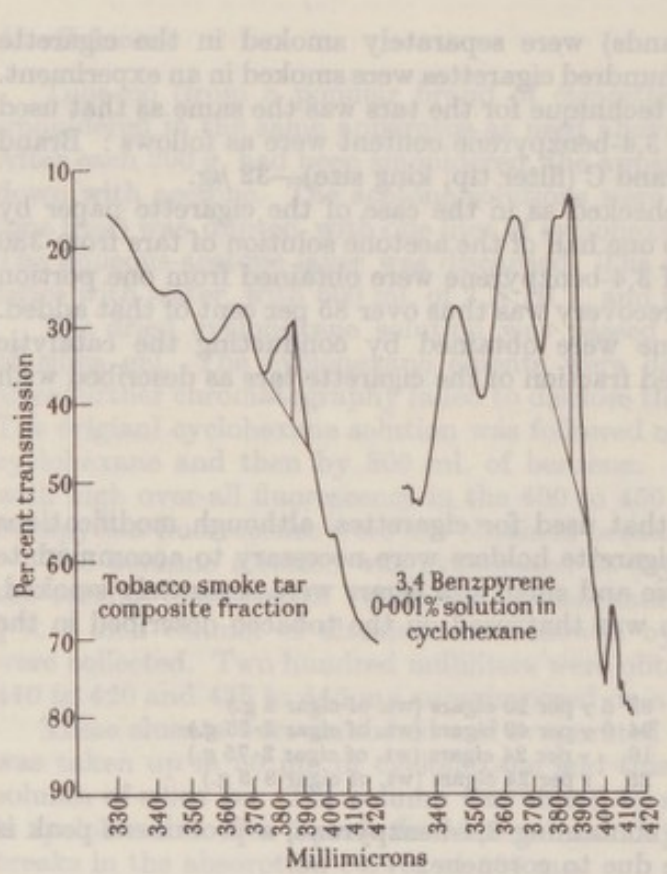


FIG. 9.—UV absorption.

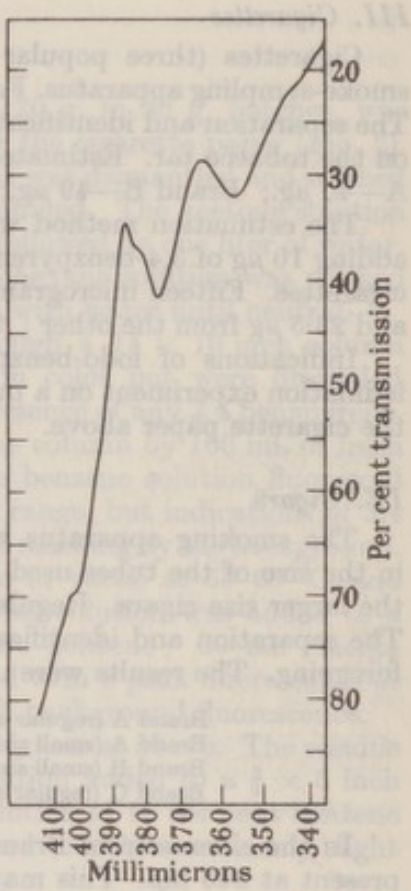


FIG. 10.—UV absorption; cigarette tars composite fraction.

DISCUSSION

The identification of 3,4 benzpyrene in the tars from the smoke of cigarettes, tobacco, and cigarette paper is based on four considerations.

1. The fluorescence spectrum. The original crude spectra of the first investigators in this field could hardly be considered identification, since they reported 3 bands in the wavelength region, 4000 to 4500 Å (Cook, Hewett and Hieger, 1933 ; Kennaway and Hieger, 1930). The spectra recorded with a wide slit show only 3 bands, but even so a difference is readily apparent between 3,4-benzpyrene, 1,2,5,6 dibenzanthracene, and 10 methylcholanthrene (Fig. 7). The narrow slit fluorescence spectrum discloses a still more complex spectrum for 3,4-benzpyrene (Fig. 6) ; 4 distinct peaks at 406, 408, 430, and 450 with fine irregularities in the region 410 to 420 mμ. This fine structure fluorescence spectrum of 3,4-benzpyrene was first noted by Bernard Muel and Michel Hubert-Habart at The Institute du Radium in Paris, France. The apparatus for fluorescent spectra used in that laboratory was designed by the director of the laboratory, R. Latarjet, and is capable of high sensitivity and high dispersion. Nevertheless, much more work is necessary before fluorescence itself can be considered final identification, since carefully determined spectra of many 3,4-benzpyrene-like compounds must yet be done.

2. Ultraviolet absorption spectrum. The identification using absorption spectra is more certain than is fluorescence alone. Five distinct peaks can be seen, at 405, 385, 380, 365, 347, with a discontinuity at 395 mμ. The spectra of only two rare polynuclear hydrocarbons of many that have been reported are closely enough aligned to that of 3,4 benzpyrene to raise serious doubts of the identity (9-methylanthracene and 1 : 12-benzperylene) (Friedel and Orchin, 1951). Fluorescence eliminates the latter as its fluorescence spectrum is completely different from that of 3,4-benzpyrene (Fig. 11).*

3. The indication of the presence of 6-iodo 3,4-benzpyrene in iodinated purified fractions of the tars (Tye, Graf and Horton, 1955).

4. The recovery of added quantities of pure 3,4-benzpyrene in the same eluents by the identical procedures as with the substance being investigated.

Further, there is indication of the presence of 1,2-benzpyrene (absorption peaks at 333 and 317 mμ) in the same eluents with the 3,4-benzpyrene, as would be expected. This is especially clear and pronounced in the excellent absorption curve obtained by Lefemine at the Miami Cancer Institute, on a highly purified fraction of cigarette paper tar.

The weight of a cigarette is about 1 g. The weight of the paper is 0·04 g. Assuming the paper and tobacco produce the same quantitites of 3,4-benzpyrene when smoked in a cigarette as they do when smouldered separately in the apparatus described here, the paper of a cigarette would produce 0·042 and the tobacco 0·122 γ. The total per cigarette is then 0·162 or for 400 cigarettes, 64·2 γ. This is in line with the results obtained with cigarettes smoked in the smoking machine (40 to 49·2), considering that $\frac{1}{4}$ to $\frac{1}{5}$ of the cigarettes are discarded as butts in the latter experiment.

* Since the preparation of this paper, a sample of 9-methylanthracene was obtained. The fluorescence of this compound is also very much different from that of 3,4-benzpyrene and can be used to differentiate between the two compounds.

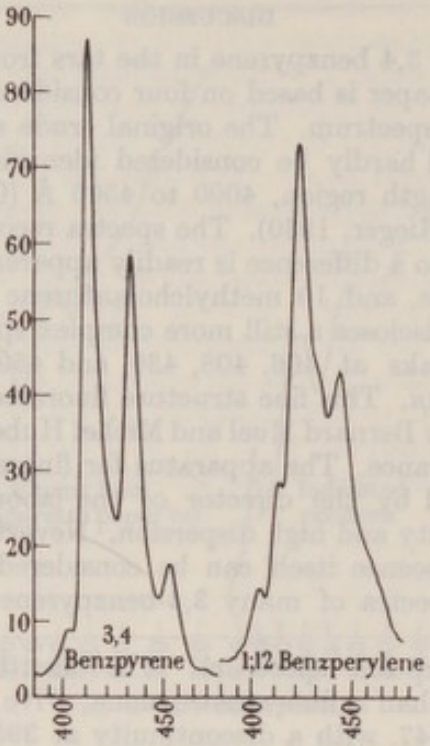


FIG. 11.—Fluorescence.

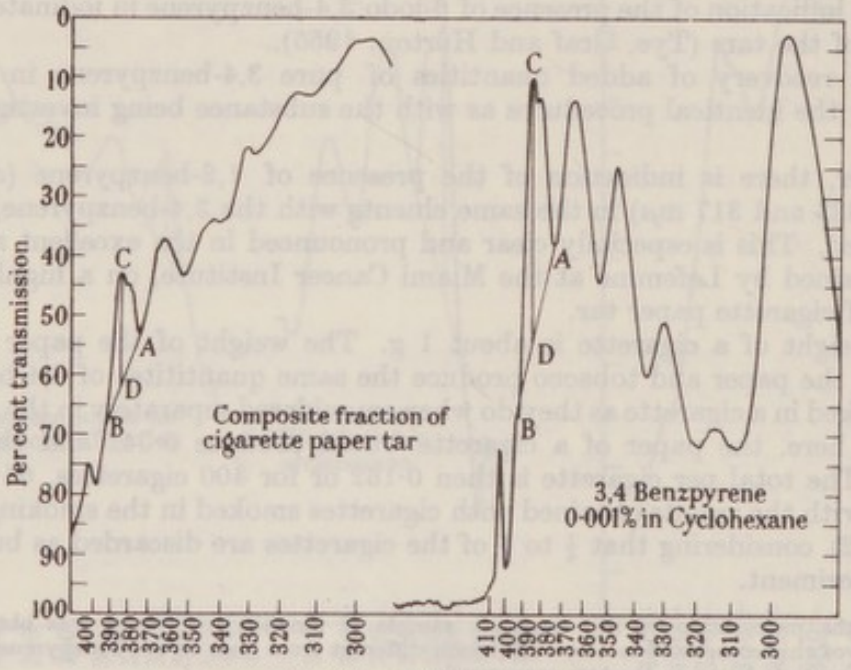


FIG. 12.—UV absorption.

The variation in 3,4-benzpyrene formation in the smoke of the different brands of cigars suggests the high value in the one brand may be a product of an additive used in this tobacco. The other brands show the expected similarity to tobacco.

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EXHIBIT 22

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THE INHIBITION OF FORMATION OF 3,4-BENZPYRENE IN CIGARETTE SMOKE*

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Received for publication May 10, 1956

IN a previous article (Cardon *et al.*, 1956) 3,4-benzpyrene was reported in the tars from the smoke of cigarette paper, tobacco, and cigarettes. Considering the possibility that cigarette smoke may be carcinogenic, as has been suggested in recent medical publications, it was considered of value to attempt the elimination of this well-known carcinogen from cigarette smoke.

As reported previously, cigarette paper produces more 3,4-benzpyrene than does tobacco per weight of material smoked. Also, the detection of 3,4-benzpyrene is simpler in tars from paper than in tobacco or cigarette tars. Our initial effort was therefore to modify the composition of the paper, thereby affecting the combustion and combustion products so that less 3,4-benzpyrene would be present in the paper smoke.

Cigarette paper was treated with aqueous and non-aqueous solutions of various chemicals, smoked, and the smoke analyzed for indications of 3,4-benzpyrene. A rapid screening method involving one chromatographic column was used. Indications of the presence of 3,4-benzpyrene were obtained from the fluorescent spectrum ; peak fluorescence at 410 and 432 mμ was considered to be due to 3,4-benzpyrene. The quantity of eluent and the intensity of this peak fluorescence was assumed to be a rough indication of the quantity present.

Using this method, many compounds were added to paper and the effect on 3,4-benzpyrene production noted. Concentrations of 5 to 15 per cent by weight of the paper were used. In general, substances which would be expected to produce acid vapors on heating in moist air, reduced the formation of total tar but left the 3,4-benzpyrene unaffected or somewhat increased. In this class are the halides of calcium, barium, magnesium, zinc, sodium, and potassium. Sulfates, nitrates, acetates, phosphates, and perchlorates did not have much effect though alkaline salts like sodium acetate and trisodium phosphate increased the production of fluorescent tars. Alcohols and organic acids were without effect.

Ammonium compounds of strong acids, i.e., phosphate, sulfate, sulfamate, and chloride, sharply reduced the production of 3,4-benzpyrene in cigarette paper tars. The nitrate was ineffective, as were ammonium salts of organic acids, presumably because ammonia is not produced by these materials when they are heated. Amines and aminoalcohols reduced benzpyrene formation but less so than the inorganic ammonium salts. The effect was roughly proportional to the "effective" ammonia content ; thus, ammonium sulfamate had the same effect as an equal weight of ammonium sulfate or twice the weight of monoammonium phosphate.

* This paper was presented at a meeting of AAAS, Atlanta, Ga., Dec., 1955.

For more quantitative data on the 3,4-benzpyrene content of the paper tars, larger batches of paper were burned as described (Cardon *et al.*, 1956) and the separation technique carried through to solutions in which the ultraviolet absorption curves gave peaks adequate for the quantitative estimation. The tar of paper containing 4 per cent or more of a diammonium salt or its equivalent contained one part or less per 15 million of 3,4-benzpyrene by weight of the paper burned. A diammonium salt content of 7 per cent reduced the 3,4-benzpyrene content to less than one part per 30 million.

Diammonium salts in the paper were unstable and lost ammonia on standing, presumably due to the presence of calcium carbonate filler in the paper. Monoammonium salts were stable. Ammonium sulfamate, although monobasic, contains two effective ammonium groups and had the same inhibiting effect as ammonium sulfate but was stable.

Tobacco containing ammonium sulfamate similarly produced less benzpyrene. Cigarettes prepared from treated paper produced less than half the benzpyrene than did cigarettes prepared from the same tobacco (not a popular brand) and untreated, paper. As previously reported (Cardon *et al.*, 1956) the relative production of benzpyrene by the paper and tobacco is approximately 8 : 1, but since the paper is 1/25 the weight of the cigarette, the expected ratio of benzpyrene from the paper and tobacco is 1 : 3. The more than 50 per cent reduction in benzpyrene formation suggests the presence of ammonium salts in the paper reduces the benzpyrene production from the tobacco.

EXPERIMENTAL

Chemicals

The solvents used were c.p. or reagent grade. The chemicals tested were c.p. except where these were not available as in some of the organic materials like ethanolamines. Reagent and technical grade ammonium salts were used indiscriminately with identical results.

Procedure

1. *Paper*

The general technique for treating the cigarette paper with water soluble materials was to dip the paper from a standard commercial roll in an aqueous solution of the desired material and pass the wet paper on a stainless steel belt under infra-red lights to dry.

The paper was burned and the tars collected in acetone as previously described (Cardon, *et al.*, 1956). The acetone solution was shaken in a separatory funnel with cyclohexane and water. The water acetone layer was discarded and the cyclohexane solution washed free of acetone with more water and dried over anhydrous calcium chloride.

The cyclohexane solution was passed through a column of activated alumina 1-$\frac{1}{2}$ inch in diameter $\times$ 4 inches long, and the column eluted with benzene. The benzene eluents were examined for the 2-peak fluorescence spectrum characteristic of 3,4-benzpyrene in dilute mixtures. Paper, the tars of which gave eluents in which this type of fluorescence was comparable qualitatively to that produced by untreated paper, was not considered further. In those cases in which the 2-peak fluorescence spectrum was present in smaller amounts of solution and was of

lower intensity, larger batches of paper were burned and the separation technique for 3,4-benzpyrene carried through to ultra-violet absorption curves and a quantitative estimate of the 3,4-benzpyrene produced per gram of paper burned was made by the technique previously described.

Soluble sulfates, nitrates, and acetates of Ca, Mg, Ba, Na, K and Zn in the paper did not reduce the benzpyrene in the corresponding tars. The halides of Ca, Mg, Zn, and Ba reduced the acetone soluble material, non-volatile at 100° C, to about 20 per cent of that formed from untreated paper, but had no inhibiting action on the formation of 3,4-benzpyrene. Alkaline salts, that is sodium salts of phosphoric, boric, acetic, and benzoic acid, increased the amounts of material with high overall fluorescence and made the detection of 3,4-benzpyrene more difficult. Repeated chromatography nevertheless showed as much benzpyrene in these tars as in tars from untreated paper.

Ammonium salts of sulfuric, sulfamic, phosphoric, hydrochloric, persulfuric, and perchloric acids caused a sharp reduction in the benzpyrene content of the tars from the papers containing them. Ammonium nitrate and ammonium acetate, which do not produce ammonia when heated, had little inhibiting effect on the benzpyrene formation. These results are summarized in Table I.

TABLE I.—*Classes of Compounds Tested**

Type of compounds.	Example.	Per cent pickup.	Remarks.
Neutral salts	Potassium sulfate	10	Definite peaks at 410 and 432.
Organic salts	Sodium citrate	11	Definite peaks.
Organic amines	Triethanolamine	10	Low peaks.
Inorganic ammonia salts	Ammonium sulfate	7	Low fluorescence. No peaks.
Inorganic oxidizing agents	Potassium persulfate	8	Definite peaks.
Organic ammonium salts	Ammonium acetate	10	Definite peaks.
Aromatic salts	Sodium benzoate	8	Definite peaks.
Inorganic acid salts	Sodium bisulfate	8·2	Definite peaks.
Dehydrating agents	Calcium chloride	15·5	Very little tars but high peaks.
Aromatic oxidizing agents	Benzoyl peroxide	9	Burns fast. Definite peaks.
Inorganic alkalies	Sodium carbonate	8	Definite peaks.
Inorganic amines	Sodium sulfamate	7	No peaks.
Complex amine salts	Nickel hexamine sulfate	10	Low tars. Low peaks.
Aromatic acids	Benzoic acid	5	Definite peaks.
Fire retardent	Boric acid	8	Low tar formation. High peaks.
Metallic salts	Sodium stannate	12	Low tars. Peaks.
Nitrogen aromatics	Pyridine	5	Peaks.
Hydroxy acids	Gluconic acid	10	Peaks in two eluents.
Fatty acid salts	Sodium lauryl sulfate	15	Peaks.
Miscellaneous	Mandelic acid	8	Definite peaks.
Miscellaneous	Chlorophyl	10	Definite peaks.

* 5 per cent solutions were used to treat the paper.

Storage tests on papers containing ammonium salts were run by periodically analyzing the paper for the ammonia content. This was done by adding a weighed sample of the paper to a 10 per cent solution of sodium hydroxide in a flask fitted with a liquid trap and distilling over the ammonia into an aqueous boric acid solution. The distillate was titrated with standard acid. To confirm the analysis,

Kjeldahl digestions of the paper were run on some samples. The results are summarized in Table II. The monoammonium salts are stable, whereas the diammonium salts lose some ammonia on standing, perhaps due to the action of calcium carbonate present as filler in the cigarette paper.

TABLE II.—*Loss of NH_3 From Treated Cigarette Paper*

Compound.	Date: 11.xi.54. % NH_3.	22.xi.54. % NH_3.	6.xii.54. % NH_3.	25.i.55. % NH_3.	11.ii.55. % NH_3.
Ammonium sulfate	1·85	1·21	1·13	—	—
Diammonium phosphate	1·45	1·28	1·12	—	—
Monoammonium phosphate	1·26	1·25	1·15	—	—
Ammonium sulfamate	0·89	0·95	0·88	0·88	0·88 and 1·78*
Ammonium persulfate	—	1.63	—	—	1·57†

* Kjeldahl digestion.
† Turns brown on ageing.

The inhibiting effect was roughly proportional to the ammonia content of the paper. This can be seen qualitatively in Fig. 1. Thus, 3 per cent ammonium sulfate is more effective than 1 per cent ammonium sulfate and 5 per cent diammonium phosphate is more effective than 3 per cent diammonium phosphate.

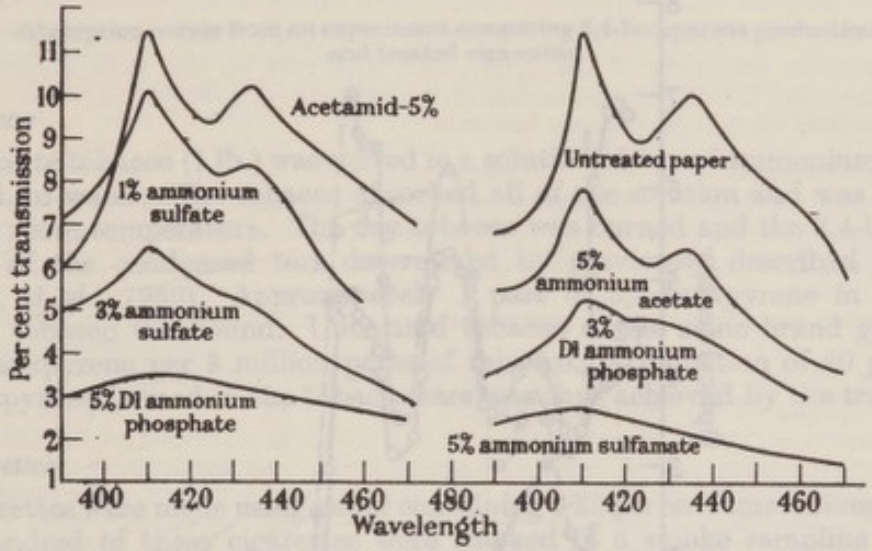


FIG. 1.—Comparative inhibitory effects of various substances on 3,4-benzpyrene.

Because of its stability and relatively high ammonia content, ammonium sulfamate was studied more extensively as an additive to cigarette paper. Papers containing various percentages of ammonium sulfamate were burned and the quantities of 3,4-benzpyrene in the tars were determined. The results are graphically shown in Fig. 2. In determining the percent reduction, a figure of one part benzpyrene per million of paper was used for untreated paper which is an average of a number of determinations. The figure for the reduction effected by the 6·7

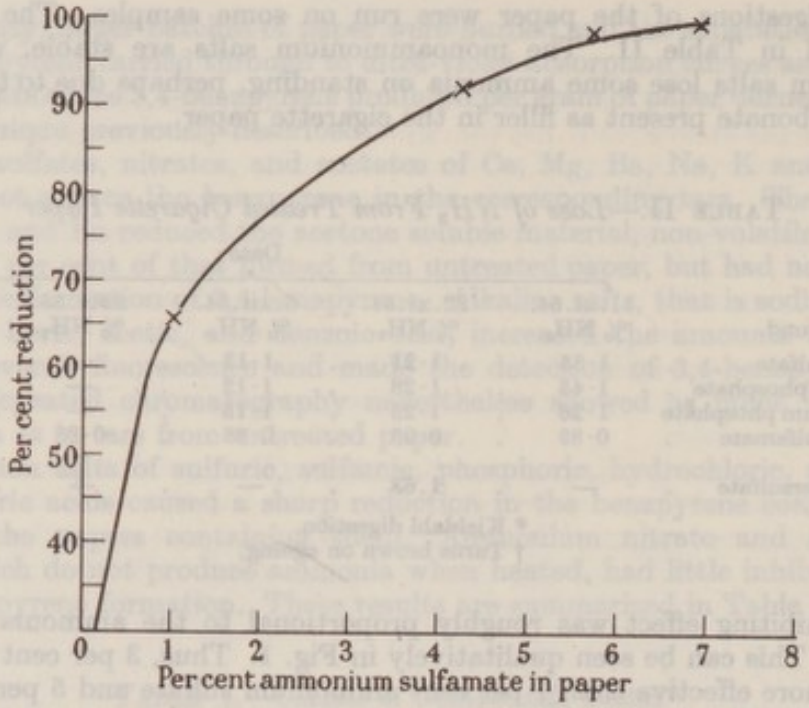


FIG. 2.—Reduction of 3,4-benzpyrene by ammonium sulfamate.

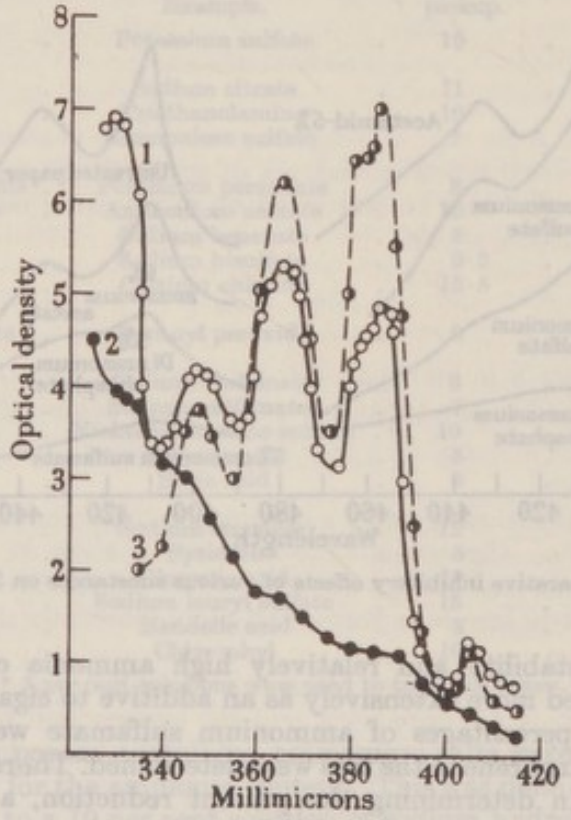


FIG. 3.—Absorption curves of the 3,4-benzpyrene from 300 g. of regular paper and from 1000 g. of paper containing 6·7 per cent ammonium sulfamate. 1. Untreated cigarette paper. 2. Treated cigarette paper. 3. 3,4-Benzpyrene (·001 per cent in isopropyl ether).

per cent paper is an estimate, as the absorption peak at 386 mμ was too low to make an accurate calculation.

Fig. 3 shows absorption curves of the 3,4-benzpyrene from 300 g. of regular paper and from 1000 g. of paper containing 6·7 per cent ammonium sulfamate.

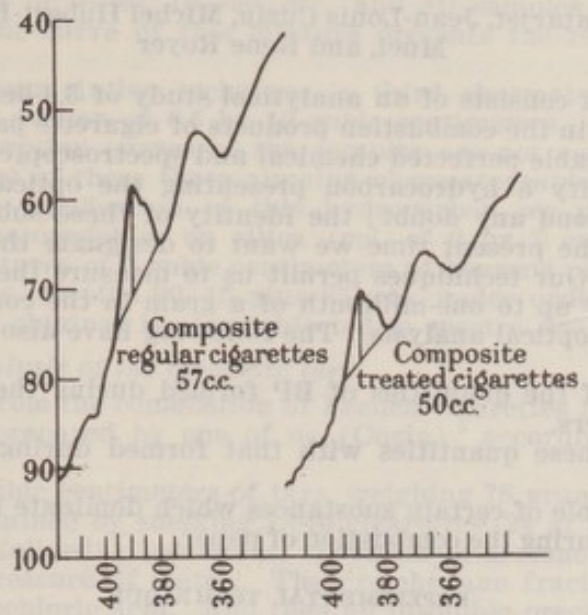


FIG. 4.—Absorption curves from an experiment comparing 3,4-benzpyrene production by regular and treated cigarettes.

2. *Tobacco*

Cigarette tobacco (1 lb.) was stirred in a solution of 9 g. of ammonium sulfamate in 50 ml. of water. The tobacco absorbed all of the solution and was then dried in air at room temperature. The dry tobacco was burned and the 3,4-benzpyrene content of the condensed tars determined by previously described techniques (Cardon, *et al.*, 1956). Approximately 1 part of 3,4-benzpyrene in 40 million parts of tobacco was found. Untreated tobacco of the same brand gave 1 part of 3,4-benzpyrene per 8 million parts of tobacco. A reduction of 80 per cent of 3,4-benzpyrene formed in the tobacco tars was thus achieved by the treatment.

3. *Cigarettes*

Cigarettes were made using paper containing 4·25 per cent ammonium sulfamate. Four hundred of these cigarettes were smoked in a smoke sampling apparatus and the tars were analyzed for 3,4-benzpyrene by previously reported techniques. The quantity of 3,4-benzpyrene formed was compared to that formed from 400 cigarettes made from the same tobacco and untreated paper. Seventy γ of 3,4-benzpyrene were found in the tars of cigarettes with untreated paper and 28 from those made with treated paper. A repeat experiment gave 67 γ and 31 γ, respectively. Fig. 4 shows absorption curves for the latter experiment.

REFERENCE

CARDON, S. Z., ALVORD, E. T., RAND, H. J. AND HITCHCOCK, R.—(1956) *Brit. J. Cancer*, **10**, 485.

EXHIBIT 23

QUANTITATIVE DETERMINATION OF 3,4-BENZPYRENE FORMED BY COMBUSTION OF CIGARETTE PAPER AND THE TOBACCO[1]

By Raymond Latarjet, Jean-Louis Cuzin, Michel Hubert-Habart, Bernard Muel, and Rene Royer

The present work consists of an analytical study of 3,4-benzpyrene, a carcinogenic hydrocarbon, in the combustion products of cigarette paper, and of tobacco. Thanks to the available perfected chemical and spectroscopic techniques we have succeeded to identify a hydrocarbon presenting the optical characteristics of 3,4-benzpyrene, beyond any doubt; the identity of these substances will be discussed later. At the present time we want to designate these substances with the letters "BP." Our techniques permit us to measure the quantities with an absolute sensitivity up to one-millionth of a gram in the concentrated solutions to be subjected for optical analysis. The following have also been found possible to investigate:

1. Comparison of the quantities of BP formed during the combustion of different types of papers.
2. To contrast these quantities with that formed during the combustion of whole cigarettes.
3. Study of the role of certain substances which dominate the quantity of pyro formations of BP during the combustion of paper.

EXPERIMENTAL TECHNIQUE

We will describe the process of combustion of paper, the condensation of tar, chromatographic analysis of the paper tar, analysis of the tars obtained from the cigarette, and, finally, spectrographic techniques of quantative determinations of BP in certain chromatographic eluants.

A. Burning the paper

Our apparatus (fig. 1) is developed according to Alvord and Cardon (1). Thirty grams of commercial cigarette paper folded into 40 layers of 75 centimeters of length was divided into 3 equal parts in the form of a reasonably tight tassel. This was firmly packed in a combustion tube. Speed of combustion is regulated by means of a water aspirator, and the degree of packing of the paper into the tube, and glass-wool plugs. The temperature reached is measured at the passage of the flame front by a nickel constantan thermocouple placed in the middle region of the combustion tube.

Three hundred grams of paper was thus burned in portions of 30 grams each. Due to the accumulation of tars, the glass-wool plug becomes less penetrating and the combustion tends to become less active as the smoldering progresses.

With the exception of the combustion tube, on the walls of which the ashes are collected, the entire apparatus is washed with a mixture of 400 cubic centimeters of acetone and 50 cubic centimeters of water. Insoluble material mixed with this solution is then filtered off. The solution is decantered with cyclohexane AR, several times. Cyclohexane washings, after being brought together, are washed with water until free of the traces of acetone and then dried over calcium chloride.

B. Chromatographic analysis of the paper tars

This process comprises the following successive operations.

A. Cyclohexane solution is transferred into a column of alumina of the size of 2 by 4 centimeters, kept at 180° C. for 13 hours. (This heating favors the fixation of hydrocarbons on alumina.) On the other hand of the column, cyclohexane is recovered, and the degree of fixation of hydrocarbons can be verified by spectrophotometric checks.

This operation is followed by a benzene (A. R.) elution, accelerated by a slight suction at the bottom of the column as provided through a water aspirator. Successive samples of 25 cubic centimeters has been collected, and the presence of benzpyrene is tested in fluorescence spectrograph. Samples containing the benzpyrene fraction are then combined together. The mixture is then evaporated in a vessel with sufficient heat to prevent any change in the residue. After the

[1] From the Bulletin on Cancer (1956), p. 180.

benzene is completely evaporated, apparatus is left for cooling without suction and the residue is taken with 20 to 30 cubic centimeters of cyclohexane.

B. This more concentrated second solution is transferred into another alumina column of lesser size than the previous (1.2 by 20 cubic centimeters). This is elueted, always in vacuum, with successively increasing benzene cyclohexane mixtures in the order of 5—95, 10—90, etc., and all samples are combined together; the fluorescent curve of this mixture presents the resemblance of 3,4 benzpyrene.

C. Using the previous elution technique, a third chromatography is undertaken on an alumina column of 0.5 by 10 cubic centimeters. At this stage the fluorescence and absorption curves of the samples are not completely purified.

D. The final product of these three alumina chromatographies contains benzpyrene. For further purification of this hydrocarbon we proceed with two supplementary chromatographies on silica (col. of 2 by 7 cubic centimeters), elueting with the mixtures of 5 cubic centimeters of benzene plus 95 cubic centimeters of cyclohexane, 10 plus 90, 15 plus 85, etc. under ordinary pressure.

The solution finally obtained is also subjected to spectro analysis.

C. *Treatment and analysis of the cigarette tars*

The tars obtained from the combustion of French cigarettes is designated here as No. 1; has been prepared by one of us (Cuzin) [2] according to an original technique (2).

We have used 93 cubic centimeters of tars, weighing 78 grams, with 24 grams of a dry material, obtained by smoking 1,040 cigarettes, 24 at a time. This tar was subjected to the following treatment. Dilution in acetone; extraction with cyclohexane in the presence of water. The cyclohexane fraction were washed with 10 percent hydrochloric acid. And then an insoluble precipitate is formed; following dryness, washing with water, then with cyclohexane. The cyclohexane fraction is subjected to a second washing with 10 percent soda. By treatment with soda another precipitate is formed which is much less in quantity than the acid wash. This precipitate is also washed. Finally the remainder of the cyclohexane extract is washed with water and dried with sodium sulfate, and concentrated. These also are chromatographed. First the chromatography is done on a 2 by 24 cubic centimeter column of alumina dried for 18 hours at 180° C. Elution with benzene under suction. All fluorescing samples are collected, combined, evaporated to dryness, and redissolved in cyclohexane.

The second chromatography on a column identical to the preceding. Elution under suction with mixtures of 5 percent benzene and 95 percent cyclohexane, 10 benzene plus 90 cyclohexane, etc.

The third chromatography on a 15 by 1 column, resembling column No. 2. A fourth chromatography on a 14 by 0.6 cubic centimeter column resembling chromatograph No. 2.

The fifth chromatography on a 7 by 0.6 centimeter silica gel column. Eluted first with 5 benzene plus 95 cyclohexane, and then with 10 benzene and 90 cyclohexane. By this technique one can obtain satisfactory samples presenting the spectro characteristics on which the actual dosage can be determined. (Quantitative research). Following the qualitative separation of the hydrocarbon which we called BP, its identity with that of 3,4-benzpyrene is affected as follows.

The sixth chromatography resembling that of the first on a column of 15 by 0.6 centimeters; elution with 5 benzene and 95 cyclohexane.

And finally the seventh chromatography identical to that of chromatogram No. 6, on which the fluorescence spectrum is finally obtained and registered.

D. *Spectrographic studies*

1. *Fluorescence.*—Precise and detailed fluorescence spectra were obtained by direct photoelectric registration through a highly sensitive receptor and a double dimension monochromotor. Of course a satisfying compromise between two antagonistic exigances, i. e., luminosity and dispersion has been done. This combination offers two major advantages over photographic spectrography: Shortens the exposure time which eliminates the changes in the substance as could be induced by excitatory UV waves during the examination; and the possibility of performing several control checks during the chromatography.

2. Once the sensitivity of the spectrophotometer is determined for a known spectral curve, the true spectrum is immediately obtained without resorting to laborious operations of photographic photometry.

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[2] Dr. Cuzin is chief of research of tobacco for French Government.

Dispersion apparatus is a subtracting type double monochromotor, built by Institute of Optics of Paris (3). It is open up to f/10, carries 2 flint prisms (angle 60°, height 6 centimeters, base 10 centimeters), and an optic mirror. The dispersion thus obtained varies between 30 A°/millimeters to 4000 A°; and this, with a 0.1 millimeter slit, permits determination of the order of 3A°. For instance we can easily separate 2 distant bands at 10 A° and 300 A°.

The receptor in a 14-stage photomultiplier (type 6685 B of the firm E. M. I.) very sensitive in the explored spectral region, having the ability to register the signals as weak as 10^{-8} amperes with a gain of 10^7 times. This multiplier is directly attached to a galvanometer.

The fluorescence of the sample is excited by the radiation of 3,650 A° delivered from a mercury vapor at moderate pressure (Mazda M. H. 125), and isolated by an ultraviolet filter of 4 millimeters thickness. A mirror concentrates the ray upon the sample. An opening at the top of parabola permits the passage of fluorescent light and reaches to the slit of monochromotor by means of a focusing lens. This way, fluorescence is excited and observed on the same side of the sample holder, eliminating the absorption of fluorescent light by the solution itself.

All the spectra mentioned in this article are true spectra, i. e., all signals on the receptor are corrected according to the spectral monochromatic sensitivity of the instrument. This sensitivity depends upon spectral variations of transmission and dispersion of monochromotor, and the sensitivity of photomultiplier. These variables have been determined individually, and final checks have been performed by the use of a source of known as spectral emission like a tungsten filament, the color of which is determined by optical pyrometer.

2. *Absorption.*—Absorption spectra of the solutions with satisfactory fluorescence have been registered by a Beckman spectorphotometer, model D. U. In a general manner, fluorescence is utilized for detection and absorption for quantitative determination.

RESULTS

A. Detection by fluorescence

Our reference spectrum of 3,4-benzpyrene (fig. 2) has been registered by using the commercial product of Eastman Kodak Co., which we purified by two successive chromatography before registration. This product shows characteristic fluorescence bands (table 1).

In order to indicate the precision of points and the sensitivity of the method, we present, also, the fluorescence spectrum of 1,2-benzpyrene, an isomer of 3,4-benzpyrene (fig. 3).

The fluorescent spectra of the following extracts have been registered, using a mixture of 90 percent cyclohexane and 10 percent benzene as final solvents.

Table I and figures 4, 5, 6 form the essential spectral data obtained. Following facts can be drawn from these:

1. The substance, designated as BP, extracted from the combustion products of cigarettes and of paper, presents the fluorescence and absorption characteristics very close, and quasi-superposable, to 3,4-benzpyrene.

2. Treatment with ammonium sulfamate hinders the formation of BP, at least to a greater extent, if not inhibits it totally. This certainly confirms the findings of Alvord and Cardon 1, who first recommended and assayed it successfully. The paper is simply dipped into a 5 percent aqueous solution of ammonium sulfamate for a few minutes and then dried. This treated paper gives an entirely different spectrum than others. Fluorescence does not reveal many of the bands of BP, but, on the other hand, displays the intense band of anthracene at 5,010 and, as a secondary irregularity, exhibits the Raman rays of cyclohexane at 4,095 A. Absorption studies confirm the massive diminution of BP and formation of anthracene.

B. Quantitative determination by absorption

Fluorescent spectra, though very favorable for detection of BP by means of the abundance of high peaks, does not render themselves for precise-quantitative determination. The intensity of the peaks are very sensitive to the presence of impurities, particularly to the presence of traces of other hydrocarbons. On the other hand, once the fluorescence characteristics of a solution is obtained, its absorption spectrum is then allowed for quantitative determination by comparing with a solution of pure 3,4-benzpyrene. For this reason, we have checked the optical density of concentration from 0.5 to 10 per cubic centimeter in a mixture of 90 percent cyclohexane and 10 percent benzene. A 5 percent cubic centimeter solution was used as standard (fig. 7). Absorption curve gives a prominent band (its maximum being at 3,045 A.), surrounded by 2 minimum

bands at 3,755 and 3,990 A. C, B, A represents three principal points of standard and C^1, R^1, and A^1 of the unknown. Abscissa passing through C and C^1 intersects the BA and B^1A^1 at the height of H and H^1. The concentration of BP then is calculated, as 3,4-benzpyrene equivalent, by means of th following formula

$$\frac{q=5C^1H^1}{CH}$$

In table II, the results of such determinations and the corresponding combustion temperatures have been exposed.

1. Paper of normal combustibility produces approximately 0.7γ of BP per gram of burned paper, and 100 cigarettes give 3γ. Paper of feeble combustibility number nine, produces three times less than paper treated with palladium.

2. Ammonium sulfamate reduces the quantity of BP formed by combustion at least 97 percent. This reduction is not parallel to a lowering of combustion temperature.

3. Quantity of BP formed from 100 cigarettes No. 1 is in order of 1.2γ. As in the case of cigarette paper No. 1, cigarette paper No. 3 produces very similar amounts of BP, corroborating that the combustion of a cigarette produces three times less BP than the amount of BP formed by the paper it contains.

DISCUSSION

Chromatographic analysis of the tars of paper or cigarettes have previously been done by several authors. So far, the presence of a dozen of more or less condensed hydrocarbons in the neutral fraction of the tar has been shown, among which 3,4-benzpyrene deserves special consideration due to its high carcinogenic potency. This piece of work is not original in its intentions. It has benefited the perfection in techniques leading chromatographic analysis, and particularly fluorescence instruments, by means of which our results escape certain criticism addressed to our predecessors. Those results will be discussed in three parts: Identity of BP and 3,4-benzpyrene; comparison of quantities formed from paper alone and from whole cigarettes; the reduction with ammonia sulfamate.

A. Is isolated hydrocarbon BP, 3,4-benzpyrene?

(*a*) Regardless of how much care has been taken, it is not sure that all the time one single hydrocarbon is chromatographed. Solution submitted to optical studies contains, even under most favorable conditions, besides the predominating hydrocarbon, other hydrocarbons in lesser concentrations. Nevertheless, three curves reproduced here, that we have mainly encountered after other numerous combustion and extractions, are only attributable to the fluorescence of a unique hydrocarbon (BP), characteristics of which might be somewhat modified by the presence of accessory traces, but not to a mixture. Mixtures manifest themselves by important fluctuations of intensity of the bands from sample to sample.

(*b*) The five fluorescent bands of 3,4-benzpyrene are steadily observed with relatively close intensitives to that of reference solution.

(*c*) The maximums of these bands reveal some displacement, signifying in fact of precision of our points at 3 A. These displacements might be interpreted by two different ways: Either BP is nothing but 3,4-benzpyrene, and its fluorescence is somewhat perturbed due to impurities, or BP is a distinct compound very close to 3,4-benzyprene like, for instance, a mono or polyalkyl derivative of it.

The fluorescence of certain derivatives of 3,4-benzpyrene has been studied by Berenblum and Schoental (4). Their spectra are characterized by displacements completely bathochromic, giving a more important band than that of principal band.

	Derivative	Displacement of the band of fluorescence (angstrom units)
5-methoxy	3, 4-benzpyrene	50
10-methoxy	do	100
8-methoxy	do	200
5, 10-dimethoxy	do	200
5, 8-dimethoxy	do	300
5-chloro	do	50
Dichloro	do	130
Trichloro	do	200

The study of absorption spectra (5) permits also to eliminate the following compounds:

	Derivative	Displacement of the band by adsorption 3845 (angstrom units)
2-methyl	3,4-benzpyrene	50
5-methyl	do	100
2-hydroxy	do	150
4-methoxy	do	100

Certainly there are other existing compounds so far not studied which give fluorescent and absorption spectra very close to 3,4-benzpyrene. A review of literature has shown us that an alkylated derivative giving the fluorescence of the nonsubstituted CH is 5-ethyl, 1,2-benzanthracene. Its bands do not separate from the corresponding bands of 1,2-benzanthracene with a bathochrome displacement of 10A. However all the bands are displaced 10A° toward the longer wavelength, and on the other hand all alkylations of this HC undergo to a bathochrome displacement as it is expected theoretically (5). Outside of this, table I shows us that:

(*a*) For a given extract, the displacements take place in all directions. For example with paper No. 2 the band at 4036 shifts toward shorter wave at 4030. and the band at 4267 shifts toward longer wave at 4282.

(*b*) For a given band displacement manifest by 2 different extracts in 2 directions. In consequence, a 10A° displacement of the five bands of the compound toward red does not change the agreement of experimental results.

Therefore we have arrived to the conclusion that the isolated HC, BP, from the extracts is 3,4-benzpyrene itself.

Observed spectral displacements should be due to the presence of impurities. The fact that absorption spectra does not agree with the 3,4-benzpyrene for the height at 3500A° is again due to the presence of these impurities which absorb UV rays at shorter wave lengths.

Naturally one cannot eliminate the possibility of dealing with a derivative too close to 3,4-benzpyrene, formally. However, such a substance has not been identified, studied, and its carcinogenic potentialities evaluated.

B. Respective contribution of paper and tobacco

The estimated amount of 3,4-benzpyrene in the neutral fraction of cigarette tar by different authors agrees satisfactorily with each other. Commis et al. (6) found 1.1γ for cigarettes; Neukomm stated (7) 2.2$\gamma \pm 0.12\gamma$ for 100 cigarettes. Our studies on French cigarettes No. 1 gives 1.2γ per 100 cigarettes.

On the other hand the results regarding the contribution of paper have been divergent. Cooper et al. (8) burning cigarettes in which tobacco was replaced by shredded paper estimated the contribution of paper alone as 0.5γ-benzpyrene for 100 normal cigarettes. Wynder et al. (8) could not detect 3,4-benzpyrene upon smouldering of paper. Burning the paper in a model as described by Alvord and Cardon which provides a combustion in a less oxygenated atmosphere at a slow rate we have been able to confirm the results of Alvord and Cardon. The quantity of pyraformed 3,4-benzpyrene is appreciably high for normal America and French papers and from each 300 grams of paper burned approximately 250γ 3,4-benzpyrene is obtained. This is equivalent to 3γ for the paper wrapping of 100 cigarettes.

INHIBITORY ACTION OF AMMONIA SULFAMATE

It is convenient, at this moment, to discuss the fact pointed out by Alvord and Cardon and confirmed by us that some ammonia salts, sulfamate in particular, reduces the amount of BP formed by the paper under our combustion conditions considerably. According to our experiments, the amount of benzpyrene falls down to 3 percent of that in treated paper.

Evidently, for chemical reasons, a destruction already formed benzpyrene cannot be called upon. Extinguishing effect of sulfamate as noticed during the burning of paper cannot be accounted for the lessening of 3,4-benzpyrene. It is quite probably, on the other hand, that an inhibition of formation of poly condensed hydrocarbon from chains of cellulose, and more generally from different constituent polyoses of the paper. It could be considered—and this suggests

interesting experiences—that at a certain level of polycondensation of aliphatic chains a blockage is exerted by the ammoniacal products; by blocking the intermediary reactive functions the condensation leading to the formation of 3,4-benzpyrene might be arrested. A support to this hypothesis can be found if one considers the high quantity of anthracene in tars derived from treated papers in which the benzpyrene is deprived (fig. 6).

It is also conceivable that ammoniacal compounds orient the pyro-polycondensation toward the nitrogenous polyheterocyclic compounds, which, by their nature, will not follow the polycyclic hydrocarbons during chromatography. Comparative studies of alkalin fractions of the tars obtained from normal and ammonia sulfamate treated papers will confirm or invalidate this second hypothesis.

Finally another hypothesis exists, although difficult to explain with our present knowledge. We have previously suggested that superoxygenation hinders the pyroformation of 3,4-benzpyrene whereas the combustion in a relative anoxic atmosphere enhances its formation. In this reaction oxygen acts by an unknown physico-chemical mechanism which should also be equally provided by nitrogen. Ammonium salts act, also, as nitrogen donors causing a hyper-nitrogenation which is also sinister than hyperoxygenation to the formation of 3,4-benzpyrene. To support of this hypothesis two of the more active products, ammonium sulfamate and urea sulfate, could be mentioned both of which contain oxygen, and they liberate nitrogen instead of ammonia while destroyed by heat, whereas the non-oxygen salts deliver more and they are less effective in prevention of benzpyrene formation. Therefore the inhibitory effect of ammonical products becomes more powerful as oxygen donors rather than nitrogen suppliers.

It could also be considered that heating of ammonium sulfamate entails the formation of low oxygen nitrogenous compounds like N_2O or NO. It is classical that these oxydes could play an inhibitory role in the Rice-Herzfeld type chain reactions in the pyrolysis of hydrocarbons; and probably the same is true for polyosic (cellulosic) chains.

These considerations bring us back to the respective contribution of paper and tobacco to the 3,4-benzpyrene content of the cigarettes. The outstanding question, evidently, consists of knowing how to compare the tars formed in the paper alone and in the cigarettes under the same conditions of temperature and oxygenation. Some fine physico-chemical measurements as it is followed by one of us (Cuzin) will permit us to answer this question.

Considering our actual experimental conditions, combustion of 100 cigarettes produces 1.2γ of 3,4-benzpyrene, however the combustion of paper wrappings of these cigarettes alone gives 3γ. Maybe tobacco contains some polyosic substances which generates 3,4-benzpyrene in paper by pyrolysis.

It is also possible that tobacco has some inhibitors for its own polycondensation and for the paper which envelopes it; these inhibitors might be multiple nitrogenous compounds formed during thermal reaction, rather than being in the tobacco.

SUMMARY

1. In the neutral fraction of the tar derived from combustion of cigarettes or from cigarette papers burned under less oxygenated conditions, a hydrocarbon is isolated which resembles to 3,4-benzpyrene by its fluorescence and absorption spectral characteristics.

2. Spectroscopic quantative dosages in different products are as follows:

Cigarette 1.2γ per 100 cigarettes

Paper of normal combustibility: 250γ per 300 gross of paper, 3γ being the quantity determined for the wrappers of 100 cigarettes.

Paper of low combustibility: 70γ per 300 grams.

Normal paper treated with palladium: 70 gamma per 300 gram.

Normal paper treated with ammonium sulfamate, less than 5 gamma per 300 grams.

3. Treatment of the paper with ammonium sulfamate diminishes the pyroformation of 3,4-benzpyrene considerably. Several mechanisms have been considered and discussed to explain the action of ammonium sulfamate.

4. This type of diminution by the presence of preformed or pyroformed nitrogenous products in tobacco causes less, 3,4-benzpyrene content of whole cigarettes.

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TABLE I.—*Analysis of fluorescence and absorption spectral curves—The spectral position of the peaks of the bands*

	Fluorescence					Absorption		
3, 4 benzpyrene	3950	4036	4087	4267	4540	3645	3845	4035
Cigarettes No. 1	3960	4035	4085	4277	4545	3630	3840	E
American paper No. 2	E	4030	E	4282	4537	3635	3840	E
French paper No. 3	E	4045	4095*	4282	4544	3635	3835	4030
French paper No. 4	3960	4040	4095*	4282	4544	E	3830	E
French paper No. 5 treated with palladium	3960	4038	4090	4272	4545	E	E	E
Paper No. 3 treated with ammonium sulfamate	----	(4010)	(4095)	----	----	(3575)	(3760)	----

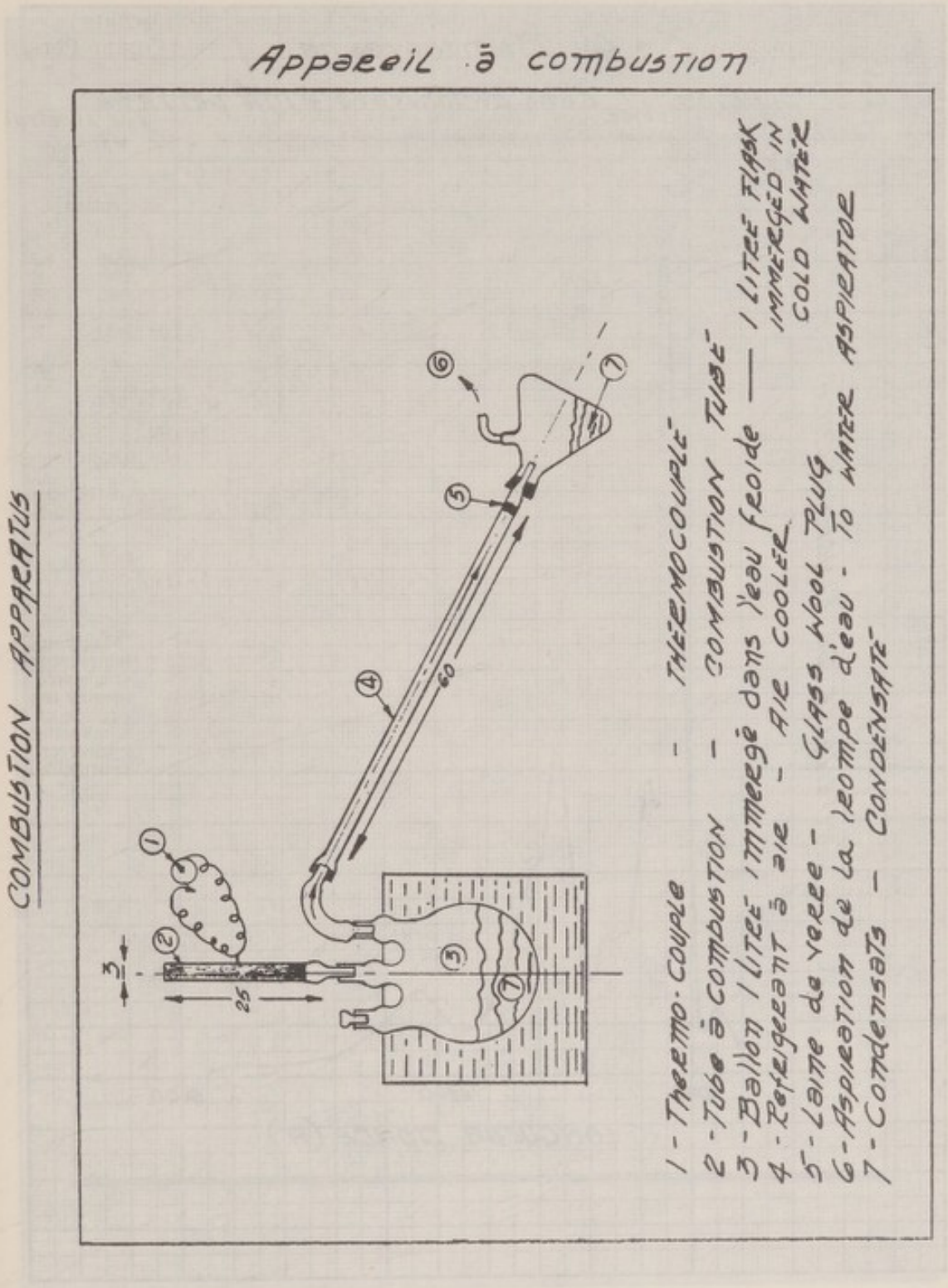
E indicates a plateau formation. Figures between ( ) indicate the other compounds like anthracene and Raman band of cyclohexane (4095).

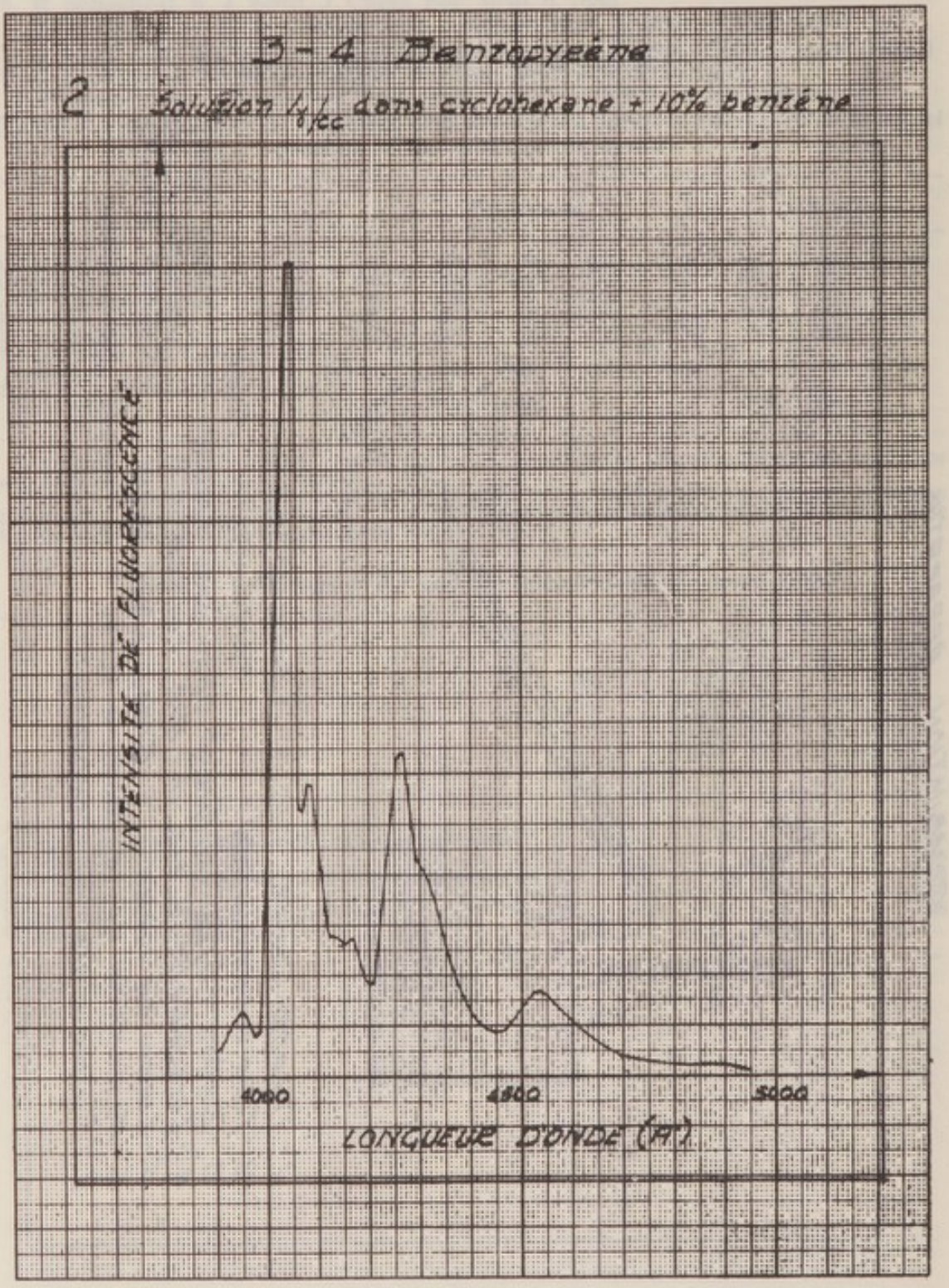
The bands marked with an * at 4095 are mainly due to BP changes masked by the principles of the Raman rays situated almost at the same region. This ray will appear also in the graphs of ammonium sulfamate treated papers following the disappearance of BP.

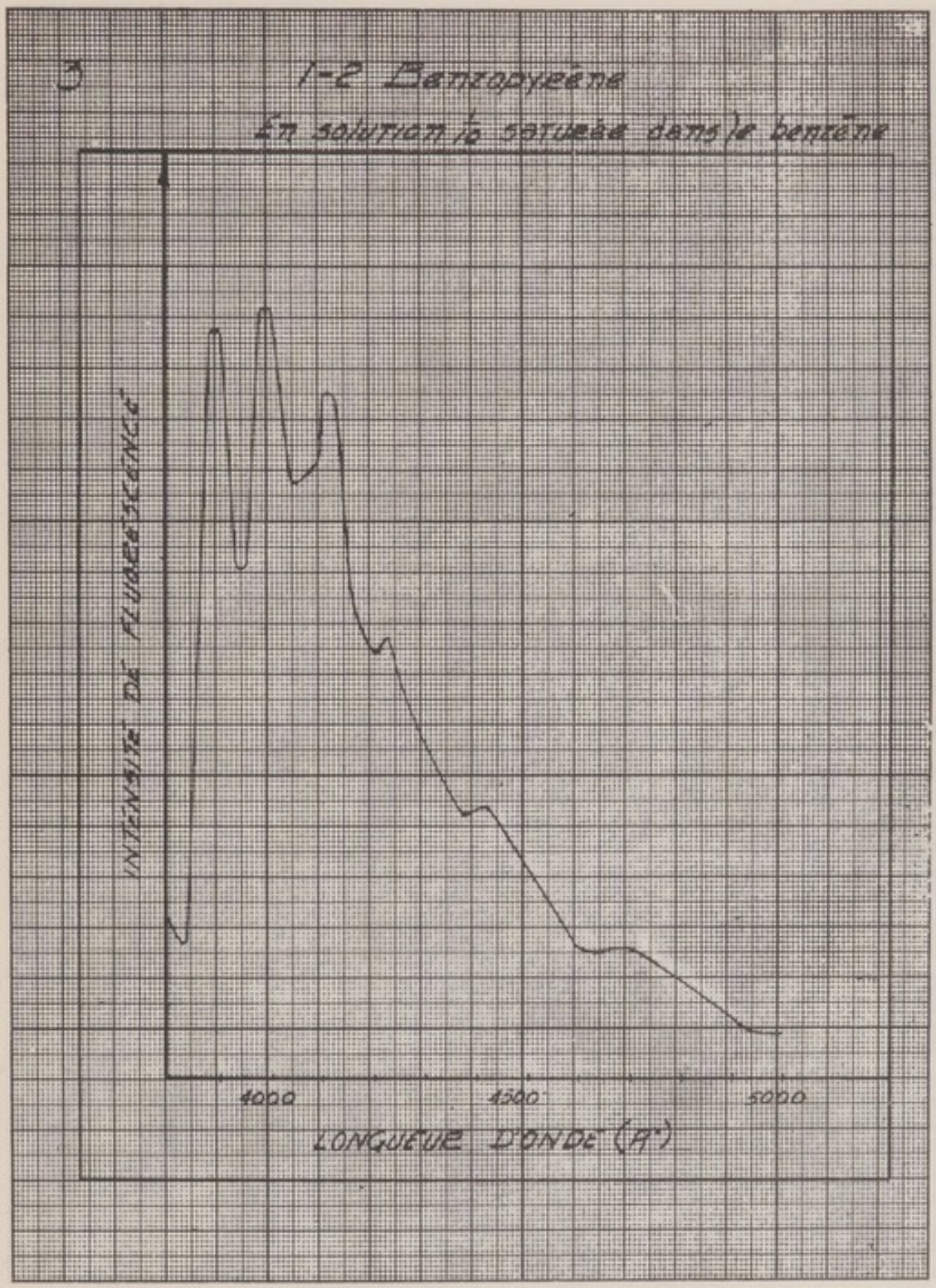
TABLE II.—*Dosage of BP and the temperature of combustion*

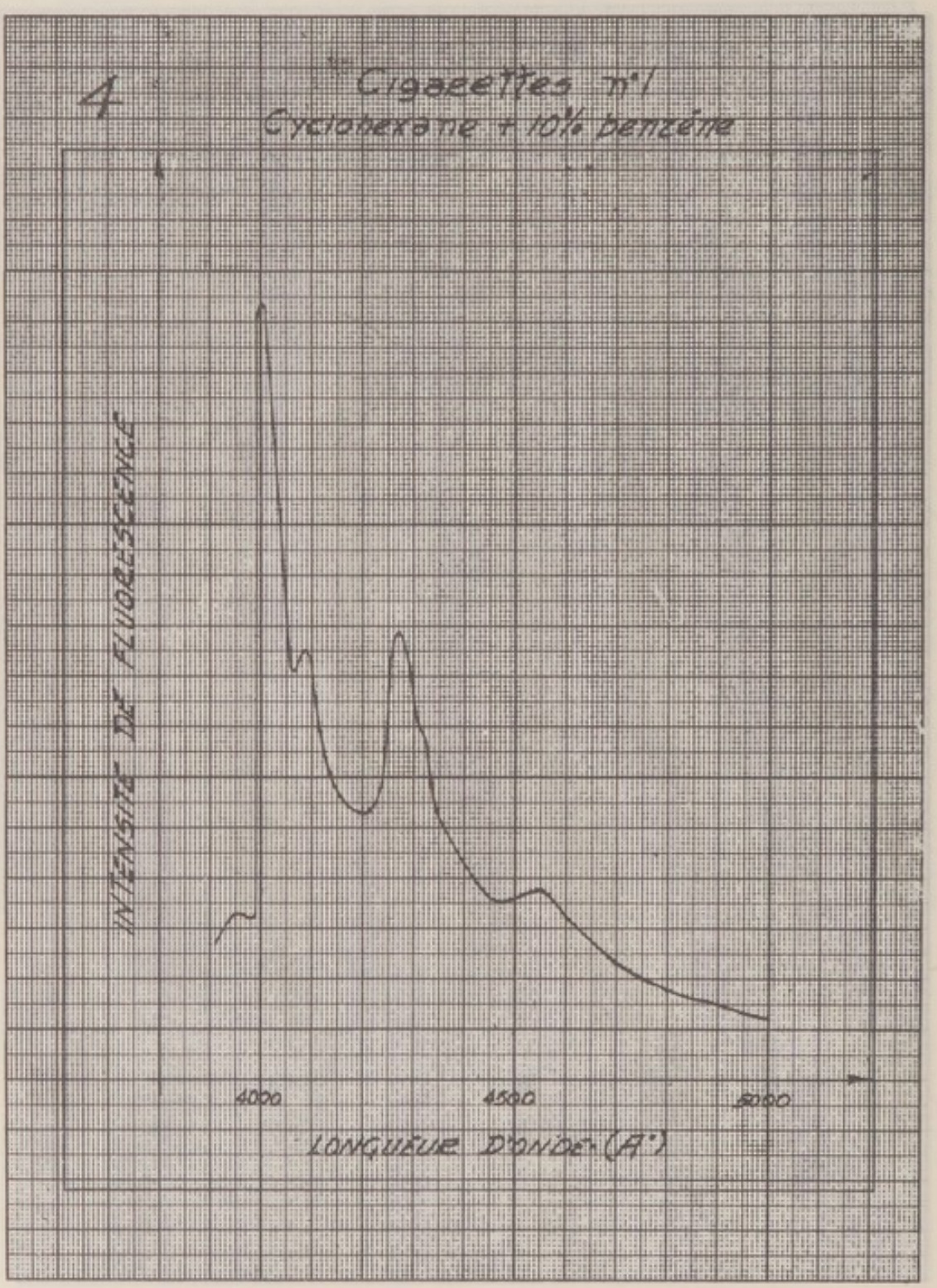
Type of paper	Quantity of BP measured in the tars produced by combustion of 300 grams of paper (γ)	Maximum temperature registered during the course of combustion (degrees centigrade)
American paper No. 2	200	884
American paper No. 2 treated with ammonium sulfamate	(1)	668
French paper No. 3	250	898
French paper No. 3 treated with ammonium sulfamate	(1)	860
French paper No. 4	70	410
French paper No. 3 treated with palladium	70	626
Cigarette No. 1 (1,040 cigarettes burned)	12, 2	----

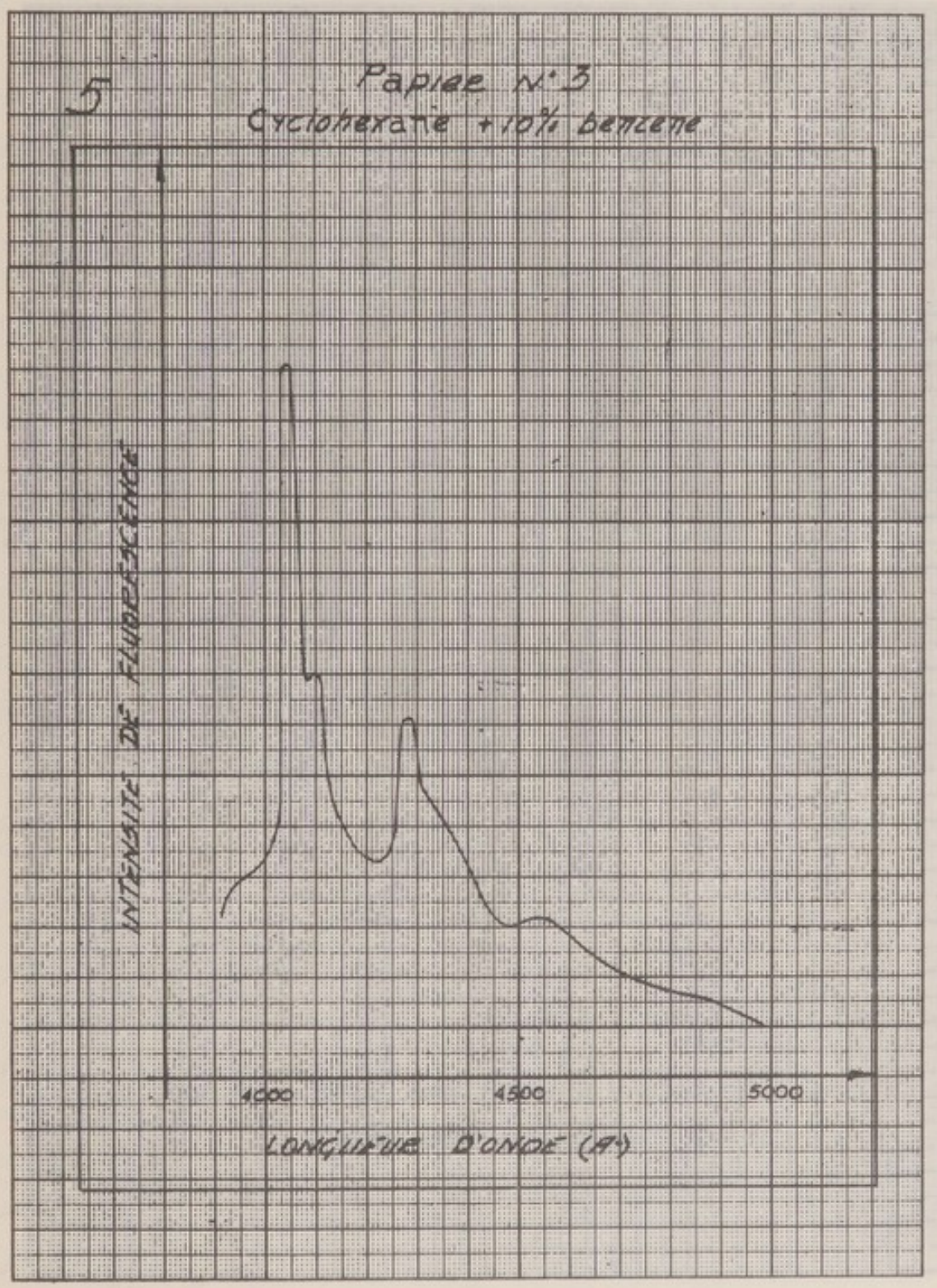
[1] Less than 5.

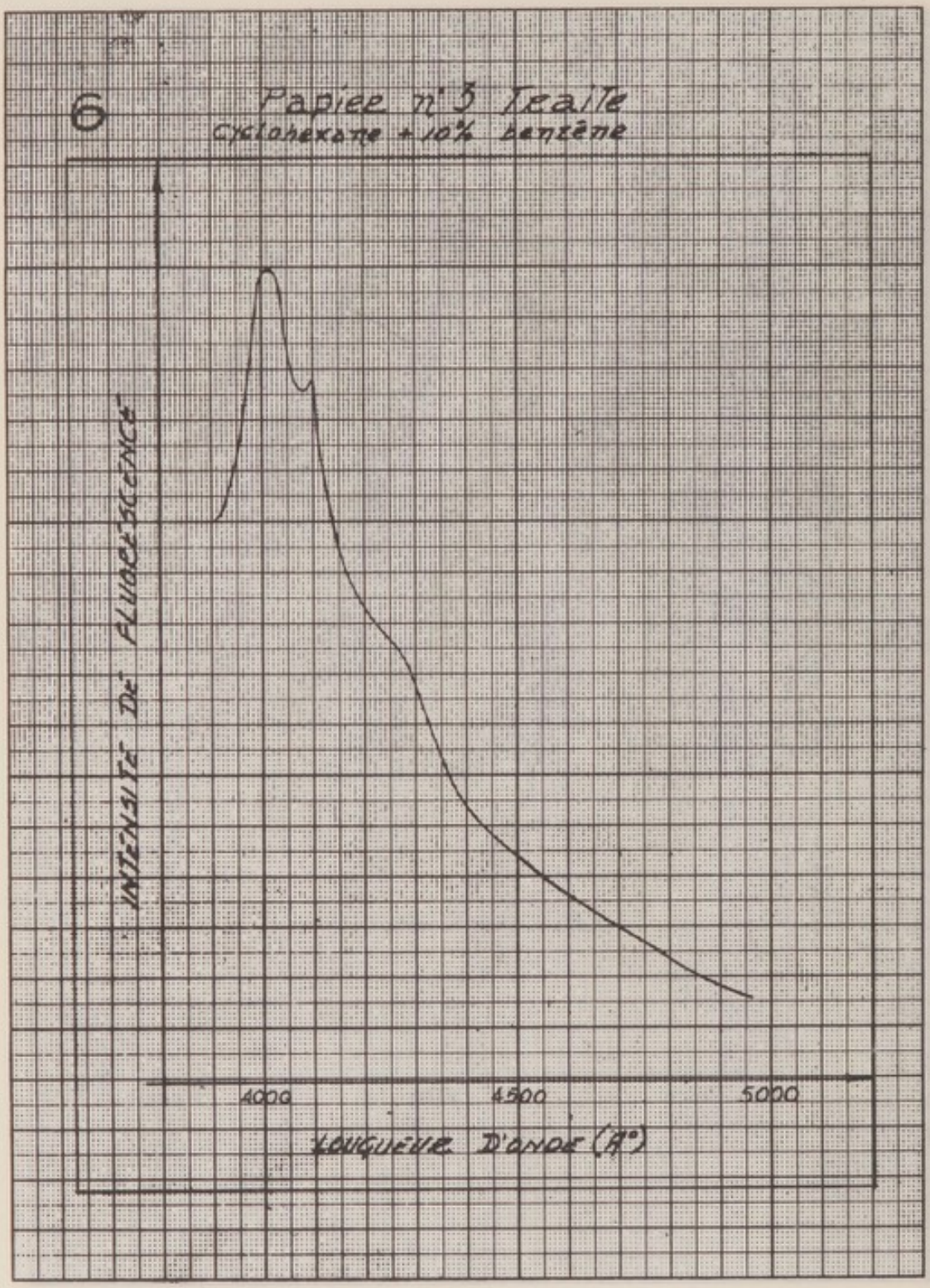

Appareil à combustion
COMBUSTION APPARATUS
1 - Thermo-couple - Thermocouple
2 - Tube à combustion - Combustion Tube
3 - Ballon 1 litre immergé dans l'eau froide — 1 litre flask immersed in cold water
4 - Refrigerant à air - Air cooler
5 - Laine de verre - Glass wool plug
6 - Aspiration de la trompe d'eau - To water aspirator
7 - Condensats - Condensate
60
25
3


3 - 4 Benzopyrène
2 Solution 1γ/cc dans cyclohexane + 10% benzène
INTENSITÉ DE FLUORESCENCE
4000
4500
5000
LONGUEUR D'ONDE (Å)


3
1-2 Benzopyrène
En solution 1/2 saturée dans le benzène
INTENSITÉ DE FLUORESCENCE
4000
4500
5000
LONGUEUR D'ONDE (Å°)


4
Cigarettes n°1
Cyclohexane + 10% benzène
INTENSITÉ DE FLUORESCENCE
4000
4500
5000
LONGUEUR D'ONDE (A°)


5
Papier N° 3
Cyclohexane + 10% benzène
INTENSITÉ DE FLUORESCENCE
4000
4500
5000
LONGUEUR D'ONDE (Å)


6
Papier n° 3 Traité
Cyclohexane + 10% benzène
INTENSITÉ DE FLUORESCENCE
4000
4500
5000
LONGUEUR D'ONDE (Å°)

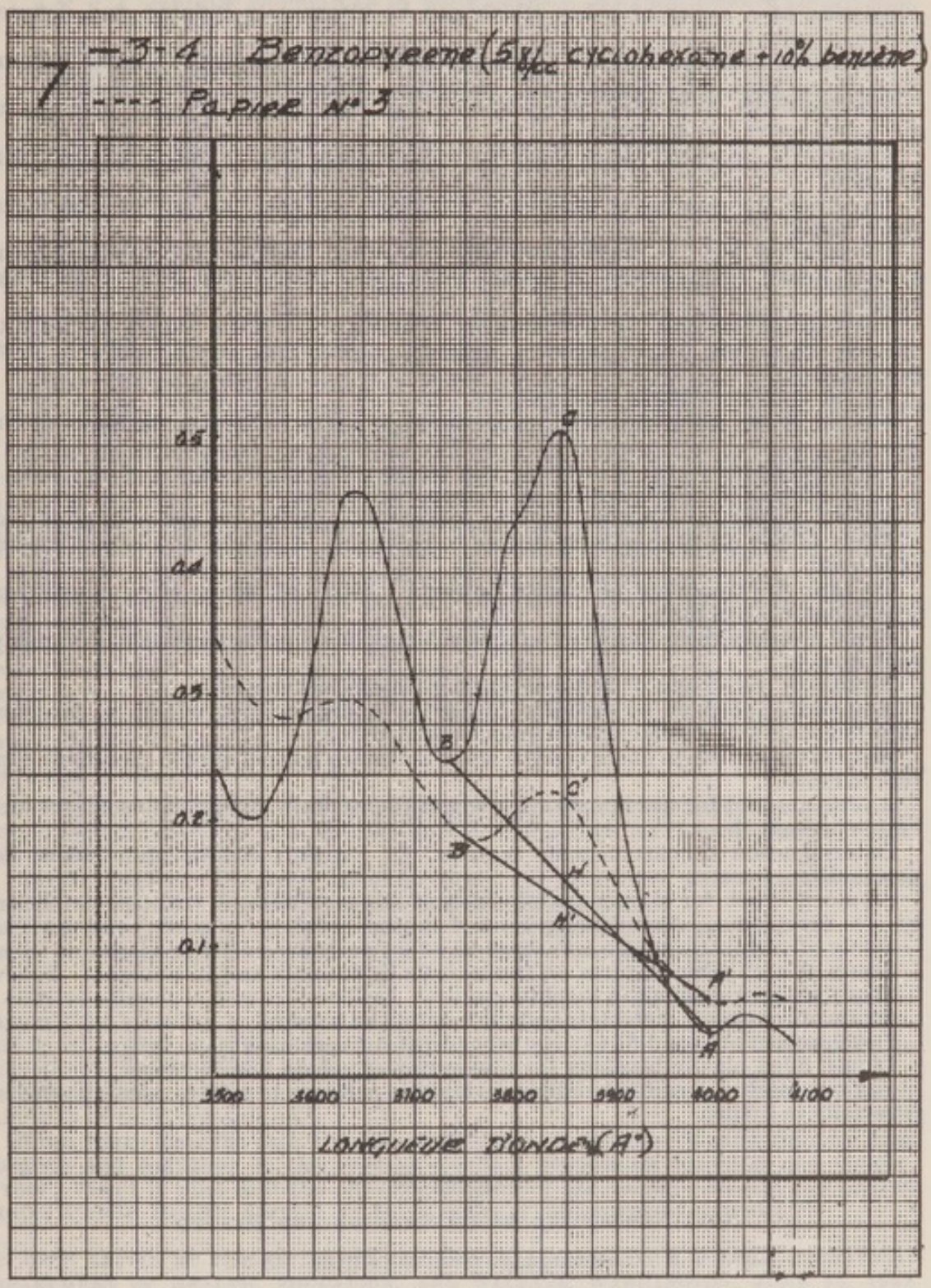

1 — 3-4 Benzopyrene (5 γ/cc cyclohexane + 10% benzene)
---- Papier n° 3
0.5
0.4
0.3
0.2
0.1
3900
4000
4100
LONGUEURS D'ONDES (Å°)

EXHIBIT 24

LIST OF MEDICAL DEVELOPMENT PROJECTS COMPLETED BY RAND DEVELOPMENT CORP.

Defibrillator—a massaging device for resuscitation in the operating room.

Alternating pressure-point mattress for prevention of decubiti—a mattress consisting of a series of tubes which can be alternately inflated and deflated automatically.

Automatic tourniquet device for applying controlled pressure on three extremities at one time and automatically changing. Has use in pulmonary edema.

Automatically controlled pump system for maintaining arterial pressure or for use in heart catheterization.

Electrically operated wheel chair run from storage battery and controlled from joystick.

Treadmill for accurately producing any given workload on patient and measuring same.

Hibernation bed. A thermostatically controlled mattress for producing any environmental temperature on patient undergoing hibernation with chlorpromazine.

Surgical instruments for momentarily shutting off arterial supply to brain to prevent thrombus from entering cerebral circulation during mitral valve operation.

Finger knife for cutting mitral valves.

Special clamp mechanism for anastomosis of aorta to the venous sinus of the heart.

Oscilloscope portrayal of heart sounds, electrocardiogram, etc.

Utilization of TV for operating-room demonstrations.

Special diaper for adult patients to prevent skin irritation and soiling of bed.

Study of bed clothing with reference to reduction of weight and better control of warmth.

Oil tank for treatment of burned patients. Study of means of elimination of waste materials in tank.

Blood pressure recording device for measuring blood pressures in small animals, such as rats.

Automatic recording of sedimentation rate photographically.

Automatic injection of atropine by spring-loaded syringe which on pressure injects needle and predetermined amount of atropine.

Respirator for use in chest operations to maintain normal carbon monoxide content in blood.

A pressure suit for use in operating rooms to maintain any given blood pressure and to prevent shock.

Determination of carcinogenic material in cigarette smoke and means for elimination of same.

Stomach brush for simple examination of stomach mucosa with special reference to carcinoma. Also development of similar brushes for other body mucosa.

Study of ultrasonics with reference to effect on cancer cells in vitro and vivo.

Study of high temperatures on tumor cells in animals and in humans.

Study of carcinogenisis in amphibians.

Study of pyrogenic materials and their action on tumors.

Development of liquid-filled plastic headrest for operating room tables.

Liquid filled plastic blankets for prevention of serum loss in burns.

Various mechanisms for handling bedridden patients to relieve nursing efforts, especially in lifting.

EXHIBIT 25

NORR NEWSLETTER ABOUT SMOKING AND HEALTH

A message from the editor

(Vol. 5, No. 1, Double Issue, June-July 1957)

FINAL REPORT OF MASSIVE SURVEY BY AMERICAN CANCER SOCIETY: DEATHS FROM LUNG CANCER 1,000 PERCENT HIGHER AMONG MEN WHO SMOKE CIGARETTES COMPARED TO NONSMOKERS

When the American Cancer Society presented its fourth and final study of 187,783 men between the ages of 50 to 70, at the annual meeting in New York early

in June of the American Medical Association, many doctors in attendance quit smoking outright, according to Newsweek. "A spectacular association has now been established between smoking and lung cancer and heart disease," declared Drs. Hammond and Horn, the authors of the report. Some of their principal findings:

Heavy cigarette smokers (2 or more packs a day) die 7 years before they would if they did not smoke.

Among cigarette smokers deaths from lung cancer are 1,000 percent higher than among nonsmokers.

Cigarette smoking raises the risk of fatal coronary heart attack by more than 50 percent.

Quitting smoking lowers the risk of premature death—even among heavy smokers who have stopped for more than 10 years the death rates were only half as large as among men who continued smoking.

Deaths from lung diseases other than cancer are 3 times as high among cigarette smokers as among nonsmokers; from pneumonia and influenza almost 4 times as high.

Death rates indicate that cigarette smokers are 7 times as susceptible as nonsmokers to cancers of the esophagus, larynx, mouth, tongue, and back of the throat.

A significant tie exists between cigarette smoking and cancer in other sites—the pancreas, death rates up 50 percent; the kidneys, death rates up 58 percent; the stomach up 51 percent; the prostate 75 percent; the bladder, 117 percent, the liver and gall bladder, 352 percent.

Of the approximately 188,000 men who had been followed up, 11,870 deaths occurred during the 44-month period of the study. Of these, 57 percent had a hisory of cigarette smoking. In the entire study only 4 nonsmokers, by microscopic examination, were shown to have died of primary lung cancer. Of the 51 who died of stomach ulcer, every one had been a smoker, mainly of cigarettes.

More than 5,200 deaths (45 percent), of the men who died during this study, were due to coronary artery disease. The death rate increased steadily with the daily consumption of cigarettes, it was found. Deaths described as due to myocarditis, cardiac failure, cardiac degeneration, and similar terms showed "a positive association" with cigarette smoking. The death rate for pipe and cigar smokers was only slightly higher than for nonsmokers. It was estimated by the researchers that 31,000 people will die this year in the United States from lung cancer, 26,000 of them men.

Of things to come

In the cigarette-health issue, as predicted in the "confidential advice on coming developments" given by the Nation's foremost business service—Babson's Washington Forecast, June 10, 1957:

"*Smoke hazard.*—Cigarette manufacturers are genuinely worried about latest medical survey * * * indicating smoking is a life shortener.

"This helps to pile up evidence against advisability of smoking * * * and tends to weaken tobacco industry's argument that nothing has really been proved.

Our prediction: Future medical reports will give further confirmation to theory that smoking is harmful to health. Percentage of smokers to total population will peak out, then start to diminish. Later on, medical education in schools will build up case against smoking.

"Twenty years from now, we believe smokers will be a relatively small minority of total population."

Three years ago, the late Dr. Evarts A. Graham, internationally famous lung surgeon and coauthor of the Wynder-Graham reports on smoking and cancer, wrote: "Isn't it time that the official guardian of the people's health, the United States Public Health Service, at least make a statement of warning?"

AN OPEN LETTER BY THE EDITOR, TO THE HONORABLE MARION B. FOLSOM, SECRETARY, DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, WASHINGTON, JUNE 25, 1957

DEAR MR. SECRETARY: Without any intention to be captious or critical, may I make public inquiry, in my capacity as editor and publisher of the Norr Newsletter about smoking and health, as to the present position of the Department of Health, Education, and Welfare on the ominous findings regarding smoking and cancer and smoking and death from coronary disease?

The British Ministry of Health, without awaiting further evidence, has announced in Parliament that "an incontrovertible association between cigarette

smoking and the incidence of lung cancer has been established." The Ministry of Education has cited the facts in an official pamphlet urging parents and teachers to warn British youth. In Sweden the tobacco monopoly has ceased all advertising and is aiding doctors and teachers in a nationwide campaign to stop smoking among schoolchildren. In Holland, following the report handed down by a commission appointed by the Director-General of Public Health, the Government is preparing energetic plans to protect youth from the menace of death and disease that often results from habitual smoking.

Nevertheless, an inquiry addressed by this editor to the Surgeon-General of the United States Public Health Service, before your incumbency into office and your appointment of his present successor, brought the incredible statement that unless a harmful association between smoking and health "could be established unequivocally through scientific proof," the Public Health Service would not be in a position to declare that smoking generally is a public-health problem.

As to "unequivocal proof," what Dr. Paul D. White, heart consultant to President Eisenhower, has remarked about coronary disease—the greatest killer of them all—certainly applies to the smoking-health problem. He has said: "Perhaps some of us shouldn't wait for scientific answers. We may be dead by the time the scientific answers come along."

You will agree, Mr. Secretary, that a no more authoritative medical journal is published in our country than the New England Journal of Medicine. On June 6 of this year, it published an editorial on smoking and health. "Sufficient evidence," it found, "has been produced from various sources to indict cigarette smoking as at least one of the factors involved in lung cancer." With so many human lives at stake, the Journal declared, "the agent accused cannot stand innocent until declared guilty. It must be considered as guilty on the evidence until proved innocent."

Yet in the face of the admission recorded in the industry's own advertising that "1 out of 3," approximately 25 million smokers, were deleteriously affected by unfiltered cigarettes, in the face of universal medical opinion that smoking is injurious to youth, in the face of the mountainous findings that excessive smoking harms all bodily systems, the United States Public Health Service—an office often referred to as to the "people's physician"—made the shameful declaration that it could not even participate in an educational program that would at least alert our youth to the terrible risks of addictive smoking.

Since the foregoing declaration, the United States Public Health has before it the findings this year of the Memorial Center for Cancer and Allied Disease in New York. This greatest and largest cancer research institution in the world today reached the conclusion that "possibly 80 percent of deaths from lung cancer could be prevented"—if the cancer-producing activity could be eliminated.

It has before it the report published in March of this year by eminent scientists, organized as a Study Group on Smoking and Health by the American Heart Association and the American Cancer Society, which declares that "the sum total of scientific evidence establishes beyond reasonable doubt that cigarette smoking is a causative factor in the rapidly increasing incidence of human epidermoid cancer of the lung," and that the evidence of a cause-effect relationship is adequate for considering the initiation of public-health measures.

Your Public Health Service has before it the conclusive proof presented this month by the American Cancer Society at the annual meeting of the American Medical Association that deaths from lung cancer are 1000 percent higher among men who smoke cigarettes than among nonsmokers, that cigarette smoking raises the chance of a fatal coronary heart attack by at least 50 percent, that heavy cigarette smokers die 7 years before they would if they did not smoke.

The Public Health Service long has had before it the evidence that millions of our youth are being tricked into cigarette addiction by the false promise of "health protection" which filter tips hold out.

This editor was the first to expose this heartless fraud in his article, The Great Filter-tip Hoax in the Christian Herald more than 9 months ago, September 1956.

At this very writing, Reader's Digest, in its July 1957 issue, reports to the Nation scientific proof of my charges. It discloses that laboratory determination of the tar and nicotine in the mainstream smoke of popular brands shows that some of the leading filter tips actually contain more tar, more nicotine than nonfiltered cigarettes by the same makers.

It is hard to believe that such deliberate flaunting of medical findings, such frightening irresponsibility toward the life and death problem before the health community of the Nation, would have occurred if the United States Public Health Service had reported the facts to the public.

It has been said that the Holy Roman Empire was not holy, nor Roman, nor empire. It would be sad if the fathers and mothers of the Nation should come to feel that the Department of Health, Education, and Welfare represents neither health, nor education, nor welfare in the urgent call to protect our youth from cigarette addiction.

ROY NORR,
Editor, Norr Newsletter about Smoking and Health.

A research worker writes us

"Animal experimentation is the very basis of modern medical science, but not for tobacco scientists who find nothing in statistical, clinical, and experimental findings that associate smoking with cancer." Reminds me of a medical autocrat who expostulated with the great Pasteur.

" 'Bah, you're using rabbits whose resistance is naturally low,' he cried. 'The rabbit is a melancholy animal to whom life is a burden and who only asks to leave it.' "

EXTRA! BRITISH GOVERNMENT TAKES UNEQUIVOCAL ACTION FOR AN EDUCATIONAL CAMPAIGN AGAINST PERIL OF SMOKING

Just 2 days after we had made an appeal to our own health guardians to set our public-health apparatus in motion so that youth may be saved from the menace of cigarette addiction, the British Government decided to take all-out action to warn the British people of the smoking peril. It had been content hitherto to stand upon the statement that an "incontrovertible association" had been established between smoking and cancer.

But on June 27 J. K. Vaughan-Morgan, Parliamentary Secretary of the Ministry of Health, told the House of Commons that the latest report of the British Medical Research Council concluded that cigarette smoking is a direct cause of lung cancer. The Research Council is Britain's highest authority on medical matters. It allocates both Government and private research funds under the authority of the Privy Council. The Council's findings were based on 19 investigations during the last 8 years made in Britain, United States, West Germany, the Netherlands, Switzerland, Norway, and Finland.

Mr. Vaughan told the Commons that the "Government feels that it is right to assure that this latest authoritative opinion is brought effectively to public notice that everyone may know the risks involved in smoking." Among the steps that have been or are to be taken:

1. The immediate distribution of the Council's report to local health authorities by the Ministry of Health;
2. Thorough publicization of the report by the state-run British Broadcasting Corp.;
3. Local health authorities—"responsible for health education as a means of prevention"—to be asked to take appropriate steps to inform the general public.

The Council's report declared that 1 out of every 8 heavy cigarette smokers would die of lung cancer, whereas only 1 in 300 nonsmokers would die of the disease. It found that men who stopped smoking even as late as their early forties would reduce their chances of getting lung cancer by at least one-half.

The British Ministry of Education

Even before the publication of latest British medical research report, had taken steps to urge teachers and youth leaders to do what they could to stop young people from starting the cigarette habit. In the Ministry's Health Educational Pamphlet (No. 31) under the chapter, Drugs, Alcohol and Tobacco, it was declared:

"Adults can help younger people best by their own example, by not encouraging them to feel that to smoke is to be grown up; more generally, too, they can help to make it clear that some control or restraint is not the same as submission to external authority, but represents something positive that comes from within."

The Government health pamphlet gave the latest information then available with regard to the findings about smoking and cancer. It declared that the British Medical Research Council had stressed that the fact that a casual agent had not yet been recognized should not be allowed to obscure the finding that there is statistically an incontrovertible association between cigarette smoking and the incidence of lung cancer.

"The evidence that led to the passing of legislation concerning benzol, analine and a host of other dangerous industrial solvents is of exactly the same type as

that which incriminates cigarette smoking as a factor in the genesis of carcinoma of the bronchus." British Journal of Clinical Practice.

THE FILTER-TIP CIGARETTE: THE SALVATION OF THE TOBACCO INDUSTRY

* * * and, it might be added, the greatest crime ever committed against public health by the cigarette cartel * * * is treated in Reader's Digest (July issue) under the title, "The Facts Behind Filter-Tip Cigarettes." It is "must reading," for the complete laboratory findings it gives about filter-tip brands. The essential facts were first revealed in the NORR Newsletter many months ago and in the editor's article, "The Great Filter Tip Hoax," published in the Christian Herald, September 1956.

We said that helpless smokers were being led to increase their death risks from cancer by the fake claims of "health protection" proclaimed in filter-tip advertising.

We said that the amount of nicotine and tars in some of the filter-tip brands was decidedly higher than in some of the regular brands.

We said the leading makers were using harsher, more nicotine and tar-filled tobaccos for their filter, so as to make the "real tobacco taste" come through to the smoker.

The Digest reports a survey which shows that 70 percent of those who have switched from regular to filter-tip cigarettes said they had done so for "health protection."

The Digest reveals that the smoker gets from 5 to 18 percent more tars, from 29 to 33 percent more nicotine from some of the most popular filter-tips.

The Digest finds that "not only did they adopt filters which, in most cases, are little more than mouthpieces; they also began loading their brands with heavier tobaccos."

In other words, without a single Government agency to raise a warning finger, leaders in the industry are deliberately, studiedly using the cruel hoax of filter tips to maintain their hold on the cigarette addict, despite 25,000 deaths a year from smokers lung cancer and many thousands more from coronary disease. "The filter-tip cigarette has been the salvation of the tobacco industry," Reader's Digest says.

As in goats, so in men

A paper on the use of nicotine in immobilizing semiwild goats recently appeared in the Journal of the American Veterinary Medical Association. The specific action of nicotine on these animals: "Within 30 to 90 seconds after drug administration the goats showed signs of apprehensiveness and excitement. Some remained standing and bleated incessantly. Others bolted from 10 to 15 yards and then fell, and a few simply laid down, and appeared to await paralysis."

From Circulation (published by the American Heart Association, February 1957): "The primary action of nicotine consists of a transient stimulation, followed by depression of all sympathetic and parasympathetic ganglia."

Cigarette prices go up 1 cent a pack * * * as up go deaths from cancer and cardiovascular disease. Now more millions can be poured into gigantic TV and other advertising campaigns to drown out the warning voice of science.

Soothing the smoker into lung cancer

You would think, wouldn't you, now that the hucksters have found a way to give the cigarette addict "smooth, smooth, smooth smoking," now that smoking

is "so mild, so cool, so sweet" with the new filter-tip cigarettes, now that the smoker can "enjoy the gentle pleasure" of "fine tobacco," the drug vendors of the cigarette industry would rest on their oars.

But no. For the ill or well, the diseased or healthy, nothing must be left undone to mask the truly irritating effects of even the "best" tobacco. So the industry is plunging into a menthol mist. The fifth mentholated cigarette has now been placed on the market. For, as one trade commentator writes, "menthol cigarettes enjoy their strongest sales from persons suffering from a cold"—and apparently it would be just too bad if cold victims stopped smoking even temporarily * * * thus avoiding smoker's cough, chronic bronchitis, and the possibility of respiratory cancer eventually.

SOME JOTTINGS AND THOUGHTS ON THE TOBACCO HABIT

An estimated 300,000 Americans died from coronary artery disease during 1956. How many could have been saved from premature death by the simple step of giving up smoking?

Dr. Clarence Cook Little, chairman of the cigarette industry's "scientific board," has declared: "It cannot be said that smoking has been absolved from suspicion (with regard to the incidence of lung cancer) but neither have the charges that smoking has a role in lung causation been proved." Question: How much hotter should purgatory be made for the cigarette pushers who despite their acceptance of the fact that cigarettes have not been absolved from "suspicion," continue their monstrous campaign for cigarette addiction?

Federal and State tax receipts for tobacco products during 1956 nearly reached the $2,185 million mark. Plenty, one would think, for Government to help tobacco farmers prepare their land for other crops.

Time for fathers and mothers of the Nation to shake off their feeling of frustration with regard to tobacco advertising. Only 3,400 complaints about advertising of any kind are said to be received by the Federal Trade Commission as a yearly average. What would happen if 340,000 or 3,400,000 complaints would pour in on the FTC because of the sinister cigarette advertising?

Three out of four fatal air crashes have been found, after inquiry, to be due to "pilot error." How many such air disasters might have been caused by smoker-pilots who suffered temporary blindness, a spasm of the blood vessels in the eyes often caused by nicotine amblyopia?

Every year another 500,000 Americans are stricken by cancer. Every year another 250,000 die, including thousands who die from smoker's lung cancer who could have been saved by giving up smoking.

The tobacco industry has a gleam in its eye for the some 70 million babies that will have been born at the end of the 1940–60 period. And that's the challenge which our youth leaders must meet.

It's in prevention, not cure that our immediate hope lies in cancer control. The 10th progress report of the Sloan-Kettering Institute issued this summer says: "The harsh truth is that there is no cancer-curing drug right now * * *. No man can guarantee that this ugly killer's threat, now aimed at one in every four Americans, will be banished this year or next year or in 10 years."

Victor Borge, the fantastically successful entertainer in the theater and on television, tells this one in his memoirs recently published in the Saturday Evening Post:

Midway in one of his TV shows he was interrupted by an announcer. "Here, Victor, won't you have one of our smooth, mellow, tender smokes?"

"No, thanks," said Victor.

The announcer paled and abandoned his script.

"What?" he snapped. "Why not?"

"Because my doctor told me to quit," said Victor, coughing loudly.

He was never again invited to perform on that show.

Reader's Digest

December 1952

ARTICLES OF LASTING INTEREST • 31st YEAR OF PUBLICATION

The Real Meaning of Christmas . *Illustrated London News* 1
We're Selling America Short . . . . . *American Scholar* 4
Cancer by the Carton . . . . . *Christian Herald* 7
This, Too, Is Infidelity . . . . . *Your Life* 9
He Wanted to Fight for Unc . . . . . *Redbook* 12
Are We *All* Destined t . . *Charles Stevenson* 17
A Giant . . . Life 21
England . . . litan 25
We Ha . . .
. . . eports 29
. . . thly 32
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Pla
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. . . k 86
. . . s 89
Hero . . . y 91
Two Hu . . . Coal . . . e 96
What You Should Kn . . . s . . . .Coll's 100
Train to Genoa . . . . . .This Week 105
Every Dog a Gentleman . . . . . *Dickson Hartwell* 109
Germany's Lonely Jews . . . . . .*Look* 113
"Not Charity, But a Job" . . . . . .*Christian Advocate* 117
The Reindeer As He Really Is . . . *United Nations World* 121
Jungle Doctor . . . . . *Saturday Evening Post* 124
Network to Catch International Crooks . . . . *Cavalier* 128
The Splendor of December Skies . . *Donald Culross Peattie* 132
The Man Who Dreamt Winners . . *"Tell Me the Next One"* 136

Book Section Cyclone in Calico . . *Nina Brown Baker* 141

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Cancer by the Carton

Condensed from Christian Herald

Roy Norr

Recent medical researches on the relationship of smoking and lung cancer

Cancer by the Carton

Condensed from Christian Herald

Roy Norr

FOR THREE DECADES the medical controversy over the part played by smoking in the rise of bronchiogenic carcinoma, better known as cancer of the lung, has largely been kept from public notice. More than 26 years ago the late Dr. James Ewing, distinguished pathologist and leading spirit in the organization of the American Association for Cancer Research (now the American Cancer Society), pleaded for a public educational campaign.

"One may hardly aim to eliminate the tobacco habit," he wrote in his famous essay on cancer prevention, "but cancer propaganda should emphasize the danger signs that go with it."

No one questions that tobacco smoke irritates the mucous lining of the mouth, nose and throat, or that it aggravates hoarseness, coughing, chronic bronchitis and tonsillitis. It is accepted without argument that smoking is forbidden in cases of gastric and duodenal ulcers; that it interferes with normal digestion; that it contracts the blood vessels, increases the heart rate, raises the blood pressure. In many involvements of heart disease, the first order from the doctor is to cut out smoking immediately.

But what gives grave concern to public-health leaders is that the increase in lung-cancer mortality shows a suspicious parallel to the enormous increase in cigarette consumption (now 2500 cigarettes per year for every human being in the United States).

The latest study, which is published in *The Journal of the American Medical Association* (May 27, 1952), by a group of noted cancer workers headed by Dr. Alton Ochsner, former president of the American Cancer Society and director of the famous Ochsner Clinic in New Orleans, discloses that, during the period 1920 to 1948, deaths from bronchiogenic carcinoma in the United States increased *more than ten times*, from 1.1 to 11.3 per 100,000 of the population. From 1938 to 1948, lung-cancer deaths increased *144 percent*. At the present time

Christian Herald (October, '52), copyright 1952 by Christian Herald Assn., Inc., 27 E. 39 St., New York 16, N. Y.

cancer of the mouth and respiratory tract kills 19,000 men and 5000 women annually in the United States.

"It is probable that bronchiogenic carcinoma soon will become more frequent than any other cancer of the body, unless something is done to prevent its increase," is Dr. Ochsner's conclusion. "It is frightening to speculate on the possible number of bronchiogenic cancers that might develop as the result of the tremendous number of cigarettes consumed in the two decades from 1930 to 1950."

A survey recently published by the United Nations World Health Organization cites the conclusion of an investigation carried out by the Medical Research Council of England and Wales that "above the age of 45 the risk of developing the disease increases in simple proportion with the amount smoked, and may be 50 times as great among those who smoked 25 or more cigarettes daily as among nonsmokers."

A study of 684 cases, made by Ernest L. Wynder and Evarts A. Graham for the American Cancer Society and published in the AMA *Journal*, May 27, 1950, stated this conclusion: "Excessive and prolonged use of tobacco, especially cigarettes, seems to be an important factor in the induction of bronchiogenic carcinoma."

More recently Wynder, now associated with Memorial Cancer Center in New York, expanded the statement: "The more a person smokes the greater is the risk of developing cancer of the lung, whereas the risk was small in a nonsmoker or a light smoker."

In his summary *Some Practical Aspects of Cancer Prevention*, Wynder lists tobacco as the *major factor* in cancer of the larynx, the pharynx, the esophagus and the oral cavity. "In 1926," he points out, "Ewing wrote that 'though a great body of clinical information shows that many forms of cancer are due to preventable causes there has been little systematic research to impress this fact on the medical profession or to convey it to the public.' This was true then, as it is today."

After a study of world-wide medical opinion, Wynder reaches the same conclusion arrived at by Ewing 26 years ago. "Cancer of the lung," he reports, "presents one of the most striking opportunities for preventive measures in cancer."

Cancer workers want something done, and done now on the basis of present clinical knowledge, to alert the smoking public.

EXHIBIT 27A

(Not printed at Government expense)

Congressional Record

United States of America

PROCEEDINGS AND DEBATES OF THE 85th CONGRESS, FIRST SESSION

The Great Filter-Tip Hoax

EXTENSION OF REMARKS
OF
HON. RICHARD L. NEUBERGER
OF OREGON

IN THE SENATE OF THE UNITED STATES

Thursday, January 17, 1957

Mr. NEUBERGER. Mr. President, I ask unanimous consent today to have printed in the Appendix of the RECORD an illuminating and informative article entitled "The Great Filter-Tip Hoax," from the September 1956 issue of the Christian Herald, one of our leading periodicals in the realm of religious discussion and spiritual issues. Author of the article is Roy Norr, whose specialty is the study of tobacco and its effect on human health.

Mr. President, I particularly call Mr. Norr's factual and convincing article to the attention of the Senate, because magazines and newspapers today are crammed with advertising which seeks to convince men and women that the smoking of cigarettes will, somehow, contribute to success and happiness. Thousands of costly radio and television programs aspire to the same goal—the exaltation of smoking. As we drive along the highway, God's scenic wonders of mountain and hillside and meadow are blotted out by innumerable billboards heralding the marvelous benefits of cigarette smoking.

We know, of course, that this is nonsense. Cigarette smoking, far from being beneficial, is dangerous to human health. Repeated tests and studies have demonstrated that it may even induce that deadliest and most ominous of all diseases, cancer—cancer which is localized in the lung and, in most instances, is hopelessly and agonizingly fatal.

The extensive advertising of cigarettes is cleverly calculated to appeal to young people. Young men are influenced by commercials showing famous baseball stars and other athletic heroes smoking cigarettes. The implication is that cigarette smoking has perhaps contributed to prowess on the baseball diamond or football field.

Then, young girls see in magazines and newspapers so-called testimonials from glamorous movie stars. And again the implication is unmistakable that cigarette smoking has been a factor in the beauty of some female movie star or in the handsome attributes of some male movie star. Of course, from a clinical and medical viewpoint, quite the opposite again is patently true. Smoking never contributed advantageously to the physical appearance of anybody.

For all these reasons, Mr. President, I urge Members of the Senate to read the article from Christian Herald of September 1956 in which Roy Norr points out that "lung cancer—mostly smoker's lung cancer—will kill more than 30,000 men and women here this year, according to figures of the American Cancer Society."

Mr. Norr's article also debunks the widely advertised notion that filter tips miraculously terminate the harmfulness of cigarettes. At first, the tobacco companies insisted that cigarettes were not harmful. Then, they switched their claims and boasted that filters ended the dangers of smoking. But Mr. Norr emphasizes that "every adult smoker will have to decide whether the risks of smoking and cancer, smoking and heart disease, are worthwhile taking."

Mr. President, I believe Mr. Norr has done a service in writing this striking article and the editors of that fine magazine, Christian Herald, have performed a public service in publishing it.

There being no objection, the article was ordered to be printed in the RECORD, as follows:

THE GREAT FILTER-TIP HOAX

Are hordes of hapless smokers—like the trusting children who followed the Pied Piper of Hamelin—being sold down the river by the song-and-dance advertising for filter cigarettes?

Is the industry deliberately downgrading filtration to let the "rich, full flavor" of tobacco come through and thus halt health-conscious millions from giving up smoking?

Will the implied promise of "health protection" draw our still hesitant youth into the relentless cigarette habit?

Do filter tips offer any real protection?

In the answer to these questions lies the most serious health problem ever faced by the American family. Fortunately, the latest medical evidence and the record of the industry speak for themselves. They point to filter claims—with few exceptions—as the greatest advertising hoax ever put over in the cigarette industry.

The almost imperceptible movement to offer filter tips as a cure-all for the smoker's respiratory symptoms has grown into a panic rush. Not so long ago, when the health risks of smoking were not so widely accepted, filter-tip cigarettes represented barely 1 percent of the industry's production. For this year it is estimated they will reach 40 percent.

Polio, a sinister health threat that strikes terror into the heart of every family, at peak has produced a death rate of no more than 2,500 annually. Lung cancer—mostly smoker's lung cancer—will kill more than 30,000 men and women here this year, according to figures of the American Cancer Society.

But cancer is a long and slow-developing disease and many, many times that number of victims will drop dead from a heart condition in which smoking plays a role; die in a road crash caused by a sudden blackout in the smoker's vision (nicotine amblyopia, doctors call it); or even suffer a fatal fall in the bathtub, before the half-life cycle of lung cancer has completed its course. Men, like mice, often die of poisonous nicotine before the lethal tobacco tars have taken their toll.

Nevertheless, this is what you read in the cigarette advertisements, hear on the radio and see on television:

"High filtration, to help you keep your smoking moderate." "Real filtration." "Effective filtration." "Travels the smoke further, filters the smoke." "Gives you natural, self-filtering action."

There is no mention of what or how much filtration filters out, no reference to health, nicotine, or tars. Psychologically, there is no need to repeat the health slogans of the previous campaign:

"No other filter tip takes out so much nicotine and tars." "Filters out so much harmful smoke that it also filters out the worry in every puff you smoke." "Extra length helps filter out nicotine and tars." "For the greatest health protection in cigarette history—the one filter that really works."

All the Big Six have to do after brainwashing the smoker with the massive affirmation of their multi-million-dollar advertising campaigns is to follow the Russian, Pavlov, who used to set his dogs to salivating by the mere ringing of a bell which associated the sound with feeding time.

They can merely claim filtration—particularly "high" filtration, "real" filtration, "effective" filtration—to cause the smoker to associate this with assurance against sudden death from heart disease or the slow torture of lung cancer—just as "salt-low" or "sodium-low" foods were accepted by unfortunate heart sufferers as sufficient indication of a "safe" diet, until manufacturers were forced to label their products with actual salt content.

The medical background against which the present cigarette campaigns are being conducted leaves little room for doubt of tobacco's guilt. "Incontrovertible" is the association between smoking and lung cancer, according to the announcement in Parlia-

ment early this year by the British Minister of Health. "Only those who are really determined to do so can argue their way around that conclusion," declared the London Times, demanding a national effort to alert youth to the danger. The Health Minister said that two cancer-producing substances in cigarette smoke already had been identified by British research workers.

Word now comes of an announcement in the British Parliament that the commercial broadcasting companies have decided not to include advertisements for tobacco in their programs.

In Sweden too, following a survey this year which showed an alarming increase of cigarette smoking among schoolboys and schoolgirls, the Swedish tobacco monopoly ceased advertising all cigar, cigarette, and tobacco products, and foreign manufacturers followed suit.

"Tobacco smoke is unquestionably and significantly related to increased lung cancer incident," and the risk is doubled and even tripled for smokers who are exposed to polluted atmospheric conditions, was the finding announced in June after an extended survey by Dr. Clarence A. Mills and his fellow scientists at the University of Cincinnati. In the same month a statistical study reported by the National Cancer Institute, based on a survey of 40,000 people in this country, found that "the weight of evidence favors the conclusion that the excess lung cancer risk among cigarette smokers, demonstrated in special study groups, occurs in the total smoking population as well."

Life magazine, reporting on an important paper presented to the Third National Cancer Conference in Detroit, said, "In the longstanding controversy over whether cigarette smoking causes cancer, the new report makes the case against cigarettes more convincing than ever." The study was by Dr. Oscar Auerbach and coworkers of the Veterans' Administration Hospital at East Orange, N. J., who examined 29,000 microscope slides of lung tissue from 150 corpses and came to the conclusion that the degree of lung cancer damage corresponds with the number of cigarettes smoked daily. Dr. Charles S. Cameron, medical director of the American Cancer Society, said that these findings were "the very evidence skeptics demanded."

At the 150th annual meeting of the New York State Medical Society this summer, Dr. Paul A. Bunn, an authority on chest diseases, listed smoking as a disease. "Smoking," he said, "is a disease certainly, probably a foolish one, unquestionably a common one, and presumably a dangerous one, leading to a variety of pulmonary ailments."

No authority claims that smoking is the only cause of lung cancer, or that all or most smokers succumb to the disease, or that sufficient human evidence is available to prove, in the strictest scientific sense, that smoking, even heavy smoking, is a primary cause of smoker's lung cancer. Dr. David D. Rutstein, Harvard professor of preventive medicine, summed up the latter point in the statement at the 52d annual meeting in New York of the National Tuberculosis Association in May of this year: "Epidemiologically," he declared, "the case is clear. There is a link between smoking and lung cancer. Nevertheless we do not know the cause of cancer of the lung any more than Jenner knew the cause of smallpox."

(His reference was to the fact that in the prevention of smallpox it was sufficient for the English physician, Edward Jenner, to observe that milkmaids who had an infection of cowpox on their hands were immune to smallpox, from which observation he founded his famous vaccination theory which through time has saved millions of lives.)

Added to the findings established by independent statistical studies here and in many other leading countries are the accumulating laboratory results that tighten the link between smoking and cancer. Investigations by cancer workers of the Sloan-Kettering Institute in New York have confirmed the findings of Dr. Evarts A. Graham of St. Louis, first surgeon in the world to remove a cancerous lung and leave his patient living: that tars from tobacco smoke can cause skin cancer in mice and when painted on the ears of rabbits. Experiments are now underway with dogs and monkeys.

Dr. George Wright, professor of chemistry at the University of Toronto, and his staff have chemically broken down the whole tar into its acid, base, and neutral components. Cancer workers under the direction of Dr. Ernest L. Wynder, head of the section of epidemiology of the Sloan-Kettering Institute of New York, have found that the latter show the greatest cancer-producing activity, it was announced this year. Dr. Wright has further broken down the neutral portion into six fractions, one of which it was found is the truly active fraction. This, in turn, is composed of hundreds of other elements, and the scientists are implacably following every clue in the chemical jungle.

On the basis of statistical and experimental evidence unnumbered thousands of our young men and women are imperceptibly dying from smokers cancer—to be buried 15, 20, 25 years from now. Bronchogenic carcinoma is an insidious disease. "If only cigarettes killed outright with the suddenness of a motor accident," sighs the British Medical Journal, the voice of the British Medical Association, "it would be much easier to bring the thing home to the smoking public."

Tobacco apologists who say that, at worst, tobacco must be a "very weak carcinogen"—it takes so long for the disease to manifest itself—have been jolted by the publication of the remarkable theory of cancer cell formation developed by Prof. Otto Warburg, director of the famous Max Planck Institute for Cell Physiology in Germany. Discussing the production of cancer by the intermittent irritation of the mucosa of internal organs, Professor Warburg points out that "frequent small doses of respiratory poisons are more dangerous than a single large dose, where there is always the chance that the cells will be killed rather than that they will become carcinogenic."

Of even greater menace to life and health than cancer is the relation of smoking to circulatory disease. Medical opinion is increasingly insistent that to make your heart last longer you had better give up smoking. In May of this year the British Medical Association through its official *Journal* propounded thirty-one questions on the subject of smoking to the famous research team of Prof. Bradford Hill of the British Medical Research Council and Dr. Richard Doll.

One question was: "Are there any other diseases (than cancer) thought to be associated with cigarette smoking?"

The answer was that coronary thrombosis (which is killing so many Americans), Buerger's disease, duodenal and gastric ulcers, amblyopia and chronic bronchitis are among the diseases thought to be associated with smoking. The mammoth statistical study made by the American Cancer Society disclosed that the death rate for heart disease was 95 percent higher among cigarette smokers than among nonsmokers.

A query in a recent issue of the Journal of the American Medical Association, "What are the effects of smoking on the cardiovascular system?" brought this answer: "There is definite evidence that smoking two-thirds of two standard-sized cigarettes will produce an average increase in systolic blood pressure of 29 millimeters of mercury, and in diastolic blood pressure of 14 millimeters of mercury, an average increase of 36 beats per minute in the pulse rate, and in some instances a change in the T-wave of the electrocardiagram of normal persons." The effect on normal persons, mind you.

Earlier, the American Heart Association cautiously announced that it was now satisfied "there is evidence, supported by clinical observation in a large number of cases, that tobacco smoking is harmful is certain diseases of the peripheral blood vessels of the arms and legs." President Eisenhower's heart consultant, Dr. Paul Dudley White, who finds no proved cause for coronary attack, in his latest booklet, "Heart Disease Caused by Arteriosclerosis," urges that no one recovering from a coronary attack should go back to smoking. "Tobacco," he has said in an interview, "is a noxious weed and an unnecessary source of irritation to people's stomachs, lungs, blood pressure and cardiac rhythm."

These are the stark medical facts against which we must judge the filtertip campaigns to reassure youth about smoking, to lure back the 2 million or more smokers who have quit for health reasons, and to increase the per capita consumption of cigarettes.

Filtration, real filtration, effective filtration insofar as tobacco tars are concerned, is not denied as a future possibility, whatever may be said about present filter devices.

The Sloan-Kettering Institute stated in its annual report published in 1955: "At the present time, the action or efficacy of the various filters now widely used on cigarettes is not clear. The effort needed to establish their effect is justified by public concern with the subject of lung cancer and the use of cigarettes."

The British Medical Association this year submitted this question, among others, to the Doll-Hill research team: "Does the use of a cigarette holder or filter diminish the risk of getting lung cancer?" The answer was that their observations indicated that some protection is afforded by cigarette holders or filtertips, although the figures were too sparse to draw a firm conclusion.

Dr. Evarts A. Graham, of St. Louis, told a medical body this spring, "If anyone thinks he can safely smoke twice as many cigarettes by using filters he is badly mistaken." He added: "My own opinion is that the only good filter is one that won't let any smoke come through."

Dr. Alton Ochsner, the first eminent specialist to declare that sufficient evidence existed for a casual relationship between smoking and lung cancer, when asked if present filters help, said: "Yes—to sell cigarettes."

On the question of whether filtertip cigarettes filter out enough nicotine to reduce health risks of smoking on the circulatory system, authoritative medical opinion is in full agreement. The answer is "No!"

Dr. Irving S. Wright, one of the Nation's top authorities on vascular disease, has said: "There is absolutely no evidence that there is any protection in terms of vascular disease from filtertip brands. On the other hand, there is quite strong evidence that such protection is completely lacking."

Dr. Grace M. Roth, of the Mayo Foundation has reported: "Whether the tobacco is denicotinized or whether the tobacco originally is low in nicotine, apparently the content of nicotine in the cigarette must be decreased more than 60 percent before the vascular effects of smoking fail to appear or are only slight."

In the race to assure the smoking population that it can have "health-protecting" filters—and tasty nicotine and tars, too—the giant cigarette companies have left nothing undone to downgrade any possible filter-tip effectiveness. A dab of cotton, a bit of cellulose, some loosened mineral sub-

stance at the cigarette tip—anything will do.

The same as to advertising, which a prominent editor has called "studied lunacy." For example, one cigarette claims it offers true tobacco taste because it has umpty-ump thousand filters "twice as many as any other two leading filter-tip brands." In other words, the more "filters" the merrier for nicotine and tars to be ingested into the human system.

It is not to be denied that some of the filter-tip innovators were moved by their responsibility to public health as well as by commercial opportunity. According to trade reports the courageous trail-blazer paid, through the nose as it were, for his intrepidity. His perfect filter has come—and gone. It allowed little more to get through than hot, steamy air. Shortly thereafter, the company's advertising plaintively announced that the filter had been properly loosened.

Now the race is on to produce filter-tip cigarettes with the least possible filtration, so that the smoker can bathe his lungs with satisfying whiffs of nicotine and tars but still think, because of the filter, that he is getting adequate protection. The highly advertised plans of a few years ago to produce better filtering materials so that "a counter-attack may be mounted against the smoker's growing health fears," have died aborning, and the trend is constantly to downgrade filter effectiveness.

Although no table showing the nicotine and tar content in cigarette smoke can be valid for more than the last batch of cigarettes produced (the amounts vary with seasonal and other factors and the grades of tobacco used), this much can be said, by way of indication of such downgrading, on the basis of competitive ratings published by tobacco companies and by other organizations during the past several years: From the low of 1.3 average milligrams of nicotine from the mainstream smoke of each cigarette among the leading king-size filter brands, the content has risen to 1.7 milligrams in recent months; the high from 2.3 milligrams to 2.6 milligrams.

In tar content, the averages have risen from a low of 7 milligrams for 1 cigarette brand to an average of 17 milligrams; for the high from an average of 21 to 25.2 milligrams.

In a number of nonfilter cigarettes the averages both for nicotine and tars have been decidedly lower than these.

To add insult to the injury of millions of scared smokers now rushing to the false health protection of filter cigarettes, are two other developments regarding which consumers have been kept in the dark.

One is the increasing use of harsher, cheaper tobaccos by the manufacturing industry, leaving high and dry the growers who have been encouraged to produce quality crops. The cigarette makers, it is intimated, believe they can get by with inferior quality when they use filters.

The other is a new manufacturing process which is said to utilize everything but the floor sweepings to produce a homogenized tobacco leaf. It includes scraps, stems, and damaged leaves, formerly discarded as waste, which are pulverized, mixed with a gluey substance and squeezed out in continuous rolls. Virtually every United States cigar and cigarette maker is either experimenting with this reconstituted tobacco or actually using it, Time magazine has disclosed. For a long time the process has been kept a dark secret, for fear of alarming the public, nurtured on the notion that tobacco should be rich and pure. Grower interests have denounced the product as unfit for humans. What it will mean in terms of multiplied health risks for the cigarette smoker is still to be shown.

A further movement is the production of mentholated, kingsize, filtertip cigarettes—against the day when the befuddled smoker will wake up with a rasping sore throat from the rich, mild cigarettes advertised to him now. Medically, of course, it will be just as irritating; but it will be like putting a chunk of ice in a glass of whisky, to provide the illusion of mildness and smoothness.

Every man for himself, is what medical bureaucrats say about the hazards of smoking. Unless and until the health forces of the Nation can speak in full agreement, every adult smoker will have to decide whether the risks of smoking and cancer, smoking and heart disease are worthwhile taking.

But what about youth on the threshold of the smoking age—young men and women, who look to parents, pastors, teachers, and others for some guidance? Are they to continue to be the victims of false medical claims for filter cigarettes? Surely their decision should have the help of hard facts.

Which means this at a minimum: Filter-tip cigarettes should be properly labeled for nicotine and tar content. In other words, the amount of the average nicotine and tar content in the mainstream of the cigarette carrying a filter tip (making allowance for reasonable and expected variations) should be given on the package, just as producers of salt-low foods are required to state on the label the amount of salt in a given weight of the food. And salt is neither the poisonous alkaloid that is nicotine, nor the health risk that is smoking.

Nothing could be more in the interest of public health and public safety than a demand made by you upon your representatives in Congress, and/or your State representatives, for a (1) State or congressional declaration that tobacco be recognized as the dangerous drug which it unquestionably is; (2) followed by a mandate to the Pure Food and Drug Administration in Washington, and/or to State health authorities, that cigarette makers be required to label their filter-tip cigarettes for actual filtration effectiveness.

With the medical indictment of relentless cigarette smoking growing ever blacker, with tobacco's relationship to the two greatest killers of our day becoming ever clearer, to give the benefit of any doubt to poison, rather than to people, to fail to provide youth with the information they need about the hazards of smoking, is to make a mockery of health education.

EXHIBIT 27B



Now EVERYBODY'S Getting Scared!

King-size cigarettes for king-size cancer? It's one of many new questions hurled in growing tempo at the worried tobacco industry and its satellites

By ROY NORR

A LITTLE more than a year ago, it was clear that smokers were getting frightened. Evidence pointing up a relationship between cigarettes and lung cancer set them off on a search for smoking safety that skyrocketed king-sized and filter-type cigarettes to phenomenally-fast public acceptance.

In fourteen months that the tobacco industry will never forget, new evidence has piled up.

Now, it's not only the fellow on the inhaling end of the cigarette who is scared. The fellows on the manufacturing, distributing and advertising end are scared too—into an astonishing exhibition of ducking and weaving.

Let's look at the facts.

Twelve separate and independent investigations, covering thousands of lung cancer patients, have been conducted in five different countries. Every study has pointed to the same conclusion: *The danger of developing lung cancer increases with age and in approximately simple arithmetical proportion to the amount of tobacco smoked.*

At the annual meeting of the American Cancer Society in New York in November it was disclosed that at a symposium sponsored by the United Nations World Health Organization and UNESCO at Louvain, Belgium, a resolution was adopted which stated unequivocally, "There is now evidence of an association between cigarette smoking and cancer of the lung."

Statistics show that more Americans are dying of lung cancer today than died of all types of cancer fifty years ago. Of our present population, 50,000,000 men, women and children will contract cancer, and 25,000,000 will be killed by the disease according to an official prediction made last year before a Congressional committee hearing in Washington. In lung cancer the rise is such that "it is almost as though the population had been exposed to some new hazard," it has been stated for the American Cancer Society.

The hazard did not seem so "new" to distinguished research scientists, pathologists and cancer specialists who have cried out for years that what tobacco was sowing would be reaped later in an agony of lung cancer incidence unprecedented in medical annals. Dr. Alton Ochsner, President

TOBACCO IS ON TRIAL FOR ITS LIFE—AND YOURS!

In 1952, the *Journal* of the American Medical Association noted, "Bronchogenic carcinoma has steadily increased in incidence until it is now the commonest form of cancer." As lung cancer rose, so did cigarette smoking. The disease was found to be more likely to occur among heavy smokers than among abstainers. In the face of feverish pooh-poohing propaganda from the tobacco industry, scientific experiments were launched to prove or disprove the theory that lung cancer and smoking were linked.

In October, 1952, CHRISTIAN HERALD reported the facts in the article, *Smokers Are Getting Scared!* That article, condensed and reprinted in *Reader's Digest* under the title, *Cancer by the Carton*, prodded the issue into the open. Since the article appeared, developments have been startling and rapid—set off by CHRISTIAN HERALD's opening gun.

As a continuing report to CHRISTIAN HERALD readers, we present this second article, which reveals that cigarettes have become more than suspects of murder, wanted for questioning. They are now specifically on trial for their lives—to say nothing of the lives of smokers.

The tobacco industry knows it. Desperately, they advertise, "The other fellow's brand may damage your health, but don't worry about *our* brand." The American Medical Association knows it. They have banned tobacco advertising as of January 1, 1954, from their *Journal* and their exhibit halls. The radio and TV industry knows it. They have told cigarette advertisers that "when dramatized advertising material involves statements by doctors, dentists, nurses or other professional people, the material should be presented by members of such professions reciting actual experience or it should be made apparent . . . that the portrayal is dramatized." Literally, "everybody's getting scared!"



ROY NORR

Roy Norr, author of both articles, has for many years conducted research into medical literature here and abroad on the problems of smoking and health. By profession a publicist for business, industry and education, he is also editor and owner of *Smoking and Health Newsletter*. A strategist in the public relations field, Mr. Norr directed the policy of the American candy industry in fighting the campaign, "Reach for a Lucky Instead of a Sweet," and won a victory that forced the advertising off the air.

of the American Cancer Society in 1948-49, head of the famous Ochsner Clinic in New Orleans, predicts that *by 1970 one out of every five Americans will be a victim of this dread disease unless preventive measures are taken*. He has long warned that heavy smoking is a definite causal factor in cancer of the lung.

Prof. Evarts Ambrose Graham, of the Washington University School of Medicine in St. Louis, former President of the American College of Surgeons, declares unequivocally that intemperate smoking is the cause of more than 95% of lung cancer. He is the first surgeon in the world successfully to operate for the complete removal of a cancerous lung, and was honored in 1951 by the medal of the American Cancer Society for "outstanding contribution to the control of cancer."

The establishment of a relationship between tobacco and the frightening rise of respiratory cancer, although the causal factor still remains to be proved in chemical terms, "is the most significant advance in lung cancer in recent years," writes a distinguished American scientist in a personal communication to the author. In Britain, Dr. Horace Joules, Director of the great Central Middlesex Hospital in London, goes further. The risk of acquiring cancer from smoking, he states, "is one of the most important facts which has come to light concerning cancer during this century."

Medical and cancer fund-raising interests who demand, "Arrest Cancer—It's Wanted for Murder!" have deplored the "alarmist fears" of the scientists who associate smoking with this terrible scourge. But last summer they themselves testified to "the startlingly rapid increase in deaths from lung cancer" and told Congress that lung cancer was increasing at an alarming rate.

For the Government, Dr. J. R. Heller, Director of the National Cancer Institute, at Bethesda, Maryland, made on October 30, 1953, this official statement on this agency's position:

"We agree that the evidence presently accumulated justifies the belief that heavy smokers of cigarettes are at greater risk to cancer of the lung than are non-smokers. All of the past studies, including that conducted here, have approached this question in an indirect fashion that is subject to many so-called 'jokers.' However, we will be surprised if the direct studies now under way, including one being initiated here, do not establish this belief as a fact. In our own study of smoking and lung cancer, evidence was developed that is not necessarily consistent with the idea that smoking unequivocally causes lung cancer. Until these and other questions are resolved, and until the evidence is further tested for general consistency, we regard the etiological significance of the association as unestablished, despite the undoubted high correlation between heavy cigarette smoking and the occurrence of lung cancer."

Nevertheless, leading cigarette makers, duty-bound to tell their stockholders *all* the facts, continue to circulate earlier statements from "Government scientists" saying it is unlikely that smoking plays a dominant role in the production of cancer . . . "since experimental observations place tobacco tars at best among the weak exogenous carcinogens" [cancer-producing substances].

Cigarettes, it seems, are only a little bit deadly!

The cigarette industry tried to take in stride the medical evidence that after age 45, the more you smoke, the quicker you die. It was, after all, the teen-agers to whom most cigarette advertising is directed. But the shock of the first sustained drop in domestic cigarette consumption in twenty years set bells ringing frantically. The tobacco huckster, like the smoker, is alarmed. Approximately 224 billion tax-paid cigarettes were produced for U. S. consumption in the first seven months of 1953, a decrease of about 5¾ per cent over the same period in 1952. *This, notwithstanding the fact that more than one million new smokers automatically became "of age" for prospective cigarette addiction.*

Bitter words were hurled by tobacco growers who accused cigarette advertisers of fouling the common nest. Said an irate spokesman: "A great deal of this advertising is plain silly and ridiculous when you consider the outlandish medical claims these manufacturers make in radio, television and press ad-

vertising."

He must have been thinking of such blurbs as these:

"Is your smoking associated with a nagging cough, a dragged-out feeling?"

"Do you lose your taste for food?"

"Do you have 'Smoker's Cough'?"

All are possible symptoms of malignant disease!

Take the advertising of king-sized cigarettes. The long, or king-sized, cigarette might be helpful to the insistent smoker, if honestly advertised for its filtration qualities.

But the smoker must discard his king-sized cigarette before it is half consumed if he is to get any filtration benefit at all. To advertise *that* would be giving the smoker a break. Instead, most of the Big Five say in their advertising:

Smoke king-size "for that extra measure of fine tobacco."

Smoke it "for longer smoking enjoyment."

Smoke it "to the last cool puff."

Points out the director of the Chemical Section of the American Medical Association laboratories, in a statement to the Kansas City National Better Business Bureau: "If a king-size cigarette were smoked to the same length butt as a standard cigarette, the extra length of tobacco smoked would furnish more irritants. And the smoke from the last portion of the cigarette does contain more nicotine, etc., filtered from the earlier smoke. Thus the claims of extra filtering length and extra puffs are contradictory. It is not to be expected that the irritants will be filtered out in preference to the flavor. A great many of the flavorful ingredients of the smoke are themselves irritants."

It seems to add up to: "Smoke king-sized cigarettes for king-size cancer."

The American cigarette industry is scared because it faces a public health battle which its vast advertising empire can no longer suppress. Heading the public health forces of the nation who feel there is little time to be lost in an educational cancer prevention campaign is Dr. Herman E. Hilleboe, Commissioner of Health of the State of New York. In a statement to the author, Dr. Hilleboe writes under date of September 15, 1953:

"Our research workers in this Department are of the opinion that the present epidemiological evidence regarding the relationship between cigarette smoking and lung cancer establishes this relationship as probably a causal one, and that further laboratory experimentation on this relationship, while highly desirable, is not essential to further establish this relationship. However, we very much hope that additional laboratory evidence by the production of lung cancer in animals will soon be forthcoming, if only to convince the skeptics among the profession and elsewhere."

With the handwriting on the wall growing ever plainer, what do the cigarette barons think of their quite awesome responsibility?

Do they accept or reject the medical findings concerning smoking and health?

What do they intend to do to make smoking a less deadly pastime than it appears to be?

Will they cease or continue the type of advertising that has outraged lay and medical opinion alike?

Medical men, certainly, have had all they could take. The American Medical Association, pressured by its doctor members, has banned all cigarette advertising in its various publications after January 1, 1954. Furthermore, advertisers were told, "This decision also applies to the presence of exhibits and/or lounges by cigarette, tobacco or allied products . . . at the 1954 and following conventions of the AMA."

But what about the tobacco bosses? What do they say to all this?

Paul M. Hahn, President, American Tobacco Company: Finds nothing to worry about, believes figures associating smoking with cancer unreliable, often meaningless, and says correlation does not constitute proof.

HIS views were definitely not supported by Dr. Cornelius P. Rhoads, Director of the famed Sloan-Kettering Institute for Medical Research of the Memorial Cancer Center in New York, the largest cancer institution in the world today. Dr. Rhoads revealed to a Congressional group last year that the chemicals which cause cancer in animals are being extracted from tobacco tar, ingeniously produced by a smoke machine, and predicted their identification in "the very near future." Said he, "We believe that there is a very real relationship between smoking and the occurrence of lung cancer."

E. A. Darr, President, Reynolds Tobacco Company: Denies there is any substantial evidence linking smoking to lung cancer and believes the "unsupported claims" will fall of their own weight.

For instance, he might have added, the "unsupported claims" of the massive British study published late in 1952 by Dr. Richard Doll and Prof. A. Bradford Hill of 1,465 patients with cancer of the lung and 1,465 matched controls, which evoked world-wide medical interest, received front-page headlines abroad, and was televised by the British Broadcasting Corporation, together with the blunt warning of the medical commentator: "We can conclude that heavy smoking predisposes to lung cancer."

B. F. Few, President, Liggett & Myers: Dismisses all evidence of the relationship between smoking and cancer as "unreliable."

Strangely, "unreliable" was the reason for the National Better Business Bureau's condemnation of Chesterfield advertising as "a flagrant disregard of the public interest." And it's why the Federal Trade Commission has attacked that advertising in the courts.

Herbert A. Kent, President, P. Lorillard Company: Admits alarming reports about smoking and health, but insists that no investigator has claimed conclusive results.

On the other hand, the highly regarded *New England Journal of Medicine*, Boston medical monthly, finds the evidence about cigarette smoking and lung cancer "so strong as to be considered proof within the everyday meaning of the word." *The British Medical Journal*, speaking for the British Medical Association, said that although the nature of the cancer-producing agent was not yet known, tobacco "has been incriminated as a vehicle conveying an agent responsible for a large proportion of lung carcinoma . . . we are bound to take what preventive action we can."

Dr. Philip A. Caulfield, distinguished Washington surgeon, summarizes the position of U.S. cancer workers with his statement that "the current belief in medical circles is that any irritant entering the body can lower the resistance of the tissue it irritates in such a way as to allow cancer to get a foothold. Smoking is considered an irritant of the lung and respiratory tract."

A. F. Lyon, Chairman of the Board, Philip Morris Company: Insists they are all "publicity seekers" who believe more poison than pleasure wreathes from the smoke of a burning cigarette.

Mere "publicity seekers," apparently, are such eminent figures as the Grahams, the Ochsners, Dr. Rhoads who attended the late Senator Taft, Dr. Caulfield who was Senator Brien McMahon's physician, the Wynders and others who see smoking as a factor in the accelerating epidemic of lung cancer in the United States. Perhaps President Eisenhower's doctor is also a "publicity seeker." States John Gunther in his book, "Eisenhower the Man," for years he [the President] smoked like a furnace—sixty cigarettes a day or more. At Columbia University, Eisenhower's pulse suddenly rose. On doctor's orders he stopped smoking on the instant. His pulse returned to normal within a week

and he has never smoked again since.

The position taken by the Big Five toward their stockholders is strictly ostrich. They say, it isn't so, or it hasn't been proved! Out of the other side of their mouths, however, they tell the public a different story. "Just What the Doctor Ordered!" screams L & M (Liggett & Myers) Filter-Tip advertising. "The cigarette that takes the *FEAR* out of smoking," bawls Philip Morris.

The bosses are likely to get proof that even they cannot shrug off—and sooner than they expect. Combined chemical and biological studies are under way independently to identify the cancer-producing elements (carcinogens) contained in tobacco tar. The presence of such agents has been shown in animal experiments with tobacco tars.

The race is on between science and cigarette sales—with death holding the stopwatch.

Skeptics—as well as medical stooges for the cigarette industry—have long challenged laboratory workers to produce lung cancer in animals, strictly from the inhalation of smoke. Smoking physicians said it couldn't be done; "smoked mice" now have proved that it can be done. Dr. J. M. Essenberg of the Chicago Medical School, in a paper delivered (1952) before the American Association for the Advancement of Science, announced that he had induced lung cancer in mice. Dr. Essenberg designed an automatic smoking machine with a chamber in which the animals lived and in which a cigarette was lighted every hour during twelve-hour days. The experiment showed that more tumors resulted in the lungs of smoked mice than in those of the controls.

NOT EVEN the new filter-type can prevent a coming day of judgment for cigarettes. The first report issued from the chemical laboratory of the American Medical Association at the year end is that most filters don't filter. Even the best of them remove little of the total nicotine of the smoked portion.

There isn't the slightest sign that the cigarette makers as an industry are willing to contribute to a truly impartial study and investigation of the possibility of removing or neutralizing the carcinogenic chemicals in tobacco. Our cigarette barons have been put to shame by the action of cigarette-makers in little Denmark.

A paper read at a meeting of Scandinavian lung cancer specialists last October in Copenhagen concluded that the smoking of as few as five cigarettes a day over a period of twenty years made smokers liable to lung cancer. The next day Christian Augustinus, Denmark's leading cigarette manufacturing company, made a substantial donation to the work of the Cancer Research Division of Copenhagen University.

There is no sign that the cigarette industry intends to meet squarely the public health issue before it. The issue extends beyond cancer. On the nicotine side, tobacco is an indisputable killer in many heart conditions. It has a constricting effect on the arterial system; it raises the blood pressure, increases the pulse rate, lowers the skin temperature. More persons continue to die of "heart trouble" than of all other diseases combined.

As to general respiratory disease, there is Smoker's Cough, Smoker's Throat, Smoker's Allergy, Smoker's Asthma, Smoker's Larynx, the latter marked by tumorous growths which led the *Journal* of the American Medical Association to warn opera stars (who coo on the air that they find cigarettes so good for the throat!) against the misuse of their larynx.

Some of the Big Five have completely abandoned medical and health claims since 1952, but have gone head over heels into tainted testimonial advertising. And be sure that the glamor girls and movie heroes who just love that cigarette, the popular announcers forced to turn "barkers," the athletes and coaches who sell out their eager young followings, are not directing their fire at grandpa and grandma. They're shooting at youth. The American Tobacco Company's late George Washington Hill, the arch-huckster of his day, shrank from the accusation. "I should be shocked as anybody else," he protested, "if a tobacco company should undertake to appeal to adolescents." His successors brazenly plug for the youth market.

Equally black is the record of the network bosses who allow the tobacco makers to attack the people's health through the people's air. Relentless, remorseless, ceaseless is the flood of preposterous cigarette claims pumped through the air daily into the American home.

Right now the networks are lame from the fall they took when the American Medical Association suddenly pulled the rug from under their feet by closing the AMA journals to all cigarette advertising.

It was a beautiful rug. It gave the broadcasters the excuse to say: "If it's good enough for the AMA, it's good enough for us."

Now that cigarette advertising has become too noisome to be published in the "bible" of the American medical profession, what possible justification can there be for such advertising on the air when addressed to children, youth and adults who are in no position, as are doctors, to distinguish between the true and the false in medical claims?

What now? it may be asked. Here are a few things that you can do:

(1) *Expose to your family and friends the hazards of smoking in terms of lung cancer.*

(2) *Urge your church, your parent-teacher or local educational groups to demand free time on the air to answer harmful misrepresentations in cigarette commercials.*

A recent president of Lorillard, E. M. Ganger, said: "We do not own the air we buy for our television programs. We simply rent it by the grace of the people. The public gives us its faith that whatever words and pictures we send into their living rooms, will be beneficial as well as entertaining; dependable, as well as amusing. And it is our solemn obligation to keep faith with that trust." Mr. Ganger, however, is no longer president.

(3) *Join in a round-robin to the Federal Communications Commission petitioning the commission to revoke the broadcasting licenses of stations which refuse to co-operate in the public interest, convenience and necessity.*

A similar petition more than twenty years ago forced the discontinuance of "Reach for a Lucky Instead of a Sweet" as an advertising slogan on the air.

(4) *Demand of your Congressional representatives that tobacco, a nicotine-narcotic drug, be placed under the discipline of the Pure Food and Drug Act.*

Tobacco was once listed as a drug in the U. S. Pharmacopeia, but was suddenly dropped with the explanation that it had come to be little used in medication, though it made a satisfactory bed-bug poison.

(5) *Keep abreast of the latest developments on smoking and health.*

We are a nation of snifflers, wheezers, coughers—and the greatest consumers of tobacco products in the world. And lung cancer is on the increase. The panicked cigarette industry admits, in big black advertising headlines, that more than 21 million smokers—at least one out of three—are definitely and deleteriously affected by nicotine, tars and other poisons in cigarette smoke.

One out of three—and who is the *one*? What a monstrous game of Russian Roulette smokers are playing!

Risking blowing out one's brains with a single bullet in a six-chamber gun gives at least a 5-to-1 chance.

When cigarettes are the weapon, the confused industry itself advertises only a 3-to-1 chance.

Are smokers prepared to face such odds? THE END

EXHIBIT 27C



Smokers are Getting *SCARED!*

No wonder! Lung cancer is on the increase and authorities point to smoking as a major "suspect." Here's the grim story and what you can do about it.

By ROY NORR

ILLUSTRATOR: NORMAN KENYON

THE RUSH is on! Filter-type and king-sized cigarettes, advertised as offering protection from the harmful effects of smoking, are taking king-sized bites out of leader-brand sales volume. Last year some reached within shouting distance of ten billion units, and one actually nosed out a big-name cigarette from number-five position.

It means that smokers are getting scared! They're wondering just where cigarettes come into the health picture—and more specifically and ominously, where they come into the lung-cancer picture. They have reason to be scared.

Meanwhile, with 800,000 adolescents recruited each year for addiction, with the strong, silent men of the organized medical profession standing on the sidelines on this tragic health issue, with an enormous increase in the appetite for narcotics—and tobacco is one—during this age of anxiety, the cigarette industry has had a field day. It has reached a mountainous production of 400 billion units annually. As against 46 cigarettes per capita consumed in 1903, today's annual consumption is at the rate of 2500 cigarettes per capita for every human being in the United States.

What this means to the health of the nation was recently blurted out by the industry itself! In big, black type, Lorillard advertisements introduced its filter-type cigarette by telling the world: "According to published medical findings, about one-third of all smokers react abnormally to the nicotine and tars in tobacco."

It was another way of saying that twenty million men, women, yes, and children—are deleteriously affected by smoking. No one questions that tobacco smoke irritates the mucous lining of the mouth, nose and throat, or that it aggravates hoarseness, coughing, chronic bronchitis and tonsilitis. It is accepted without argument that smoking is forbidden in cases of gastric and duodenal ulcers; that it interferes with normal digestion; that it contracts the blood vessels, increases the heart rate, raises the blood pressure. In many involvements of heart disease—the greatest killer

of modern times—the first order from the doctor is to cut out smoking immediately.

But what sends the shivers down the spine of the cigarette industry and appalls public health leaders is the dread shadow of cancer that now looms over every medical discussion of the relationship of excessive smoking to diseases of the respiratory tract. For three decades a curtain of smoke almost as impenetrable as the Iron Curtain has largely kept from public notice the bitter controversy raging within medical ranks over the part played by smoking in the dizzying rise of bronchogenic carcinoma, better known as cancer of the lung. But the curtain is now being lifted by shocking statistics.

The increase in lung cancer mortality is reaching almost epidemic proportions, according to the survey published this summer by the United Nations World Health Organization. The report cites the conclusion of an investigation carried out by the Medical Research Council of England and Wales, that "above the age of 45, the risk of developing the disease increases in simple proportion with the amount smoked, and may be 50 times as great among those who smoked twenty-five or more cigarettes daily as among non-smokers."

The latest study, published in the *Journal* of the American Medical Association (May 27, 1952), by a group of noted cancer workers headed by Dr. Alton Ochsner, former President of the American Cancer Society and director of the famous Ochsner Clinic in New Orleans, discloses that during the period 1920 to 1948, deaths from bronchogenic carcinoma in the United States increased over *ten times*, from 1.1 to 11.3 per 100,000 of the population. From 1938 to 1948, lung cancer deaths increased *144 per cent*. At the present time cancer of the mouth and respiratory tract kills 19,000 men and 5,000 women annually in the United States. In 1914 only 442 deaths from cancer of the larynx and lung were cited for men, and 166 deaths for women.

The death rate for cancer had quadrupled in the last fifty years of the nineteenth century, doubled again the last fifty years of this century, and bids fair to quadruple again in the last fifty years, according to other statistical studies. Cancer of the stomach had been the largest cause of cancer mortality for many years, but in 1949 over twice as many patients with cancer of the lung as with gastric cancer were admitted to the Memorial Hospital in New York.

"It is likely and probable that bronchogenic carcinoma soon will become more frequent than any other cancer of the body, unless something is done to prevent its increase," is Dr. Ochsner's conclusion. "It is frightening to speculate on the possible number of bronchogenic cancers that might develop as the result of the tremendous number of cigarettes consumed in the two decades from 1930 to 1950."

JUST how frightening is made evident by the mammoth research project described in the annual report of the American Cancer Society. The project now under way will determine to the Society's statistical satisfaction whether smoking is a culprit or not, in the terrifying incidence of cancer of the lung. The project involves 50,000 volunteer workers, who will undertake a monster death watch over 500,000 aging subjects throughout ten states of the Union. Each volunteer will obtain the smoking histories of ten male acquaintances above fifty years of age—relatives, friends, neighbors. Each year each "case" will be followed up. When death occurs, the cause—and if the cause should be cancer, the type of cancer—will be carefully noted.

"Cancer of the lung," the ACS declares, "is on the increase, especially in men. The death rate for this form of disease in men has tripled in the last twenty years. It is too high to be accounted for simply by the increased population and the greater numbers of older people. Many authorities have suspected smoking as the major cause."

The death watch will be continued for at least five years, by which time, it is stated, "from the large and representative 'sample' from all parts of the country, analysis should adduce the true facts."

That cancer authorities "suspect" smoking as a major cause of lung cancer is a rousing understatement, according to workers in the field. They want something done, and done now, on the basis of clinical knowledge of the subject, to alert, if not alarm, the smoking public. Leading cancer authorities point to an impressive literature on smoking in relation to cancer—largely suppressed from public notice by the advertising pressure of the cigarette industry—and to the most recent investigations of the subject undertaken here and abroad.

They refer to the study of 684 proved cases made by Wynder and Graham for the American Cancer Society and published in the *Journal* of the American Medical Association, May 27, 1950. The report ("Tobacco Smoking as a Possible Etiologic Factor in Bronchogenic Carcinoma") stated this conclusion: "Excessive and prolonged use of tobacco, especially cigarettes, seems to be an important factor in the induction of bronchogenic carcinoma."

Wynder, in his most recent observations ("Some Practical Aspects of Cancer Prevention," April 10, 1952), has expanded the thought with the statement: "The more a person smokes the greater is the risk of developing cancer of the lung, whereas the risk was small in a non-smoker or a light smoker."

For the British Empire Cancer Society, Doll and Hill, in their report, published in *The British Medical Journal* ("Smoking and Carcinoma of the Lung," September 3, 1950), made this finding: "We . . . conclude that smoking is a factor and an important factor, in the production of carcinoma of the lung."

What is true of lung cancer is true of other cancers of the respiratory tract. The latest summary of medical literature on the subject is published by Wynder, now associated with the Memorial Cancer Center in New York. His summary ("Some Practical Aspects of Cancer Prevention," April, 1952) lists tobacco as the *major factor* in cancer of the larynx, the pharynx, the esophagus, and of the oral cavity. Wynder in beginning his study, writes: "In 1926, Ewing in an article on 'Cancer Prevention,' wrote that 'though a great body of clinical information shows that many forms of cancer are due to preventable causes, there has been little systematic research to impress this fact on the medical profession or to convey it to the public.' This was true then, as it is today."

The fact that even today there is an enormous lag in the preventive side of cancer education is not denied by leading lung cancer specialists. Yet here is a reply recently received by this writer from the American Cancer Society on a question directed to this very issue: "At the present time we do not know the principal causes of cancer in the human population. The American Cancer Society is supporting research on a number of different factors, including smoking, which have been suggested or suspected as being important causes of cancer. We are not in a position to take any action until the facts are known."

From the outgoing president of the American Medical Association came

this statement: "There is still considerable dispute as to what part smoking may play in the development of cancer of the lung. When sufficient information is available on the subject to make a positive statement concerning it, it will be made. This will require a period of years to ascertain."

Nothing could better serve the tobacco industry than for the cigarette-cancer problem to be put in deep freeze for five years, while medical bureaucrats await the result of further "extensive surveys"! Meanwhile, the campaigns for cigarette addiction would continue on the air. The "testimonialiar" would reign supreme. The "Doctor" on the television screen could still prescribe his cigarettes as a remedy for Smoker's Cough. And the huckster would continue to blitz the American family with fake medical claims.

For the next five years thousands of our youth will be inducted into the army of the doomed-to-die-agonizingly, as is the way of lung cancer, because "no action can be taken until the facts are known"! Man knows as little as he did one hundred years ago of the mystery of cell growth gone on a rampage, which is cancer. But we do know much of the possible, and even probable causes of the disease, contributory or direct, in many of the forms which cancer takes.

THE persistent inertia of medical bureaucracy has helped the tobacco industry obscure the truth. The tender relationship between the medical hierarchy and the cigarette makers has been evident through many years. The "bible" of the profession, the *Journal* of the American Medical Association, in its advertising pages, was glad to accept Dorothy Lamour as an authority on cigarette "mildness"; to present the claim that "not a single case of throat irritation was due to smoking—"; and until recently to feed 160,000 doctors with the false claims of a cigarette "proved definitely and measurably less-irritating to the throat."

Apparently, however, the AMA has taken another look at the problem. Its outgoing president in a communication last February disclosed to this writer that the decision of its trustees, made two years ago, to "eliminate health claims in cigarettes" in the advertising pages of the *Journal*, was at last being put into effect. The rule had been held in abeyance, because it was felt that "forced radical change in the advertising" might influence certain pending action before the Federal Trade Commission." That was the explanation!

It is a tragic thought that the risk of lung cancer through cigarette addiction should be many times multiplied by the comfort and aid which the cigarette habit has received from organized medical sources.

Few cancer authorities whom this writer has interviewed have any doubt that a new educational campaign, if organized, with the help of independent medical opinion, the public health forces of the nation, educators, parent-teacher associations, ministerial bodies, and women's clubs, would save untold thousands of lives.

A "Prevent Cancer!" campaign to face up to the threat of tobacco is everyone's business. You can help too.

First, alert the public to the danger. The more people who point to the medical fact that tobacco smoking is listed "among the recognized and suspect sources" of cancer of the respiratory tract, the more headway prevention will make. Even if you can't do anything else, you can do this much.

Second, stop the fake testimonials. Those 800,000 adolescents who are yearly initiated into smoking are captured largely by Hollywood and radio favorites, stage and opera stars, who beg and cajole them to try their brands. They represent all that is glamour, popularity and success to their youthful followings. Legislation that would declare any health testimonial bought and paid for as patently insincere and therefore fraudulent advertising, would put the testimonal mills operating openly in Hollywood and New York out of business.

Third, rip off the mask from cigarette claims. Arthur Godfrey challenges his listeners: "I don't know what you smoke, but why don't you try to find out what's in your cigarette? You'd be surprised."

YOU would, indeed, if you knew that more than one hundred adulterants have been used during the years in the process of tobacco manufacture, and, that one—diethylene glycol—considered not harmful when used as a moistening agent in cigarettes, killed more than one hundred people when used as a solvent in a promising "medicine."

Yes, the public is entitled to full disclosure! Legislation requiring that the chemicals added be clearly stated on every package of cigarettes is long overdue.

Fourth, break the stranglehold of the tobacco huckster on the people's air. The greatest smoke barrage ever hurled at the American public has been with the help of television. You can't turn on your TV set without being told how to cure sore throat by switching your brand. Switch to what? To smooth, rich, ripe, roasted or toasted golden tobacco? No more damaging myth has ever been foisted on the public mind! The real commodity which the cigarette people sell is *smoke*—a dry, destructive distillate of tobacco leaves largely composed of carbon dioxide, carbon monoxide, nicotine, ammonia, formaldehyde, furfural, tars and formic acid.

When it comes to smoke, half-truths, misleading assertions, insincere comparatives and fraudulent claims are cheerfully tolerated in television. The public responsibility which the networks have surrendered to the tobacco sponsor is no better stated than in their industry code and their own company regulations which read:

"A television broadcaster should, in recognition of his responsibility to the public, refuse the facilities of his station to an advertiser where he has good reason to doubt the integrity of the advertiser and the truth of his advertising representations. . . . Testimonials must reflect the authenticated experience of competent living witnesses. . . . When dramatized commercials are used that involve statements by doctors, dentists, nurses, the lines must be read by members of these professions, reciting actual experiences. . . . No consideration of expediency should be permitted to impinge upon the vital responsibility towards children and adolescents which is inherent in television."

If cigarettes cannot be promoted with truth and restraint, if broadcasting is to neglect the public good, then the sole recourse of the citizenry is Congressional action.

Tell your Congressman, and your local radio and television stations!

There could be no finer crusade than the tracking down through research of the evil spirit of cancer in the immense darkness in which it still lies. *But while that is going on,* we dare not neglect prevention, where the light is bright. It makes sense to try to prevent what clinical evidence indicates as preventable cancers. Among the areas in which this possibility is greatest are the critical areas of lung cancer, mouth cancer, throat cancer.

To continue to ignore the major suspected cause—the excessive use of cigarettes—would be one of the blackest social crimes of our day.

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EXHIBIT 28

[The Wall Street Journal, Tuesday, July 9, 1957]

COSTLIER SMOKES EXPECTED TO BOOST PROFITS OF BIG CIGARETTE COMPANIES

GAINS MAY SHOW IN SECOND HALF; SALES RISE DESPITE REPORTS OF CANCER LINK

NEW YORK.—Costlier smokes are expected to boost profits for the big cigarette companies, at least temporarily.

In addition, cigarette sales are heading upward, company officials indicate, despite two recent reports which revived the controversy over the alleged link between smoking and lung cancer.

Results of the recent wholesale price increases on regular and king-size cigarettes, which went into effect June 17, won't show up in earnings for the 6 months ended June 30, tobacco officials say. The price hikes came too late in the period, they assert, and orders on the companies' books when the increases were announced are still generally being filled at the old prices. But they believe the higher prices should make a difference in the second half of this year, although they concede that certain costs, such as advertising and tobacco, may also rise.

EFFECT ON PHILIP MORRIS

"For the last 6 months of 1957 the increase could make a difference of 40 to 50 cents per share over what we might have earned" without the increase, says C. H. Kibbee, secretary and treasurer of Philip Morris, Inc. "For all 1958 it could amount to 80 cents or $1 per share over what we might have earned," Mr. Kibbee adds. In the second half of 1956, Philip Morris earned $2.23 a share, and for all 1956 it posted earnings of $4.06 a share.

Most other companies, though reluctant to spell out the possible effects on profits, agree their earnings should get a fillip from the higher wholesale prices.

Mr. Kibbee gets his estimate by figuring how many thousands of regular and king-size cigarettes the company sold in past periods and multiplying that by 35 cents a thousand, the size of the price increase.

JUDGING THE ROSY PICTURE

However, company financial officers, including Mr. Kibbee, are quick to note that such a rosy profit picture can be misleading. "It's unrealistic since it assumes that everything else will remain unchanged," says a spokesman for American Tobacco Co.

Factors which could take a bigger bite out of future profits include:

Tobacco, which accounts for about three-fourths of the cost of making a cigarette, has been getting more expensive. For example, this year's burley crop, used extensively in making filter-tip cigarettes, will be marketed in the fall, and tobacco men say they won't be surprised if the leaf price tags are higher than last year.

"There was no increase in burley acreage, and this could have an effect on tobacco prices this fall," says A. H. Galloway, vice president and treasurer of R. J. Reynolds Tobacco Co.

Advertising costs, a big item in a cigarette company budget, are also going up. One company said it was taking a look at its advertising budget for the second half of this year and may "revise it upward, since there'll be more money available."

PENNY-A-PACK PROFITS

With the price boosts, profit margins to the companies on regular and king-size brands average a bit more than a penny a pack, compared with about four-fifths of a cent before the increase. "Profit margins have been holding up nicely" on the filtered brands, says Reynolds' Mr. Galloway.

The filter cigarettes cost a bit more to produce than the regular and king-size smokes, since it takes two extra machines to put on the filter, executives claim. But, since the filter takes up space in the cigarette normally used for tobacco, there is a saving in the amount of tobacco used.

With the growth of the filter-tip cigarettes, there has been more demand for different types of burley leaves—generally those that grow higher up on the stalk—to "get more flavor through the filter," company officials say. The resulting demand has pushed prices of this type of leaf up close to the levels of the other

main tobacco staples. "The prices have tended to equalize," says William L. Perry, vice president and treasurer of Liggett & Myers Tobacco Co., Inc.

Although conceding they use "heavier bodied and higher flavored" tobacco in the filtered brands, the companies don't like to say the quality is inferior to nonfiltered blends. "Our Hit Parade blend is the same grade as used in Lucky Strike and Pall Mall," American Tobacco emphasizes.

The filtered smokes make up about 40 percent of the total cigarette market, industry observers believe, and some estimate that share may increase to near 50 percent by the year end.

The price increases have put the king-size brands in the same price range as the filters, with the regular size about a cent behind. Some tobacco officials speculate that the reduced price difference might hasten the downfall of the nonfilter cigarettes. "I don't see how the nonfilters and the filters can sell at the same price for very long," one executive confides. "It isn't realistic pricing." Prior to the increase, the filter-tip brands generally sold for about a cent more than the king-size lines and 2 cents more than the regular size.

SALES STILL ON RISE

Sales of cigarettes still appear to be on the upswing. A report in March and another earlier this month linking cigarette smoking with lung cancer "haven't hurt our sales at all," one company treasurer states.

R. J. Reynolds' sales "are up from last year," Mr. Galloway says. American Tobacco says its sales are "holding up well." O. Parker McComas, president of Philip Morris, said recently his company's sales were running about 10 percent ahead of 1956 levels.

Some of the sales increases can be attributed to the growth of the higher priced filtered brands. The Internal Revenue Service, which counts the number of cigarettes on which the manufacturers pay taxes rather than dollar volume, reports cigarette smoking for the first 4 months of this year was 5 percent ahead of the like 1956 period. This figure could be swelled a bit by new brands coming into the market. Example: P. Lorillard Co.'s Newport and Liggett & Myers' Oasis methol-filter-tip brands are being rushed to retailers.

If the better sales trend continues, it could be a record year for cigarette sales, topping the 394.1 billion cigarettes consumed in 1952. In 1953 and 1954, consumption of the smokes dropped off sharply when the reported cigarette-lung cancer link was first publicized. The Internal Revenue Service statistics show that tax-paid removals of cigarettes from domestic factories dipped to 386.8 billion units in 1953 and to 368.7 billion in 1954. The uptrend began in 1955, when 382.1 billion cigarettes were smoked, and reached 391.6 billion last year.

EXHIBIT 29

CONGRESS OF THE UNITED STATES,
HOUSE OF REPRESENTATIVES,
Washington, D. C., July 25, 1957.

Hon. JOHN A. BLATNIK,
Chairman, Subcommittee on Legal and Monetary Affairs, Committee on Government Operations, House of Representatives, Washington, D. C.

DEAR MR. CHAIRMAN: Pursuant to the authorization which you so graciously extended to me, I am transmitting herewith letter from Dr. Milton B. Rosenblatt directed to Dr. Leroy E. Burney, Surgeon General, Public Health Service, dated July 15, 1957, for incorporation in the record of the hearings which your subcommittee is presently conducting with respect to certain claims of filtered cigarette manufacturers and what action has been taken by the Federal Trade Commission in protecting the public from false and misleading advertising.

In view of the rather extensive testimony that has been received concerning the probabilities that cigarette smoking bears a causative factor relationship to cancer of the lung, and knowing that you would want to reflect as comprehensively as possible the pro and con viewpoint of the experts in the field, I believe that Dr. Rosenblatt's statement is most pertinent.

Dr. Rosenblatt is associate professor of medicine at New York Medical College and visiting physician and chief of the chest clinic at New York City Hospital.

Dr. Rosenblatt, with Dr. James R. Lisa, director of the pathology service, New York Department of Hospitals, is author of an authoritative medical book entitled "Cancer of the Lung—Pathology, Diagnosis, and Treatment," published

by the Oxford University Press, New York City, 1956. Six other medical authorities contributed chapters to this book on aspects of the disease in which they specialize.

Dr. Rosenblatt is also a fellow of the American College of Chest Physicians, the American College of Cardiology, the New York Academy of Medicine, and the American Medical Association.

The Rosenblatt-Lisa book has received favorable notice in medical and scientific journals both here and abroad.

Both Dr. Rosenblatt and Dr. Burney have approved the insertion of this letter in the record.

Appreciating your courtesy, I am,

Sincerely,

JOHN C. WATTS.

NEW YORK, N. Y., *July 15, 1957.*

Dr. LEROY E. BURNEY,

Surgeon General, United States Public Health Service,

Washington, D. C.

DEAR DR. BURNEY: In an article in the New York Times on July 13, 1957, statements from my book Cancer of the Lung were quoted in regard to your taking an official position on the etiological role of smoking in lung cancer.

There is no question of the responsibility of the Public Health Service in bringing health facts to the attention of the health professions and the public. Should not this responsibility be extended to include a presentation of all the facts from responsible sources? In an issue which seriously involves the public's welfare and which is so highly controversial, the official promotion of one point of view denies the public the opportunity to evaluate the data which reject the proposition that a causal relationship between smoking and lung cancer has been established. May I summarize some of the facts:

1. Cancer of the lung has been recognized for more than 150 years antedating the popularity of cigarette smoking by more than a century.

2. In hospitals interested in the disease, and/or with routine autopsies, there has been no real increase in lung cancer with respect to total cancers. This holds for the 19th century as well as for the 20th century. For example, in a survey of the hospitals conducted by the London County Council, the percentage of lung cancer to total cancers was 25 in 1936 and 27.1 in 1947.

3. The attempt to implicate tobacco was first made in this country by Adler in 1912. Inasmuch as cigarette smoking had not yet achieved popularity, the onus was placed on cigars. With the greater consumption of cigarettes in later years, the blame was gradually shifted to cigarettes finally culminating in the recent barrage of statistical epidemiological studies conducted on the grand and loud scale.

4. A statistical association does not imply a cause and effect relationship, particularly, when the validity of the conclusions is seriously questioned by such responsible statisticians as Joseph Berkson of the Mayo Clinic or Edward Lew of the Metropolitan Life Insurance Co.

5. Bronchogenic carcinoma has never been produced by tobacco or its products in any experimental animal despite the multiplicity of attempts. Dr. Greene, of Yale, who has devoted a lifetime to experimental cancer research found no causal relationship between smoking and lung cancer. Actually, he found tobacco tar to be among the lowest of the carcinogenic agents used experimentally. The production of skin cancer by tobacco products is of little significance, particularly, when reputable investigators could not reproduce the experiment.

6. History has repeatedly demonstrated that whenever an inaccessible cancer becomes accessible, the incidence automatically increases. Prior to 1930, the clinical facilities for the diagnosis of lung cancer were few and far between even in thoracic disease hospitals. The perfection of diagnostic tools (exfoliative cytology, bronchoscopy, radiology and exploratory thoracotomy) occurred within the past 25 years. This is obvious to all who have worked through this era. Many hospitals now perform more bronchoscopies per day than they did per year a quarter of a century ago. The records of a world-famous cancer institute show that its staff recognized only about half a dozen lung cancers annually before 1930.

7. Lung cancer is a disease of older age groups. There are more older people among us and more potential candidates for lung cancer. The total number of cases is increasing but the rate of increase is slowing down and will eventually

be stabilized like laryngeal or other upper respiratory cancers in which diagnostic methods have been long established.

8. The nasopharynx and the larynx are greatly exposed to tobacco products and if tobacco is a carcinogen for the lung, it should also act on the upper respiratory passage. However, no statistical association has been demonstrated between increased consumption of cigarettes and increased incidence of upper respiratory cancer. In 1940, the United States death rate from laryngeal cancer was 1.1 per 100,000 and in 1950, it was 1.2 per 100,000.

9. The predominance of the disease in males has lent support to the smoking theory on the assumption that men are the heavier smokers.[1] Be that as it may, women have been smoking prodigiously for a great many years. If smoking were a factor in lung cancer, the ratio between male lung cancers and female lung cancers should be gradually decreasing over the years. However, although the disease is increasing in females, the ratio is always predominantly in favor of the male suggesting that the greater susceptibility of the male is based on more basic sexual factors.

10. The prevalence of lung cancer is more related to diagnostic facilities than it is to smoking, air pollution, or other urban factors. No matter how remote the community, the establishment of a diagnostic center inevitably results in a greater incidence of lung cancer. A survey in east Pakistan disclosed 20 new cases over an 18-month period; only 2 of the new cases were heavy smokers, and these used the hukka, a water-filtered pipe. The recent increase of lung cancer throughout the world has occurred in countries where cigarette consumption has increased (United States, Canada, Italy), and it also occurred in countries where cigarette consumption remained fairly stationary (Germany, Austria, Turkey, Japan). National origins have also been implicated in lung cancer. In an epidemiological study in New Zealand, Eastcott found a higher incidence of lung cancer among those who were originally born in England, the risk of contracting lung cancer being proportionate to the years in which they were exposed to the English environment. Smoking was absolved, inasmuch as the smoking habits of both countries are similar.

11. The epidemiological studies, unsupported by corroborative experimental data, show only the number of smokers who have lung cancer. Any other conclusion is speculative. In a disease like cancer, in which so little etiological data has been established, it is presumptive to rely on epidemiological studies which are questioned by statistical experts and refuted by clinical experience.

Until the etiology of lung cancer has been firmly established, more will be gained by a circumspect point of view. Medicine has had its share of colossal blunders in the past. Not too long ago, tuberculosis was attributed to night air, and lung cancer to tuberculosis.

Respectfully submitted.

MILTON B. ROSENBLATT, M. D.

P. S.—References are available at the end of Chapter II: Cancer of the Lung, Oxford University Press, 1956.

EXHIBIT 30A

MAYO CLINIC,
Rochester, Minn., July 21, 1957.

Hon. JOHN A. BLATNIK,
Chairman, Legal and Monetary Subcommittee,
House Committee on Government Operations,
Washington, D. C.

SIR: I refer to your request, transmitted through Mr. Curtis Johnson, to appear before your committee on July 23, 1957. I regret that professional obligations do not permit me to attend this hearing. In lieu of my personal appearance, I am hereby transmitting this statement.

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[1] Twenty-six million adult male regular cigarette smokers in United States. Fifteen million adult female regular smokers in United States.

I have studied carefully the evidence that has been advanced for the theory that smoking causes lung cancer. Attached is a paper, which I wrote 2 years ago, pointing out that the evidence put forward up to then was inconclusive. Furthermore, there were some aspects of the findings which indicated that the statistical association of smoking and lung cancer might be spurious, due to selection and invalidity of the data. Since then, more data have appeared, which I have studied carefully. I am, myself, engaged in some related studies, which are not yet ready for publication. I find no reason to modify my previous opinion that the evidence, taken as a whole, does not establish, on any reasonable scientific basis, that cigarette smoking causes lung cancer. On the contrary, I have found more and stronger reasons to doubt this conclusion.

With regard to the contrary opinion expressed by some statisticians, I have full respect for their sincerity and general competence. My criticism is that they have not allowed time for careful, critical analysis of existing data, let alone time in which to consider results of investigations now in progress. Consider only the following facts: The second report of the American Cancer Society study, on which the opinion that smoking causes lung cancer largely rests, has as yet been published only in newspaper form, and will not appear in a technical journal, for scrutiny of scientists, until fall. Then, again, the United States Public Health Service now has underway an extensive statistical study on the subject, for which it has not yet issued even a preliminary report.

The problem of cancer is one of the most baffling ever attacked by science. Time and time again, conclusions as to its cause or cure have been prematurely published, only to be disproved later. This not only discredits science and confuses the public, but does immeasurable harm in creating an atmosphere in which true scientists cannot work effectively. I have no interest except the advancement of scientific knowledge by research. I should not wish to obstruct its progress, or to jeopardize my own reputation to boot, by questioning the conclusion that smoking causes lung cancer, if I did not sincerely believe that there is serious doubt as to the scientific validity of that conclusion.

While I have this opportunity to address your committee, I take the liberty of presenting another thought. I have every respect for the wisdom of this committee, and I consider it commendable in every way that it should be trying to get the facts about smoking and lung cancer, especially since these are germane to the question of cigarette advertising. However, since this is a scientific question, I respectfully call to your attention the fact that we have in this country a body to which one naturally turns for an authoritative opinion on a scientific problem, namely, the National Academy of Sciences. I, therefore, suggest that this committee could obtain a more competent estimate of the scientific evidence linking smoking to lung cancer than is now available, by requesting such an evaluation from the National Academy of Sciences.

A copy of this statement is being sent to Dr. Detlev Bronk, President of the National Academy of Sciences.

Yours respectfully,

JOSEPH BERKSON, M. D.,
Mayo Clinic, Rochester, Minn., Section on Biometry and Medical Statistics.

Enclosure: Published paper, Proceedings, Mayo Clinic, July 27, 1955.

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JOSEPH BERKSON

Book Notices

THE STATISTICAL STUDY OF ASSOCIATION BETWEEN SMOKING AND LUNG CANCER

Joseph Berkson, M.D., Section of Biometry and Medical Statistics

> "In a very real sense the excellence of a person as a statistician may be said to depend largely upon his native ability by some obscure sixth sense to avoid the fallacy of observational selection." Edwin B. Wilson[1]

When I encountered the first of the series of statistical studies on the association between smoking and lung cancer which have recently appeared,[2,3] it immediately recalled to me a prior investigation on the association between tuberculosis and cancer.[4] My thinking on the present question has developed out of a careful study which I had made of the older work. I shall take the liberty of elaborating my analysis by way of reference to that investigation as a base, not only because in the circumstances it is natural for me to do so, but also because I believe it will be instructive.

The investigation referred to was founded on an observation, first made by the renowned pathologist Rokitansky and noted also by others, that tuberculosis and cancer occurring together in the same individual appeared to be very rare. A study was instituted using the records of the Pathological Laboratory of the Johns Hopkins Hospital. The records of 816 cases were isolated in which at necropsy some form of malignant neoplasm had been found. The question was, in the words of the author, "To what extent, in point of frequency, was tuberculosis, active or healed, associated with the presence of malignant neoplasms in the 816 cases of malignancy comprised in this

1. Wilson, E. B.: Morbidity and the Association of Morbid Conditions. J. Prevent. Med. 4: 27-38 (Jan.) 1930.
2. Cutler, S. J.: A Review of the Statistical Evidence on the Association Between Smoking and Lung Cancer. J. Am. Statist. Assoc. 50:267-282 (June) 1955.
3. Dorn, H. F.: The Relationship of Cancer of the Lung and the Use of Tobacco. Am. Statistician 8:7-13 (Dec.) 1954.
4. Pearl, Raymond: Cancer and Tuberculosis. Am. J. Hyg. 9:97-159, 1929.

autopsy material? Were tuberculosis lesions, active or healed, found at autopsy more or less frequently in this group of 816 cases of malignant neoplasms, than such lesions would be expected to be found in an equal number of non-cancerous persons, of the same ages, sexes, and races as the 816 persons composing the cancerous group?"

To answer this question a control group of necropsied persons was set up with which the cancerous group could be compared, in the following manner: The entire set of records pertaining to noncancerous persons, that is the whole of the body of cards carrying the necropsy records minus the 816 cards comprising the cancerous group, were arranged in order of date of necropsy, and similarly the 816 cards of the malignancy group were arranged in order of date of necropsy.* Starting with the earliest card in the cancerous group, the first card in the noncancerous group was found which corresponded in respect to race (colored or white), sex and age, and similarly corresponding cards in the noncancerous group were found for the second and succeeding cards in the 816 of the malignancy group. As the author expressed it, "The final outcome of this procedure was to give 816 cards pertaining to persons who had no malignant tumors at death, but who had, as a group, precisely the same sex, color and age distribution as the 816 persons, taken as a group, who did have malignant tumors at death. Furthermore each person in the control group died at about the same date as his cancerous partner in the group of malignant cases. . . . the control group is selected to agree in one-to-one correspondence with the malignant group in respect of sex, color, age, and date of death. . . . But the composition of the group is completely random in respect of pathological lesions, save for the fact that it contains no case of malignant neoplasm."

The whole weight of the presentation is placed on the presence of a control. At various points in the article the author takes up the possible unrepresentative character of the data as a whole in one or another respect, but always this is met with the argument that the controls as well as the malignancy group are equally unrepresentative. Thus there is some discussion of the possibility that the percentages of tuberculous lesions are perhaps comparatively low both in the malignancy and in the control group. The author points out that the number of cases in which any lesion is found indicating the presence of tuberculosis depends on the zeal with which the search for such lesions is conducted. As respects this he says: "From the point of view of the present study the question of the meticulousness of the search for minute healed lesions of tuberculosis has no relevance. The logical procedure in this study has been throughout to compare specifically diseased (malignancy) and control groups. The procedure of

*For brevity I am presenting only the essential elements of the procedure, not necessarily the actual details of how it was carried out.

the pathologists in looking for and recording tuberculous lesions, active or healed, has been uniform in both groups. . . . Only if it were differential could the absolute number of tuberculous lesions found be significant in the present study."

I have quoted somewhat extensively from the author, to make clear a particular point which it is important for me to establish. Note how analytically careful the author is, how he stresses the importance of a properly constituted control, how he rests his case on the establishment of such a control and how he finally summarizes the objective attained. What Pearl has done here is a paradigm of what, according to widely held opinion, are the essentials for a perfectly performed experiment of statistical comparison. He takes two samples from the identically same population, at random otherwise than that they have been stratified to be comparable in every relevant respect except one, namely that in the one group each individual has a malignant neoplasm, and in the other no individual has a malignant neoplasm. Then he is to compare the two groups in respect of tuberculosis, and if a material difference is found—so the argument goes—what can it be attributed to except that there is some association between cancer and tuberculosis?

What then were the findings as respects tuberculosis in the two groups? The answer is that active tuberculosis was found in only 6.6 per cent of the 816 persons having malignant tumors and in 16.3 per cent of the 816 persons without malignant tumors but of the same race, sex and age as the former group; active tuberculosis occurred more than twice as frequently in the controls as it did in the malignancy group. When the data were segregated into subgroups it was revealed, in the words of the author, ". . . with the utmost clarity and precision, that in each decade of age, and in each sex and race division of the material, the percentage of persons showing active tuberculous lesions at autopsy is markedly higher in the control group than in the malignancy group."

The meticulous analyst, having conclusively shown that tuberculous lesions were less frequent among the cases with malignant tumors than among cases without malignant tumors, says that something else should be looked into. He poses the converse question to that first considered: Are malignant tumors more or less frequent among those with active tuberculous lesions at necropsy than they are among those with no tuberculous lesions at necropsy? After a pertinent analysis he concludes: "The answer is precise and unequivocal so far as concerns the 1,632 autopsies here studied. In each decade of age, over the whole life span, cancer or other malignant tumors occur less frequently in those with active tuberculosis, than in either the non-tuberculous or those with old healed lesions."

The apparently unequivocal conclusion from the study, that tuberculosis and cancer are biologically antagonistic, was accepted with utmost seriousness. An experiment with animals was set up and an extensive program of treating cancer patients with tuberculin was instituted.

Now I shall quickly come to the first anticlimax of the story. Even before the definitive publication of the study, objections were advanced as to the validity of the comparison which had been made. So far as I know and understand, although the critical arguments took different specific forms, essentially they had to do with one central point. Since the comparison was of concomitant lesions found at death, consideration had to be given to the duration of the diseases in question, for an individual had to live long enough with one disease to contract the other if the presence of both was to be found at necropsy. These objections were more intensely discussed after the publication, and as a result of these and perhaps also on the basis of evidence that appeared from experiments, on which Pearl always laid the greatest stress,[5] the author was convinced that the original conclusion was doubtful and he issued what amounted to a retraction, from which I shall briefly quote.[6a] "[A possible interpretation] is that the result is purely fortuitous, the infrequency of association arising from the assumed fact that the time relations of the disease between onset and death are such as to make impossible the complete freedom of joint association which is an implicit postulate of the simple probability theory. Or putting the point less formally, it can be alleged that the reason why persons with cancer are clinically found to have florid tuberculosis less frequently than persons without cancer . . . is because the cancer kills them before there is time for florid tuberculosis to develop."

Although retrospectively I agree that the conclusion reached by Pearl in his first investigation is not correct and that some *ad hoc* criticisms of it which were expressed have force, still, on the basis of very generally accepted principles of statistical procedure it seems to me that he was invulnerably right. If in two "cohorts" of a population, differentiated in respect of only one relevant characteristic x, the finding of an unquestionable difference between the two, in the relative frequencies of a character y, establishes association between x and y, irrespective of the character of the population itself, then Pearl's investigation did establish negative association between cancer and tuberculosis and in fact it was an impeccable example of such a demonstration. It is on the validity of this general principle that the

5. Berkson, Joseph: Review of Introduction to Medical Biometry and Statistics by Raymond Pearl. J. Am. Statist. Assoc. 37:145-147 (Mar.) 1942.
6a. Pearl, Raymond: A Note on the Association of Diseases. Science 70:191-192 (Aug. 23) 1929.

most frequently quoted studies on smoking and cancer rest the conclusion that there is an association between smoking and cancer of the lungs.* If on the other hand the population itself must meet some particular criteria, then I submit that no studies have been made directed at discovering just what these criteria must be, still less have they been definitely set down. I have my own idea, but I recognize that it is a personal opinion, without general statistical authority.

My own idea is that, if an essential biologic association is to be established as a definitive scientific conclusion, that is to say, if it is to be considered *"proved"* the population must not be anything else except an *experimental population.* An association found in a purely statistical investigation made on an existent population, by which I mean an investigation which is retrospective as regards either of the variables concerned, however strongly it may suggest association as a *presumptive* conclusion, is tentative until it is corroborated fully by means of experiment. I am not here referring to "association" in a purely statistical, descriptive sense. If proper study of a given population shows that there is positive correlation between stature and weight, then it is a descriptive fact that tall individuals in that population are on the average heavier than short individuals. But there is no concluding even here that there is a necessary biologic relation between stature and weight; we do not know for instance that the correlation would exist if the population were placed on a different diet. Here I am only restating what Pearl himself asserted:[6a] "Perhaps in the long run it will appear that the chief usefulness of the statistical technique in methodology of science is the not unimportant one of suggesting problems and lines of attack upon problems which must finally be solved, if they ever are solved, by the application of the methods of experiment and observation, or a close and integrated correlation of these methods with the statistical to reach a common end."†

And now I conclude briefly in an autobiographical vein with my analysis of Pearl's study. At the time the study was made I thought

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*Pearl's set up fully complies with the requirements laid down by Cutler,[2] who, commenting on some published studies showing association between smoking and cancer, says: "The sampling techniques used in these case history studies were generally not sophisticated. It may be that the lung cancer cases studied were not representative of all persons with lung cancer and that the controls were not representative of the general population, but these two requirements are not essential. To study the relationship between smoking and lung cancer it is sufficient that the lung cancer cases and controls be drawn from the same population."

†Another quotation from Pearl[4] (1929) is pertinent to the subject under present discussion: ". . . it is of interest to note that as the frequency of the incidence of fatal tuberculosis of the lungs has declined in recent years there has occurred a marked increase in lung cancer, which has been so notable as to attract the attention of pathologists generally."

In considering the significance of the putatively real increase of the rate at which lung cancer is developing in the population, Cutler[2] says: "This increase cannot reasonably be attributed to genetic change in the human population and therefore must be due to environmental factors." It is not obvious why it must be. Individuals reaching the adult cancer ages in current times are, in respect to their constitutional resistance to disease, greatly different from individuals formerly attaining these ages. The marked lowering of death rates in the early ages of life that characterizes the hygiene of the present era has resulted in having in the adult population individuals who in former years would

the logical development on which it was planned was cogent, and when the critical argument was presented on which Pearl's retraction is based, I thought *that* was cogent. But I later came to doubt this also! The particular criticism made was centrally based on the fact that the population concerned was a population of the dead; it was death from cancer that prevented later contraction of tuberculosis, otherwise the tuberculosis would have been found with its representative frequency—that was the argument. I resolved never to study association of diseases in a dead population.

Several years after the events referred to—in the early thirties—I found myself in the position of statistical consultant in a large medical institution. I was amazed at how soon after I took up my duties, and with what frequent recurrence, I was asked questions referring to association between diseases. I believe it was in 1935 that the particular event occurred which I wish to relate. A physician came to me with an idea with which I had by now become generally familiar. He said that he had the impression that cancer of the stomach occurred rarely in persons who had duodenal ulcer, and suggested that we might study the pathologic records statistically to check on the validity of his impression. In this particular instance, after I recited the difficulty about working with a dead population, I suggested that we might make the statistical study using the clinical diagnoses made on examination of living patients, of which we had a very complete cross-index file. It was while working out a numerical example, in which I began with a hypothetical general population in which there was not any association but only random frequencies, that I became conscious of a difficulty that prompted me to review the elementary factors that enter into the comparison by which association is established. I reached the conclusion that there is a fallacy in studying association of diseases in the living as well as the dead clientele of a hospital population, and that basically it was on this fallacy that Pearl's investigation of an association between tuberculosis and cancer had foundered.

Thereafter, when association of diseases was broached as a subject for possible investigation, I presented the possibly fallacious character of the conclusion that could be reached if hospital populations, dead or living, were taken as a base. As I mentioned before, the suggestion of

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have died in infancy or youth. It is likely that they carry with them a measure of the lower resistance to disease which was the constitutional basis of their former early death. Frost,[6b] in a beautiful and important paper, has shown how high mortality from tuberculosis in later life is related to *escape* from excessive mortality in earlier life. This effect undoubtedly is present with other diseases and especially with diseases of the same tissue resistance. It is entirely possible and even likely that at least part of the increase in death rate from lung cancer which has been recently noted is attributable to deaths in adulthood from this disease of individuals who have not been eliminated, as in former years they would have been, by death in early life from tuberculosis or some other pulmonary malady.

6*b*. Frost, W. H.: The Age Selection of Mortality From Tuberculosis in Successive Decades. Mulbank Memorial Fund Quarterly *18*:61-66 (Jan.) 1940.

association between diseases was periodic and my lecture on the subject became routine. In 1946 my recital impressed one of my hearers and I was urged to publish the analysis. It happened that the case presented referred to a possible positive association between diabetes and cholecystitis, and it was in terms of this example, which was of then current interest, that I did publish it,[7] but what I had in my mind was tuberculosis and cancer. The point of my analysis in detail can be studied in the article. For the moment it will suffice to say that, if the subpopulation of the hospital which is used for the comparison of the incidence of a disease y in a group x and its control not-x is not representative in the ratio of the marginal totals of x and not-x of the corresponding ratio in the general population, then, except under special circumstances, association will appear in the hospital population even if none exists in the general population from which the hospital population is drawn.

Although my article[7] has been referred to in the literature on cancer and smoking as possibly relevant in evaluating those studies referred to as "retrospective," in which patients in a hospital having lung cancer were compared in respect of incidence of smoking with a control group from the hospital not having lung cancer, it has not been cited so far as I know, in connection with the prospective studies which have been published, in which individuals differentiated initially as regards smoking and not smoking are followed to ascertain the relative rate of death from cancer. It was Professor Donald Mainland[8a,b] who suggested to me rather pointedly what I had thought of only vaguely—that it *might* apply also to the prospective studies. We may think of the population sampled in these studies as replacing the hospital population, so far as it is not the entire reference population in which the study of association is the real objective but only a sample of it. In the prospective study of Doll and Hill,[9] for instance, the population actually composing the material of comparison is only a certain portion of the physicians registered at the time of inception of the study and these are only a portion of the general population. In the prospective study of Hammond and Horn[10] the population concerned in the analysis is an unknown fraction of the potential of solicited friends of member workers of the American Cancer Society who answered the questionnaire addressed to them respecting their smoking habits. On more serious consideration than I had first given the idea, it suggested itself to me that a simple mechanism may be

7. Berkson, Joseph: Limitations of the Application of Fourfold Table Analysis to Hospital Data. Biometrics Bull. 2:47-53 (June) 1946.
8a. Mainland, Donald: Personal communication to the author.
8b. Mainland, Donald: The Risk of Fallacious Conclusions From Autopsy Data on the Incidence of Diseases With Applications to Heart Disease. Am. Heart J. 45:644-654 (May) 1953.
9. Doll, Richard and Hill, A. B.: The Mortality of Doctors in Relation to Their Smoking Habits: A Preliminary Report. Brit. M. J. 1:1451-1455 (June 26) 1954.
10. Hammond, E. C. and Horn, Daniel: The Relationship Between Human Smoking Habits and Death Rates. J.A.M.A. 155:1316-1328 (Aug. 7) 1954.

operating which will produce spurious association in the selected population similar to that referred to in the study of association of diseases in a hospital population. An artificially simple example of such an effect is the following:

Suppose there is some reference population which we wish to study in order to ascertain whether there is association between smoking and death rate. We will suppose that in this population there is in fact no association, but that this is unknown to us. We visualize the reference population as composed of two groups:

Group I: This is the element of the population which at the time of the beginning of the study is in various degrees of serious ill health; for the most part these individuals are destined to die within a year. If the symptoms of an individual in this group are sufficiently prominent, such an individual may eliminate himself or be eliminated from the investigation deliberately or tacitly by the investigator. It is assumed that an individual belonging to this group is eliminated or not eliminated from the investigation entirely on the basis of the condition of his health, and quite independently of whether he is or is not a smoker. That is, for this group of individuals with ill health, health is, so to speak, dominant over any other factor that may in general affect whether an individual is selected for the investigated population. We will postulate for a hypothetical example that group I comprises 3 per cent of the reference population, that the death rate in the year for this group is 99 per cent and that only 50 per cent of group I are recruited into the investigation.

Group II: This is the remaining portion of the reference population and the individuals comprising it are free of serious ill health, in the sense that no individual in this group is experiencing symptoms which could be the basis of his elimination from the investigation. Some few members of this group do die during the year from diseases which at the time of the survey were not exhibiting prodromal symptoms, and arbitrarily we set the death rate for this group in the example at 0.03 per cent. In this group II, we assume, whether an individual comes into the investigation or not depends solely on whether he is a smoker, and we will assume that there is a greater tendency for a smoker to eliminate himself than for a nonsmoker to do so. Of the nonsmokers in this group, we will say that 99 per cent respond, while of the smokers in this group only 65 per cent respond and are included in the investigation.

We consider a reference population of which 80 per cent are smokers and in which the over-all mortality rate for the year is 3 per cent. For this population there is given in table 1, A and B respectively, the constitution of the reference population and that of the selected population. A summary of the comparison is given in table 2.

Table 1
Statistical Association Produced by Interaction of Competitive Risks of Selection: Hypothetical Case

A—Reference population, cohort of 100,000

Smoker	Exposed			Deaths			
	I	II	Total	I	II	Total	Rate per cent
No	600	19,400	20,000	594	6	600	3.0
Yes	2,400	77,600	80,000	2,376	24	2,400	3.0
Total	3,000	97,000	100,000	2,970	30	3,000	3.0

B—Selected population, expected number

Smoker	Exposed			Deaths			
	I*	II	Total	I	II	Total	Rate per cent
No	300	19,206†	19,506	297	6	303	1.6
Yes	1,200	50,440‡	51,640	1,188	16	1,204	2.3
Total	1,500	69,646	71,146	1,485	22	1,507	2.1

*Of group I individuals (the ill) in the reference population, 50 per cent are recruited, independent of whether they are smokers.
†Of group II's 19,400 nonsmokers, 99 per cent are recruited.
‡Of group II's 77,600 smokers, 65 per cent are recruited.

Table 2
Summary of Comparison, Reference and Selected Populations, From Table 1

	Reference population, per cent	Selected population, per cent
Proportion of reference population	100	71
Proportion of smokers	80	73
Death rate, over-all	3.0	2.1
Among nonsmokers	3.0	1.6
Among smokers	3.0	2.3

It is to be observed that while there is no association between smoking and death rate in the original reference population, an appreciable positive association appears in the selected population, the death rate for smokers being 2.3 per cent while that for the nonsmokers is 1.6 per cent. The origin of the appearance in the hypothetical table of spurious correlation, it should be noted, is not a supposed tendency operating directly for an individual to eliminate himself for reasons of ill health with greater probability if he is a smoker than if he is a nonsmoker; the individuals who eliminate

themselves for reasons of ill health are randomly taken from the population so far as smoking is concerned. Rather it is the simultaneous operation at different intensities of the selection on both smoking and deaths. I do not mean that it is solely by exactly this mechanism that selection can produce spurious correlation in prospective studies. It is only a "statistical model," and has been presented because wide acceptance of the prospective studies as probative appears to be based on the idea that with this method of investigation no fallacy is possible. More broadly considered, wherever it is found that selection is operating, it is gratuitous to *assume* that selection does not affect differentially different strata of the population sampled, and therefore one must be prepared to find differences between corresponding strata in the sample, even if there are none in the original population.

Table 3
Cigaret Smoking: Data of the American Cancer Society Study[10]

Age, years	Percentage	
	Current cigaret smoker	Cigaret smoker some time in life
50-54	51.7	66.6
55-59	45.0	60.5
60-64	37.0	51.6
65-69	28.0	40.7
Summary totals		
50-59	48.5	
50-69	42.8	57.4

The earmark of selection is the appearance in the selected table of an unrepresentative proportionality in the constituents of the marginal totals In the hypothetical case represented in the table we see that the proportion of smokers is smaller in the selected population than it is in the parent reference population and that also the proportion of deaths is smaller. How do these matters stand in the data of the American Cancer Society study of Hammond and Horn,[10]* from which these authors have concluded that an association between smoking and cancer of the lungs has been *proved*, an association which they attribute to a positive causal relationship? Since cigaret smoking is most clearly indicted in their study, pipe and cigar smoking being

*This study is chosen as the one to examine because it is a prospective study where competitive selection hitherto has been thought not to be relevant, because it provides the most detailed findings of any article published to date, particularly as respects age-specific death rates and because it is the one from which the most definite conclusions have been drawn.

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less if at all involved, and also because statistics on cigaret smoking are more easy to come by, I shall concern myself only with this form of smoking.

In the study of Hammond and Horn, data are available for current cigaret smokers and also for men who ever were cigaret smokers in their lives. In table 3 are given percentages representing the prevalence of cigaret smoking among males comprising the population of the study of Hammond and Horn, as extracted from their data. For males in the decade of age 50 through 59 years the prevalence of current cigaret smokers is 48.5 per cent.* In contrast with this a Gallup poll[11] for June, 1954, gives as a national average 57 per cent for current cigaret smokers among males in this age group.† In the total population of the American Cancer Society study, which is composed of white males in the age range 50 through 69 years, the proportion of current cigaret smokers is 42.8 per cent. Surveys referring to all males for various localities in the United States show a median of from 60 to 65 per cent (these, however, must be presumed to refer on the average to younger men than represented in the study from the American Cancer Society). The proportion of men in the American Cancer Society study who reported themselves as having been cigaret smokers at some time in their lives is 57.4 per cent. As against this, the Bureau of Research Information estimates that for the general population this figure is about 75 per cent.[11] Although statistics for exact comparisons are not available, it appears to me that there is little doubt, on the basis of what has been noted, that in the response to the call of the workers of the American Cancer Society for volunteers to enter the projected survey on smoking and cancer there was a "tendency" for cigaret smokers not to enter themselves, with the result that the sampled population is "selected," that is unrepresentatively weighted with nonsmokers of cigarets.

Now we will consider the other variable concerned, deaths. For this purpose I have assembled in table 4 a comparison of the death rates in the population of the American Cancer Society study, calculated on an annual basis, and those for United States white males for 1952, which are the latest rates available in the detail required. This table gives a comparison for deaths from all causes and also for specific causes so far as these can be evaluated from the data reported in the publication of Hammond and Horn. It is seen that, without exception, the age-specific death rate from all causes, and from each specific

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11. Clarke, A. G.: Bureau of Research Information, New York. Personal communication to the author.

*Calculated by dividing the number given in the study as currently smoking cigarets (their table 5) by the total number in the corresponding age group (their table 3).

†This poll excluded from the count of cigaret smokers any individuals who were also cigar or pipe smokers; the percentage of 57 is therefore to be considered an underestimate of current cigaret smoking in this age group of males, in the sense in which it is represented in the quoted statistics of the study of Hammond and Horn.

Table 4
Comparison of Death Rates for the United States With Those in the American Cancer Society Study*

Age, years	Death rate, per 100,000 per year														
	All causes			Cancer, lungs			Other cancer			Coronary heart disease			Other diseases		
		A.C.S.			A.C.S.			A.C.S.			A.C.S.			A.C.S.	
	U.S. white males	Total	Non-cigaret smokers	U.S. white males	Total	Non-cigaret smokers	U.S. white males	Total	Non-cigaret smokers	U.S. white males	Total	Non-cigaret smokers	U.S. white males	Total	Non-cigaret smokers
50-54	1,206	838	602	50	32	6	148	100	91	426	371	230	582	335	274
55-59	1,891	1,345	1,072	85	61	19	259	178	158	701	614	440	846	491	454
60-64	2,793	1,925	1,444	114	56	6	406	298	225	1,018	851	592	1,255	719	622
65-69	4,089	2,905	2,662	127	79	45	609	416	358	1,438	1,247	1,115	1,915	1,163	1,144

*The annual death rates for the population of the American Cancer Society study were calculated by assuming a uniform exposure of 20 months (median) for all individuals in that population. The rates for United States white males are for 1952 obtained from reference 12 supplemented by a special tabulation referring to deaths from lung cancer for which I am indebted to Mr. I. M. Moriyama.

cause, is materially lower for the population of the American Cancer Society study than it is for the corresponding white male population of the United States. I have included in the tabulation the death rates of the nonsmokers of cigarets of the American Cancer Society study. These death rates are seen to be strikingly low compared with those of the United States white male population. The nonsmokers of cigarets in the population of the American Cancer Society study are evidently a lot of phenomenally hardy men. One may reasonably conclude from these comparisons that in the response to the call for individuals to enter the survey, men in relatively poor health tended to be excluded, so that the investigated population is selected favorably as respects death rate from all causes, and from specific causes including deaths from cancer.

Thus the data of the American Cancer Society study taken as a whole exhibit prima facie evidence that they have been subjected to a kind of selection which can produce association in the data studied, such as in fact was found in these data, even if the association does not exist in the primary reference population.

The authors of the study themselves appear to be conscious of the fact that selection in their data may be operating which can produce an artificial appearance of association. In the course of describing their procedure they say: "In planning the study, we had anticipated that deaths that occurred during the first few months would have to be excluded from the analysis in order to avoid the theoretical possibility of a bias influencing the relationship between death rates and smoking habits. In order to test for this, we have made an analysis separately for deaths that occurred in each of three six month periods as well as . . . prior to May. In all four periods, regular cigarette smokers had higher death rates than men who had never smoked. Because of this finding, it was decided to base the present report on all deaths. . . . As a matter of fact, the relationships between cigarette smoking and death rates were greater in the last two six month periods than in the earlier periods."

I do not believe that in the last part of the quoted passages, the authors meant to imply that cigaret smoking, as a direct or mediate cause of death, was increasing in severity over the several months reviewed. It appears rather that their observations provide pointed evidence that artificial selective factors were operating in the study which could effect an apparent association between cigaret smoking and death rate. Whatever were the unknown selective forces which caused the relationship of smoking and death rate to appear to be

12. U. S. Department of Health, Education and Welfare, Public Health Service, National Office of Vital Statistics: Deaths and Death Rates for 64 Selected Causes, by Age, Race, and Sex. In: Vital Statistics-Special Reports: National Summaries. 40:57-103 (Dec. 21) 1954.

increasing in the last two 6-month periods, these factors were presumably operating also at the beginning of the investigation. If they could bring about an apparent increase of association, it is possible that they may be responsible for the appearance of an association in the first instance, and that what is exhibited in the findings of the study is an elaboration of the detailed effects of this selection. The fact that the exact mechanism of such selective association is not readily visualized is not an adequate reason for considering the suggestion of its possible existence to be—as it has been characterized—"farfetched." The operation of selective forces in statistical investigations can be a very subtle process, and no factual studies have been made to answer the question of their possible importance for the present situation. Opinions expressed of their irrelevancy appear to be based wholly on conjecture.*

Nor is it conclusive that the considerable number of statistical studies that have been published[2,3] all agree in showing an association between smoking and cancer of the lungs. On the contrary, undeviating consistency of statistical results all in support of the same conclusion is in some circumstances the hallmark of spurious statistical correlation. If correlation is produced by some elements of the statistical procedure itself, it is almost inevitable that the correlation will appear whenever the statistical procedure is used. It was notable at the time of issue of Pearl's report showing a negative correlation between tuberculosis and cancer, previously referred to, that the negative correlation was shown without exception, not only in the data as a whole, but in each subgrouping by race, sex and age.† The question naturally posed itself: If the antagonism between tuberculosis and cancer is so consistently and strongly operative that it is obvious whenever statistical data are assembled for examination, even admittedly imperfect data, why is the corresponding pathologic process not evident in individual cases? I confess that I entertain similar misgivings with respect to the significance of the uniformly consistent statistical results showing an association between smoking and cancer, so out of line with lack of parallel biologic findings in individuals. I shall return to this point later.

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*The analytical statistical problem dealt with can be considered as an aspect of the general problem of "competitive risks" as formulated by Fix and Neyman[13] and by Neyman.[14] The mathematics of the problem seems to have received little attention; indeed even the existence of the problem is not widely appreciated.

†The finding itself was later corroborated by other workers[15] but with a different interpretation.

13. Fix, Evelyn and Neyman, Jerzy: A Simple Stochastic Model of Recovery, Relapse, Death and Loss of Patients. Human Biol. 23:205-241 (Sept.) 1951.
14. Neyman, Jerzy: First Course in Probability and Statistics. New York, Henry Holt and Company, 1950, 350 pp.
15. Carlson, H. A. and Bell, E. T.: A Statistical Study of the Occurrence of Cancer and Tuberculosis in 11,195 Postmortem Examinations. J. Cancer Research 13:126-135 (July) 1929.

ANCILLARY EVIDENCE

The investigations under consideration had their origin in an idea that there is an association between smoking and cancer of the lungs; and although now a possible relationship of smoking to coronary heart disease and even to other diseases is being broached, the relation to lung cancer is still generally the one of central current interest. In the hypothetical development of the origin of the selective association described above, specific diseases are not involved except so far as some causes of death will, more than others, have prodromal symptoms that have some particular reference to the investigation in hand and so can be the basis for the selection into or out of the experimental population.* It is pertinent therefore to examine the data of the American Cancer Society study, as regards the relation of smoking history to deaths from other diseases than lung cancer. In table 5 is shown a comparison, between the cigaret smokers and nonsmokers of cigarets, of death rates from all causes and from various specific causes. An examination of the table shows that the death rates, not only from cancer of the lungs but also from cancer other than cancer of the lungs and from coronary heart disease, are greater among the smokers than among the nonsmokers of cigarets.† Perhaps more interesting, however, is the fact that the death rates from causes other than either cancer (of any organ) or coronary heart disease are also higher for the cigaret smokers than for the nonsmokers of cigarets.‡ In short, so far as the published data of the American Cancer Society study make it possible to tabulate death rates by specific diseases, it appears that the death rates from each specific disease are higher among the cigaret smokers than among the nonsmokers of cigarets.

Do then the statistics of the American Cancer Society study support the hypothesis that cigaret smoking causes cancer of the lungs? In one sense they do, since the age-specific death rates from cancer of the lungs for the cigaret smokers in the population are higher than the death rates from cancer of the lungs among the nonsmokers of cigarets. In another sense, the results do not corroborate this specific idea, for they prove too much. If the finding of a higher death rate

*Deaths from accidents, for instance, could not artificially appear to be related to smoking by way of the selective mechanism which I described, since one could not have a present selection on the basis of an unknown occurrence of the future.

†A comparison with respect to death rates from cancer other than lung cancer, and for coronary heart disease is given by Hammond and Horn. The death rates from causes other than cancer or coronary heart disease were computed from data in their publication.

‡This fact, as well as some other statistics given in the present paper, has already been noted by Arkin.[16] The present analysis, however, was completed before the appearance of his publication and anything in common between them has been arrived at independently.

16. Arkin, Herbert: Relationship Between Human Smoking Habits and Death Rates: An Analysis of the American Cancer Society Survey. Current Med. Digest 22:37-44 (Apr.) 1955.

from cancer of the lungs among smokers in the population studied is proof that smoking causes cancer of the lungs, the finding that the death rate is higher for cancer other than cancer of the lungs is proof that smoking causes other cancer too. And since the death rate is

Table 5
Death Rates, Cigaret Smokers and Nonsmokers, Specific Diseases, American Cancer Society Study*

Age, years	Death rate, per 100,000 per year											
	All diseases		Cancer, lungs		Other cancer		Coronary diseases		Other diseases			
	Cigaret smokers		Cigaret smokers		Cigaret smokers		Cigaret smokers		Cigaret smokers			
	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes		
50-54	602	955	6	46	91	103	230	442	274	365		
55-59	1,072	1,523	19	89	158	192	440	727	454	515		
60-64	1,444	2,375	6	103	225	367	592	1,095	622	811		
65-69	2,662	3,259	45	127	358	502	1,115	1,439	1,144	1,191		

*Twenty months' exposure (median) assumed for all individuals in the American Cancer Society study. The rates for cancer of the lungs and for "other diseases" were calculated from figures given in the publication of the study.[10]

higher for coronary heart disease, smoking causes coronary heart disease; and since it is higher for all other specific diseases for which the statistics have been studied, smoking causes some or all of these diseases. It does not seem unfair to say that, so far as the American

Cancer Society study is concerned, the hypothesis of causation of cancer of the lungs by smoking stands or falls with the conclusion that smoking causes also other cancer and also coronary heart disease and also other diseases than either cancer or coronary heart disease. Indeed the question raised by the findings in the American Cancer Society study of higher death rates among cigaret smokers is not, "Does cigaret smoking cause cancer of the lungs?" so much as it is, "What disease does cigaret smoking not cause?"

The association with cigaret smoking of deaths from other diseases than lung cancer is shown in the American Cancer Society study, not only in the finding at all specific ages of a higher death rate from these other diseases for smokers than for nonsmokers but also in a

Table 6

Death Rates, Other Causes Than Cancer or Coronary Heart Disease, in Relation to Amount of Current Cigaret Smoking, American Cancer Society Study*

	Death rate, per 100,000 per year			
	Age, years			
Cigaret smoking	50-54	55-59	60-64	65-69
Never smoked	295	460	586	1,078
Less than ½ pack a day	353	434	920	1,223
½ to 1 pack a day	368	498	812	1,310
1 pack or more a day	372	613	865	889

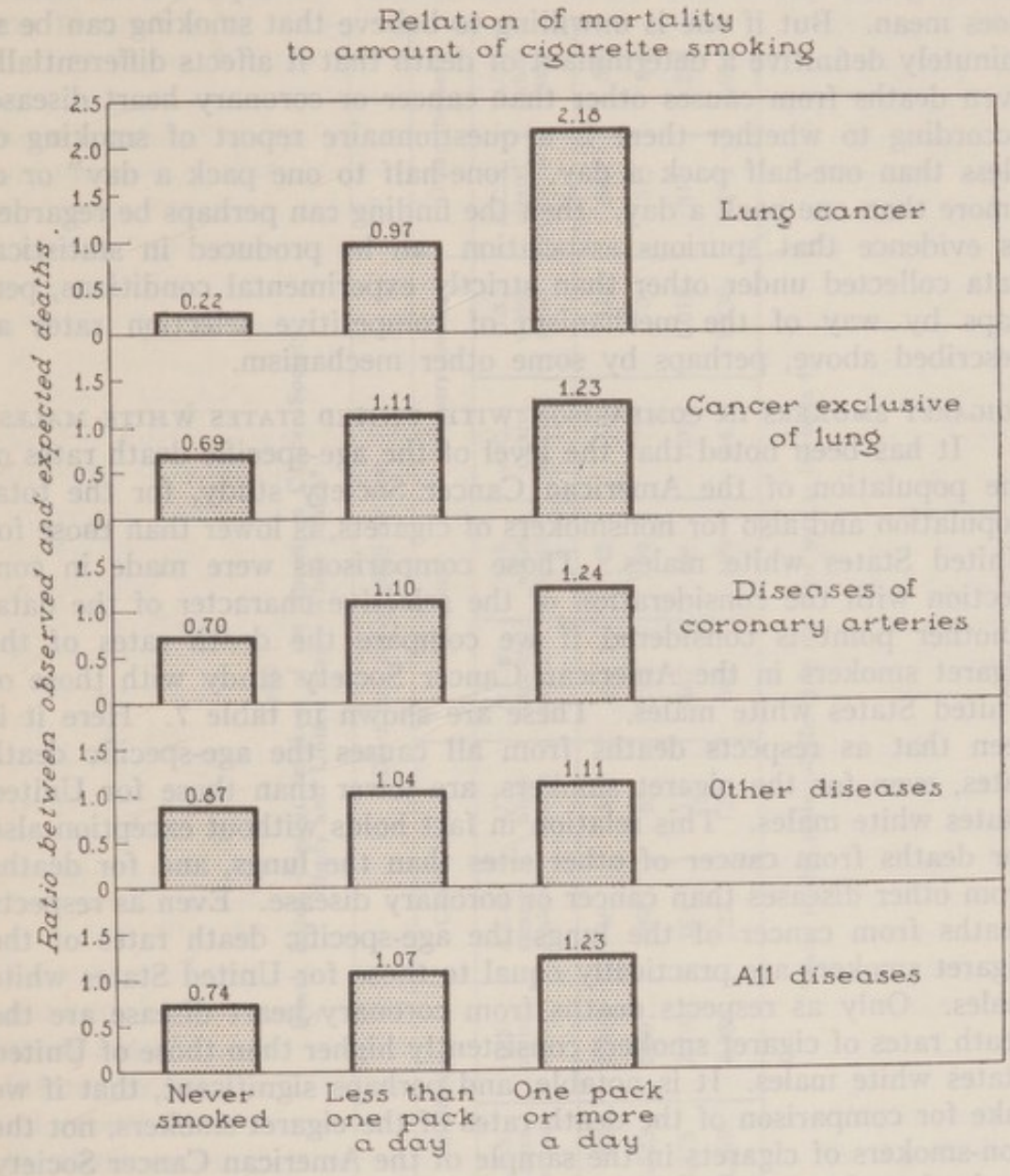
*Twenty months' exposure (median) assumed for all individuals. The calculations of the death rates in this table are based on the individuals who reported themselves as current cigaret smokers, and those who reported themselves as never having smoked, as are the similar calculations in the publication of Hammond and Horn. The rates for the group "Never smoked" are not the same as those given in table 4 and table 5 for non-cigaret smokers, because the last include smokers of cigars or pipes or both.

positive correlation between the death rate and the *amount* of smoking. In table 6 is shown the death rate from "other causes," for the group "never smoked" and for each of the three classes of amount of current cigaret smoking recorded in the American Cancer Society study, for each 5-year age class of the data. It will be seen by examination of the details of the table that the death rate increases steadily with increase of amount of smoking, in each age group, with a few exceptions that reasonably may be attributed to sampling fluctuation.

The increase of death rates with increase of amount of reported cigaret smoking may be exhibited clearly by use of the index employed by Doll and Hill,[9] consisting of the ratio of the total observed to expected deaths, for each class of amount of smoking. These are shown in the figure. It may be seen that the deaths from cancer of other sites than the lungs, from diseases of the coronary arteries and from "other

diseases," as well as deaths from lung cancer, all show a continuous rise of death rate with increase of amount of smoking.

Doll and Hill[9] reasonably considered the finding of association between death rate and *amount* of smoking to be the strongest evidence



Increase of mortality with increase of amount of current cigaret smoking in data of the American Cancer Society study.[10] Mortality measured in terms of ratio between observed and expected deaths. (Method of Doll and Hill)[9] Amount of smoking in three groupings; the amounts measured as "less than ½ pack a day" and "½ to 1 pack a day" have been combined because of a curious reversal of regression in the original data as respects these two amounts, in the case of deaths from cancer. (The regularity of the reversal in the specific age groups and the fact that it occurs only with the cancer deaths suggest a clerical transposition.)

A positive regression of mortality with amount of smoking is shown for all specific disease groups, including other diseases than cancer or coronary heart disease.

of a real association, and from general intuitive considerations this would appear to be cogent. The appearance in the American Cancer Society study of a positive correlation between amount of cigaret

smoking and death rate from causes other than lung cancer, including deaths from cancer of other sites, coronary heart disease and other diseases, would from such considerations be considered cogent statistical demonstration of a meaningful, even causal, association between smoking and death from these other causes. Perhaps this is what it does mean. But if one is unwilling to believe that smoking can be so minutely definitive a determinant of death that it affects differentially even deaths from causes other than cancer or coronary heart disease, according to whether there is a questionnaire report of smoking of "less than one-half pack a day," "one-half to one pack a day" or of "more than one pack a day," then the finding can perhaps be regarded as evidence that spurious association can be produced in statistical data collected under other than strictly experimental conditions, perhaps by way of the mechanism of competitive selection rates as described above, perhaps by some other mechanism.

CIGARET SMOKERS IN COMPARISON WITH UNITED STATES WHITE MALES

It has been noted that the level of the age-specific death rates of the population of the American Cancer Society study, for the total population and also for nonsmokers of cigarets, is lower than those for United States white males. Those comparisons were made in connection with the consideration of the selective character of the data. Another point is considered if we compare the death rates of the cigaret smokers in the American Cancer Society study with those of United States white males. These are shown in table 7. Here it is seen that as respects deaths from all causes the age-specific death rates, even for the cigaret smokers, are lower than those for United States white males. This relation in fact holds without exception also for deaths from cancer of other sites than the lungs, and for deaths from other diseases than cancer or coronary disease. Even as respects deaths from cancer of the lungs the age-specific death rates of the cigaret smokers are practically equal to those for United States white males. Only as respects deaths from coronary heart disease are the death rates of cigaret smokers consistently higher than those of United States white males. It is notable, and perhaps significant, that if we take for comparison of the death rates of the cigaret smokers, not the non-smokers of cigarets in the sample of the American Cancer Society study, but the comparable general United States population, the only specific disease that shows death rates consistently unfavorable to cigaret smoking is coronary heart disease, and this is the particular disease group for which there is independent biologic evidence that cigaret smoking may be deleterious. For each of the specific diseases except coronary heart disease, the cigaret smokers of the American Cancer Society study show equal or more favorable rates than the comparable United States white male population. It suggests itself that perhaps the general United States population is a better "control"

group with which to compare the death rates of the smokers than is the sampled population of nonsmokers of cigarets in the American Cancer Society study, the death rates of which are so extraordinarily low as to allow little doubt that it is very highly selected.

Table 7
Death Rates, Cigaret Smokers of the American Cancer Society Study and United States White Males*

Age, years	Death rate, per 100,000 per year									
	All diseases		Cancer, lungs		Other cancer		Coronary diseases		Other diseases	
	A.C.S. cigaret smokers	U.S. white males	A.C.S. cigaret smokers	U.S. white males	A.C.S. cigaret smokers	U.S. white males	A.C.S. cigaret smokers	U.S. white males	A.C.S. cigaret smokers	U.S. white males
50-54	955	1,206	46	50	103	148	442	426	365	582
55-59	1,523	1,891	89	85	192	259	727	701	515	846
60-64	2,375	2,793	103	114	367	406	1,095	1,018	811	1,255
65-69	3,259	4,089	127	127	502	609	1,439	1,438	1,191	1,915

*Rates in this table calculated in the same way as for table 4.

It has been suggested, on the basis of the findings in the publication of Hammond and Horn, that an active educational campaign be conducted to warn the American public that cigaret smoking exposes them to an increased hazard of developing cancer. Such a proposal

seems poorly founded when an examination of the data on which the conclusions of the study rest discloses that the cigaret smokers in the American Cancer Society study are enjoying equal or lower death rates from cancer than the general public is experiencing, and that their over-all death rate is also lower. Calculations which have been made of the number of lives which could be saved in a year if all men stopped smoking cigarets are based on the fallacious practice of applying the difference between the death rates of strata observed in the population of the American Cancer Society study to the United States population, the general death rates of which are materially different. It would seem more cautious, not to say more logical, to assume that whatever selective forces were responsible for the difference evident between the death rates of the investigated population taken as a whole and the United States population, affect also the *difference* of death rates between selected strata of the population. Why should a complex mechanism of selection which effects a falsification in representing the incidence of deaths in each of two groups concerned in an investigation act in a way so co-operative with the objectives of the investigating statistician as to render the *difference* between the false death rates a correct reflection of the difference between the true death rates? An examination of the history of science would hardly disclose nature to be so helpful in balancing out the errors of observational data.

BIOLOGICAL CONSIDERATIONS

A disquieting element in the array of observations which have been assembled pointing the finger of accusation at smoking as a cause of lung cancer is that it is so ample, yet it is so exclusively statistical. There are lacking observations of the pathologic process of which the statistics are only the supposed reflection. If smoking causes cancer in many individuals it does so in each one of these individuals separately and by way of some material physicobiologic process. One would expect a body of clinical and pathologic observations to have accumulated over the years suggestive of such a process. The notable lack of these, taken together with the abundance of statistics, should, in view of previous similar experience, arouse suspicion that we may be dealing with a purely statistical phenomenon related to the way the statistics were obtained.

If cigaret smoking is so effective in causing lung cancer that one can see its evidence statistically so impressively in so short a time as 20 months as it is on view in the report of Hammond and Horn, then we can reason that it is a very potent cause. Why then cannot lung cancer easily be produced experimentally? If, to counter this question, it is argued, as it has been argued, that the cancer produced in the short period of observation is the result of a cumulative effect of many years of exposure to smoking, then one wonders why pathologists have

not reported, from the enormous number of necropsies with careful tissue examination which have been performed over the years, evidence of a profound chronic process which retrospectively could be allocated as the precursor of smoking-produced cancer.* Since the American Cancer Society study shows cancer other than lung cancer to be associated with smoking, it is not only in the lung but in all cancer-susceptible tissues that such changes should be found. It is of course possible further to argue that the widespread cellular damage by smoking that is going on for years, is of a fundamental biochemical nature not visible to the pathologist and not yet discovered by the chemist, and that animal experiments are futile because humans are the only animals susceptible to smoking-produced cancer. But such explanations are hardly more than a restatement of the paucity of direct evidence. Moreover, even if the process of causation of cancer by smoking is "on the average" very long, say many years, still we should expect on the basis of observations of other biologic characters, that the period of incubation would be variable and that at least in a small proportion of cases it would be very short. Why then over the years have we not seen individual case reports of the appearance of fulminant cancer in association with acute exposure to smoking? I mention these few isolated questions merely to illustrate the general fact that clinical experience with individual patients has not produced independently an impression that cancer in patients is associated with smoking. If one can imagine all the statistical evidence, including that from vital-statistics reports, not to have appeared then I believe that there is nothing substantial in the record of clinical or pathologic observations on individuals that would have suggested smoking to be the cause of cancer. In these circumstances it seems premature to conclude definitely that it is the cause.

Actually the American Cancer Society study does not point specifically to association of smoking and cancer, for all specific diseases for which the number of cases permits examination show association, exhibiting a larger death rate among smokers than among nonsmokers. Therefore, if the association found is not statistically spurious and is to be explained as a biologically causative effect, it is not on these findings specifically a carcinogenic effect but something which influences broadly whatever may increase the susceptibility of the organism to fatal disease. Now, the most important known "cause" of cancer and some other diseases, notably those of the cardiovascular system, is *age*. We might say speculatively that smoking accelerates the rate of living and advances age and age causes cancer. The supposed effect of smoking, if it exists, may be to stimulate those trophic

*Since this writing there has come to my attention some unpublished work by Auerbach and also by Ryan and McDonald, presenting observations on epithelial changes in the larynx and lung that are of interest in this connection.

processes, of which little is known, that constitute the biology of aging. The idea is not entirely implausible or without support in existing literature. It is in keeping with Pearl's idea that duration of life is inversely related to the *rate of living*[17] and with Pearl's own study of the effect of heavy smoking on longevity.[18]

WANTED—AN ADEQUATE PROGRAM OF EXPERIMENTAL VERIFICATION

In most recent reviews of the statistics on smoking and cancer, the studies referred to have been divided into two types which have been designated respectively as "retrospective" and "prospective." This distinction is made, I presume, having in mind the development of cancer. In the retrospective studies, when the investigation begins, the cases with cancer are already developed and designated, while in the prospective type the cases with cancer are still to be assigned. In the retrospective studies the cases are primarily differentiated as to cancer and secondarily investigated as to smoking; in the prospective studies the cases are primarily differentiated as to smoking and secondarily investigated as to the development of cancer. The retrospective type of study generally has been recognized to contain certain particular difficulties for inference, of which the prospective type is free. I do not doubt that the prospective type of study does mitigate some important objections which have been raised against the retrospective type of study. In a crucially important sense, however, both of these types of study are retrospective. If in the prospective type of study—exemplified in those of Doll and Hill[9] and of Hammond and Horn—the designation of the cases with and without cancer is "prospective," the designation of each individual as to whether he is a smoker or a nonsmoker is not prospective, for this is already accomplished at the initiation of the study. It is just this which opens the way to "selection."

The type of study which is genuinely prospective is the experimental study, for here one begins with neither variable predetermined, but instead with the entire group of individuals undifferentiated in respect of either of the two variables, the association of which is under investigation. Separation of individuals is then made in respect of one variable—the putatively "causal" variable—*at the will of the experimenter,* and according to well-defined statistical principles of randomization. The procedure is described in detail with great acumen by Hill[19] for the study of association of therapeutic effect and the administration of specified drugs. The great care of the planned systematized randomization employed in such investigations involves considerable, sometimes very formidable, practical difficulties, and it

17. Pearl, Raymond: The Rate of Living. New York, Alfred A. Knopf, 1928, 185 pp.
18. Pearl, Raymond: Tobacco Smoking and Longevity. Science 87:216-217 (Mar. 4) 1938.
19. Hill, A. B.: The Clinical Trial. New England J. Med. 247:113-119 (July 24) 1952.

would hardly be insisted on if it were not essential for a valid statistical inference of association implying causation of therapeutic effect. If, in the prospective studies on the association between smoking and the development of cancer of the lungs, randomization has not been employed as respects the smoking variable, it is not because randomization is less necessary here than in clinical trials for the critical evaluation of the scientific significance of the results, but only because of the practical difficulties involved. It seems at the moment quite outside the realm of practicality to perform an experiment in which persons randomly designated are subjected to various degrees of smoking, with prospective observation on the development of cancer.

But in science there is no substitute for experiments. It is a central purpose of this essay to urge that an adequate program of experimentation with animals be accomplished, before definitive conclusions are drawn regarding the association between smoking and cancer. What is to be considered an adequate program of experiments will bear much thinking about, and by persons more competent and more experienced than I am as regards the technical aspects of the problem, but what I have in mind as a minimum is a forthright *ad hoc* check on the prospective investigations of Doll and Hill, and of Hammond and Horn. It is notable that these investigations, which have provided affirmative evidence, involved large numbers of individuals, and that they were pursued with the methods of statistical epidemiology. If they are to be verified experimentally it suggests itself that a similar procedure should be employed applied to animal populations. I refer specifically to "inhalation" experiments biologically simulating the exposure of the respiratory system to cigaret smoking in different intensities, which is represented in the prospective studies referred to as different amounts of cigaret smoking and resulting—it has been inferred from these studies—in the production of correspondingly greater frequencies of cancer.

The variety or varieties of animal to be used is one of the questions on which counsel will be wanted from specialists, but since mice are known to develop cancer of the lung as well as cancer of other sites[20] and some small-scale experiments with these animals show promise of significant results,* I should think mice would be satisfactory experimental animals. Careful statistical control should, of

*For instance, in a report by Campbell[21] 74 mice exposed to diluted cigaret smoke and tarry matter were compared with 76 control mice. For the exposed mice the mean duration of life was 417 days and 24 per cent were found with primary lung tumors, while for the controls the mean duration of life was 427 days and 14 per cent developed primary lung tumors. The number of animals used is small and details respecting the pathologic character of the tumors are wanting, but the general results of the experiment are corroboratory of the thesis that cigaret smoking decreases longevity and increases the incidence of lung cancer.

20. Stewart, H. L.: Pulmonary Tumors in Mice. In Homburger, Freddy and Fishman, W. H.: The Physiopathology of Cancer. New York, Paul B. Hoeber, Inc., 1953, 93-112 pp.
21. Campbell, J. A.: The Effects of Exhaust Gases From Internal Combustion Engines and of Tobacco Smoke Upon Mice, With Special Reference to Incidence of Tumours of the Lung. Brit. J. Exper. Path. 17:146-158 (May) 1936.

course, be exercised, with an adequate number of animals used with each of several intensities of smoking exposure, stratified possibly on some pertinent biological variables such as age and weight. Time of death with pathologic findings at necropsy would be the dependent variables which, subjected to life-table analysis, could summarize the results.

An experimental program of appropriate dimensions doubtless is not something to be undertaken out of hand, but neither is it anything that can be considered prohibitively difficult. As I have explained previously, I do not believe that from the statistical studies so far accomplished one can conclude definitively that smoking causes cancer, or even that necessarily it is "associated" with it. On the other hand, at the very least these studies pose a strong presumption that smoking *may* cause cancer and/or *may* otherwise be deleterious to health and longevity. Considering that we may have here a clue to the etiology of human cancer and in view of the social importance of even the belief that it causes cancer, something more seems in order than sporadic efforts on the part of isolated individuals who happen to be particularly interested, but who are not necessarily adequately equipped with the required laboratory facilities or even by themselves with all the technical knowledge required for a comprehensive experiment. Aside from the direct interest of such an experiment in relation to the interpretation of the statistical studies under discussion, the opportunity to perform a study in experimental epidemiology involving possibly an environmental factor affecting the "rate of living," possibly an etiologic agent of cancer, seems unusual in its potentiality for elucidating a basic problem in human ecology.

In reviewing the present paper before it was dispatched to the printers, I realized that I may have overelaborated some of the points and produced an unbalanced impression. I do not fail to appreciate the great pioneering effort which the statistical studies on smoking and cancer represent. It was not intended to denigrate them, so far as they are considered tentative evidence that there is some relation between the two. My thesis is only that it is unwarranted to conclude from them that a meaningful association already has been proved beyond doubt, as some writers have asserted and as appears to be widely accepted in the United States.[10,16] Much less do I believe that causation has been established. I think that the possibility that selection is the source of the observed association has not been given sufficient weight, and that much more work must be done, and time allowed for its evaluation, before a responsible definite opinion can be had as to the precise significance of the findings.

APPENDIX NOTE 1. OTHER STUDIES THAN THAT OF THE AMERICAN CANCER SOCIETY

It has been mentioned in the text that the study from the American Cancer Society was taken as the basis for the present analysis because it is the prospective study which is most amply provided with detailed data, of any published up to the time of the present writing. The conclusion I have drawn from consideration of that study is that, since the sample was not obtained by appropriate methods of randomization, it is possible that a selected sample was obtained in which association between smoking and deaths from lung cancer has been produced by the interaction of competing selective risks as described or by some other selective mechanism. The detailed data of the American Cancer Society study contain evidence supporting the hypothesis that that is what may have happened. How do other reports which have appeared come out when they are inspected with these considerations in mind? The study most directly to the point is the prospective study of Doll and Hill,[9] which antedated that of the American Cancer Society and which, together with the retrospective study of the same authors,[22,23] is in many ways more carefully and critically analyzed by the authors than is the study of Hammond and Horn. The question to be considered is "Does evidence appear of selection on the pertinent variables, smoking or death or both?"

1. *General Measure of Selection.*—The questionnaire inquiring about smoking history ". . . was sent on October 31, 1951, to 59,600 men and women on the *Medical Register*. Of the 41,024 replies received, 40,564 were sufficiently complete to be utilized."[9] Thus the usable records referred to 68 per cent of the population designated for study. The present (preliminary) report of Doll and Hill concerns itself only with 24,389 men aged 35 years and above, who answered the questionnaire, and it is possible that this subgroup represents a different proportion of the original population. But the percentage of 32, representing the fraction of the population in which association was to be studied, that failed to enter the investigation provides a measure of the potential of selection present in the study.

2. *Selection on Deaths.*—Doll and Hill found that the smokers in the sampled population experienced higher death rates from lung cancer than did the nonsmokers. But so far as the level of death rates for the total sampled population is concerned, the authors recognize that there has been selection favoring lower death rates among the doctors, and offer an explanation of how this has come about. They say:

22. Doll, Richard and Hill, A. B.: Smoking and Carcinoma of the Lung: Preliminary Report. Brit. M. J. 2:739-748 (Sept. 30) 1950.
23. Doll, Richard and Hill, A. B.: A Study of the Aetiology of Carcinoma of the Lung. Brit. M. J. 2:1271-1286 (Dec. 13) 1952.

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"Why should the rates for the doctors be so much lower? One important reason . . . is, we believe, that doctors who were already ill of a disease likely to prove fatal within a short space of time would have been disinclined, or indeed unable, to answer our inquiries. In other words, we should learn of their deaths, but we would have no corresponding completed questionary on our files. That this may well be true is shown (*a*) by the relatively low death rate from all causes that we have recorded—namely, 14.0 per 1,000 per annum, against 24.6 per 1,000 for men of all social classes in the same age group in the U.K. in 1951, . . ." So far as deaths from specific diseases are concerned, a comparison is made by Doll and Hill[9] with respect to deaths from cancer of the lungs among men aged 45 through 74 years as experienced in the prospective study and as estimated from their retrospective study among residents in Greater London in 1950. The standardized death rate per 1,000 men from cancer of the lungs for physicians in the prospective study was 0.73; for the residents of Greater London it was estimated as 1.97. So far as the latter can be taken as representative, the death rate from cancer of the lungs, as well as the total death rate, in the sampled population is smaller than that of the comparable general population. Indeed even among the heavy smokers (25 gm. + per day) the total death rate 16.3, and the death rate from lung cancer, 1.45, are smaller than the respective rates for the general population. Thus it is seen that, as is the case with the sampled population of the American Cancer Society study, the sampled population of the prospective study of Doll and Hill has been subjected to severe selection in favor of low death rates.

3. The Smoking Variable.—The prospective study of Doll and Hill[9] shows 87.3 per cent of smokers of all forms of tobacco and 72.7 per cent of cigaret smokers among the physicians in the sampled population. The corresponding percentages shown in their retrospective study[23] in the control population of patients without lung cancer are considerably higher, being respectively 95.5 and 89.0. Part of the difference shown may be due to differences as to social class, age and sex between the populations as well as to differences in definition of smokers used in the different studies. However, it is characteristic of the studies published on the association between smoking and cancer that the percentage of smokers indicated in the controls varies over a wide range. Among 14 retrospective studies reviewed by Cutler,[2] the percentage of smokers among the controls varies from 69 to 95. It appears that whether or not an individual is recorded as a smoker in these studies depends on the circumstances of the study. It is not unreasonable to suggest that whether an individual records himself on a questionnaire as a smoker also is dependent on circumstances, and I do not see how one can be sure that this is independent of the general health of the individual or of the appearance of the presence of a

disease which, it is known to the recorder, is under investigation. Specifically, however, I do not find evidence of selection as respects smoking in the prospective study of Doll and Hill. On the contrary, in their retrospective study[23] these authors quote the results of a social survey of Greater London which shows 87.9 per cent smokers, which, while it is low in comparison with the 95.5 per cent smokers shown among the controls of that retrospective study, agrees well with the 87.3 per cent shown in the prospective study. It has been suggested that the prospective studies provide an opportunity to validate the questionnaire reports of smoking habits, if in the case of each individual deceased, a "retrospective" inquiry is made. There is need to get some light on why the percentages of smokers reported among the controls in the retrospective studies are so different from those reported in the prospective studies.

4. Other Diseases Than Lung Cancer.—In the analysis of the data provided in the publication of Hammond and Horn, it was found that the smokers in the population died at higher rates than the nonsmokers, not only from lung cancer, but also from other cancer, from coronary heart disease and from diseases other than any of these. The prospective study of Doll and Hill agrees in showing an association of smoking and death from lung cancer and from coronary heart disease, but no association is found with deaths from cancer other than lung cancer, respiratory diseases other than lung cancer, cardiovascular disease other than coronary heart disease or diseases other than any of these diseases. This result is more in keeping with the hypothesis of a specific action of smoking in causation of disease, and it may be that selective association exists in the sampled population of the American Cancer Society and not in the sampled population of the prospective study of Doll and Hill. Nevertheless the material differences between the study of Hammond and Horn and that of Doll and Hill as regards association between smoking and diseases other than lung cancer and coronary heart disease, unless one is to believe that smoking affects very differently American males generally than English male physicians specificaliy, demonstrate that the appearance of association between smoking and disease, in statistical studies, can be influenced by the circumstances in which the sample is taken.

APPENDIX NOTE 2. CHECK ON SELECTION HYPOTHESIS

Evidence on whether or not the association shown in the prospective studies of Doll and Hill[9] and of Hammond and Horn has been produced spuriously by selection will perhaps be provided in the projected study on the records of the Veterans' Administration. If this study will deal with a predesignated cohort of veterans, if these are traced completely or practically completely, if the results show the death rates for the group as a whole to be fairly comparable with the

rates for United States males of the same age distribution, if the percentage of cigaret smokers agrees fairly well with general statistics as shown by independent studies of cigaret smoking, and if association between cigaret smoking and death rate from cancer is found, then this will provide evidence that the source of the observed association in the present studies is not a selective mechanism of the kind I formulate.*

Perhaps useful relevant information can be obtained also if a subsidiary detailed study is made on the records of the study of Doll and Hill[9] in respect of those physicians on the registry who did not fill in the questionnaire addressed to them regarding their smoking history. What was the death rate of these compared with those who did? Could something in the way of a special investigation be made on a sample of these cases to ascertain the percentage of smokers among them to be compared with the percentage found for the physicians included in the study?

A check will be available when prospective studies have been continued long enough. Experience of insurance companies indicates that selection effected by initial medical examination, and by self-selection is "worn off" in about 3 to 5 years.[24] After about that time we may expect that the age-specific death rates of the sampled population as a whole should approximate those of the general comparable population. If after, say, 5 years have elapsed since the beginning of the study, comparison is made of the death rates of smokers and nonsmokers over a 2-year period, referring only to the individuals who have survived 3 years, and the differences between smokers and nonsmokers are found to be just as large as earlier, it will be reasonable to conclude that the importance of selection in the sense discussed in the text is negligible. In this connection I hope it is not amiss to suggest that when sufficient data will have been accumulated in the prospective studies, the results will be presented in the form of a life-table analysis. At present in the prospective studies[9,10] the death rates are computed by dividing observed deaths by total number of corresponding individuals entered in the population. While it is not to be supposed that there is any important difference in exposure times of the smoking and nonsmoking groups compared, it would seem preferable

*It is to be hoped that all the necessary effort will be made in this investigation to secure a complete follow-up. Otherwise, even if its findings should be similar to those of other prospective studies, a cloud of doubt will hang over it because of the possible effects of selection. It is desirable also that some validation of the smoking questionnaire be attempted, and at least for a subsample some data be obtained on the relative socioeconomic characteristics of the populations which are compared, as suggested by Arkin.[16]

24. Jordan, C. W.: Life Contingencies. Chicago, Society of Actuaries, 1952, 331 pp.

to use an analysis that takes account of period of exposure,[25,26] and which at the same time presents the annual death rates in a way in which they are immediately seen in relation to time of entry into the investigation.

BOOK NOTICES

"An Atlas of Congenital Anomalies of the Heart and Great Vessels" by J. E. Edwards, T. J. Dry, R. L. Parker, H. B. Burchell, E. H. Wood and A. H. Bulbulian (Springfield, Illinois, Charles C Thomas, 1954) is reviewed in Prensa Médica Mexicana *20*:20-21 (Jan. 31), in Circulation *11*:993 (June) and in The Ohio State Medical Journal *51*:514 (June) 1955.

"Clinical Diagnosis" by E. G. Wakefield (New York, Appleton-Century-Crofts, Inc., 1955) is reviewed in Hebrew Medical Journal *1*:124, in the Bulletin of the Summit County Medical Society *30*:45-46 (May), in Postgraduate Medicine *17*:A-114 (May), in the Journal of the American Medical Association *158*:617 (June 18) and in the Journal of the Arkansas Medical Society *52*:20 (June) 1955.

"Collected Papers of the Mayo Clinic and the Mayo Foundation" (Philadelphia, W. B. Saunders Company, 1954) is reviewed in Archivos Médicos de Cuba *6*:48 (Jan.-Mar.) 1955.

"The Mayo Clinic Diet Manual," Ed. 2, by the Committee on Dietetics of the Mayo Clinic (Philadelphia, W. B. Saunders Company, 1954) is reviewed in the A.M.A. American Journal of Diseases of Children *89*:767 (June) 1955.

"Diseases of the Skin," Ed. 8, by O. S. Ormsby and Hamilton Montgomery (Philadelphia, Lea & Febiger, 1954) is reviewed in The Medical Journal of Australia *1*:320 (Feb. 26) and in the New England Journal of Medicine *252*:1055 (June 16) 1955.

"Peripheral Vascular Diseases," Ed. 2, by E. V. Allen, N. W. Barker and E. A. Hines, Jr. (Philadelphia, W. B. Saunders Company, 1955) is reviewed in the Hebrew Medical Journal *1*:123, in the Delaware State Medical Journal *27*:91 (Apr.), in the Bulletin of the Johns Hopkins Hospital *96*:229 (May), in the Journal of the American Medical Association *158*:148 (May 14), in the Journal of the Arkansas Medical Society *51*:300 (May), in Postgraduate Medicine *17*:A-110; A-114 (May), in the Texas State Journal of Medicine *51*:282 (May), in The West Virginia Medical Journal *51*:35-36 (May), in GP *11*: 145-146 (June), in the Journal of Medical Education *30*:365-366 (June), in Medical Annals of the District of Columbia *24*:314 (June) and in the American Heart Journal *50*:161 (July) 1955.

25. Berkson, Joseph and Gage, R. P.: Calculation of Survival Rates for Cancer. Proc. Staff Meet., Mayo Clin. 25:270-286 (May 24) 1950.
26. Berkson, Joseph and Gage, R. P.: Survival Curve for Cancer Patients Following Treatment. J. Am. Statist. Assoc. 47:501-515 (Sept.) 1952.

EXHIBIT 30B

[From the Washington (D. C.) Post, July 14, 1957]

SMOKING AND CANCER

Your newspaper might well have given a first-page position on July 7 to the most important and excellent article by Nate Haseltine on the problem of smoking and health. This is not just a problem of the medical world; it is everyone's problem, and all persons should have their attention called to its dangers.

Mr. Haseltine pointed out that Great Britain has seen fit to assume a proper role in the control of this health hazard. Specific legislative action is being taken by the British Government to bring to the attention of their people, especially the young people who have not as yet begun the smoking habit, the facts as to the dangers of cigarette smoking.

Action by appropriate agencies in this country has not been taken. This is even more difficult to understand when it is realized that a large part of the research upon which Britain took her stand against smoking was carried out in this country by American investigators.

Also, it is hard to understand how the Government and voluntary health agencies could have requested a panel of topflight scientists to evaluate the cigarette smoking health problem and then fail to take action on their report. This report emphasized that there was a casual relationship between heavy cigarette smoking and lung cancer, and that further research was not necessary to further establish this, and that appropriate public-health measures should be taken to control this hazard.

The appropriate Government health agencies and the American Cancer Society have failed to take any positive action. No concerted effort is being made to bring to the teen-age groups the facts regarding the dangers of heavy cigarette smoking, while, on the other hand, no effort is being made to curtail the heavy advertising activities of the cigarette companies.

No effort is being made to indicate on the merchandise itself that the enclosed cigarettes are harmful if more than a very few are smoked every day. This seems difficult to understand when even the toothpaste tubes are made to carry warnings against their use by children if they contain fluorides. There seems to be a certain unwillingness of those who should shoulder responsibility to stand up and be counted.

Certainly, Great Britain has less to lose by a curtailment of tobacco sales than does this country, because most tobacco is imported into Great Britain. Certainly, curtailment of tobacco use in this country would have a considerable effect on the tobacco farmer, the tobacco industry, and all related industry, including even TV entertainment. However, if this is the only reason for our failure to act, one must wonder if we are not really becoming a soft, luxury-seeking nation, if we can fail to act to protect the health of our young people.

Why should it not be required: (1) That the story of the health hazards of cigarette smoking be accurately presented to all our high-school students? (A good film on this is greatly needed and none is available.)

(2) That a warning as to the hazards of heavy cigarette smoking be printed plainly on each package of cigarettes?

(3) That cigarette advertising be made to include some adequate reference to the health hazards associated with heavy cigarette smoking?

Unless the Government and appropriate voluntary health agencies take such steps, they must consider themselves responsible—not for the epidemic of lung cancer which is already here, but for the continuation of the epidemic in future years.

No person who dies of lung cancer in years to come should be able to say to any of us—"I didn't know that heavy smoking was dangerous. If I had known, I'd never have begun to smoke."

CALVIN T. KLOPP, M. D.

(Dr. Klopp is director of the George Washington University Cancer Clinic.)

EXHIBIT 30C

[From the Washington Daily News, July 18, 1957]

B. B. D. & O. drops Reader's Digest

CONFLICT OF (TOBACCO) INTEREST

NEW YORK, July 18 (UP).—The advertising agency of Batten, Barton, Durstine & Osborn, Inc., has given up the account of the Reader's Digest, which it has held for 28 years, because of "an obvious conflict of interest" between it and another client, the American Tobacco Co.

The magazine published last month an article which questioned the effectiveness of filter-tip cigarettes with an analysis showing how much tar and nicotine was present in the smoke of various brands of cigarettes.

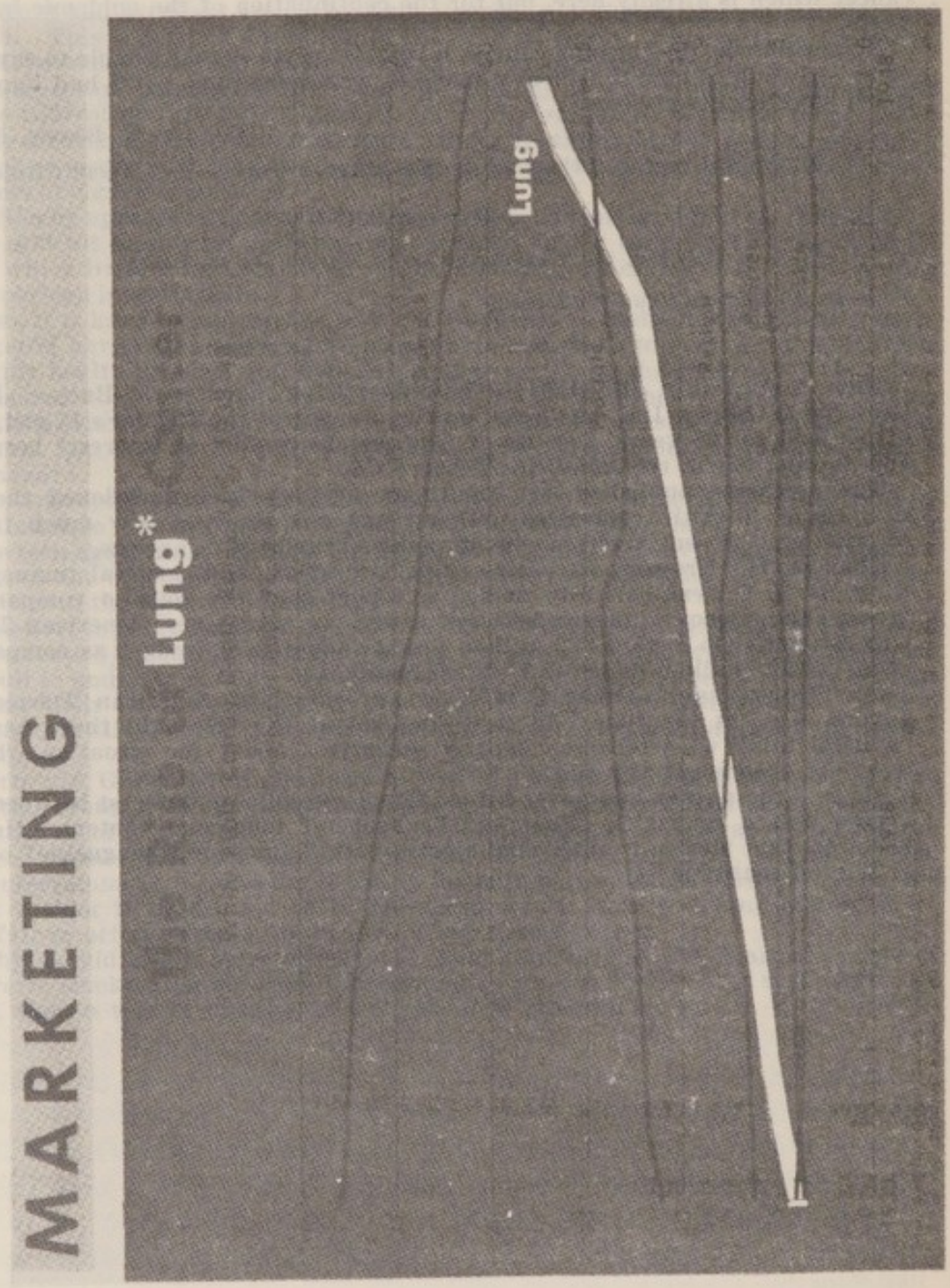
Charles H. Brower, executive vice president and general manager of B. B. D. & O., emphatically denied a report that the tobacco company had ordered the agency to choose between it and the magazine. American Tobacco was estimated to have a $22 million annual advertising budget as compared to about a $1.25 million budget for the magazine.

Mr. Brower said neither Paul Hahn, president of American Tobacco, nor anyone else "at the time told us to get rid of it." He said there had been "a little talk" with tobacco-company executives about the situation, "but we were not ordered at any time."

He said the agency had never before found a conflict of interest between other clients and a magazine client on the basis of magazine content. He said, however, that such a conflict might arise with "almost any magazine" over an article "of that type."

EXHIBIT 301

[From Business Week, December 5, 1953]



CIGARETTE SCARE: WHAT'LL THE TRADE DO?

The slow fuse that has been burning under the cigarette industry for several years reached the powder last week.

The sputtering began in earnest a couple of weeks ago when it began to be apparent that this year's sales of cigarettes are going to run about 1 percent behind last year's (Business Week, November 21, 1953, p. 140). What made this stand out is the fact that for 20 years the cigarette industry has had a charmed life of steadily increasing sales and markets.

In the background.—The unexpected setback brought to the surface an ugly phrase that had been lurking underneath people's consciousness for several years—lung cancer. To many observers it had looked very much as though the chief reason for the setback was the spreading fear that cigarettes cause lung cancer, a fear that has made a lot of people cut down their smoking.

Then the top blew off:

Reader's Digest, a longstanding foe of the industry, published an article called "Can the Poisons in Cigarettes Be Avoided?" which bluntly stated that "used to excess tobacco may . . . even shorten life."

Time magazine came through with an article on cancer research that points a finger straight at cigarettes as a major cause of lung cancer, the incidence of which has quadrupled during the 20 years that cigarette consumption has shot up from 100 billion to about 400 billion a year (Business Week, December 27, 1952, p. 41).

Silence ends.—These fast-paced events loosened up for the first time official tongues of the tobacco industry, which up until now has preserved a rigid silence on lung cancer.

The first to speak was Paul M. Hahn, president of giant American Tobacco Co. (Lucky Strike, Pall Mall, Herbert Tareyton). He issued a tempered statement "of reassurance to the public." The burden of it was that the case against cigarettes has not been proven, either statistically or in the laboratories. He scored "much loose talk on the subject as reported in the press during the recent months."

E. A. Darr, president of R. J. Reynolds (Camel, Cavalier), made in essence the same case but added a testy remark about lung cancer research: "One of the best ways of getting publicity is for a doctor to make some startling claim relative to people's health regardless of whether such statement is based on fact or theory."

Philip Morris (Philip Morris, Dunhill, Parliament) subscribed officially to the sentiments of Hahn. By late last week, however, the other three major companies—Liggett & Myers (Chesterfield, Fatima), P. Lorillard (Old Gold, Kent, Embassy), and Brown & Williamson (Kool, Raleigh, Wings, Viceroy)—had not been heard from.

To many observers the actions of the cigarette companies had the air of haste and improvisation. But the significant point is that, however belatedly, the cigarette industry has recognized that a problem exists. What it does not say, certainly not publicly, is that it faces what is perhaps potentially the gravest problem besetting any industry since prohibition shut down the liquor business.

As if to nail the point down, Government figures on September tax payments on tobacco sales to wholesalers, out this week, showed a decline from last year's figures for the third consecutive month. The dip: 5 percent.

I. WHERE IS THE GUILT?

The first question that arises is, of course, that of the extent to which cigarettes really can be held responsible for that sinister rising curve in the incidence of lung cancer (chart, p. 58).

In the past 4 years the scientific evidence has been mounting. In 1949 and 1950 four reports were published by different groups drawing the same conclusion—that a relationship existed between smoking habits and the occurrence of lung cancer in males. A few weeks ago, Dr. E. A. Graham of Washington University School of Medicine in St. Louis added fuel to the fire by announcing that he had produced a skin cancer in mice by using tobacco tars from a cigarette-smoking machine.

This makes a very strong case against the cigarette industry. Few scientists seem to doubt now that some relationship does exist. Assuming—as the industry does not—that they are correct, the major questions still being posed are:

What is the nature of the relationship between cigarettes and cancer?

What is the cancer-producing material in cigarettes?

Can the cancer-producing agent be removed?

What can be done about present smokers—will it do them any good to stop smoking?

Getting the answers to these questions is not easy. Trying to pin down just what the relationship between cigarettes and cancer might be is far more difficult than determining that such a relationship is evident. Scientists would like to know, for instance, whether the seriousness of the cancer depends on the total number of cigarettes smoked or whether it is an accident that might just conceivably occur from any single cigarette.

Mysteries.—In trying to find out what in cigarettes may cause cancer, the researchers run up against the entire field of cancer research (Business Week, November 7, 1953, p. 142). Not very much is known about cancer. Less is known about the agents that may cause it or how they work. In trying to isolate the cause in cigarettes, the scientists run smack into this entire ignorance of the behavior of the cancer cell.

Assuming that they will succeed in finding the chemical or product that is the cause, if a single cause exists, getting it out of the tobacco may be difficult or simple. Some scientists have suggested that arsenic in the tobacco from bug sprays may be the cause. If this were true, eliminating it would be relatively easy. If, however, it should be a more subtle chemical making up part of the tobacco plant, removal could be much more difficult.

Wide check.—About 2 years ago, the American Cancer Society tackled the problem of finding out what relationship existed between cigarettes and cancer. Over 200,000 males between 50 and 69 were asked to volunteer for the ACS study. All were believed to be healthy when the project started. These volunteers filled out an extensive report on their smoking habits.

Each year for the next 3 or 4 years, ACS is checking to find if any of the 200,000 have died or are sick. Medical records are being used to determine any clinical reports of lung cancer in the death or sickness.

Statisticians at ACS hope that this mass of data will give them a good scientific correlation between smoking habits and cancer occurrence. They hope that it will answer the question as to whether lung cancer depends on the total number of cigarettes smoked. Meantime, ACS denies that it has issued a condemning report, which is being blamed by Wall Street for the present plight of tobacco stocks. Its first report on the smoking survey won't be due until 1955.

If the ACS or similar studies show that the incidence of cancer depends on total number of cigarettes, it won't make much difference to the man of 50 who has been smoking for 30 years. But it may make a whale of a lot of difference to a teen-ager just getting started on the habit.

Of course, cigarettes aren't the only possible cause of the growing lung cancer death rate. Researchers will also have to study such other suspects as automobile exhaust fumes, various chemicals, and air pollution. Separating cases caused by different agents will not be easy.

II. DECLINE OF SMOKING

Whether or not the recent dropoff in sales can be wholly attributed to the fear of lung cancer, there is little doubt that it has had a growing and significant effect on smokers and the public in general.

The fear first began spreading back in 1950 with an article called Cancer by the Carton that ran in Reader's Digest. In 1952 the British Medical Journal published an article that reverberated around the world. It was a study of 1,465 lung-cancer patients, and it showed a close correlation between heavy smoking and the disease. The retreat from smoking was on.

The extent of the retreat is unknown, since there is no known research into the change in smokers' habits. What evidence there is, though indirect, suggests a widespread effect on smokers.

One important piece of evidence is the growth of long cigarettes and more recently of the filter tips. Long cigarettes will account this year for about 23 percent of all cigarettes sold. Filter tips, and infinitesimal part of the market a couple of years ago, will account for about 3 percent. The significance lies in the fact that both types of cigarettes have been pushed heavily by the cigarette makers on the protection they afford against tars and nicotine.

Other scraps of evidence come from the increased sales of Denicotea and other cigarette holders offering some measure of filtering, of books on how to stop smoking, of pills that are supposed to help break the habit.

Still further evidence comes from everyday experience. Everyone must know half a dozen people who have just given up smoking or who are trying to.

Fear of revolt.—The cigarette industry scoffs at such unscientific evidence, but it is having nightmares about what such a popular revolt—based on scientific evidence or on none at all—can do to the industry.

As one official of a cigarette company put it last week, "If every smoker in the country smoked one cigarette less a day, it would knock 5 percent off our sales."

In a way, the vulnerability of the industry stems from its very success. There are no less than 63-million smokers in the United States today, according to the commonly accepted estimate. That means practically everyone who can reasonably be expected to smoke does so. The big gains of the industry over the past decades were scored by the social revolution that emancipated women and made them cigarette smokers by the hordes. (The direct appeal to women in cigarette advertising appeared back in 1929, amid a storm of disapproval.) Now, because there are no large segments of nonsmokers left, the industry would have to count on population growth for any further gains in its market.

Recruiting.—But perhaps new recruits won't come along so fast as heretofore. The youngsters coming of smoking age are part of the slim "hollow generation" to begin with, and what if parents, churches, schools, and other authorities gang up to keep them from smoking as long as possible?

What is a lot of older smokers cut it out or cut down?

The possibilities can be glimpsed from the example of one less cigarette per day per smoker. That alone would mean a drop of some 3,150,000 packs a day, or about $700,000 in total retail volume. On a yearly basis this would mean that each smoker would spend about $4 less than the $97.54 that he spent at retail in 1952 according to the figures of the National Association of Tobacco Distributors.

This raises a further conjecture: What will happen to some millions in advertising revenue that cigarette makers put out yearly?

III. INDUSTRY REACTION

Faced with these horrendous possibilities, what has the cigarette industry done?

Until now it has done virtually nothing, hoping to ride out the gathering storm. This is implicit in Paul Hahn's comment last week:

"At one time or another within the past 350 years practically every known disease of the human body has been ascribed to the use of tobacco. One by one these charges have been abandoned for lack of evidence."

In other words, as you gather even more clearly from private conversations with cigarette officials, they were expecting to weather this storm, too, and based their long silence on this premise. Fear also played a part. Cigarette people will admit that they were afraid that if their own company made any statement about lung cancer the only effect on the public consciousness would have been to connect disease with their brand.

Research.—Until last week the industry didn't see fit, therefore, to reveal that it has been putting any money into cancer research.

Now, it has raised a corner of the curtain. The industry says that it is financing research, both within its own laboratories and outside in independent institutions, on lung cancer. But it clams up on the details. Just who is getting the money and for what remains an almost complete mystery.

You can put a finger on only a couple of projects. Time magazine reports that one unnamed company is filtering money through the Damon Runyon Memorial Cancer Fund into a major cancer research project at New York University. Business Week has also been able to run down one case. It has ascertained that American Tobacco is contributing $15,000 as an annual grant to the Medical College of Virginia to study the constituent parts of various types of tobacco smoke. But this project is under the resident professor of pharmacology and is strictly not a cancer project.

Other leads offered privately by the industry have come to nothing. For example, there are reports that Duke University has been given cigarette-company money specifically tagged for cancer research, but the university categorically denied the reports last week.

Contradictions.—Even the stories from within single companies are contradictory. The president of one of the Big Six says—off the record—that his company "spends a hell of a lot on its laboratories" and puts other money into independent research. But his own director of research says—off the record—that "we are doing practically nothing but a little spadework in our own laboratory."

Faced with a serious threat, the cigarette industry just can't seem to get together.

IV. ANTITRUST FEARS

The industry's failure to get together goes back to its early troubles. The trust busters broke up the great tobacco trust years ago, then nailed down their victory in 1941 with the famous Lexington case, based on alleged conspiracy (BW–Nov. 8, 1941, p. 17). The terms of this decision were so strict that, as one man in the industry puts it, "If we bump into the official of another company in a public washroom we have to make sure an impartial third party is present before we say hello."

The cigarette companies say these strict stipulations and the fear of further antitrust action are what prevent them from getting together.

Hush-hush move.—Actually, something of this nature is afoot, though no one outside a small circle will say just what is going on. Here is another half-raised curtain. One source says that the key to the whole thing lies in getting the Government to relax the terms of the Lexington decision sufficiently to permit a foundation to be set up, the first project of which would be a research program. But reporters in Washington and Kentucky are unable to discover any overt moves by the cigarette industry to get the courts to issue such an order.

However, still other sources hint that there is a possibility that the industry will follow a technique it used several years ago in setting up a joint foundation to fight cigarette taxes. This organization, the National Tobacco Tax Research Council, is supported by money from cigarette companies but is under the aegis of the industry's middlemen, the National Association of Tobacco Distributors.

V. ADS COME HOME TO ROOST

The cigarette industry is unfortunate at this juncture in having not just one but two albatrosses around its neck. Besides its antitrust record it also has its advertising.

Cigarettes offer the classic case, studied in every business school in the country, of how a mass-production industry is built on advertising.

Unfortunately, the cigarette companies achieved much of this remarkable result by screaming at the top of their lungs about nicotine, cigarette hangovers, smoker's cough, mildness, and kindred subjects.

Narrowing down.—The situation arose by a very natural process. In the beginning there were many companies and many brands. Then, as time and competition had their way, there were few companies and many brands. Finally, the cigarette companies found that competition required them to throw all their distributive and advertising resources into one cigarette in order to gain and keep market dominance.

By an equally logical and empirical process the companies also hit on the theme that sold the most cigarettes. From the early 1930's on, this meant almost solely one thing—sell health. Who began it is a moot point today, but the modern era began around 1933 with such slogans as "Chesterfields are milder," "Nature in the raw is seldom mild" (Luckies), and "Not a cough in a carload" (Old Gold).

Eventually this led to the avalanche of advertising that today features doctors, filters, tars. It has culminated with Philip Morris "fear" advertising (Business Week, November 14, 1953, p. 54) and the graphic television commercials for Kent showing how cigarette smoke stains filter paper.

Guilt complex.—Why has the industry persisted in this negative form of advertising even when, as tobacco growers and others complain, it hurts the trade by making people conscious that cigarettes can be harmful?

The reason is contained in a piece of motivation research recently made by the Chicago Tribune, which discovered that consumers think that "Cigarettes are considered morally and physically wrong, but their use is justified by the psychological satisfactions they provide."

The cigarette companies knew this years ago. They sensed that the majority of adult Americans have always been uneasy smokers, thanks to an inherited puritanical feeling about cigarettes and conviction from the state of their own bronchial tubes that cigarettes just aren't good for you. In their own way the cigarette people tried to reassure the smoker that everything was all right—just as long as he smoked X brand, which, of course, is milder, easier on the throat, and so forth.

The raucous claims of the cigarette companies rubbed a lot of people the wrong way. The Federal Trade Commsision, beginning in the early 1940's, tried to

eliminate the claim that one brand is less irritating than another, with very small success.

Doctors irked.—Now doctors are burned up at cigarette advertising, particularly because of the recent flood of so-called men in white television commercials, wherein announcers wearing white coats and other symbols of the medical profession plug cigarettes. The Journal of the American Medical Association recently called this a "cheap attempt to mislead." This sort of thing, plus a general reaction to all health claims in cigarette advertising, led to the recent AMA ban on cigarette advertising in its publications after January 1. The group denies that the move has any tie with the cancer problem.

So you have the anomalous picture of the advertising for one brand (say, Kents) tearing down by implication another brand made by the same company (in this case, Old Gold).

Why didn't the industry put a stop to the whole thing? Cigarette people will give you two good reasons:

The industry notes that where companies drop the health theme their sales go to pot. They point to the famous case of Philip Morris, whose sales shot up steadily under the impact of its original advertising theme that it was less irritating than other cigarettes (BW–Nov. 17, 1952, p. 73). The trade dates its slide-off as a brand from the time it switched from health themes.

No one will put his guns on the table until the others do so. "Why should we stop unless our competitors do?" said one man last week.

The question now arises whether the industry can go on advertising in the way it has, after publicly admitting that there is fear about lung cancer.

Ignoring the thing doesn't make sense, now that the cat is out of the bag. Neither does pounding harder on the health theme, which could drive away even more smokers than the critics say have already been driven away.

VI. COURSES OF ACTION

What alternatives are there?

The answer depends somewhat on whose advice you seek. Psychologist Ernest Dichter, who has counseled cigarette makers on motivational research in the past (BW–Jan. 23, 1951, p. 68), thinks that no matter what happens, people will probably go on smoking. He reasons that they will figure something like this: If I give up this there are a lot of other things I do—drink, overeat, strive for success—that I ought to cut out, too. However, Dichter thinks that right now the smoker is going through a process of reexamining his addiction. On that basis this is his advice:

"The advertiser who will help the smoker in his reexamination of smoking will have the edge. He must prove that he is on the side of the smoker."

Stress on filters.—Aside from Dichter's suggestions, here are two other possible tacks that have been suggested:

Put a greater stress on filter-tip cigarettes.

Push moderation for the sake of public relations.

There is one final possibility. As one cigarette man put it, "If we are guilty and they find out what causes cancer we'll remove it from cigarettes."

EXHIBIT 30E

BOOXBAUM & BOOXBAUM,
New York, N. Y., July 15, 1957.

Hon. JOHN A. BLATNIK,
Washington, D. C.

DEAR HONORABLE SIR: I read that you are about to begin the investigation of the claim that filters in cigarettes protect smokers from specific hazards. Undoubtedly, you are referring to lung cancer and coronary conditions. I believe that the information that I have on hand may be of some interest to you.

Presently, I am a practicing attorney. Prior to entering into the legal profession, I was a practicing physician for 15 years. I am fully cognizant of the claims of the American Cancer Society as well as those of the tobacco industry.

Three years ago, I incorporated a research laboratory whose main purpose was to eliminate the chemicals responsible for the irritants contained in ciga-

rettes. Its chemist had devoted his lifetime in the research and having exhausted his moneys, and being on the verge of success, he induced my client to finance the final research. All of the laboratory work was performed in European countries due to his inability to secure the cooperation of owners of pilot plants. Corporations equal to DuPont and International Paper cooperated with the chemist. It was determined that the tarry substance present in cigarettes was benzpyrene. This chemical when rubbed into the skin of mice and other animals produced cancer. It was also found that benzpyrene was present in cigarettes in the following percentages—about 96 percent in the cigarette paper and about 4 percent in the tobacco. This confirms the findings of the American Cancer Society, as well as other research units, that pipe and cigar smokers are less prone to cancer than those smoking cigarettes. Medical research has also found that cigarettes made with rice paper relieves coughs and other respiratory conditions common to the heavy smokers.

The aim of the new corporation was to produce cigarette paper made wholly from pure tobacco. Samples were brought back to the United States. Three cigarette manufacturers in the United States made several thousand packages of cigarettes using the all tobacco fiber paper. They requested certain changes to be made such as—increased combustibility and porosity. This was accomplished, however, the chemist was informed off the record, that they could not use the paper nor advertise its noncancer quality (absence of benzpyrene).

There are only two manufacturers of cigarette paper in the United States which supply the entire cigarette industry. The all-tobacco fiber paper was chemically analyzed and numerous tests were made and compared with the paper now in use. The last test and comparisons were made by Mr. Lafemine of the Miami Cancer Institute. His report, based upon numerous tests, revealed a complete absence of benzpyrene from the all-tobacco cigarette paper. When made into cigarettes and analyzed, and compared to the present cigarettes, the chemical findings revealed about 6–7 percent benzpyrene and about 90 percent benzpyrene present in the cigarettes on the market today.

Incidentally, Mr. Lafemine and the Miami Cancer Institute, were the first ones to find that benzpyrene was the causative factor in cigarettes in the production of cancer. This was more than 4 years ago. I was informed by Mr. Lafemine that he could not secure any tobacco magazine to publish his findings.

Should you desire any further information regarding the various research laboratories as to the findings of this benzpyrene as being the only factor in the causation of cancer and its presence in the cigarette paper now in use, I will gladly furnish you the findings of both England, France and other countries. The use of filters is not and will not benefit the smoker. Ninety percent of the trouble could be eliminated by the use of the all-tobacco paper, a good filter may eliminate some small percentage, how much, I cannot state because I have no records before me.

I hope that the contents of this letter may be of some assistance to you.

Very truly yours,

MAXWELL M. BOOXBAUM.

EXHIBIT 31

STATEMENT OF DR. V. STEFAN KRAJCOVIC, IN RE USE OF FLEXIBLE CARBON AS A CIGARETTE FILTER WHICH WILL REMOVE ALL TARS AND OTHER NOXIOUS ELEMENTS SUSPECTED OF INCREASING THE INCIDENCE OF LUNG CANCER

Flexible carbon, produced from vegetable fibers through an invention (patent applied for) of William T. Soltes, has been described by a nationally known carbon expert as the most promising answer to the problem concerning the probability that cigarette smoking is accelerating cases of lung cancer in the United States, Great Britain, and other parts of the world where cigarettes are in general use.

Unlike other filters, flexible carbon has been adjudged by the carbon expert who is making an extensive investigation, Col. Harry N. Hardsog, as: "Almost too good for any filtering purpose, in that it can remove almost any airborne impurity."

Continuing with Colonel Hardsog's findings: "Cigarette filters can be made to any desired specifications of filtering action. * * * Of course, it is activated carbon in a sense, and has an affinity for absorbing anything that is not air. In a cigarette, it is particularly useful in absorbing the condensed matter resulting from smoking. Its absorption rate is not entirely dependent upon

quantity, but rather on the amount of impurities even though it weighs but one-half the usual filter, which absorbs 27.9 milligrams as compared with 84.1 milligrams registered in tests of flexible carbon (relating to the same weight of tobacco)."

According to Hardsog's report, the flexible carbon filter is 300 percent more efficient than the best filter in use today, and will more than satisfy the demands of scientists who testified before the committee for a reduction of 41 percent of the tar, nicotine, etc., contents recommended for popular brands of United States manufactured cigarettes.

As Mr. Soltes' representative, I and my associates have met with considerable resistance from leading cigarette-manufacturing companies of the United States, when I have attempted to interest them in examining the possibilities of finding a really safe filter for cigarettes.

The tobacco companies—which, in almost every case, have expressed an interest in flexible carbon—have, so far, not come forward to fully examine the possibilities of this new product.

I have also been slightly discouraged by the apparent lack of interest on the part of the Health, Education, and Welfare Department, and its related agencies—including the National Cancer Institute—in thoroughly investigating this new form of carbon, which to many persons who have examined it, shows great promise as an agent which can answer some of the more troublesome problems involved in the current tobacco controversy.

May I suggest that, as many witnesses have indicated before your committee, air pollution which results from automobile, bus, etc., exhausts, incinerators, release of factory gases, etc., could be a very potent factor in the increasing incidence of lung cancer in industrial areas?

In many ways, the tests on flexible carbon show an even greater promise for the eventual solution of problems, such as those encountered in Los Angeles, Calif., Pittsburgh, Pa., and other cities where smog and other situations caused by temperature peculiarities and industrial growth, appear to be causing health problems.

I am convinced that your committee is as much interested in the related possible effects on the increasing incidence of lung cancer in the United States, as it relates to general air pollution, as it is in the main investigation into cigarette smoking.

Flexible carbon, according to Col. Harry N. Hardsog and other experts in this field, can be more quickly, and easily produced as—

(1) Dust filters for air conditioning. Requires only packaging after its characteristics for this purpose have been accurately established.

(2) Cigarette filters. These may be mdae to any desired specification of filtering action. Carbon filters gain weight and the usual filters do not.

(3) Odor filters are feasible, since carbon absorbs odors. May be applicable to auto air systems to eliminate the most annoying road fumes.

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