Yellow fever in the Gambia / by G.M. Findlay and T.H. Davey.

Reprinted from the
Transactions of the Royal Society
of Tropical Medicine and Hygiene.
Vol. XXX. No. 2. pp. 151-164
(Issued July, 1936)

YELLOW FEVER IN THE GAMBIA.

II.—THE 1934 OUTBREAK.

BY

G. M. FINDLAY

AND

T. H. DAVEY.

Wellcome Bureau of Scientific Research, London; and Sir Alfred Jones Research Laboratory, Liverpool School of Tropical Medicine, Freetown, Sierra Leone.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE
MANSON HOUSE, 26, PORTLAND PLACE, LONDON, W.1

TRANSACTIONS OF THE ROYAL SOCIETY OF
TROPICAL MEDICINE AND HYGIENE.
Vol. XXX. No. 2. July, 1936.

YELLOW FEVER IN THE GAMBIA.

II.—THE 1934 OUTBREAK.

BY
G. M. FINDLAY
AND
T. H. DAVEY.*

Wellcome Bureau of Scientific Research, London ; and Sir Alfred Jones Research Laboratory, Liverpool School of Tropical Medicine, Freetown, Sierra Leone.

In a previous communication (FINDLAY and DAVEY, 1936) a brief historical account was given of the various outbreaks either of yellow fever itself, or of diseases closely resembling yellow fever, that had occurred in the Gambia since its discovery by Europeans 480 years ago.

In the present communication observations are recorded on the epidemic that occurred in Bathurst during the last 3 months of 1934.

A few preliminary details in regard to the town may not be without interest.

Bathurst, the capital and only town of any size in the Gambia, was founded in 1816 on the island of Banjola, thereafter renamed the island of St. Mary. The site of the town was apparently selected on strategic rather than on sanitary grounds for the island, 13° 27′ N. latitude, 16° 34′ W. longitude, is in reality little more than a sand-bank approximately 3½ miles long and 1¼ miles broad. The island lies 18 miles from the mouth of the River Gambia which is here some 2½ miles wide ; it is bounded on the north by the main stream of the river and is separated from the mainland to the west by a narrow channel, Oyster Creek. The island has an area of approximately 2,500 acres, but a large part is swampy and during the rainy season water-logged, since the highest point on the island is only about 6 feet above sea-level, while much of the island is from 3 to 4 feet below sea-level. Even in the middle of the dry season subsoil water can therefore be reached by digging down a few feet. As a result

*We have to express our sincere thanks to His Excellency Sir ARTHUR RICHARDS, K.C.M.G., Governor and Commander-in-Chief of the Gambia, without whose sympathetic aid the investigations here described could not have been carried out. Our thanks for many kindnesses are also due to Captain H. OKE, M.C., Colonial Secretary of the Gambia, the Commissioners of Provinces, Dr. H. J. BERMINGHAM, Acting Principal Medical Officer of the Gambia, Dr. LOCKHEAD and Dr. SCOTT-JOHNSTON, as well as to Mr. ERIK JONSON for permission to publish the aerial photographs of Bathurst.

of the physical configuration (Fig. 1) efficient drainage of the island is at present almost impossible, the difficulties being increased by the fact that, apart from an occasional shower in January, the rains are confined to the months of June, July, August and September. Though the rainy season begins and ends with tornadoes, from a half to a third of the year's rains occur in August.

Considerable variation is found in the annual rainfall ; in 1905 for instance, 66·07 inches fell, but in 1913, only 23·68 inches ; the average is about 45 inches.

No correlation exists between the incidence of yellow fever and the annual rainfall either for the year in which yellow fever occurred or for the preceding year. The possibility that certain general conditions were indirectly responsible for the epidemic is, however, suggested by the fact that during the last 3 months of 1934, outbreaks of yellow fever also occurred in and around the town of Kano, Northern Nigeria, in Hill Station, Sierra Leone, and in an isolated prospecting camp in the Gold Coast.

Wide diurnal variations in temperature, sometimes amounting to 30° F., occur during the dry season in the Gambia. The harmattan blows intermittently from the Sahara from December to April.

The population of Bathurst, as given in the 1931 census, was 14,370. In 1840, 24 years after the foundation of the town, the population was only 2,825 Africans and 36 Whites. The increase has been due not to an excess of births over deaths, for only in 1909, for the first time on record, was the number of births slightly in excess of the number of deaths, but rather to immigration from the adjacent countryside.

Although Bathurst was carefully laid out with broad straight streets, the increased population has resulted in very considerable overcrowding (Fig. 2). Complete segregation of Europeans, advocated on numerous occasions, is under present conditions clearly impossible and in addition, many of the houses, more than a hundred years old, are unsuitable for Europeans. Even in 1927, two fresh European houses were erected in the heart of the African quarter.

The principal agricultural export, on which the prosperity of Bathurst and the Protectorate depends, is the ground nut (*Arachis hypogaea*).

The Yellow Fever Outbreak in 1934.

For the first 9 months of 1934, as judged by the monthly returns of deaths (Table I), there was no abnormal degree of sickness among the African population of Bathurst. On 6th August, however, an African female, F.R., aged 32, Aku tribe, was admitted to hospital with fever, severe headache, pains across the small of the back and aching in the limbs. The urine contained albumin : no malarial parasites were found in the blood and nothing abnormal in the stools. The pulse was not characteristically slow. The temperature returned to normal on 17th August without a definite diagnosis having been reached. The patient's blood, when tested 5 months later, was found to protect in a dilution of 1 in 128. At some time, therefore, she had certainly suffered

Fig. 1.

Fig. 2.

Fig. 1.—The Island of St. Mary, Gambia.
Note the swamps surrounding Bathurst.

Fig. 2.—Bathurst, Gambia.

To face page 152.

from yellow fever though whether this was in August, 1934, cannot, of course, be determined.

In September the number of deaths showed a slight, though hardly a significant, increase over the figures for 1933 and 1932—thirty-six as compared with thirty and twenty-four respectively. During October, November and December, however, the number of deaths both of adults and of infants under five showed a definite increase, more marked among adults than among infants. (Table 1.)

TABLE I.

DEATHS IN BATHURST DURING 1932, 1933 AND 1934.

	Under 5 Years.			Over 5 Years.		
	1932	1933	1934	1932	1933	1934
January	4	—	3	22	26	22
February	—	1	—	21	12	17
March	—	—	1	13	23	24
April	1	—	—	20	14	20
May	2	2	—	27	20	29
June	3	1	3	14	20	25
July	4	1	2	14	17	16
August	11	6	4	19	24	17
September	5	4	8	19	26	28
October	8	3	10	19	24	32
November	3	2	5	24	22	29
December	1	1	3	19	22	46
1st quarter of year	4	1	4	56	61	63
2nd " "	6	3	3	61	54	74
3rd " "	20	11	14	52	67	61
4th " "	12	6	18	62	68	107
Total for year	**42**	**21**	**39**	**231**	**250**	**305**

Unfortunately, many Africans dying in Bathurst are unattended medically, so that a majority of the diagnoses are approximate only. Two deaths, however, were certified as due to uraemia, one to acute nephritis and one to acute malaria.

Case 1. The first patient (Table II) suspected as having died from yellow fever was taken ill on the morning of 3rd October and died 3 days later. She dwelt in an old house in Buckle Street, unscreened as are most of the European dwellings in the town. Deaths from yellow fever had occurred in the near neighbourhood in 1928 and in 1911. In fact the whole distribution of the cases in 1934 is strikingly similar to that of the 1911 outbreak.

TABLE II.

CASES OF YELLOW FEVER NOTIFIED IN BATHURST.

(OCTOBER, 1934—JANUARY, 1935.)

Case No.	Sex.	Nationality.	Date of Onset.	Date of Death.	Duration of Illness in Days.
1	F	French	3.10.34	6.10.34	3
2	M	British	31.10.34	5.11.34	5
3	M	„	8.11.34	12.11.34	4
4	M	„	14.12.34	19.12.34	5
5	M	African	22.12.34	25.12.34	3
6 ?	F	„	1.1.35	Recovered : protection test negative	

Case 2. The second case, also fatal, did not occur till 4 weeks later. The patient died on the 5th day of illness. The residence of this patient was on the Marina quite close to the bungalow of Case 4. The third patient resided within 100 yards of Case 1. He became sick 8 days after the onset of illness in Case 2, and died on the 4th day of illness. An interval of 32 days elapsed between the death of Case 3 and the appearance of the fourth case. The fourth patient died on the 5th day of illness.

The clinical symptoms and pathological changes in these four patients leave little doubt that they died from yellow fever. The same certainly does not apply to Cases 5 and 6. The history of these patients is briefly as follows :—

Case 5. J.G., African male, aged 29, was admitted to the Victoria Hospital, Bathurst, complaining of enteritis. Repeated examinations of his stools did not reveal the cause of the intestinal symptoms. On 22nd December, he was given carbon tetrachloride by mouth ; the same evening his temperature rose to 101·4° F., but returned to normal on the following day though he complained of general aching and intermittent headache. On 24th December, his temperature again rose and he began to vomit coffee ground material at intervals. His urine contained albumin and granular casts, and during the last 40 hours of his life only 8 ozs. of urine were passed. He died at 11.55 p.m. on 25th December.

Postmortem.—The conjunctivae were icteric, the subcutaneous fat orange coloured : the liver was pale, with haemorrhagic patches under the capsule : the spleen was enlarged and congested : the kidneys pale. The histological changes were such as might have occurred in acute carbon tetrachloride poisoning. Definite evidence that death was due to yellow fever is thus lacking.

Case 6. V.M., African female, aged 6, was admitted to the Victoria Hospital early in December, 1934, with ulcers on the legs and arms. On 1st January, 1935, she complained of abdominal pain and vomited clear fluid in large quantities : the temperature was 101·4° F. The fever reached 104·4° F. on 4th January, and was normal 6 days later. On the 3rd day coffee ground matter was vomited and on the following day the conjunctivae were icteric. Albumin and casts were present from the 2nd to the 9th day of illness. Blood was removed 18 days after the onset of illness and tested for immune bodies to yellow fever ; none were present. It is therefore probable that this illness was not yellow fever.

Although only the above six cases were notified as having actually, or probably, suffered from yellow fever there is evidence which strongly suggests that at least six other Europeans suffered from yellow fever during the months of October and November. With one exception all were resident on the Marina, in close proximity to the quarters occupied by Cases 2 and 4. The symptoms in these six cases were attributed either to influenza or to malignant tertian malaria though parasites were not found. The clinical picture in all cases was very similar, fever, headache, aching in the back and legs and vomiting, though coffee ground material was absent. In three out of the four cases examined albumin was present in the urine. The temperature fell to normal usually about the 6th or 7th day of illness and convalescence was uneventful. In one case the skin was said to have had a slight icteric tint and in three the pulse during early convalescence was slow. Clinically, therefore, the cases bore considerable resemblance to mild cases of yellow fever.

Additional, though not conclusive, evidence that yellow fever was the disease from which these six Europeans suffered was obtained from an examination of their bloods for the presence of immune bodies to yellow fever.

Blood from five of these patients, examined early in January, showed immune body titres, by the mouse protection test, of 1 in 256 in one case, 1 in 128 in three and 1 in 64 in one. In November, in common with 131 other inhabitants of Bathurst, these five persons received an injection of mouse brain infected with neurotropic yellow fever virus, the virus having been " attenuated " for 4 days at 22° C. No reactions followed these injections. An examination of sera from sixty-five other persons inoculated with this " attenuated " virus 2 months after the injection failed to reveal the presence of immune bodies which were thus present only in the sera of the five persons who had suffered from illnesses suggestive of yellow fever before their inoculation. It seems probable therefore, that the prophylactic injection of " attenuated " yellow fever virus given in November contained no living virus and was thus devoid of immunizing action. This conclusion is strengthened by the fact that one of those receiving the prophylactic inoculation subsequently contracted yellow fever and died (Case 4).

In the case of the sixth person suspected of having had yellow fever, it was only possible to examine the blood 3 weeks after he had been further injected

with yellow fever virus and immune serum. At that time his serum protected in a dilution of 1 in 128.

The evidence thus suggests that among Europeans the outbreak was more extensive than was at first suspected. The same appears to have been true also in the case of Africans. Four African servants, employed by G.P. (Case 4), were found to have suffered during October and November from febrile attacks with headache and vomiting. G.P.'s personal servant, who was thought to have suffered from influenza, was personally tended by his master. He and his three fellow servants all showed immune bodies to yellow fever in their bloods. In the same way an African servant in the employ of two of the suspected cases was also found to have suffered from " fever " in November. He, too, protected against yellow fever.

In order to throw further light on contact infection, sera were examined from eleven young adult Africans who either had suffered from fever during the previous year or during the same period had been in close contact with a fatal case of acute illness. Of these eleven Africans, five showed immune bodies to yellow fever in the blood serum.

Thus, including V.R., of seventeen Africans who had either suffered from undiagnosed fever during 1934, or had been in contact with a fatal case of acute illness, eleven or 64·7 per cent. protected against yellow fever. As controls to this series, bloods were obtained from twenty Africans, permanent residents of Bathurst, 18 to 40 years of age, who had not suffered from any illness during 1934 and as far as they knew, had not been in contact with any acute illness ; eight or 40 per cent. protected against yellow fever.

The figures both for adult contacts and controls are thus higher than those obtained by BEEUWKES and MAHAFFY (1934) who found that among twenty-three children from Bathurst six or 26 per cent. were positive, the youngest positive donor being 5 years of age.

YELLOW FEVER IN THE GAMBIA PROTECTORATE.

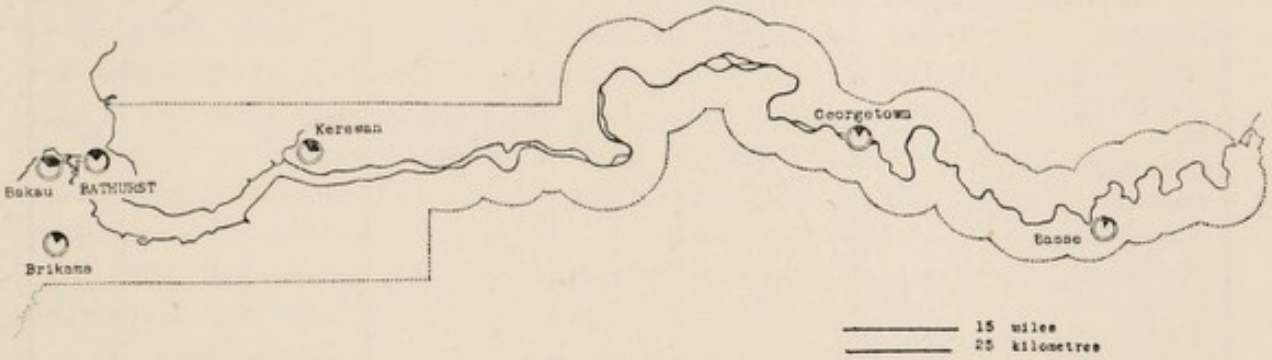
While recurrent outbreaks of yellow fever have been a feature of the history of Bathurst since its foundation 120 years ago, no epidemic of yellow fever has been recorded in the same period in the Protectorate. In 1860, however, three medical officers are said to have died from fever in the same house at MacCarthy Island after 5 days illness; and in 1911, FLOURENS (1913) records that two customs officials died in French territory on the frontier of the Gambia.

Evidence that yellow fever is certainly endemic in many villages in French Senegal, in the neighbourhood of the Gambia, was provided by the survey conducted by STÉFANOPOULO (1933) who found positive sera in Saboya in Senegal not far from the northern frontier of the Gambia.

BEEUWKES and MAHAFFY (1934) also found that of fourteen adults from Georgetown 35 per cent. were positive, while of thirty-one bloods from children

from the same town 23 per cent. were positive, the youngest positive donor being 10 years of age. These bloods were, however, co.lected from boys at the Armitage Schools, MacCarthy Island, and as their homes were widely scattered, no exact evidence was obtainab!e as to the distribution of yellow fever in the Gambia Protectorate.

In order to throw further light on the problem of yellow fever in the Protectorate, bloods were obtained from the following localities : Bakau, Brikama, Kerewan, Georgetown and Basse. The position of these villages is shown in the accompanying sketch map from which it will be seen that the Gambia forms a narrow enclave into French territory. With the exception of Bakau, all the villages are surrounded by orchard bush. Great care was taken to obtain



MAP OF GAMBIA.

Outer circles represent adults ; inner circles represent children ; black sectors indicate the proportion with protective sera.

bloods only from persons who had been born and bred in the respective towns. At least forty bloods were obtained from each town, the population being divided into four age groups, (1) under 10 years, (2) 10 to 20 years, (3) 20 to 40 years, (4) above 40 years. At the same time efforts were made to obtain bloods as often as possible from blood relations in order to determine whether there was any evidence of the infection having a familial or house distribution.

The results of the survey are shown in Table III. In all the five localities bloods were found containing immune bodies against yellow fever, the total percentage of positives varying from 20 per cent. in Basse to 33·8 per cent. in Brikama. In children under 10 years of age the highest percentages were at Bakau and Kerewan—36 per cent. Bakau, the village at Cape St. Mary, is, of course, only 7 miles from Bathurst and in daily communication with it : in addition, a number of Government bungalows are built on the cliff-edge at Cape St. Mary within 100 yards of the village, so that there are thus many opportunities for infection to pass from Bathurst to Bakau and *vice versa*. There is, however, no record of any case of yellow fever ever having occurred in Bakau.

TABLE III.

YELLOW FEVER IMMUNE BODIES IN THE BLOODS OF AFRICANS FROM THE GAMBIA.

Locality.	Age Groups.									Percentage Protecting.		
	Under 10 Years.			10–20 Years.		20–40 Years.		Above 40 Years.		Under 10 Years.	Over 10 Years.	Total.
	P	NP	Approximate Age in Years of Youngest Positive.	P	NP	P	NP	P	NP			
Brikama (South Bank Province)	3	11	8	1	9	5	5	4	6	21	33	33·8
Baku (Cape St. Mary)	4	7	4	1	13	5	4	3	2	36	32	30·7
Kerewan (North Bank Province)	4	7	5	3	8	4	8	1	9	36	24	27·0
Georgetown (MacCarthy Island)	2	10	7	2	12	4	8	6	4	16	33	29·0
Basse (Upper River Province)	1	9	9	2	13	2	10	5	7	10	23	20·0

P = protection ; NP = no protection.

At Georgetown and Basse, the number of children under 10 with positive sera was small, only one, a boy of approximately 9 years of age, being found in Basse and two of 7 and 8 in Georgetown. On the other hand, the number of positive bloods in these up-river towns was high in the age groups 20 to 40 and 40 upwards. Kerewan, in the North Bank Province, was the only town in which it was possible to examine bloods from females. Of seventeen tested five were positive, three between 20 and 40 years, one between 10 and 20 years and one only under 10. No evidence could be obtained indicating any close domiciliary or family relationship between those with positive sera in any of the localities investigated.

MOSQUITOES IN THE GAMBIA IN RELATION TO YELLOW FEVER.*

Although time did not allow of extensive collecting, it was possible to determine the presence in the Gambia of three species of Culicidae not previously

*In this connection we desire to thank Dr. F. W. EDWARDS of the British Museum (Natural History) and Mr. B. JOBLING, Entomologist to the Wellcome Bureau of Scientific Research, for their kindness in identifying the mosquitoes collected in the Gambia.

recorded from this area, *Anopheles rufipes* Gough from Keruan, *A. mauritianus* Duruty and D'Emmerez, and *Mansonia africana* Theo. from Kauur. A complete list of the Culicidae so far recorded from the Gambia is given in Table IV. The following culicine species are known to transmit yellow fever by their bites, *Aëdes aegypti* L., *A. luteocephalus* Newst, *A. vittatus* Bigot, *A. simpsoni* Theo., *Culex fatigans* Weid., *C. thalassius* Theo., *Eretmopodites chrysogaster* Graham and *Mansonia africana* Theo. In addition, the following species, while unable to transmit yellow fever by biting, can, nevertheless, harbour the virus for considerable periods—*Aëdes irritans* Theo., *A. nigricephalus* Theo., and *Mansonia uniformis* Theo.

Eight known transmitters of yellow fever are thus present in the Gambia. The most interesting fact, however, in regard to the distribution of these vectors of yellow fever is the rarity of *Aëdes aegypt* outside Bathurst. On the Island of St. Mary, *A. aegypti* was readily found and was undoubtedly the commonest yellow fever vector present, though *Culex thalassius*, which together with *A. irritans* breeds in the innumerable crab holes, was also numerous. In the Gambia Protectorate, however, there is only one record of *A. aegypti* at at MacCarthy Island. In our own brief survey, *A. aegypti* was again absent. This does not, of course, mean that it cannot be found by intensive search, but it does suggest that possibly *A. aegypti* does not play the predominant role in transmitting yellow fever in rural areas in the Gambia.

The possibility that other mosquito vectors may play a part in transmitting yellow fever in the Gambia raises the further question whether any animals form alternative hosts for the virus of yellow fever, as is possibly the case in South America (SOPER, 1935). A certain number of bloods were obtained from monkeys caught in the neighbourhood of Bathurst, while a limited number of bloods from domestic animals were also examined for immune bodies to yellow fever. The results which have already been discussed by FINDLAY, STÉFANOPOULO, DAVEY and MAHAFFY (1936), showed that two sheep gave positive bloods while the monkey bloods were negative. In other areas in Africa—the Gold Coast, French Guinea and the Belgian Congo—positive primate bloods were obtained. It is therefore possible that in rural areas a supply of infected mosquitoes, other than *A. aegypti*, may be maintained by feeding on certain wild or domestic animals.

DISCUSSION.

The facts described in connection with the incidence of yellow fever in the town of Bathurst and in the Gambia Protectorate raise two important questions : (i) What is the explanation for the occurrence at intervals of some years of explosive epidemics of yellow fever in an urban community ? (ii) What is the explanation for the apparent occurrence of yellow fever in rural areas in the complete absence of epidemics as shown by the presence of yellow fever immune bodies in the sera of persons living in these areas ? If an answer

TABLE IV.

MOSQUITOES IN THE GAMBIA.

FAMILY CULICIDAE.

Species.	Date.	Locality.	Collector.
SUB-FAMILY ANOPHELINAE.			
Nyssorhynchus pharoensis Theo.	1902	Bathurst	Dutton
" "	1911	Gasan	Simpson
Anopheles funestus	1902	Bathurst	Dutton
" " var. *subumbrosa* Theo.	"	"	"
" " *umbrosa* Theo.	"	"	"
* " *costalis* Loew.	1902	"	"
" "	1911	MacCarthy Island	Simpson
" "	1924	Bathurst	Innes
" "	1934	"	
" "	"	Keruan	
" "	"	Kauur	
" *rufipes* Gough	"	Keruan	
" *mauritianus* Duruty and D'Emmerez	"	Kauur	
SUB-FAMILY CULICINAE			
Culex annulioris var. *gambiensis* Theo.	1902	Bathurst	Dutton
C. duttoni Theo.	"	"	"
"	1911	"	Franklin
"	1924	"	Innes
C. invidiosus Theo.	1902	"	Dutton
†*C. fatigans* Wied.	1902	"	"
†*C. thalassius* Theo.	1924	"	Innes
"	1934	"	
C. nebulosus Theo.	1929	"	Innes
C. poecilipes Theo.	1902	"	Dutton
"	1934	Basse	
C. tigripes Grandpré	1911	Bathurst	Franklin
"	1924	"	Innes
C. decens Theo.	1911	Brikama	Simpson
†*Aëdes aegypti* Linn.	1902	Bathurst	Dutton
" "	1911	"	Simpson
" "	1922	"	
" "	1924	"	Innes
" "	1934	"	
" "	1912	MacCarthy Island	
**A. irritans* Theo.	1929	Bathurst	Innes
"	1934	"	
†*A. luteocephalus* Newst	1929	"	Innes
"		Bakau	"
†*A. vittatus* Bigot	1902	Bathurst	Dutton
"	1929	"	Innes
†*A. simpsoni* Theo.	"	"	"
**A. nigricephalus* Theo.	"	"	"
†*Eretmopodites chrysogaster* Graham	"	"	"
**Mansonia (Mansonioides) uniformis* Theo.	1902	"	Dutton
" " "	1911	Kulari	Foster
M. africana Theo.	1934	Kauur	

† Can transmit the yellow fever virus by its bite.

* Can only transmit the yellow fever virus on injection.

could be found to these questions it would go far to solve much that is now obscure in the epidemiology of yellow fever.

So far as Bathurst is concerned, there is no proof either that yellow fever is constantly present in the town or that it is introduced by shipping from other ports on the coast. The only external source of infection would thus lie in the surrounding country. All the evidence, however, points to the fact that the 1934 epidemic was of a typical urban character. There is therefore no need to invoke factors other than (1) the virus, (2) *Aëdes aegypti* and (3) the non-immune human being. The occurrence of an epidemic might conceivably be due to variation in one or more of these three factors.

In the case of certain viruses such as those of influenza and foot and mouth disease, it has been suggested that periodic variations in virulence may occur. The evidence, however, is by no means conclusive and in the case of the yellow fever virus there exists no clear indication of any periodic variation in virulence.

It is possible that periodic variations may occur in the numbers of the mosquito population. It is now known that, in association apparently with climatic cycles, there occur cycles in the population of animals as diverse as silver foxes, lemmings and gall-midges. The numbers of any one species gradually rise to a peak ; then, as a result of food shortage and disease, fall with startling rapidity. In the case of field voles (*Microtus*) in which a 3 to 4 year cycle exists it is now possible to predict with considerable accuracy the times when such population peaks are due (Elton, Davis and Findlay, 1935). Whether mosquitoes pass through similar cycles is unknown, as experimental observations are entirely lacking : on *a priori* grounds, however, there is a strong probability that such cycles do occur.

The third factor in the genesis of recurrent urban epidemics is the presence of non-immune human beings. It has previously been pointed out that in the case of Bathurst increase of population takes place not by an excess of births over deaths, for the reverse is usually the case, but by immigration from the country. A number of these immigrants will be already immune to yellow fever but, if it be assumed that the percentage of immunes among the immigrants is less than among the urban inhabitants, while the percentage of active immunes is also less among the entrants by birth than among the departures by death, it follows that in the course of a few years the proportion of non-immunes in the urban population will gradually rise. If infection is then introduced from the country an epidemic will occur. It must be recognized that the individual carrying the infection need not necessarily be a virulent case of yellow fever : he might well be a case of inapparent infection. In this connection, it must also be remembered that many species of animals both wild and domestic which do not exhibit any symptoms of yellow fever after inoculation may nevertheless allow the virus to circulate in their blood for some days : if brought into the town during this period they would provide a source from which urban mosquitoes could become infected.

One other point in connection with the possible introduction of yellow fever from the country to the town appears worthy of further consideration. In connection with certain malaria outbreaks it has been shown that prior to their onset an immigration of anopheline mosquitoes occurs. Whether similar immigrations of aedine mosquitoes take place is as yet unknown.

From all the evidence it is suggested that yellow fever is not endemic in the town of Bathurst, but that the virus is periodically reintroduced, either by means of an infected human being, in the blood of an infected animal or possibly even by the immigration of infected mosquitoes. Whether an epidemic actually occurs will depend on the proportion of non-immunes in the population and on the stegomyia index.

To explain why no outbreak of yellow fever has ever been recorded in the Gambia Protectorate, though from 18 to 33 per cent. of the inhabitants possess immune bodies to yellow fever, requires a consideration of the divergences shown by SOPER (1935) to exist in South America between urban and jungle yellow fever.

Before discussing these divergences, however, it should be emphasized that no serious criticism has been levelled against the high degree of specificity of the mouse protection test. Of the diseases with which yellow fever might be confused clinically, malaria, blackwater fever, dengue, Rift Valley fever, epidemic catarrhal jaundice and influenza, all yield negative protection tests. BOYÉ (1935) has recently suggested on somewhat slender evidence that " red fever " (fièvre rouge du Congo) may give a positive protection test. Apart from the fact that red fever with its conspicuous symptoms has never been recorded from areas such as the Anglo-Egyptian Sudan where sera give a positive test for yellow fever, a recent observation by one of us (G.M.F.) has shown that serum from a fully authenticated case of red fever, for which we are indebted to Dr. C. C. CHESTERMAN, did not show the presence of immune bodies to yellow fever virus. There is thus little doubt that the high percentage of positive sera found in the Gambia Protectorate is evidence that infection with the virus of yellow fever has occurred.

The fact that no clinical cases have been noted may be due to a combination of circumstances such as :—

(*a*) The racial resistance of the African to yellow fever as a result of which the disease is usually mild or inapparent and the mortality rate low.

(*b*) The possibility that in certain cases immune bodies may be transmitted hereditarily from mother to child : during the period of passive immunity the child might be bitten by infected mosquitoes, thus acquiring an active immunity without clinical symptoms.

(*c*) The entire absence in African villages of any record of deaths.

(*d*) The possibility that in many African villages infection is acquired not in the house but in the fields as a result of the bites of certain non-domestic

mosquitoes. If solitary cases occurred only at irregular intervals, there would be little chance of their being seen by European observers.

Now the characteristics of jungle yellow fever, as described by SOPER (1935), are :—

(i) The disease is endemic, explosive outbreaks do not occur.

(ii) The disease is not transmitted by *Aëdes aegypti.*

(iii) The disease does not attack whole families and is contracted not in the house but in the field.

(iv) Young adults are, therefore, more likely to be attacked than small children.

(v) Immune bodies are found in the bloods of monkeys obtained from endemic areas, infection being maintained in the absence of susceptible human beings.

It is obvious that a close similarity exists between South American jungle yellow fever and the conditions existing in the Gambia Protectorate. Further observations are obviously required before it can be said that typical jungle yellow fever actually exists in Africa: in particular, evidence must be obtained that yellow fever is occurring in the complete absence of *Aëdes aegypti.* It is however certain that the epidemiology of yellow fever in Africa is far more complicated than was at one time supposed. If jungle yellow fever, transmitted by non-domestic mosquitoes, actually exists, preventive measures of value in combating urban yellow fever are of little avail. Personal prophylaxis by artificial immunization is at present all that can be attempted. In the Gambia this has been carried out since the greater part of the European population has now been actively immunized. The results of this immunization will form the subject of a further communication.

So far as urban outbreaks are concerned, the reduction to and maintenance of the mosquito index at zero is sufficient to guard against the possibilities of reintroduction of the virus.

CONCLUSIONS.

1. The epidemic of yellow fever that took place in Bathurst in 1934 is described. Evidence is brought forward to suggest that the cases actually diagnosed were but a part of those that actually occurred.

2. Although yellow fever epidemics have never been described among Africans living in the Protectorate, a survey of the Gambia shows that in a number of villages from 20 to 33 per cent. of the inhabitants possess immune bodies to yellow fever.

3. A list of the culicine mosquitoes recorded from the Gambia is given.

4. The urban type of yellow fever seen in Bathurst is contrasted with the rural type found in the Protectorate. The possibility that the rural type of yellow fever in Africa is similar to the jungle yellow fever of South America is discussed.

REFERENCES.

BEEUWKES, H., & MAHAFFY, A. F. (1934). The past incidence and distribution of yellow fever in West Africa as indicated by protection test surveys. *Trans. R. Soc. trop. Med. Hyg.*, xxviii, 39.

BOYÉ, L. (1935). La " fièvre rouge " congolaise et le test de protection amaril en Afrique equatoriale française. *Bull. Off. int. Hyg. publ.*, xxvii, 1319.

ELTON, C., DAVIS, D. H. S., & FINDLAY, G. M. (1935). An epidemic among voles (*Microtus agrestis*) on the Scottish border in the spring of 1934. *J. Anim. Ecol.*, iv, 277.

FINDLAY, G. M., & DAVEY, T. H. (1936). Yellow fever in the Gambia. I—Historical. *Trans. R. Soc. trop. Med. Hyg.*, xxix, 667.

FINDLAY, G. M., STÉFANOPOULO, G. J., DAVEY, T. H., & MAHAFFY, A. F. (1936). Yellow fever immune bodies in the blood of African animals. Preliminary observations. *Ibid.*, xxix, 419.

FLOURENS. (1913). Note sur la dernière épidémie de fièvre jaune au Senegal. *Rev. Méd. Hyg. trop.*, x, 31.

SOPER, F. L. (1935). Rural and jungle yellow fever. A new public health problem in Colombia. (Lecture before the Faculty of Medicine of Bogota.) Bogota : Editorial Minerva, S.A. (translated from *Rev. Hyg.*, 1935, iv).

STÉFANOPOULO, G. J. (1933). Quoted by BOYÉ, L. Les cas européens de fièvre jaune en Afrique occidentale française pendant l'année 1932. *Bull. Off. int. Hyg., publ.*, xxv, 1015.

PRINTED IN GREAT BRITAIN.

H. R. GRUBB, LTD.,
PRINTERS,
POPLAR WALK, CROYDON.