An attempt to ascertain the absolute toxic possibilities of fritted lead glazes ; White lead and plumbism / by W. Thomason.

With the Author's Compliments.

26.

An Attempt to Ascertain the Absolute Toxic Possibilities of Fritted Lead Glazes.

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White Lead and Plumbism.

BY

W. THOMASON.

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XX.—An Attempt to Ascertain the Absolute Toxic Possibilities of Fritted Lead Glazes.

By W. THOMASON (Lambeth).

SINCE the introduction of fritted lead glazes, and particularly since the classification of these into sections by the Government method of extraction in the cold with ·25 per cent. HCl, there has been a certain divergence of opinion as to the efficacy of fritting as a means of preventing plumbism. In my view there is no warrant for this divergence, since, quite apart from the merits or demerits of the Government method of classification, the returns of lead cases from such factories as are working under the 5 per cent. standard of solubility showed no cases of plumbism for years amongst workers in the prepared glazes—such case as did occur was in course of preparation of the frit—and the information available from the Continent is to a similar effect.

This was the position up to the beginning of 1909, when curiously enough a case—and a fatal case—occurred at the Lambeth works of Messrs. Doulton & Co., Ltd., and the subject was a man who for some years had worked in lead glazes of solubility under 5 per cent.

The circumstances really bear out the opinion that these glazes are relatively innocuous, though at a first glance the opposite would, of course, appear to be the case.

The worker had for many years previous to the introduction of fritted lead been subject to lead poisoning from raw lead glazes, and had on several occasions been off work seriously ill with colic, which was undoubtedly set up by the raw lead—his gums were black and his teeth loose, and he would have been removed to other work had not the fritted lead glazes been introduced. From the time these glazes were in use he had no colic, though it was in this way that previously the lead had affected him, and though he was occasionally off work with illness the certificate was never that of lead poisoning, but of hemorrhage of one form or another, until twelve months before his death, when *previous* lead poisoning was adduced as the reason for the hemorrhage.

At this point he was taken from dipping and placed in another part of the works—light outside work—and he held this position until the time of his death, which took place about twelve months after he had been in contact with lead in any form. The immediate cause was hemorrhage on the brain, following on Bright's disease, following on lead poisoning—not recent poisoning.

All active symptoms of lead poisoning disappeared from the introduction of the fritted glazes. The abdomen at the time of the autopsy was grey on the exterior, and the bowels themselves were black. I shall refer to this fact later on, as I think it has a certain bearing upon the deductions to be drawn from the experiments I have made.

At the present time it is the custom to classify glazes by means of a method which in itself conforms to few of the conditions which exist in the human animal. The acidity of the extracting solution is approximately that of the gastric juice of the healthy human animal, but the temperature at which the extraction is conducted is not the body temperature, and the HCl used in the extraction admittedly does not fully represent the gastric juice. The time of contact is perhaps a fair statement of the average period which food remains in the stomach; though this period is so variable as to be hardly capable of an exact statement.

In the light of these dissimilarities it is not unreasonable to draw a *prima facie* conclusion that the solubility in the laboratory and in the human system are not comparable, but if we look somewhat more deeply into the matter we shall see that these *prima facie* conclusions receive little further support.

In the first place, if the extraction be continued for twice the prescribed time, or longer, relatively little increase in soluble PbO is obtained.

Secondly, if extraction be continued for half the prescribed period, almost as much lead becomes soluble.

Thirdly, if the strength of the acid be increased from ·25 per cent. to 2·5 per cent. but little extra solubility is obtained.

Hence we are partially entitled to draw the conclusion that the want of similarity between the Government method of extraction, and extraction in the stomach, cannot be so very different since the portions dissolved are fairly constant under very varying laboratory conditions.

These known facts are interesting in themselves, but apart from the corroboration to be found in the records of plumbism in potteries they would be insufficient, and accordingly I set myself to ascertain, if possible, what is the actual difference between the Government and the animal system methods.

The first point to be examined was the effect of acids other than HCl, of same equivalent strength, upon the same frit in the same degree of fineness—this by reason of the fact that the gastric juice consists, apart from the enzymes which it contains, of lactic, acetic, and hydrochloric acids.

The following table shows the solubility of a particular majolica glaze in these acids at 15°, and at 37·7—40°—also the effect of mixed acids.

TABLE A.

Agitation for 1 hour, and standing in contact with acids for 1 hour in all cases. Acid equivalent to ·25 per cent. HCl.

	Hydrochloric acid.	Acetic acid.	Lactic acid.	Mixed acids
PbO ..	15° 37·7° 2·02% 3·66%	15° 37·7° 1·96% 3·27%	15° 37·7° 2·28% 3·53%	15° 37·7° 2·01% 3·40%

It is therefore permissible to infer that the small amounts of acids, other than HCl, present in gastric juice are unimportant, and that the use of HCl alone in laboratory practice is sound in this respect. From Table A we find that on an average the solubilities of the lead frit at 15° and at 37·7° are as

$$2{\cdot}07 : 3{\cdot}46 = 1{\cdot}00 : 1{\cdot}67 = 3 : 5.$$

The action of butyric acid which occasionally occurs in the stomach in very minute quantities was not ascertained, because of its offensive smell and from the uncertainty as to its presence.

The next point was to ascertain the action of a solution of pepsin in ·25 per cent. HCl on lead frit. Here as a matter of convenience the material was frit only, not a completed glaze. Side by side with these, experiments were made with a solution of acid, pepsin and egg albumen, and the results are as follows:

TABLE B.

500 c.c. ·25 per cent. HCl in all cases.

	1 hr. and 1 hr.	1 hr. and 1 hr.	1 hr. and 1 hr.	2 hrs. and 2 hrs.	2 hrs. and 2 hrs.	2 hrs. and 2 hrs.
Pepsin	None	None	4 grms.	None	4 grms.	4 grms.
	15°	37·7°	37·7°	37·7°	37·7°	17 grms. Albumen
PbO	2·35%	4·54%	3·98%	5·76%	5·00%	4·55%

Conclusions.

The acceleration due to rise of temperature is somewhat above that previously found on complete glaze, and is as 1·93 :

1·00. The extra solubility set up by doubling the period of action is 1·27 : 1·00 in absence of pepsin, and 1·26 : 1·00 in presence of pepsin.

The effect of pepsin which was intentionally used in great excess is slightly to retard action, the solubility being as ·88 : 1·00, and if nitrogenous matter also be present ·79 : 1·00.

At this point I had some communication with physiologists who were approaching the matter from the same standpoint as myself, and from the point of the action upon cats. I was informed that the foregoing experiments were really of inconsiderable value since they had been conducted in acid solutions equivalent to ·25 per cent. of HCl, whereas pepsin was most active in ·15 per cent. HCl solution $\left(\text{really } \frac{\cdot 13 \text{ HCl}}{\cdot 02 \text{ Lactic}}\right)$ hence the pepsin had not had a proper chance to do its work. I pointed out that digestion had, as a matter of fact, gone on since the flocculent portions of the albumen had been rapidly cleared up, but to make matters quite certain I repeated some of the experiments with results as follows:—

TABLE C.

Two hours shaking and two hours standing in all cases.

	500 c.c. ·13 HCl ·02 Lactic acid at 37·7°	500 c.c. ·25% HCl 37·7°	·13 HCl / 500 c.c. ·02 Lactic acid — ·15 HCl equivalent. ·5,000 grm. pepsin at 37·7°
PbO ...	5·40	5·76	5·30%

The action therefore is slightly less than in the old series with HCl of ·25 per cent. strength, and here we have evidence that the acid alone in the constituent we must consider. In this experiment the pepsin not being used in the same excess has not retarded quite so much as before, but most emphatically had no accelerating effect, which is precisely what the chemist would expect, though medical men starting from different premises might hold different views. The argument used to me was the fibrin argument—fibrin being quite insoluble in dilute HCl, but rapidly attacked by a solution of pepsin in the same acid, an argument which it is straining somewhat to extend to a powdered glass. Here, and in all subsequent experiments, the pepsin is that described as of 5,000 test, meaning, I believe, the number of times its own weight of albumen which it can digest.

In the foregoing the food used had been nitrogenous only, since it is from this that the most probable effects would be obtained; but further experiments were made with a mixture of bread and milk as representing carbohydrates, fats and proteids, though of course not to the extent in which they occur in actual life.

The proportions used and the lead soluble are as in Table D.

TABLE D.

Period = Two hours and two hours in all cases, and acidity equivalent ·13 HCl% to ·02 Lactic acid.

—

·15% HCl.

Temperature 37·7°—40°.

1	2	3
1·000 grm. frit ·5000 ,, pepsin 200 c.c. milk 300 c.c. weak acid 20 grms. stale bread Solution divided into two parts and examined for lead colorimetrically and gravimetrically. Colorimetric = 5·00% PbO. Gravimetric = 5·33% PbO. Mean = 5·13% PbO.	— ·5000 grm. pepsin 200 c.c. milk 300 c.c. weak acid 20 grms. stale bread	1·000 grm. frit ·5000 pepsin 500 c.c. weak acid Gravimetric only, and organic matter destroyed by oxidation with HCl + $KClO_3$ PbO = 5·86.

We therefore find that the food substances, in this instance as in the case of albumen only, produce a slight retarding effect upon the solubility of the frit. Column 2, Table D, will be referred to later, and column 3 is merely a control.

I have found it impracticable to reproduce the conditions of one extraction exactly on a later occasion, and have consequently always made use of a blank or control in each separate experiment. Hence, each table must be considered by itself, and not in conjunction with others. The difficulty is chiefly that of maintaining an absolutely regular temperature.

The colorimetric estimation as above was conducted on quite ordinary lines with SH_2 as a blackening agent, but was distinct in that the solution used in matching the tint was that obtained from column 2, Table D. By this means any possible inaccuracy arising out of the organic matters in solution was avoided. The lead was a standard solution of $PbCl_2$, and I have found that with these precautions the method is capable of reasonably accurate results—that is, as accurate as any

colorimetric method can be, and capable of discriminating up to one part in about twenty-five. One other point is to be observed, namely, that the SH_2 solution shall be added to the $PbCl_2$ solution, not the reverse, precisely as Nessler's reagent is added to the ammonium chloride solution in water analysis.

We have now seen that pepsin and food stuffs both somewhat reduce the action of plain acid upon frit, and that accordingly a pure acid of proper strength *can* be used as a guide to the action of actual gastric juice, but it may be asked how this retarding action is set up. Is it a secondary action resulting in re-precipitation of lead which has been taken into solution in the acid, or is it merely mechanical? To decide this, I took 100 c.c. of clear solution from column 2, Table D, and digested at body temperature with 25 c.c. $PbCl_2$ solution as used in the colorimetric estimation described above. Digestion continued for four hours, and the solution at end of that time was perfectly clear, suggesting mechanical action only. This point is a trifle more fully dealt with in portion of paper on white lead solubilities.

In this way I endeavoured to deal with the gastric digestion of lead frits, but physiologically the digestion does not end here. So far as most foods are concerned, it is now recognised that much of the digestion and more of the absorption goes on in the intestine, and it has been argued that from this cause the mere extraction with HCl is insufficient, since in the intestine the digestion occurs in a different solution and for a longer period.

The fact is that the essential change is fron an acid to an alkaline solution + a change of enzyme, since pepsin is inactive in alkaline solution. This fact discounts very largely all attempts to bring intestinal digestion into the discussion, since the probabily of action by weak alkaline solutions is remote.

TABLE E.

1	2
1·0000 grm. frit	200 c.c. milk
200 c.c. milk	300 c.c. water
300 c.c. water	20 grms. bread
20 grms. bread	25 c.c. pancreatic extract
25 c.c. pancreatic extract	·5000 grm. Na_2CO_3
·5000 grm. Na_2CO_3	
PbO in solution = ·40%	

However, here are the actual results obtained by matching as nearly as possible the conditions in the intestine. In earlier attempts a dried pancreatic extract was used, but the results

are not recorded, since the activity of the enzyme could not be quite relied on. Subsequently a liquid pancreatic extract was used.

Digestion continued for four hours with repeated agitation, and the solution from column 2 was used in matching the tint by $PbCl_2$ solution. A further attempt was made in which the digestion was continued for six hours and filtration proceeded overnight, but it was found that the reaction of the solution had gradually changed, and in the morning it was faintly acid. Under these conditions the solubility rose to ·60 per cent.—in each case quite trifling, and probably that portion of the frit which is immediately attacked by acid in gastric digestion, that is loosely held PbO.

It might have been interesting to see if any PbO at all is extracted by above solution from a frit already exhausted by HCl, but the amount extracted under recorded conditions was so small as to make this unnecessary, particularly in the light of what is to follow.

We now know that practically no further extraction goes on in the intestines after a frit has reached this point from the stomach, and the question now arises as to the action of the alkaline pancreatic extract upon such $PbCl_2$ as may be dissolved in the stomach but not absorbed before being carried forward into the intestine. To ascertain this, 100 c.c. of column 2, Table E, was digested four hours at body temperature with 25 cc. $PbCl_2$ solution = ·1 grm. PbO. The solution becomes turbid, but no very obvious precipitation goes on during this digestion. Fifty c.c. of the above 125 c.c. was filtered and the lead estimated by destroying the organic matter with HCl + $KClO_3$ as before, precipitation with SH_2, and weighing as $PbSO_4$.

50 c.c. mixed solutions	= ·0400 grm. PbO.
In filtrate after digestion	= ·0331 ,, ,,

Therefore 83 per cent. of the PbO is not precipitated by an excess of Na_2CO_3 in presence of the organic matter from food and pancreatic extract, so it would appear that most of the lead which reaches the intestine may actually be absorbed in the intestine. We must remember, however, that in all the experiments which have gone before, the medium separating soluble from insoluble has been filter-paper, whereas in the animal it is a less permeable tissue.

This closed experiments of the filter-paper series. All the foregoing experiments being subject to the objection I have named the investigation was carried a step further, and semi-permeable media used in separating the solids from materials in solution.

This means, of course, that the figures previously obtained cannot be exceeded, but may be reduced, since the poisoning effect of lead salts in solution is dependent upon the amount absorbed from the stomach and intestine into the system.

We are no longer concerned by the degree to which lead frit dissolves in acid—this we know—so in the following experiments the lead was introduced as $PbCl_2$ solution, and digested with HCl, pepsin and food at 37·7° in a dialyser, using parchment as the membrane.

Here we have rapid demonstration of the activity of the pepsin since at the end of four hours the membrane was perforated at several points and reduced to a pulpy condition throughout, hence it was necessary to have recourse to parchment paper as the medium. The figures are as follows:—

TABLE F.

25 c.c. $PbCl_2$ solution = ·1000 grm. PbO.
100 c.c. milk.
150 c.c. dilute acid.
10 grammes bread.
·250 grm. pepsin.

At the end of 15 minutes lead could be identified in the surrounding liquid, and at the end of four hours it was very prominent there. This establishes that lead rendered soluble by the stomach *can* pass as chloride into the system through the walls of the stomach. We do not know, however, that all such soluble lead is actually absorbed, in fact, since it is known that most of the absorption of foods goes on in the intestine, we may infer that much of the $PbCl_2$ is carried with the food into the intestine, and we have therefore to ascertain what goes on in this organ in respect of already soluble lead salts.

The mixture as in F was made and digested in a flask for four hours at body temperature. At this point it was halved, and both portions rendered neutral with Na_2CO_3, using methyl orange as indicator, and checking the neutralization by

TABLE G.

Digested two hours with vellum as membrane.
No lead in exterior liquid, but strong reaction for chloride with $AgNO_3+HNO_3$.

TABLE H.

Digested four hours with parchment paper.	Digested four hours with parchment paper.
No lead in exterior liquid, but very strong reaction for chloride.	No lead in exterior solution.
	Large amounts of chlorine.

spotting on litmus paper. To one of the neutralised portions, marked "G," ·25 grm. Na_2CO_3 was added in excess +7 c.c.

pancreatic extract, and the mixture digested in a dialyser having a vellum membrane, and again in a parchment paper dialyser.

The other neutralised portion, marked "H," was rendered alkaline by the addition of $NaHCO_3$, equivalent to ·25 grm. Na_2CO_3, and subsequently treated as G.

Longer contact than two hours digested the vellum, so we have evidence of the activity of the pancreatic extract. That the dialyser was in working condition is evidenced by the strong reaction for chlorine in exterior liquid, and by the strong yellow colour assumed by this liquid owing to transmission of methyl orange used in neutralising the interior liquid.

We see from the foregoing results that no lead from a lead chloride solution rendered alkaline with Na_2CO_3 or $NaHCO_3$ in presence of food stuffs and enzyme can pass the semi-permeable walls of the intestine, although such lead is not actually precipitated and will pass a filter-paper to a very considerable extent—such lead is in fact colloidal and will pass out of the system in the solid excreta. In my opinion this colloidal state of the PbO accounts for the black condition of the bowels of the man to whom I referred earlier in this paper. The lead is unable to pass the bowel-wall, so remains in the tissue or on its inner surface, and is slowly blackened by the sulphide in the system.

Reviewing the foregoing figures, we find that:—

(1) A rise of temperature from 15° to 37·7° produces a growth of solubility equal to a ratio of 5 : 3 or thereabouts.

(2) Presence of pepsin equal to that in the gastric juice produces a slight retardation approximating to 2 per cent.

(3) Even comparatively small amounts of foodstuffs produce a retard of 5·86 to 5·16=about 12 per cent., and this effect would be the greater the fuller the stomach up to a reasonable limit, as is shown more fully in paper dealing with white lead.

(4) Solution of lead stops from the moment the frit enters the intestine.

(5) Absorption of lead chloride does on in the stomach.

(6) No absorption of lead salts goes on in the intestine, though here most absorption of food takes place.

The toxic effect of glaze is entirely a matter of stomach action.

The increase on the cold extraction figures is to be discounted by the pepsin and food contents of the stomach. It is further subject to reduction by the fact that the smaller portions of the solid contents of the stomach are more rapidly transmitted to the bowel than are the coarser portions, hence two hours contact is certainly too long.

It is also to be remembered that the dust of fritted lead falls more rapidly from suspension in the air than does that of white lead.

Under a combination of such conditions, it appears to me that we are right in assuming the solubility of a glaze in the human stomach is effectively gauged by the method of extraction in cold solution in weak HCl, though this solution does not conform either in temperature or composition to those of the gastric juice, and that as no action goes on in the intestine the figure thus obtained is a fair statement of the toxic possibilities of a glaze.

It has been suggested by Drs. Goadby and Goodbody that, in the bowel, frit which has passed the stomach comes into contact not only with an alkaline solution, but with an excess of carbonic acid gas, and this CO_2 is supposed to effect further solution of the lead in the frit. Exactly why CO_2 should be credited with a greater power to attack a frit than has a gastric juice, I do not know, but as the suggestion has been made by physiologists, whose opinions have been asked by the Lead Committee which has just reported, it was investigated.

The same mixture of frit, food and enzyme + Na_2CO_3, as in Table E, was digested for four hours at 37·7°, and during the whole period a stream of CO_2 was allowed to bubble through the liquid. Lead oxide found in the solution amounts to ·20 per cent., or rather less than when the CO_2 was absent from the reaction.

In the *Lancet* of October 2nd, 1909, there appears an article by Drs. Goadby and Goodbody on the subject of industrial lead poisoning, and in it the authors state the conclusions at which they have arrived by the direct administration of lead oxide in one form or other to cats.

These gentlemen also started to attack the question in a similar manner to that outlined in this paper, but it is significant that no reference whatever is made to the chemical evidence in their article.

The only specific case they quote is of a cat which ate with its food white lead of as much as 1·000 gramme per day, and at the end of eight months this cat was stated to be in no way affected by the treatment. This is intended to imply that white lead is not really a poison when taken into the stomach, but the authors seem to have lost sight of the fact that Dr. Goadby, in the *Journal of Hygiene*, of April, 1909, found another cat fed on ·05 to ·10 grms. showed signs of poisoning on the tenth day, and colic occurred on the 21st day.

Beyond this one instance the authors make no reference to the accepted theory that the lead dust reaches the stomach and lungs by respiration, but confine their remarks to the effect produced on cats by inoculation of lead acetate, carbonate and bisilicate. Exactly how this bears upon the question of industrial lead poisoning they do not explain, nor do they even here give details of their work, but merely state their conclusions by saying details would be out of place in the *Lancet*; perhaps we may ask, why out of place?

Referring further to the paper by Dr. Goadby in the *Journal of Hygiene*, we find that here the author studies the effect of respired lead as compared with the same lead compound eaten with food. He shows that white lead dust may be aspirated through wash-bottles with damp walls and joined up with ¼ in. india-rubber tubing, and still make its appearance at the end of the series; but does anyone doubt that such would be the case, and does anyone doubt that a certain amount of white lead will make its way into the lungs as well as into the stomach? It has yet to be shown, however, how far such lead as reaches the lungs is absorbed there and not merely expectorated later, or remains to pigment the lungs along with the other inhaled matter, such as carbon, etc., etc., from the air In the course of his experiments Dr. Goadby ascertains the effect of gastric juice from a healthy man upon white lead, litharge, and lead sulphate. He causes this solution to act on the above lead salts and litharge at 37·7°, and finds the lead sulphate is slightly the most soluble of the three, but not wholly soluble, and hence he argues sulphuric acid is no use as a preventive of plumbism in those cases. To state that lead sulphate is as soluble as white lead or litharge in gastric juice is absurd on the face of it; but, curiously enough, the conclusion arrived at by the author is sound. though his method is quite unsound.

Looking into his figures we see that in each case 10 c.c. of gastric juice was caused to act on ·1,000 grm. white lead, etc., etc. The gastric juice would be ·15 to ·25 per cent. HCl, and if we assume it to have been ·20 per cent. we shall not be far wrong. 10 c.c. of this acid = ·02 grm. HCl = ·06 PbO = ·07 white lead. Hence the reason why he did not get complete solution of his litharge and white lead is the fundamental one that equivalents prevented him, his acid being insufficient for the purpose he put it to.

—*Read March 12th, 1910; Received April 8th, 1910.*

XXI.—White Lead and Plumbism.

By W. THOMASON (Lambeth).

THE foregoing experiments concern themselves chiefly with such glazes as have their lead fritted, though, of course, the conclusion as to non-absorption of lead in the intestine bears on lead from all sources.

In the following I have endeavoured to show as far as it is possible in laboratory practice what will go on if raw lead glazes gain access to the stomach, and, as before, the conditions are made to embrace a stomach without food, and a stomach containing considerable amounts of food, which food also is of a variable character.

In the first case solutions of HCl (·15 per cent. and ·25 per cent. HCl) were made to act on white lead in absence of food or pepsin.

At body temperature it was found that white lead in either of these acids is completely dissolved in ten minutes. The quantities taken were ·200 grm. white lead and 500 c.c. solution, or a somewhat higher proportion of lead to acid than is adopted in the usual Home Office classification of glazes, but still providing an ample surplus of acid over base.

Secondly, the same experiment was carried out with the above acids in presence of pepsin, and no appreciable retard in solution was observed. In these cases it was quite obvious when the white lead had dissolved, since the solution was clear and the progress of the reaction could be observed. Hence it is evident that under industrial conditions, which presuppose a very small amount of lead to be present in the stomach at one time, and, in absence of food, solution must proceed rapidly and completely.

In a further experiment the white lead was raised to 2·000 grm. – 500 c.c. acid (·15 per cent.) = about a 30 per cent. excess of acid over the equivalent lead present, and about 75 per cent. of the above large amount dissolved in two hours—that is to say, in this period the solution of the white lead proceeded to the point at which about 66 per cent. of the acid was saturated. Such conditions can scarcely be called industrial, and the point is raised merely to show that in plain acid and pepsin solution, which fairly well represents gastric juice, the presence of very little such solution will be sufficient to ensure the solubility of the small amounts of lead which, industrially, are possible at one time in the stomach.

Proceeding to a consideration of the effect of gastric juice upon white lead in the presence of foodstuffs, we find it is not by any means so simple a process as in the preceding case—we find the effect varies with the proportion of acid, food, and lead present.

TABLE A.

200 c.c. milk
300 c.c. weak HCl
20 grms. bread
·5 grm. pepsin
} ·25% HCl, digested two hours, of which one was with agitation

·5000 grm. raw earthenware glaze	·5000 fritted earthenware glaze
PbO in solution = ·088 grm. = 17·6% = 94% of PbO present	PbO in solution = ·018 grm. = 3·6% = 18·5% of PbO present

Here, with a low lead content, but still very high from an industrial point of view, and a large amount of acid and not very much food, the solution of the glazes are just about in accordance with the solubilities of the glazes in ·25 per cent. HCl cold. Under Home Office conditions the solubilities are 3·2 per cent. and 17·0 per cent.

If, however, we take a considerably larger amount of the glazes and reduce the acidity to ·15 per cent. HCl, maintaining the food at same relative amount as before, we get:—

TABLE B.

100 c.c. milk
150 c.c. weak HCl
10 grms. bread
·25 „ pepsin
} ·15 per cent. HCl, digested two hours

1·25 grms. fritted glaze	1·25 raw glaze	·25 grm. fritted glaze
PbO in solution = ·016 = 1·28% PbO in solution on total PbO = 6·6%	= ·028 = 2·24% = 12·0%	= ·008 = 3·2% = 16 5%

Here the increase of lead operated upon, though in all cases insufficient to approach neutralisation of the acid, has a very considerable effect on the case. The lead dissolved from 1·25 grms is only twice that removed from ·25 grm of the same glaze, whilst the raw lead glaze is not twice as soluble as the fritted, whereas in the presence of the smaller amounts of lead —that is, under more approximately industrial conditions—the solubility of the raw is nearly five times that of the fritted.

Obviously, then, dosage has an effect on the relative poisoning possibilities of raw and fritted glazes, and it must not be forgotten that in industrial poisoning the conditions as to lead present are nearer to Table A than Table B.

At this point it may be worth while to state that throughout these experiments it has been found that white lead when acted upon as such is distinctly less soluble than is the same amount contained in a glaze. The reason is the purely mechanical one of aggregation by the unmixed white lead, and my meaning will be illustrated by the following:—

TABLE C.

100 c.c. milk
150 c.c. weak acid
10 grms. bread
·25 „ pepsin
Digestion two hours, as before.

·05 grm. Lambeth white lead	·05 grm. Stafferdshire white lead	·25 grm. raw earthenware glaze, practically equivalent in PbO to ·05 grm. white lead
Lead in solution = ·024 grm. = 48%	·024 48%	·036 14·4%
PbO soluble on PbO present = 56%	56%	77%

Such being the case it will not be quite correct to assume in the feeding of animals that the same result must arise from the treatment with white lead as such as with the same amount of the lead in glaze form.

We have seen enough now to show the necessity for something rather more systematic in the study of these solubilities, and the subject was approached considering its three possible variables separately.

The effect of varying the amount of white lead, keeping food and acid constant.

TABLE D.

100 c.c. milk
150 c.c. weak acid } ·15% HCl
10 grms. bread
·25 „ pepsin
Digestion two hours, as before.

White lead	= ·05 grm.	= 1·000 grm.	1·500 grm.
Lead oxide soluble ...	= ·024 = 48%	= ·051 „ = 5·1%	·070 „ 4·66%
PbO soluble on PbO present	= 56%	= 6·0%	5·4%

The increase of soluble lead here, as in case of glazes, is shown not to be in proportion to the amount of PbO present, even though at the end of the reaction a large amount of free acid remain in the liquid. This is no doubt due to two causes, namely, the ratio of surface to volume which is greater in the smaller amount of PbO, and again to the fact that, in the presence of food, a concentration of PbO in solution begins to cause a retarding film on the remaining white lead. This point is referred to later.

The effect of increasing the acidity of the solution keeping food and lead constant.

TABLE E.

1·000 grm. white lead
100 c.c. milk
150 c.c. weak acid
10 grms. bread
·25 ,, pepsin
Digestion two hours, as before.

Acidity = ·15% HCl.	Acidity = ·25% HCl.
PbO in solution = ·05 = 5% = 5·8% on PbO present.	PbO in solution = ·500 = 50% = 58% on PbO present.

Table E shows the rapid increase in solubility in an increasedly acid solution. This is *quite contrary* to the position with lead frit where the extra acidity has little effect.

The effect of increasing the foodstuffs, while keeping acid and lead constant:—

TABLE F.

1·000 grm. white lead.
Acidity = ·15% HCl in all cases.
Digestion two hours, as before.

	250 c.c. milk 20 grms. bread ·25 pepsin	100 c.c. milk 150 c.c. water 10 grms bread ·25 ,, pepsin	50 c.c. milk 200 c.c. water 5·0 grms. bread ·25 ,, pepsin
PbO in solution	= ·010 = 1·00%	= ·040 = 4·0%	= ·286 = 28·6%
PbO soluble on PbO present	= 1·16%	= 4·6%	= 33·3%

The retardation produced by the food is here evident.

These results might be stated in curves, but, as the points of observation are only three, the intermediate positions would not be accurately stated by this means.

It now remains to decide what is the particular constituent of the food which causes the retardation, and how it brings it about.

The food consists of carbohydrates, fats, and proteids, and to introduce the necessary variation on this the milk was in one instance replaced by *separated* milk, and in another the milk was entirely removed.

The results are stated in Table G.

TABLE G.

1·000 grm. white lead
10 grms. bread
·25 „ pepsin
Acidity ·15% HCl
Digestion two hours, as before.

100 cc. milk 150 cc. weak acid	100 cc. separated milk 150 cc. weak acid	No milk 250 cc. weak acid
Lead oxide in solution = ·070 grm. = 7·0%	·070 7·0%	·800 80%
PbO in solution on PbO present = 8·2%	8·2%	93%

In the separated milk there was an almost entire removal of fat, and in the absence of milk a large drop in the proteid content. The only conclusion which can be reached is that the fat and carbohydrates produce no retarding effect, and that this effect is entirely a function of the proteid.

As to the rationale of the retardation.

TABLE H.

100 c.c. milk 100 c.c. weak acid 50 c.c. $PbCl_2$ solution = ·2000 grms. PbO 10 grms. bread ·25 „ pepsin Digest 2 hours as before.	100 c.c. milk 100 c.c. week acid 10 grms. bread ·25 „ pepsin Digest 2 hours, add 50 c.c. $PbCl_2$, and again digest.
PbO in filtrate = ·037 = 18·5%	PbO in filtrate = ·080 = 40%

We now know that the precipitating proteids are mainly those originally present in the milk, and are not the products of digestion. The retardation is therefore either a result of films of protecting proteid compounds on the surface of the white lead, or a reprecipitation of the lead from solution. The former is almost certainly the case, since apparently unaltered white lead can be seen in the digestion at the end of the operation.

It has been established that alcohol is an accelerating agent in plumbism, and the foregoing experiments were repeated in the presence of alcohol, as follows:—

TABLE J.

1·000 grms. white lead 100 c.c. milk 150 c.c. acid ·15% HCl 10 grms. bread ·25 „ pepsin	1·000 c.c. white lead 100 c.c. milk 130 c.c. acid ·15% HCl 20 c.c. rectified spirit 10 grms. bread ·25 „ pepsin

Digestion two hours, as before.

PbO in solution =	·030 grms.	= ·020
	= 3·0%	= 2·0%
PbO soluable on PbO present =	3·5%	= 2·3%

Alcohol, though it exerts an accelerating effect, does so, not through enabling a larger amount of PbO to be dissolved in the stomach under otherwise constant conditions, but by reducing the resistance of the system to the lead present, or by preventing its elimination.

So far the experiments have been made upon that theoretically perfect food for man—bread and milk—but it can hardly be held that these constitute the regular food of the average lead worker; so to meet his case a further series were arranged in which the proteid should be in excess, and derived from the much more usual flesh sources.

The results obtained are rather important, as showing how the health of a worker may be influenced by his diet.

As a preliminary I took the course of substituting the milk by egg albumen in one case, and an excess of mutton in the other.

As a control I had the usual digestion of milk and water and bread going side by side with the new experiments, and the results are as follows:

TABLE K.

1·000 grms. white lead.

Digestion two hours, as before.

100 c.c. milk 150 c.c weak acid ·15% HCl 10 grms. bread ·25 „ pepsin	·15% 250 c.c. weak acid containing 30 grms. egg albumen 10 grms. bread ·25 „ pepsin	250 c.c. weak acid ·15% 50 grms. mutton 10 „ bread ·25 „ pepsin
PbO in solution = ·060	= ·700	·002
= 6·0%	= 70%	·20%
PbO in solution on PbO present = 7·2%	= 82%	·23%

The albumen has practically no effect, and is certainly much less efficient than are the proteids of milk, whilst the actual flesh food has produced an extraordinary reduction in the soluble PbO. This aspect of the case was followed further, using varying quantities of food, and of different qualities.

The results are as follows:—

TABLE L.

1 grm. white lead
250 c.c. ·15% HCl
10 grms. bread
·25 „ pepsin
Digestion two hours, as before.

Grammes of mutton	50	33	22	11
Lead in solution	=·002	·300	·500	·700
	=·20%	30%	50%	70%
On PbO present	=·23	35%	58%	82%

TABLE M.

1·000 grm. white lead
250 c.c. ·15% HCl
10 grms. bread
·25 „ pepsin
Digestion two hours, as before.

Grammes of beef	50	33	22	11
PbO in solution	=·002	·15	·40	·60
	=·20%	15%	40%	60%
On PbO present	=·23	17·5	46	70

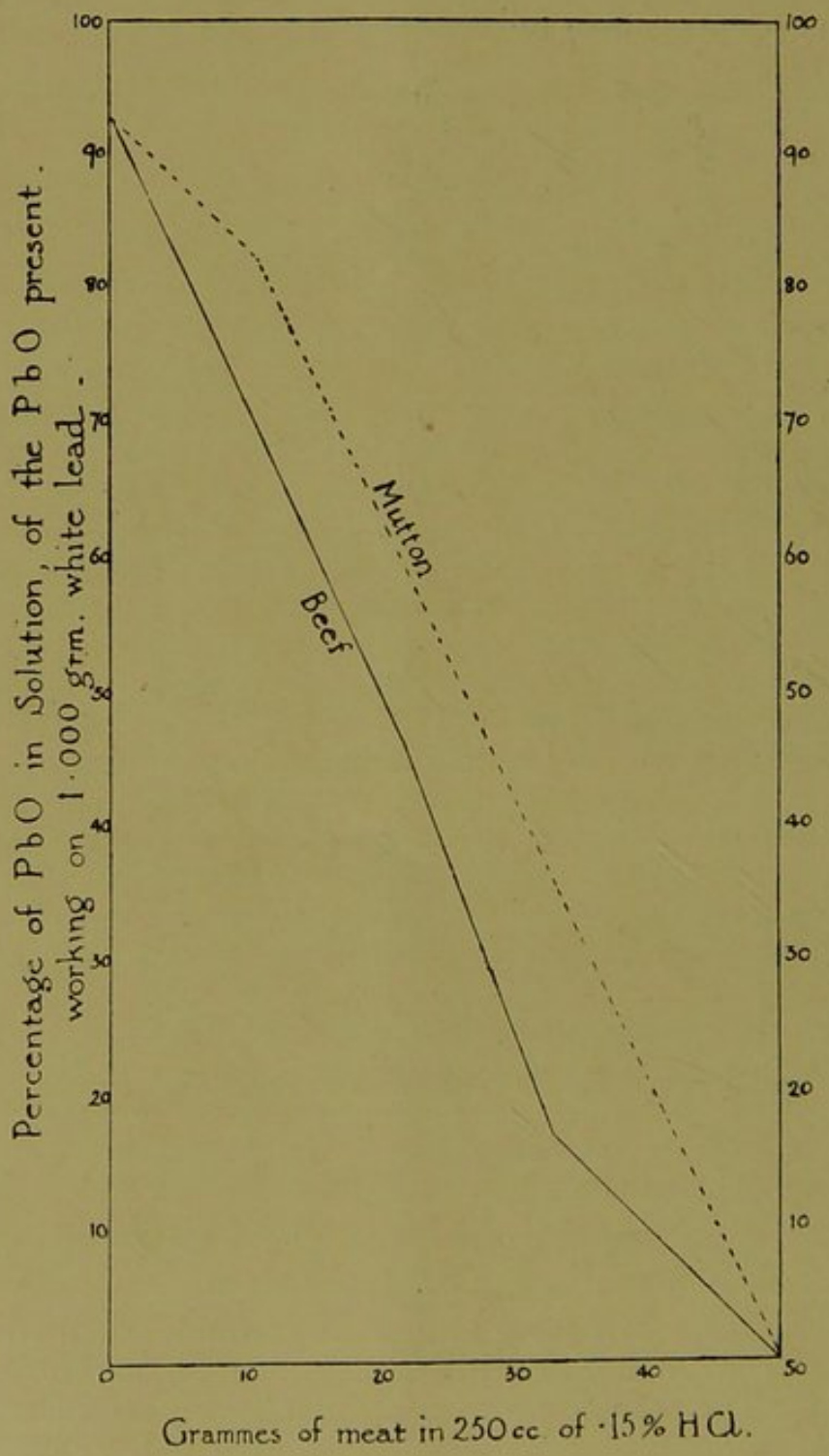
TABLE N.

1 grm. white lead
250 c.c. ·15% HCl
10 grms. bread
·25 „ pepsin
70 grms. plaice
Digestion two hours. as before.

	Cooked	Raw
Lead in solution	=·002	·0012
	=·20%	·12%
On PbO present	=·23	·14

In the three foregoing tables the result is much the same, but more complete than with milk; the rationale, however, is different. The soluble proteids are but slowly dissolved in the acid, and before this is complete the white lead has been almost wholly dissolved. A subsequent reaction, however, results in the very complete reprecipitation of the lead as a flocculent ppt.

Curves shewing the fall in Soluble PbO with the rise in Proteids in the Stomach.



In the foregoing experiments I have endeavoured to trace the probable fate of white lead in the human stomach under such a variety of conditions as must cover all circumstances under which workers in pottery processes can find themselves.

In my opinion the almost complete precipitation of lead in the stomach caused by proteid foods is sufficient to explain the non-effect of white lead eaten with food by the cats used in Mr. Goadby's experiments without having recourse to the much less probable theory of lung absorption being the main source of infection.

Also, I think it a fair conclusion that much of the so-called special susceptibility of females to lead poisoning is largely a matter of their diet, since their food practically always is less in proteid content than is that of males living in similar circumstances.

With regard to the lung theory of lead absorption, as mentioned before, Mr. Goadby considers his figures in *Lancet* and *Journal of Hygiene* are evidence of the paramount importance of this. His reasons are mainly the non-effectiveness of lead taken with food into the stomach, as mentioned above, compared with the effect of smaller amounts of white lead inhaled; but the figures showing precipitation of practically all lead salts by proteid foods in the presence of a large excess of acid makes it obvious that the possible poisoning effect of white lead eaten with this proteid food must be very trifling when we consider how small must be the secretion of gastric juice in the course of one meal in so small an animal as a cat compared with the 250 c.c. of acid used in the artificial extraction.

In the case of inhalation such lead as reaches the stomach will probably find this organ only partially filled with food, and the lead will, in consequence, the more readily dissolve.

Another point upon which too much stress can be laid is the amount of white lead eaten, quite apart from the precipitating effect of the proteid food.

In the experiments to which I refer the amount administered with food was as high as one gramme per meal. Let us suppose for a moment that the amount could have been raised to 100 grammes, and the apparent non-poisoning effect of white lead through the stomach would have appeared more pronounced than ever. The fact is, of course, that both one gramme and the imaginary 100 grammes are equally excessive, since the poisoning is governed by the amount of gastric juice secreted, not by the weight of white lead admitted, and that a dosage beyond a certain point can have no effect.

However, there is the possibility of lead reaching the lungs,

and if there its only chance of passing into solution depends upon the formation of a soluble lead bicarbonate under the action of the CO_2 of the lungs. Such being the case, I have attempted to ascertain the extent to which white lead and lead bisilicate may be taken up in the lungs.

For this purpose two lead compounds in varying amounts, suspended in water at 37° to 40°, were exposed to the action of an excess of CO_2 for varying periods.

It is found that as a fact both the lead salts are acted upon to a certain degree, but that this degree is very small compared to the gastric extraction under as nearly as possible parallel conditions.

TABLE O.

250 c.c. water, 37°C, 40°C in all cases, and stream of CO_2 over whole period.

1·000 grm. white lead. 2 hours in contact with CO_2.	·25 grm. earthenware glaze, 6 hours contact with CO_2	30 grammes white lead 1 week's contact
PbO in solution in 250 c.c. = ·0008	·00066	·00066
PbO in solution to PbO present = ·09%	1·5%	·002%

We find in all these cases that the PbO in solution is practically constant, hence it is evident that the reaction is soon stopped by the H_2CO_3 solution becoming saturated with lead bicarbonate, and this point appears to be reached when a concentration of ·0027 grm. PbO per litre is reached.

Similar results have been obtained with bisilicate. It must be remembered that no attempt has been made to reproduce the slightly alkaline and mucous condition of the lungs and air passages, so that probably in the animal itself the action of the CO_2 is much less rapid than in the above experiment; but even if we accept the above as showing the actual speed of solution we see that it is much behind that of the same amount of white lead in ·15 per cent. HCl, even in the presence of considerable amounts of foodstuffs, when the solubility in the same period is not 1·5 per cent. but 14·4 per cent.

To the best of my belief it is established that though respiration is the means by which lead poisoning is set up it occurs mainly through the stomach, since the main evidence of the lung theory rests on the presumed similarity in action of lead taken into the stomach with much food, compared with that in the absence of food or very little—a similarity which has been shown not to exist.

Reviewing the results obtained the position is briefly thus:—

(1) The percentage solubility of white lead in the stomach in the presence of food is the greater the less the amount of lead present.

(2) The solubility of lead is inversely as the proteid food present.

(3) The solubility of white lead is directly as the acidity of the solution—in this it is much more susceptible to change than is lead bisilicate.

Some of the points of this paper have been investigated by Bedson in 1891, but, though his conclusions are stated, details of his work are not given in any papers which are accessible, and in any case there has been no attempt at quantitative statement of the facts. I do not suppose my figures are absolutely as they would be found in the human body, were examination of this possible; but, subject to correction, I consider the conditions have been met to a degree sufficient to make the above a fair if not an absolute statement of the case.

It may be held that the diminution of solubility of white lead in the presence of proteid foods affords a means of checking plumbism without any of the disadvantages attending the use of frits, and on the face of it the matter is worth a more systematic trial than it has been given in the past.

It must, however, be remembered that the least satisfactory results have been obtained on the smaller quantities of lead, and that in industrial poisoning the amount of PbO actually present in the stomach and lungs in one day is less than the smallest I have examined. As against this is the fact that the artificial juice employed is certainly in excess of that produced in the body.

An average man will be found to inhale in the course of one hour about 18 cubic feet of air. This air, in a very badly constructed factory, may contain as a maximum about ·3 milligramme of Pb = 2·7 mgrm. per nine hour day = about 3 mgrm. PbO. Of course in most cases the actual quantities inhaled are much less than the above, but it is shown how small really are the amounts of lead which may cause poisoning, and in interpreting laboratory results these figures should be borne in mind.

In such experiments it is certainly a mistake to place the lead salt in the position of being in excess of the acid present in the stomach.

In conclusion, I wish to express my thanks to Messrs. Doulton & Co. for permission to carry out these investigations.

—Read March 12th, 1910.

DISCUSSION.

Mr. BERNARD MOORE:—I am sure the best thanks of the Society are due to Mr. Bishop for reading this paper. Having heard it for the first time, and not having had the opportunity of looking through it at all, it is one which I should not care to attempt to criticize this evening, except with regard to some of the remarks that are made in the early part of it. Mr. Thomason is evidently a very strong advocate of fritted lead, and it seems to me that he has twisted his facts somewhat to fit in with that theory. He commences by saying that there have been no cases of lead poisoning in the English factories employing lead of low solubility. I think that if he has been careful in making that statement he ought at the same time to have told us on what figures he bases such an assertion; as a matter of fact I think the actual number of lead workers in British potteries is something like 5,500. The number of lead workers upon which Mr. Thomason's figures are supposed to be based in under 200. Moreover, is it not a fact that there have been no cases of lead poisining in factories using lead of low solubility. There are, I believe, four deaths. These were not, however, all attributable to dipping. My recollection is that among the cases was one aerographer and one glazemixer. (The glazemixer should, I think, go to the credit of fritted glaze.) Then again the workers employed in those factories utilising fritted lead are not medically examined. Do you think that that dipper to whom Mr. Thomason refers, who had lead colic five years before his death, and had been off his work on account of illness from time to time—many times—suffering from hemorrhage in some form or another, would have been allowed to go on working if he had been employed in a factory using ordinary glaze. There is not the slightest doubt in the world that he would have been suspended long before. I presume the practice in vogue in other factories is in vogue at Lambeth, that is, that the workers in fritted lead are not examined. This is undoubtedly a great mistake, and I think it is one that will be remedied, because it is injurious and fatal to the trade that things should be backed up by arguments that are not sound. It is not sound to compare a factory where employees are not medically examined with a factory whose employees are examined. We know very well that there are many workers who would go on working except for the monthly examination which detects cases of lead poisoning often in their very early stages. I cannot think that the paper should

go on without some remarks being made with regard to these phases of the subject.

Of course with regard to the fact that food retards the toxic action of lead this is no new discovery, but has been taught by the best toxicologists for many years. Working on an empty stomach is one of the worst things a lead-worker can possibly do. Milk has been advocated as a specially good food in this connection, and I was interested to see the effect that mutton had in the experiments. I did not know anything about that before. I wish, however, that Mr. Thomason had given us the effect, say, of onions. I say this seriously, gentlemen. There is rather a remarkable fact that the Italians, who are quite large workers in white lead factories about London, suffer very much less than Englishmen, and this has been attributed by a medical man who has given a very great deal of study to the question, and who is in practice there, that their immunity from attack was due to their free use of onions. The Italians have also very much better teeth as a rule than the average British worker, and that probably has very much to do with it also. There was one other thing. I do not think that we ought to ignore some evidence that weighs very much with me, very much more than the figures that have been put before us to-night, with regard to the action of fritted lead. I dare say most of you know Dr. McAldowie, who gave evidence at the arbitration before Lord James some years ago. Dr. McAldowie went through an experiment that I could very well understand, whereas I cannot grasp all that has been given us this evening. Dr. McAldowie took a grain of lead every morning before breakfast on an empty stomach for a fortnight, producing a mild attack of lead poisoning, and then curing himself. He then waited a certain length of time and repeated his experiment, but with fritted lead, and the fritted lead was four or five days longer before it produced the effect, but when it *was* produced the effect was very much more severe and was accompanied by lead symptoms. That is a very important point. I know nothing about medicine, and should never have dreamt of speaking on a subject of this kind, but I was certainly struck with Dr. McAldowie's evidence, and he was seriously of the opinion that the accumulation of small doses of fritted lead was very severe in its consequences, and that fritted lead was more dangerous than raw lead by reason of the greater accumulation of small doses which, while they might not produce colic, acted more insidiously and produced final injury of a more serious character. I hope that in making these remarks I shall not be understood to be in any way antagonistic to fritted lead. I advocate extended experiment

with it as one possible means of reducing plumbism, but I think it is a great mistake to ride a thing of this kind to death, and to say that fritted lead will entirely stamp out plumbism, when we have so little evidence in this direction. We want to know what is the truth of the matter, and I do ask you in the meantime to keep an open mind on this subject. Accumulate evidence and try to find out what is the real truth, and do not try to take one side only to the total exclusion of the other. I do think that in a paper that is read before a scientific society that both sides ought to be put before us.

Mr. J. G. ROBERTS:—A paper of this kind is one of those papers which one ought to have in his hands a week before coming to a meeting to discuss it. From what I have gathered whilst the paper has been read I should certainly be inclined to question some of Mr. Thomason's experiments. For instance, he seems to come to the conclusion that no lead can pass the membrane of the intestines. He seems to base that opinion upon the fact that none passed through a filter paper which he was able to precipitate with sodium carbonate. The conditions of the human stomach after all cannot be imitated or reproduced by filter paper. The organs are very much more complex. I was interested to hear what Mr. Moore had to say about the effect of food. So far I have only had the misfortune to come across one case of lead poisoning, and that man was a very poor eater. A medical friend also assured me that bad eaters always suffer the most. It seems that these experiments are very suggestive, but at the same time they need just as careful repetition as getting up a recipe for a pottery body before you can place much reliance upon them. It is difficult enough to perform experiments in inorganic chemistry, and repetition is essential; but here there is greater need for repetition in order to assure oneself that the results obtained are concordant. Many of the results are perhaps pretty much what one would expect to find. We should certainly expect that milk would have a retarding effect upon the action of lead. Still one wants to read the paper thoroughly and digest it before passing much comment upon it.

Mr. D. W. F. BISHOP, M.A.:—I was going to mention the point that Mr. Moore mentioned, that it is interesting to trace that accumulated small doses such as you get with fritted lead are worse in their consequences than less frequent and larger doses such as obtained with raw lead. I think it is hardly permissable for Mr. Thomason to infer and argue from one single case of lead poisoning met with at Messrs. Doulton & Co.'s Lambeth Works. There is also a remark in the paper that

the toxic action of a glaze is entirely the effect of stomach action. That, I think, is a medical question, and it seems to me rather a broad statement to make. Is the presence of lead going to have no action on the lungs and chest except when it passes through them to the stomach? Is it going to have no effect on the nervous system? It seems to me that this is a question which needs investigation, and that there is no evidence in the paper to support the view. The paper further states that there is a rapid increase in the solubility of raw lead in an increasingly acid solution, whereas the contrary is experienced with a lead frit, where the extra acidity has but little effect. I could not see any evidence to prove that with regard to a lead frit, and I should like to know whether this is the case, whether if you increase the acid you would not increase the solubility with a lead frit in the same way as you increase the solubility with white lead.

Mr. A. HEATH:—It seems to me that Table G, where figures are given showing the effect of increasing the acidity of a fritted lead compound would have been very much more convincing if the milk had been taken out and replaced with weak acid If the result had then been the same as in the previous experiment the fact would have been very much more conclusive.

Mr. W. THOMASON (communicated):—Mr. Moore is right in supposing that I advocate the use of fritted lead, which I believe to be in all respects a substitute for white lead, except that its use introduces certain difficulties in manipulation, which, however, are not insuperable. This, however, is not the subject of the papers under discussion.

I did not state there had been no cases in factories employing fritted lead. Taking Mr. Moore's remarks in sequence, I refer him to the Annual Report of the Chief Inspector of Factories for 1908, page 11. I think he will find there corroboration of my statement as to the cases of lead poisoning to which he takes exception.

Mr. Moore contradicts me as to the cases, and says there were four deaths. He has, however, the grace to say that they were not *all* attritutable to dipping, so perhaps it may interest the Society to learn the details of the cases.

The report reads, "In nine potteries the 5 per cent. limit is in force under Rule 23a, adopted prior to 1904 in five of them, the other dates being 1905, 1906, 1907. There was *one* case of plumbism in 1908, the only cases reported since 1904; a man aged 46, employed 26 years dipping tiles," this is the man whose case I quote.

The next paragraph goes on, "Lastly there are twenty potteries working under the 5 per cent. limit as regards glaze, Schedule B not being affixed. In the last three years—1906-1908—three *cases* of lead poisoning have been reported from two potteries, *but none among the workers in glaze*; the persons attacked were respectively a colour-blower, a saggar-washer, and a manager of a decorating department who weighed out the colours!" I have ascertained that *none* of these three were fatal. So much for Mr. Moore's assertion that there have been four deaths. Further, Mr. Moore knows that *at least* two out of the three cases mentioned not only are not workers in low solubility glazes but are employed in either raw lead or fritted lead of such high basicity as to make them all easily soluble in gastric juice.

Of course the workers in raw lead exceed those in fritted lead because the potter is the most conservative man in the world, but statistics are not invalidated from that cause since the return is in percentages of the persons employed, and the number of workers in fritted lead (accepting Mr. Moore's figures as about 200) is quite sufficient, coupled with the years during which the frit has been used, to give a proper percentage return.

Mr. Moore works himself into a state of virtuous indignation in respect to these statistics, and states that the workers are *not* medically examined! He also charges me with suppressing half the facts! Has Mr. Moore forgotten that the *women* workers during this period have been medically examined—why did he omit to mention this fact? If the one case of lead poisoning which really might be construed as poisoning by fritted glaze had been medically examined *before* the adoption of these glazes, he would have been certified as suffering from plumbism occasioned by the use of raw lead glaze.

All this, however, is beside the point, and I should not have referred to it had not my critic made it imperative for me to do so. The paper was, as it emphatically states, an attempt to show the toxic qualities of a fritted lead glaze apart from any statistics, and in respect of this portion of the paper Mr. Moore is singularly quiescent. When he does refer to it he again takes a limited view. He refers to my results obtained with mutton, quite ignoring the essential fact that it is the proteid content not the *name* of the food which is of importance. It is quite true that the effect of food on plumbism has been inquired into before, but I incline to the belief that the relative values of food had not been established at all conclusively.

Mr. Moore refers to the supposed fact that Italians have better teeth than Englishmen, and ascribes a certain immunity from lead poison to this condition. This is a typical raw lead red herring. Perhaps he is right, but all this merely goes to prove that by taking a narrow view of the case, as Mr. Moore has done in this discussion, we shall not advance the question one inch. We must get rid of the personal factor and consider the statistics broadly, and in a sufficient number of cases to eliminate possible differences as to teeth and more important matters connected with the individual, or else we must approach it as I have endeavoured to do, by laboratory means.

Mr. Moore refers to Dr. McAldowie's evidence before Lord James' arbitration, and, being present as a spectator, I heard the evidence, which appeared to me most unconvincing. The witness gave himself lead poisoning by taking one grain of raw lead before breakfast each day for a fortnight and then he "cured" himself—that is, the allowed the *symptoms* to abate—and then repeated the dosage with fritted lead and got more serious symptoms; but whether he would have got these symptoms had he not previously poisoned himself with raw lead will never be known.

Cannot Mr. Moore see that by following Dr. McAldowie's evidence to its proper conclusion you should not attempt to protect the workers *either* by *exhaust fans*, etc., or by *fritting* (since both these methods have the one aim of preventing poisonous lead from obtaining access to the human system). No matter what method you employ you still have some lead left, and consequently you are in either case laying the worker open to "the greater accumulation of small doses, which, while they might not produce colic, acted more insiduously and produced final injury of a more serious character." Yet Mr. Moore is an active supporter of any system which seeks to prevent plumbism by sanitary means, but sees positive danger in fritting.

Finally, perhaps Mr. Moore will point out to me where I suggest fritted lead "will entirely stamp out plumbism." I think anyone who has read the paper under discussion—if it has not been read, why attempt to discuss it—will agree that a *certain* toxic effect is ascribed to fritted glazes and that both aspects of the case have been put before the Society.

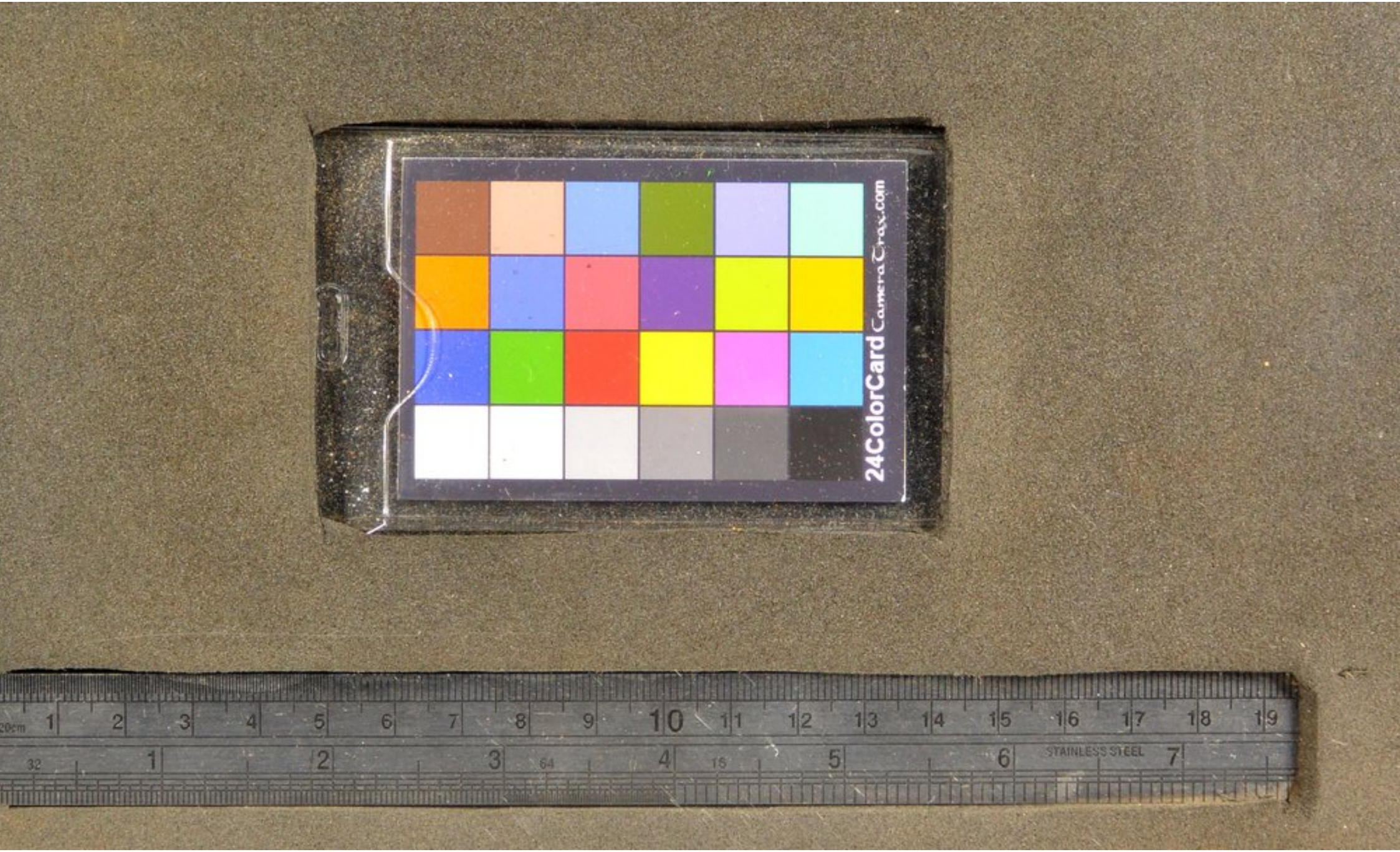
I think Mr. Roberts must have misunderstood the intention of the paper—probably through not having had an opportunity of going through the results at all closely. He will find that my conclusions that lead cannot pass the membrane of the intestines are *not* based upon any filter-paper experiments, as he assumes, but upon dialyser through vellum

and parchment paper. The filter-paper experiments were open to the objection he alludes to, and to get rid of this objection the dialyser series were carried out. By all means check the results I have obtained, and in doing so work nearer to tl actual body conditions if you can. For myself, I have endeavoured to protect my work against experimental error.

Mr. Bishop was good enough to read my paper, and he has had something of a thankless task. As I stated in reply to Mr. Moore I do not wish to draw any conclusions from the one case of plumbism which I have met in pottery. I prefer to stand by the laboratory results. I shall welcome and endeavour to reply to any criticism levelled at them if it is of a sufficiently specific character. The statement that the toxic effects of a glaze is entirely one of stomach action follows from the previously arrived at figures as to the behaviour of lead in stomach and intestines. The statement is not made to exclude the possibility of passing through the lungs, but differentiates between the stomach and intestine. The part played by the lungs I endeavoured to deal with in another part of these papers—that on white lead. Of course *all dust* has an action on lungs and chest. The increase in solubility of lead frit and white lead are dealt with in Table C, first paper, and Table E, second paper.

It is curious to find Mr. Bishop as well as Mr. Moore harping on the supposed evil results of working in fritted lead. I should very much like to know by what means they are able to "trace" these results, since the Government returns do not bear out the statement even in women workers who *are* examined. Also I should like to know why the infections from raw lead are *less* frequent than from fritted.

I regret I am unable to follow Mr. Heath's remarks as to Table G.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