A FOOD POISONING OUTBREAK AT BRIGHTON.

By WILLIAM G. SAVAGE, M.D., B.Sc. (Lond.),
County Medical Officer of Health, Somerset,

and DUNCAN FORBES, M.D.,
Medical Officer of Health, Brighton.

An outbreak of food poisoning which occurred at Brighton in November, 1917, showed several features of unusual interest which makes its publication of value. While the epidemiological inquiries and pathological investigations were carried out separately a coherent narrative is best obtained by combined presentation. The pathological investigations were carried out for, and at the request of, the Local Government Board.

The outbreak occurred in the Royal Sussex County Hospital which at the time contained 369 residents, 227 being patients and 142 staff. Twenty-eight persons suffered from food poisoning, 24 of whom were patients and 4 members of the staff. Two deaths occurred but both were patients already suffering from severe diseases.

PARTICULARS AND CLINICAL FEATURES OF THE OUTBREAK.

The persons affected were all attacked on November 28th and 29th, the interval between the onsets of the first and last cases being 48 hours. The onset was sudden, the principal features being abdominal pain, vomiting and diarrhoea; headache was frequent but usually not severe. The temperature was raised in practically all cases, at times to over 104° F. Rigor was reported in a few cases.

Incubation Period. If, as supposed, the patients were infected at dinner on the 27th November then the incubation periods (in hours) are as follows: 7, 18, 19, 21, 23, 23, 25, 25, 25, 26, 27, 31, 31, 31, 32, 33, 41, 45, 46, 47, 55, 55.

In this connection the effects of aperients are of interest. Of the 18 persons, who partook of fried fish in Ward D, 10 out of 13 who had no aperient on the evening of the 27th were attacked whilst only 2 of the 5 who had aperients suffered. Unfortunately evidence from Ward V is not available but that from Ward D points to the advisability of the administration of an aperient if food poisoning is suspected.

VEHICLE OF INFECTION.

The abruptness of the outbreak and the other features clearly pointed to food poisoning as the cause, although other possibilities were investigated. It will be unnecessary to detail all the different inquiries made in these directions or as regards articles of diet. The only article of food common to all, or almost all, the sufferers was fried fish while the time of onset pointed to the incriminated food having been taken on the 27th. On that day the patients' midday meal was principally of fish; 122 patients in five wards had fried fish, 70 had steamed fish and 35 had other diets. The 24 patients attacked belonged to two wards (V and D) and 23 of these patients are known to have had fried fish. In Ward D 18 patients had fried fish and 13 of these were infected; in Ward V, 21 had fried fish and 10 of these were infected although less severely than the patients in Ward D. The remaining patients did not have fried fish.

Of the patients in the affected wards 59 per cent. of those who ate fried fish were attacked, while of 14 patients who had not partaken of fried fish but were on other diets only one was attacked.

Although there seems to be no doubt that fried fish was the chief vehicle of infection it may also have been carried by other foods as one nurse (a severe case) and one patient said they had not partaken of fried fish. It has to be remembered, however, that the inquiry as to cause was made on the 29th November and 1st December, and when any large number of persons are affected mistakes in recalling diets are sure to be made, also there is the chance of infection of one food stuff by another, the fish and meat for the day's consumption, both before and after cooking, being in close contact.

BACTERIOLOGICAL EVIDENCE AS TO THE CAUSE OF THE OUTBREAK.

The material examined consisted of blood and internal organs of one fatal case, blood-serum from a number of the cases, excreta specimens from a few cases. Unfortunately none of the fish was available for bacteriological examination.

Fatal case (F. F.). Age 30, a soldier. Died December 8th. *Post-mortem* performed by Dr Galt, Pathologist to the Hospital, 20 hours after death and to whom we are indebted for the following particulars.

The small intestine showed only slight evidences of irritation and contained no blood. From the ileo-caecal valve to the rectum the colon showed acute ulcerative colitis. The mucous membrane at parts was

destroyed, but there was no evidence of extension of the ulceration into the muscular wall. Much bloody mucus was present throughout the colon. There were no other pathological lesions of moment, but the above is of importance in view of our findings since Dr Galt's view is definitely that the cause of death was ulcerative colitis of long standing, and that his death was not directly traceable to the outbreak.

This patient took oxalic acid 5 years previously and had suffered from colitis ever since, and was admitted in June 1917 with intractable diarrhoea with some blood. Later much mucus with a little blood in the stools.

The material received by one of us for examination consisted of a piece of spleen, some heart blood and pieces of the ileum and colon. No organisms of the Gaertner or Proteus groups were isolated from the ileum or colon. From the spleen in pure culture and from the heart blood, mixed with a few *B. coli* and streptococci (both no doubt a *post-mortem* invasion), a bacillus was isolated which for convenience we will call "Brighton." The characters of this Brighton organism were as follows:

A short bacillus with rounded ends, non-sporing, Gram negative but staining well by ordinary dyes. When first isolated showed only moderate motility but active movement after one or two days' artificial cultivation. White, circular, rapidly growing colonies on neutral-red-lactose-bile-salt agar. Uniform turbidity in broth. Bluish translucent growth upon gelatine slope without liquefaction. No indol produced in peptone water media. In litmus milk first some acid production then marked alkalinity. It fails to produce either acid or gas in lactose, salicin, saccharose or raffinose media. In glucose it produced when isolated acid but only a bubble of gas, while in dulcite, mannite and glycerine it produced acid but no gas. Its reactions with other sugars and alcohols were not tested for about two months. It then fermented sorbite and maltose with acid and gas production, produced acid and slight gas in galactose and xylose while it failed to produce either acid or gas in inulin, adonite or amygdalin. Its pathogenicity to rodents was well marked. 2 cc. of a 24 hours broth culture injected intraperitoneally into a guinea-pig (weight 230 grams) killed it in less than 18 hours. The inoculated bacillus was recovered in pure culture from the spleen, liver and heart blood.

The organism as isolated differed from members of the true Gaertner group, such as *B. enteritidis* or *B. paratyphosus* β in its failure to produce gas in glucose (only one bubble of gas after repeated tests), mannite and dulcite.

Excreta from four cases. Unfortunately the samples had to be sent for examination just before Christmas and there was great delay in transmission, only being received four days after collection. In view of this delay and the fact that they were samples collected over three weeks after the attack no conclusions can be drawn from our failure to isolate the "Brighton" organism or members of the typhoid, Gaertner and dysentery groups. The patients having left the hospital it was not found possible to obtain fresh specimens.

Sera from cases. The fact that the "Brighton" organism was isolated from the internal organs of the case by no means proves its association with the disease, especially since this patient was suffering from long old standing colitis with dysenteric symptoms. An extended series of serological tests were carried out to try and clear up this question.

Table I gives the agglutination reactions with the serum of a number of persons who were attacked with food poisoning symptoms, and one (H.) who handled the fish.

TABLE I.

Sera from sufferers from the food poisoning outbreak and from Miss H.

Bacillus tested	F.F.	Col.	W.	B.	C.	P.	O.	H.
B. brighton	100	1000	300	200	200	200	–	100
B. enteritidis	100	1000	300	200	200	300	–	100
B. paratyphosus β	100	100	30	50	50	50	–	30
B. suipestifer	100	100	30	200 (*p*)	200 (*p*)	200 (*p*)	–	–
B. typhosus		300		500	200	300	–	
B. dysenteriae (Flexner)		300		200 (*p*)	100 (*p*)	30	–	
,, (Shiga)		–		–		–		

The figures give the limits of positive agglutination reactions.
(*p*) indicates the reaction was partial with the highest dilution.
– = no reaction; usually in 1 : 30 dilution.

The agglutination results are very interesting but in themselves not conclusive. It will be noted that all the sera except one agglutinate "Brighton" bacillus in at least a 1 per cent. dilution, but on the other hand other bacilli, especially *B. enteritidis* and *B. typhosus*, are equally agglutinated. The question at once arises whether any of these positive results can be ascribed to the effects of inoculation against typhoid, paratyphoid or dysentery fevers singly or combined. Specific inquiry showed that F. F., C. and O. were soldiers and had been inoculated, but P. and B. were civilians, while Col. and W. were nurses in the Hospital, and H. a kitchen maid. None of these five had been inoculated.

TABLE II.

Absorption tests, i.e. agglutination tests with the respective sera after absorption by the bacilli indicated.

	Bacillus	P. 50	P. 100	P. 200	B. 50	B. 100	B. 200	Col. 50	Col. 100	Col. 200
After absorption by Brighton bacillus	Brighton	–	–		–	–		–	–	
	Enteritidis	–	–		–	–		–	–	
	Paratyphoid β	–	–		–	–		–	–	
	Typhoid	–	–		–	–		–	–	
	Flexner	–	–		+ *p*	+ *p*	+ *p*	+	+	+
After absorption by *B. enteritidis*	Brighton	+ *p*	–	–	–	–		–	–	
	Enteritidis	–	–		–	–		–	–	
	Paratyphosus	–	–		–	–		–	–	
	Typhoid	–	–		–	–		–	–	
	Flexner	+ *p*	–	–	+	+	+	–	–	
After absorption by *B. typhosus*	Brighton	+	+ *p*	–	+	+		+	+	–
	Enteritidis	+	+	+ *p*	+	+		+	+	
	Paratyphosus	–	–		–	–		–	–	
	Typhoid	–	–		–	–		–	–	
	Flexner	+ *p*	–		+	+	–	+	+	+
After absorption by *B. paratyphosus* β	Brighton	+	+	–	+			+	+	–
	Enteritidis	+	+	+ *p*	+	+	+ *p*	+	+	–
	Paratyphosus	–	–	–	–	–		–	–	
	Typhoid	+	+	–	+	+	+	+	+	–
	Flexner	+	–	–	+ *p*	+ *p*		+	+	+

Dilutions of 1 : 50, 1 : 100 and 1 : 200 employed.

The agglutination tests are clear evidence of infection and the facts suggest that this was due to infection with "Brighton," the agglutination results with other bacilli being due to the well-known associated agglutinin reactions (co-agglutinins). To settle this point the series of absorption tests set out in Table II were undertaken. The sera selected were cases uncomplicated by any inoculations. Apart from the Flexner results, which are anomalous, the absorption tests show that absorption with either "Brighton" or *B. enteritidis* removes the whole of the agglutinins, while on the other hand absorption with *B. typhosus* or *B. paratyphosus* β, while removing all agglutinins for these organisms largely fails to remove the agglutinins for either the Brighton bacillus or *B. enteritidis*. These results show definitely that the positive agglutination results with these sera are due to infection either with Brighton bacillus or with *B. enteritidis*, and they suggest that these two bacilli are almost identical in their serological behaviour. This important

https://doi.org/10.1017/S0022172400007282 Published online by Cambridge University Press

point was cleared up by immunizing a rabbit with the Brighton bacillus. The serum, withdrawn January 14th, gave the following results:

TABLE III. *Strains tested.*

Dilution	Brighton	H.	*B. enteritidis*	*B. paratyphosus β*	*B. suipestifer*	*B. typhosus*	*B. dysenteriae* (Flexner)
1 : 200	+	+	+	−	−	+	+
1 : 500	+	+	+	−	−	+	−
1 : 1000	+	+	+			+	−
1 : 2000	+	+	+			−	−
1 : 5000	+	+	+			−	
1 : 10,000	−*a*	−*a*	−			−	
1 : 15,000	−	−	−			−	

These results show that the Brighton bacillus and *B. enteritidis* are serologically identical, while the positive reactions with *B. typhosus* explain the positive reactions met with in the sera of patients.

THE SOURCE OF INFECTION OF THE INCRIMINATED FOOD.

From the above considerations it is evident that the outbreak was due to infection by a Gaertner group bacillus, allied to but not identical with *B. enteritidis*, and that the vehicle by which the bacillus was conveyed to the sufferers was by means of fish eaten after frying. The next point to determine was the means whereby the fish became infected.

All the evidence is to the effect that the fish as delivered was fresh and in good condition and no objections to it were raised by anyone. This fact, together with the length of the incubation periods, the isolation of a Gaertner group bacillus and the presence of specific agglutinins in the blood, quite negatives any possibility of the outbreak being due to decomposing food.

A second possibility, that the fish was infected with this bacillus on delivery in Brighton from Milford Haven on November 24th, is rendered extremely unlikely by the fact that up to the evening of the 26th it was stored with the fish for sale in the shop, indeed half of the same consignment was sold to private customers and there was no report of consequent illness.

The evidence therefore points to the fact that the place of infection must be looked for either at the fishmonger's or subsequently at the Hospital. The treatment of this batch of fish was as follows: On the evening of the 26th the fish for the Hospital (ling and cod) was sliced by the manager and stored by itself in a sink in the basement of the shop; the ice in which it was stored was broken on the floor in the basement close to all kinds of empties and under the prism-light grating of

the shop entrance through which water percolated at times. In the morning the sliced fish was washed before being sent to the Hospital. At the Hospital the chance of contamination did not appear great, as the fish after delivery in the morning between 9 and 10 a.m. was cooked from 10.30 onwards. Immediately before being cooked the larger pieces of fish were further sliced with a rather blunt knife and each slice was dipped in flour before it was placed in the heated fat in which it was fried. The fat remained unchanged throughout the cooking. The handling of the fish (slicing and flouring) and the cooking were mostly done by a kitchen maid (H.).

It is of considerable interest to note that this particular fish consignment was eaten partly as fried fish, partly as steamed fish. Seventy persons ate this fish steamed and none were affected. Doubtless the temperature of steaming would be sufficient to kill any Gaertner bacilli if present and this would account for the immunity.

On the other hand, if the fish had been infected with this bacillus while at the fishmonger's we should have expected (even with the comparatively cold weather) considerable multiplication and some production of toxins. Gaertner group toxins are notoriously heat resisting and would not be destroyed by the steaming. Such toxins would have produced at least some cases of illness and that none occurred is evidence, though by no means conclusive evidence, that infection was of later date, i.e. probably on the Hospital premises.

BACTERIOLOGICAL EXAMINATION OF THE FOOD HANDLERS.

The sera of all, or at least the most important, of those persons who had handled the fish over the suspected period were bacteriologically investigated. The blood of the man who almost solely handled the fish outside the Hospital and of the cook at the Hospital both gave quite negative results as regards agglutination tests against a number of bacterial strains, but that of the third person, a Miss H., who was kitchen maid at the Hospital gave definite positive results (see Table I). We were fortunately able to obtain a specimen of her excreta on Jan. 8th and from this seven organisms were isolated which were identical with one another and with the Brighton bacillus.

This strain which we call "H" agrees in every particular with the Brighton bacillus and shares its peculiarities, except that one or two of the subcultured colonies gave a bubble of gas as well as acid in mannite media. The agglutination reactions obtained with this bacillus with the

serum of the immunized rabbit (see Table III) are identical with those of the Brighton bacillus and complete the proof as to their identity.

In view of its intrinsic importance it may be pointed out that the rather peculiar deviations of these two strains from the typical Gaertner type make it absolutely certain that these two organisms are identical.

The presence of the Brighton bacillus in the excreta of the kitchen maid H., combined with the positive agglutination reaction, at once raises the question as to whether this girl was the cause of the infection of the fish or was herself a victim of the food poisoning and so became infected with this bacillus. The importance of this point was obvious to us and we made the most careful inquiries to clear it up. We ascertained the following:

(*a*) Miss H. is positive she did not eat any of the fish and indeed disliked fish. On that day all the fish cooked was sent to the wards and all of it was eaten, none being returned to the kitchen.

(*b*) She did not suffer from symptoms of food poisoning or illness of any kind during the outbreak. This fact was ascertained by Dr Galt before she was found to harbour the bacilli and was reinvestigated by one of us again after the bacilli were detected.

(*c*) She had abundant facilities for contaminating the fish if she was a carrier since she actually sliced and floured the fish which proved to be infective.

We are therefore in a position to affirm that the available evidence strongly suggests infection of the food at the Hospital and that the source of infection of the fish was the kitchen maid (Miss H.) who was the one person who especially handled the fish and in whose excreta was found a bacillus identical in all particulars with the bacillus causing the outbreak.

THE SOURCE OF INFECTION OF THE FOOD CARRIER.

We next endeavoured to carry our inquiries a step further and ascertain when and how Miss H. became infected and a carrier. In this inquiry we met with but little success. This girl was only 17 at the time of the outbreak, and since she left home, a period of two years, had been in service at the Hospital. She had not been off duty except for the usual holidays and neither she nor her mother could remember any illness from which she had suffered.

If, as we conclude, she had been a carrier for some time it is interesting, but not remarkable, that she should not have caused any other outbreaks in view of her favourable opportunities to infect food. This

is probably explainable from the well-known intermittency of infection of carriers, while a large part of the food she handled was cooked before use. Further of course a carrier is only likely to infect when the bacilli have gained access to her hands from accidental or uncleanly self-infection and this may have been a matter of great rarity in this case.

REMARKS AND CONSIDERATIONS OF A NUMBER OF POINTS OF SCIENTIFIC INTEREST.

(1) The bacillus isolated while clearly of the Gaertner group when isolated did not agree in its cultural characters with any known food poisoning strain. The differences were, however, of degree only, did not involve any added characters and were merely in the direction of diminished fermentation power. Thus the three substances, glucose, mannite and dulcite, were split up with only acid production but not with the formation of gas also. There was however evidence of very slight gas production and in view of this and for the above reasons we considered that the cultural differences were probably due to transient retardation of fermentation powers. Some mutation of characters is a well-recognized property of this group.

Our surmise was confirmed as after artificial cultivation in litmus milk and other media for several weeks and then replating and retesting moderate gas production with glucose and dulcite and well-marked gas production with mannite were obtained in both the Brighton and H. strains.

At the same time it is of great scientific interest to note this abnormality of characters when isolated, especially as it was equally well marked in the carrier strain and in the bacillus recovered after passage through a guinea-pig.

(2) The fatal case investigated (F. F.) was ill at the time of the outbreak with colitis and therefore the bacillus isolated could not be accepted at once as having anything to do with the outbreak. This patient not only ate the suspected fish but had a rise of temperature and other symptoms, coincident with the other cases. His blood gave a well-marked reaction to the Brighton bacillus and also to *B. enteritidis* (see Table I). The other facts prove that this bacillus was not one peculiar to and associated with his condition of colitis but was the cause of the outbreak.

One other patient who ate the fish and had symptoms died and *post mortem* his death was found to be due to malignant ulcerative colitis. This case was not bacteriologically investigated.

The fact that the only fatal cases suffered from inflammatory conditions is of especial interest. The ulcerative bowel condition would predispose at least to infection and probably to a fatal infection. Meyer[1] in America has quite recently reported a case which bears upon this point. In this case a young man became infected with *B. enteritidis*, recently isolated from a calf dysentery case, used for feeding calves in an experimental investigation upon calf dysentery. A number of other laboratory workers were equally exposed to infection, the material being handled in a very careless manner, but he only was attacked, evidently because at the time he was suffering from mucous colitis.

(3) The considerable degree to which the blood of the affected persons also agglutinated *B. typhosus* and other strains, and the fact that some of the patients were inoculated soldiers, added a complication which necessitated the use of absorption tests to clear up the matter. The investigation very clearly shows the value of these absorption tests.

(4) Food poisoning outbreaks definitely traced to human carriers are extremely rare. In several Continental outbreaks this source has been suggested but for none of those which we have been able to read is the proof established or even more than a reasonable possibility.

In the 79 outbreaks in Great Britain reported by one of us[2] to the Local Government Board, in only one outbreak (Wrexham 1910) is a human carrier advanced as the cause and source of infection. In this case the evidence is inconclusive owing to an organism described as *B. paratyphosus β*.

In the Brighton outbreak the bacillus isolated is clearly a slightly abnormal strain of *B. enteritidis*, an organism which only sets up food poisoning outbreaks and never long continued infections such as paratyphoid fever or enteric fever. It is not a natural inhabitant of the human or animal intestine and its presence there can only be ascribed to an infection with food containing these organisms. That a food poisoning outbreak should be traced to a human carrier of this organism is therefore both a unique and a specially interesting fact. We know of no other definite case in the literature of food poisoning.

Its importance made us especially careful to satisfy ourselves that Miss H. was a carrier and not infected during the outbreak. Since the bacilli were only looked for and found after the outbreak we cannot express absolute certainty as to the matter, but the evidence we have

[1] *Journal of Infectious Diseases*, 1916, Volume XIX, p. 700.

[2] Savage, 1913, Report to the Local Government Board on Bacterial Food Poisoning and Food Infections.

adduced, we think, very strongly favours the view that she was an actual carrier.

The blood of this girl H. still gave a well-marked positive agglutination reaction with "Brighton" bacillus in 1 to 50 dilution and an indefinite reaction in 1 to 100 dilution on both Feb. 13th and March 22nd, 1918. We failed however to find this bacillus in her excreta in specimens collected on both these dates.

(5) We have no difficulty in explaining why only a portion of the consumers of the fried fish were attacked as doubtless the cooking was sufficient to sterilize the fish in most cases. The most difficult part of the outbreak to explain is the survival of any bacilli after the cooking to which they were exposed. The length of the incubation period and other facts make it evident that the infecting bacilli did survive and it is not an outbreak due to heat resisting Gaertner group toxins.

We have been able to elicit a few further facts which bear upon this point. It has already been mentioned that the consumers of the steamed fish and those of fried fish other than to Wards V and D escaped. It is evident therefore that there must have been some factor or factors which prevented efficient sterilization of these particular batches of fish. We have ascertained that on the 27th the frying fat did not cover the fish slices as usual and in addition the cooking was hurried because of late delivery. The fish for Ward D, in which the most severe cases occurred and the greatest percentage were infected, was cooked first and was kept warm on a hot-plate for over an hour, thus encouraging multiplication of any bacilli not destroyed in the cooking, whilst the fish served to Ward V was cooked last and in a hurry, being much underdone, the patients having difficulty in separating the flesh from the bones.

While the temperature of cooking of fried fish is high it is very superficial and these factors must have been sufficient to prevent the killing of all the bacilli.

We are indebted to Dr Galt, Pathologist, and to the resident medical staff of the Hospital, for the pathological and other particulars of the cases and inquiries made amongst the Staff, to Dr Harriette Chick of the Lister Institute for kindly testing our strain upon a number of sugar-alcohols which we had not at our disposal, and to Dr MacFadden of the Local Government Board for permission to utilise the pathological data contained in our report to the Board.