THE ABERDEEN OUTBREAK OF MILK-BORNE GAERTNER ENTERITIS, JULY, 1925.

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(With 2 Charts.)

An epidemic of enteritis due to infection of milk with a Flexner type dysentery bacillus, causing over 1000 cases and 72 deaths, occurred in Aberdeen in 1919, and has previously been recorded in the *Journal of Hygiene* (1). Another milk-borne outbreak of enteritis, causing 110 cases and no deaths, occurred in 1923, and has similarly been recorded (2). The cause of this outbreak was not determined, but was assumed to be due to a living bacillus of unrecognised type. In July, 1925, there occurred a further extensive outbreak of enteritis, which has been proved to be due to infection of milk with *Bacillus enteritidis* Gaertner, and as the infection has been proved to be of bovine origin, the Gaertner bacillus having been isolated from the udder and flesh of the diseased cow, as well as from the infective milk, it appears advisable to record the outbreak in considerable detail.

Epidemiology.

On July 7th, 1925, it became evident that a widespread outbreak of acute enteritis had developed suddenly in Aberdeen, the first of the cases developing in the early hours of the morning. An increasing number of cases continued to be reported during July 8th, and thereafter the outbreak terminated.

In all, 497 persons were ascertained to have suffered from the enteritis, 158 cases having occurred on July 7th and 339 on July 8th. Of the 497 persons who contracted the enteritis, 262 were males and 235 females—that is, 53 per cent. males to 47 per cent. females. As to age incidence, 2 per cent. of the patients were under 2 years of age; 6 per cent. 2–5 years; 15 per cent. 5–15 years; 23 per cent. 15–25 years; 28 per cent. 25–45 years; 21 per cent. 45–65 years; and 5 per cent. 65 years and over.

There was no evidence that any of the 497 cases were due to contact infection, the infection in each household having obviously a common source, and the absence of contact infection was further indicated by the fact that not a single case of enteritis originating after July 8th was discovered.

Incubation Period. Detailed investigation revealed the fact that the incubation period of the enteritis varied from 2 to 48 hours, the average incubation period being 19 hours.

Symptoms. The symptoms in all the cases were remarkably uniform, varying only in degree of severity, and presenting the classical features of a Gaertner enteritis. The symptoms were ushered in with headache, nausea, shivering and epigastric pain associated with rapid rise of temperature to a maximum of 103 or 104° F., with pulse and respiration rates in the region of 130 and 30 respectively. The temperature Charts I and II are typical.

The increasing nausea rapidly culminated in the almost simultaneous onset of vomiting and diarrhoea, associated with much abdominal pain and cramp in the back of the legs and in the lumbar region. The vomiting and diarrhoea rapidly became of an urgent description, the patients "going at both ends" either time about or both together, and with but brief intermissions. The more

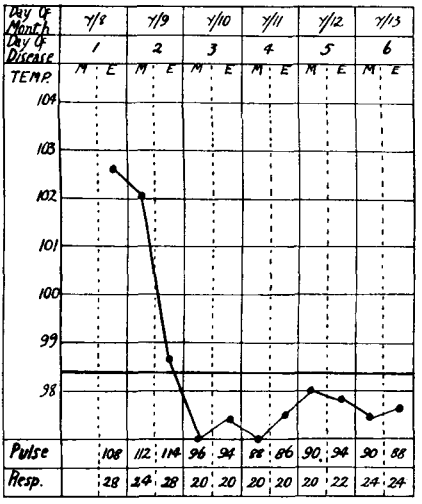

Chart I. Milk poisoning due to *Bacillus enteritidis* of Gaertner, 1925.
Case—M. M. (female).

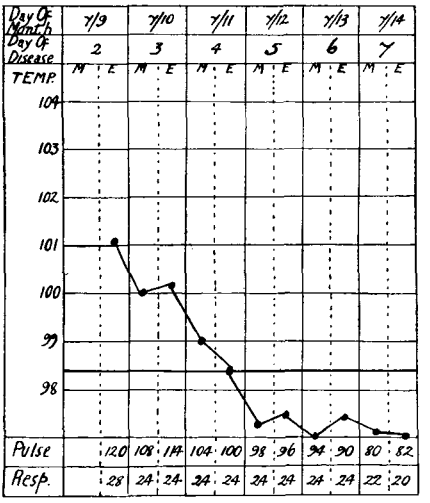

Chart II. Milk poisoning due to *Bacillus enteritidis* of Gaertner, 1925.
Case—J. L. (female).

acute cases passed two or three loose green stools containing much mucus, but no blood, every hour during the first day of illness. In this condition the patients lay in bed, with anxious expression, thickly furred tongue, offensive breath and congested fauces. The headache and nausea were continuous. In the intervals of vomiting and diarrhoea the slightly tumid abdomen was tender to palpation over the two upper quadrants.

The duration of illness was remarkably uniform in all the cases, the acute symptoms having an average duration of forty-eight hours. During the first day the symptoms were of the urgent nature indicated, and during the second day the high temperature and pulse rate declined, coincident with the gradual cessation of vomiting and a diminution of the diarrhoea. By the third day the acute symptoms had subsided, temperature and pulse were normal, vomiting and diarrhoea had ceased, and the only symptoms complained of were those of weakness and epigastric pain. At this stage the patients appeared

worn out, they had no desire for food which appeared to increase abdominal discomfort, and the desire for rest without interruption was obvious. Complete recovery took place rapidly, the patients being ready for discharge from hospital within five days of the onset of illness. No cutaneous rashes, meningeal symptoms, ocular or other paresis appeared in any of the cases. In the mildest cases nausea, abdominal discomfort and slight diarrhoea were the only symptoms.

Treatment. In the absence of a specific therapy the treatment was symptomatic, and consisted mainly of nursing devices. The elimination of the Gaertner bacillus and its toxins was accelerated by the administration of sodium sulphate, 2 drachms two hourly, and water was given in great abundance to facilitate vomiting and relieve dehydration.

Mortality. Only one death occurred, in an invalid aged 85 years. All the other patients were completely recovered within a week.

Milk Infection. On first inquiry it was not clear that milk was the vehicle of infection, since it was early ascertained that the milk supply to the infected families was being obtained from four apparently separate sources. Further inquiry revealed the fact that one of the four dairymen supplying the milk retailed 35 gallons of milk direct from his farm in the vicinity of the city, the milk being that of a herd of 21 cows, and that this farmer was supplying the other three retail dairymen whose milk supplies were also implicated with 7 gallons, 8 gallons and 16 gallons of milk respectively. These three retail dairymen were receiving milk from other and separate sources, but it was ascertained that only the milk from the farm in question caused the poisoning. Fortunately, these dairymen retailed the different milk consignments separately and without bulking the milk. No other food was common to the infected households. Lists of all customers supplied by the farmer and by the three retail dairymen were obtained and all were visited, and it was ascertained that a total of 1016 persons were consuming the milk, although only 497 of these persons contracted the enteritis. Detailed investigation revealed the fact that the milk that caused the enteritis was the mid-day and evening milk of Monday, July 6th.

As regards the source of infection at the farm, it was ascertained that a cow suffering from induration of the udder had been removed from the dairy herd and isolated in a field a week prior to the onset of the enteritis in the city, and that the milk was stripped daily from this cow by the farmer, who also bulked the milk of the herd, although he did not take part in the actual milking. Unknown to the Health Department, this ailing cow which had developed symptoms of septicaemia, was slaughtered on the day following the outbreak of the enteritis, but later in the day the carcase was seized by the City Veterinary Inspector and condemned as unfit for human food. This action revealed the source of the carcase, and specimens of the udder and flesh of the cow were accordingly made available for bacteriological examination.

It having been ascertained that the milk from this farm was the source of

the infection, the remaining twenty cows of the herd were examined by the City Veterinary Inspector on the day the ailing cow was slaughtered, and all the cows appeared to be in good condition. In the absence of any symptoms of illness in the cows, the Veterinary Inspector proceeded to obtain specimens of milk, blood and faeces from all the cows for bacteriological examination.

On the evening of the day of the outbreak of the enteritis in the city, the source of infection had been traced to the farm, and it was found on inquiry that the farmer and his family, nine individuals in all, were wholly responsible for the production of the milk. All the nine individuals were found to be in good health, and no history of recent enteritis could be elicited. In this connection, however, it is important to indicate that it is our experience in investigating this and similar outbreaks of milk-borne disease that the sense of personal responsibility on the part of the farmer and his financial concern are such that he immediately adopts a policy of reticence which defeats the object of the inquiry. In this connection an educational campaign among dairymen is an urgent requirement, with a view to making them appreciate the fact that *Salmonella* milk infections are liable to occur even in a well-conducted dairy, and that within certain limits the dairyman cannot be censured. In the absence of any evidence of infection in the nine individuals concerned in the production of the infected milk specimens of blood and faeces from each of the individuals were obtained for laboratory examination.

The widespread nature of the enteritis having made the milk supply suspect from the first, it was possible to obtain three separate samples of the infective milk for bacteriological examination from the houses of patients suffering from the enteritis. One such specimen was obtained from a household in which the milk had been partaken of for supper on July 6th, after which the family retired to bed, and the onset of the enteritis had prevented the supper-table being cleared in the morning. In two other households of railwaymen on the night shift, the milk was taken with porridge at 8 a.m. and 11 a.m. respectively on July 7th, and was still on the table and so available for bacteriological examination some 11 hours later, when the onset of enteritis in the two families instigated an appeal to the Health Department for assistance.

Hospital Arrangements. With a view to the intensive clinical and bacteriological investigation of the enteritis and in order to provide nursing services in cases of urgent necessity, sixteen cases were admitted to hospital.

Specimens for Laboratory Examination. The following specimens for laboratory examination were obtained:

(*a*) *Material obtained from Enteritis Cases.*

25 samples of faeces from patients in the height of the enteritis.

6 samples of vomit.

10 samples of faeces from convalescent cases of enteritis.

3 specimens of blood for blood culture from patients early in the acute stage of the enteritis.

21 samples of blood from enteritis cases in the tenth day of illness (for purposes of agglutination tests).

(*b*) *Material obtained from Source of Infection.*

3 samples of the infective milk from the homes of persons who suffered from the enteritis.

Samples of blood from each of the nine members of the farmer's family (for the purpose of testing for the presence of agglutinins).

Samples of faeces from each of the nine members of the farmer's family.

Samples of milk from each of the twenty cows in the dairy herd.

Samples of blood from each of the twenty cows in the dairy herd.

Samples of faeces from each of the twenty cows in the dairy herd.

A piece of the udder of the ailing cow which had been slaughtered.

A piece of the flesh of the ailing cow which had been slaughtered.

Preventive Measures. In consultation with the County Medical Officer of Health, all milk from the farm was stopped as from Wednesday forenoon, July 8th, since no practicable means were available for pasteurising the milk. This embargo was raised on Monday, July 13th, when the laboratory investigation had failed to produce evidence of a carrier condition in any of the milkers or in the cows. Later examination of the detailed evidence showed that the only infected consignments of milk were those of the mid-day and evening milkings of July 6th, and that food poisoning from this source thereafter ceased.

Bacteriological Investigation.

A. *Specimens obtained from Patients.* Within a few hours of the onset of the outbreak specimens were received at the Laboratory. Twenty-five specimens of faeces were obtained from individual patients and sixteen of these showed numerous colonies of a non-lactose fermenting organism when plated on McConkey's medium. Six samples of vomit were obtained and two of these yielded practically pure cultures of the same type of organism. Ten specimens of faeces were later obtained from the same patients after a period of forty-eight hours, but the non-lactose fermenting types had entirely disappeared.

The organisms obtained from the faeces and vomit produced the typical reactions of the paratyphoid group on the initial sugars, were actively motile, and were agglutinated to the full titre of a Gaertner Serum (1 in 2000) and not at all by a 1 in 50 dilution of sera prepared from *B. paratyphosus* A, *B. paratyphosus* B Schottmüller, *B. aertrycke* Mutton and from *Salmonella* Type Newport. Later, many strains of the organism were tested on an extended series of sugars and the following reactions were obtained after a ten days' period of incubation; neither acid or gas was produced in lactose, salicin, saccharose, inosite and raffinose, while acid and gas were produced in glucose, mannite, dulcite, sorbite, trehalose, rhamnose, maltose, xylose, and arabinose; milk showed slight acidity at first, but this was followed by a definite alkaline reaction. Blackening of lead acetate medium rapidly occurred and no indol production could be detected in peptone water.

Absorption of agglutinin tests showed that the isolated strains had the

power to completely absorb the agglutinins from a Gaertner serum, and the stock *B. enteritidis* Gaertner strain completely removed the agglutinins from a serum prepared against one of the strains isolated from the faeces of a patient.

In order to facilitate the investigation of the outbreak sixteen of the more severely ill cases were admitted to Hospital. Blood cultures obtained from three of these cases in the acute stage remained sterile and the blood serum gave no agglutination against the stock strain at this period. After an interval of from 7 to 10 days the sera from eighteen individuals, including further specimens from the three cases from whom blood serum had already been obtained, were tested against formalised broth cultures of *B. paratyphosus* B, *B. enteritidis* Gaertner (stock strain), the causative strain, and the whole of the types of food poisoning group as classified by Schütze (3). The actual strains had been previously obtained from the National Collection of Type Cultures. With the exception of five sera, agglutination was only obtained against *B. enteritidis* Gaertner (stock strain), and *B. enteritidis* Gaertner (causative strain) in dilution varying from 1 in 50 to 1 in 800, the agglutination titres against the stock strain being consistently lower than those obtained against the causative strain.

In five cases, however, various agglutination reactions were obtained as set forth in the following table:

Strains	Agglutination titres of cases				
	No. 2	No. 4	No. 6	No. 8	No. 13
B. paratyphosus B Schottmüller	100	25	0	0	100
B. enteritidis Gaertner (Stock)	50	50	200	200	50
,, ,, (Causative)	100	100	400	800	50
Salmonella Type Stanley	50	50	0	0	100
,, ,, Reading	25	50	0	25	100
,, ,, Newport	50	25	0	0	50
,, ,, Aertrycke (Mutton)	25	50	100	50	100
,, ,, Binns	25	50	0	0	50
,, ,, Hirschfeld	0	100	25	0	50
,, ,, Arkansas	0	100	50	50	25
,, ,, G.	0	50	0	0	25

Previous inoculation with T.A.B. vaccine might have accounted for the presence of various agglutinins in three cases but no history of previous inoculation or enteritis could be obtained from the other two. As a control to these observations ten sera were obtained from normal individuals and tested against the same suspensions of the various organisms. One serum only was found to agglutinate all the types of the food poisoning group in a dilution of 1 in 50 with the exception of Type G and *B. enteritidis* Gaertner. Five of the patients' sera which gave high agglutination titres were absorbed with emulsions of both strains of *B. enteritidis* Gaertner. The agglutinins were completely removed.

The pathogenicity of a strain for various animals was tested. Intraperitoneal inoculation of three guinea-pigs and three mice with a living emulsion caused death in 24 to 72 hours. Six white rats, two rabbits, six guinea-pigs and twelve

white mice were fed with emulsions of the organisms. The rabbits and white rats remained unaffected, but all the mice died at varying period from 48 hours to 14 days after feeding, and the guinea-pigs died within a fortnight. *B. enteritidis* Gaertner was recovered post-mortem from the blood and various organs of these animals.

B. *Investigation of the Sources of Infection.* As it was evident that milk was the only common food consumed by the patients, every endeavour was made to obtain samples of the infecting milk. Three such samples were obtained in the homes of certain patients and from all three specimens numerous colonies of *B. enteritidis* Gaertner were isolated.

At the farm from which the milk was supplied a thorough bacteriological investigation of the farmer's whole family and his dairy cows was made. Blood was obtained from nine members of the family and tested against the formalised emulsion of the various types of the food-poisoning group. Agglutination was obtained only against both strains (stock and causative) of *B. enteritidis* Gaertner by two of the sera. The serum from the farmer himself agglutinated both strains in a dilution of 1 in 25, while the serum from a daughter aged 12 years agglutinated both strains in a dilution of 1 in 100. The faeces from the nine members of the family were examined but no Gaertner bacilli were obtained.

One sample of milk and two specimens of faeces from each of the twenty cows in the herd were examined without definite result. It was then ascertained that a cow which had been ailing for a week had been sent to be slaughtered. The carcase was condemned and from the flesh and mammary gland of the animal (every precaution being taken to avoid external contamination) scanty colonies of a non-lactose fermenting organism were obtained. These strains were found to give sugar, agglutination and absorption reactions,

Table I. *Organisms.*

Cow no.	*B. enteritidis* Gaertner (Stock)	*B. enteritidis* Gaertner (Causative)	Salmonella: Type Stanley	Type Reading	Type Newport	Type *aertrycke* (Mutton)	Type Binns	Type Hirschfeld	Type Arkansas	Type G.	*B. paratyphosus* B Schottmüller	*B. paratyphosus* A.	*B. typhosus*
1	0	0	0	0	50	0	0	50	50	100	50	0	0
2	0	50	100	0	0	0	0	0	50	0	0	0	100
3	0	50	0	0	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0	0	0	0	0	0
5	0	50	50	0	50	50	50	50	0	0	200	0	0
6	0	0	50	50	50	50	0	100	50	100	100	0	0
7	0	0	0	50	0	50	0	0	50	100	0	0	50
8	0	100	100	100	200	200	100	200	200	100	200	0	0
9	0	50	50	0	0	0	0	0	50	0	0	0	50
10	0	0	0	0	50	0	0	0	50	0	0	0	0
11	0	0	0	0	50	0	0	0	100	0	0	0	50
12	100	50	50	100	50	50	0	50	50	50	50	0	100
13	100	400	50	50	0	50	0	0	50	0	50	0	100
14	0	100	0	0	0	0	0	0	0	0	0	0	0
15	50	50	200	0	50	50	0	0	50	0	50	0	100
16	50	100	100	50	50	50	0	50	100	0	50	0	100
17	0	0	0	0	50	0	0	0	100	0	50	0	50
18	0	50	50	50	50	50	0	0	200	0	50	0	50
19	100	50	50	0	0	0	0	0	100	0	0	0	50
20	0	50	50	50	50	0	0	0	100	0	0	0	50

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identical in every way with those of the stock strain of *B. enteritidis* and with the strains of the organism isolated from the patients and the milk. A sample of blood from each of the cows in the herd was now obtained and the serum was tested for agglutinins against the emulsions of the various types of organism. The results are set forth in Table I, the lowest dilution of the serum tested being 1 in 50.

The examination of the sera from the cows was therefore of very indefinite value, and the conclusion to be drawn was either that normal agglutinins were present in the sera of many cows in fairly high dilutions, or that certain of these animals suffer at some time or other from infections due to the organisms of the typhoid paratyphoid group.

Blood was now obtained from twenty heifers and bullocks of various ages on being slaughtered, these animals having been chosen at random and having no connection with the infected farm, and the serological results obtained are as follows:

Table II. *Organisms.*

Bullock no.	*B. enteritidis* Gaertner (Stock)	*B. enteritidis* Gaertner (Causative)	Salmonella: Type Stanley	Type Reading	Type Newport	Type *aertrycke* (Mutton)	Type Binns	Type Hirschfeld	Type Arkansas	Type G.	*B. paratyphosus* B Schottmüller	*B. paratyphosus* A.	*B. typhosus*
1	0	0	0	50	0	0	0	0	0	0	0	0	100
2	0	0	100	0	0	0	0	0	0	0	0	0	100
3	0	0	50	0	50	50	0	200	50	0	50	0	50
4	0	0	0	0	0	50	0	100	0	0	0	0	0
5	400	0	0	0	0	0	0	100	0	0	0	0	50
6	0	0	0	0	0	0	0	50	50	0	0	0	0
7	100	100	0	0	50	0	0	100	50	0	0	0	0
8	0	0	0	50	100	100	100	100	100	50	50	0	0
9	50	0	50	50	100	100	100	100	100	0	50	0	50
10	50	100	100	100	200	50	100	100	200	50	50	0	0
11	50	0	0	0	0	0	0	0	0	0	0	0	0
12	0	100	0	0	0	0	0	0	200	0	0	0	0
13	0	0	0	0	0	50	0	0	50	0	0	0	0
14	0	0	0	0	0	0	0	0	200	0	0	0	50
15	0	50	0	0	0	50	0	0	0	0	0	0	50
16	0	0	0	0	0	0	0	0	50	0	0	0	0
17	0	0	0	0	0	0	0	0	0	0	0	0	0
18	0	0	0	0	0	0	0	0	0	0	0	0	0
19	50	200	100	50	50	50	50	100	400	100	200	0	50
20	0	0	0	0	0	0	0	0	0	0	0	0	0

It will be seen that the reactions obtained from the sera of a random sample of bullocks and heifers are very similar to those obtained from the sera of the cows in the dairy herd under investigation.

Blood samples were next obtained from nine calves varying in age from 3 to 21 days. With these sera no agglutination was obtained against any of the strains in a dilution of 1 in 50. It would thus seem likely that certain of these animals suffer at some period of their life from infections due to organisms belonging to the typhoid paratyphoid group.

As a matter of interest, the sera of sheep, pigs, wild rats, wild mice, and guinea-pigs were then investigated and the results obtained are given in the following tables:

Table III. *Serological reactions obtained from the sera of sheep.*

Sheep no.	*B. enteritidis* Gaertner	Salmonella: Type Stanley	Type Reading	Type Newport	Type *aertrycke* (Mutton)	Type Binns	Type Hirschfeld	Type Arkansas	Type G.	*B. paratyphosus* B Schottmüller	*B. paratyphosus* A.	*B. typhosus*
1	0	0	0	0	0	0	0	400	0	0	0	0
2	0	0	0	0	0	0	0	0	0	0	0	0
3	0	0	0	0	50	0	0	100	0	0	0	0
4	50	0	0	0	0	0	0	0	0	0	0	0
5	50	50	100	0	50	0	0	400	0	0	0	0
6	0	400	0	0	0	0	0	0	0	0	0	400
7	0	50	0	0	0	0	0	100	0	0	0	200
8	0	0	0	0	0	0	0	50	0	0	0	0
9	0	0	0	0	0	0	0	100	0	0	0	0
10	50	0	0	0	0	0	0	100	0	0	0	0
11	50	0	50	0	0	0	0	100	0	0	0	0
12	50	50	0	0	0	0	0	100	0	0	0	50

Table IV. *Serological reactions obtained from the sera of pigs. Lowest dilution used* 1 *in* 50.

Pig no.	*B. enteritidis* Gaertner	Salmonella: Type Stanley	Type Reading	Type Newport	Type *aertrycke* (Mutton)	Type Binns	Type Hirschfeld	Type Arkansas	Type G.	*B. paratyphosus* B Schottmüller	*B. paratyphosus* A.	*B. typhosus*
1	50	0	0	0	0	0	100	0	0	0	0	50
2	0	0	0	0	0	0	100	0	0	0	0	0
3	50	0	0	0	0	0	100	0	0	0	0	0
4	50	100	100	100	100	100	100	100	50	100	0	100
5	100	0	0	0	0	0	0	50	0	0	0	100
6	0	50	50	50	50	50	0	0	0	0	0	100
7	50	0	0	0	0	0	0	0	0	0	0	0
8	50	50	0	50	0	0	0	50	0	0	0	50
9	50	0	0	0	0	0	0	50	0	0	0	0
10	50	50	0	50	0	0	0	0	50	50	0	0
11	100	50	50	50	50	50	50	100	0	0	0	50
12	50	50	0	0	0	0	0	0	0	0	0	0

The blood sera from twelve rats were next examined and one specimen only agglutinated two strains—Type Stanley and *B. typhosus* in a dilution of 1 in 100. The sera from ten wild mice and from ten guinea-pigs did not produce agglutination, the lowest titre of the sera from the mice being 1 in 50 and from guinea-pigs 1 in 10.

In addition the faeces from twelve sheep, twelve pigs, twelve rats, ten mice and ten guinea-pigs were examined for organisms of the paratyphoid group. Only one specimen from a rat gave an organism which, culturally and serologically, gave the typical reactions of *B. enteritidis* Gaertner.

Discussion.

It has been shown that the 497 cases of enteritis occurring in Aberdeen in July, 1925, were caused by the *B. enteritidis* Gaertner which was isolated from (*a*) the vomited material and faeces of patients suffering from the enteritis, (*b*) the infective milk as obtained from three households infected with the enteritis, and (*c*) the udder and flesh of a cow from the dairy herd producing the milk, the cow having suffered from induration of the udder and later a septicaemia.

It might be suggested that a human carrier transmitted the infection, since the blood serum of the farmer agglutinated both strains (stock and

causative) of *B. enteritidis* Gaertner in a dilution of 1 in 25, and the serum of his youngest daughter, aged 12 years, agglutinated both strains in a dilution of 1 in 100. As opposed to this view it is to be noted that on occasion Gaertner agglutinins in as high a dilution as 1 in 100 may be found in the blood serum of apparently normal individuals, that there were no symptoms or history of enteritis in the farmer and his daughter, and that the examination of their faeces was negative. In these circumstances the positive finding of *B. enteritidis* in the udder and flesh of the septicaemic cow and in the infective milk, warrants the conclusion that the septicaemic cow was the essential source of the infection.

The fact that the source of this milk-borne Gaertner enteritis has been traced back to an infected cow gives support to the hypothesis originally put forward by Savage to the effect that the Gaertner-caused food-poisoning outbreaks are due to infection of food with virulent Gaertner group organisms derived either from animals which are at the time suffering from disease due to Gaertner group bacilli, or from animals acting as carriers of these bacilli. The common view that Gaertner infections are often spread by human carriers has no foundation in fact. Reference to the literature of *Salmonella* food-poisoning shows that in only three outbreaks in this country has any evidence been obtained indicating a human source of the infection, viz. in the Wrexham outbreak (1910), the Brighton outbreak (1917), and in the Dublin outbreak (1921). The matter is fully discussed by Savage and Bruce White in the *M.R.C. Special Reports*, Nos. 91 and 92(4).

The negligible mortality of this Aberdeen outbreak of milk-borne Gaertner enteritis as contrasted with the high mortality of the Aberdeen milk-borne Flexner dysentery outbreak in 1919 is noteworthy, and is in accord with the findings of Savage and Bruce White.

The presence of agglutinins of the typhoid paratyphoid group in the sera of bovines, sheep and pigs, and their absence in young calves, would appear to indicate that these animals either obtain immunity by reason of the absorption of non-infective doses of such bacilli, or that at some period of their lives they suffer from infections due to organisms belonging to the typhoid-paratyphoid group. Since the available evidence is against the view that cattle commonly harbour *Salmonella* organisms in their intestines, the elucidation of the causes of the development of these agglutinins in cattle and other animals may provide fresh information as to the manner in which *Salmonella* infections are transmitted from lower animals to man, and may eventually show that *Salmonella* infections are essentially and primarily diseases of lower animals occurring only secondarily in man.

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