

A HOSPITAL OUTBREAK OF TYPHOID FEVER

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(With 2 Figures in the Text)

An epidemic of typhoid fever occurred in a hospital in the autumn of 1948 and involved 135 members of the staff and patients, including nineteen symptomless excretors. The majority of the cases occurred during the first 4 weeks of the epidemic, but the last case did not sicken until 7 weeks after the commencement of the epidemic.

The hospital is situated just over a mile from a town of 10,000 inhabitants. At the beginning of the outbreak there were at the hospital 359 patients and a staff of 383. The after-care of patients in the region is supervised by a staff living and feeding in a house about a mile from the hospital.

The hospital was first opened for its present purpose in 1920 in hutted buildings erected during the 1914-18 war. Subsequently, new buildings of a more permanent character were erected over a considerable area of the hospital site, and their erection had necessitated considerable adaptation of the main water and drainage plans from time to time.

In January 1948 a fire had destroyed a large part of the hospital, including some ward blocks.

The foul drainage system was connected to the main sewers of the district, but from January to March 1948 it had been necessary, owing to repairs to the hospital sewer, to pump the hospital sewage on to ground adjacent to the hospital which was later used for the cultivation of vegetables.

Food for patients was cooked in the main hospital kitchen, a separate kitchen in the nurses' home being used for the preparation of food for the resident nursing and domestic staff. A canteen supplied meals to out-patients, visitors and non-resident workers.

CHRONOLOGY OF THE OUTBREAK

On 8 September 1948 the physician to the hospital had under his care twelve members of the nursing staff and two patients in the hospital. They had become ill during the previous 3 days with symptoms suggestive of influenza. The suspicion of typhoid fever was confirmed serologically in six cases on 9 September, and the diagnosis was fully established on 10 and 11 September when typhoid bacilli were identified in the blood and faeces of some of these cases at the Public Health Laboratory.

Discussions took place between the hospital medical staff and the district Medical Officer of Health on 9 September. Arrangements were made for the boiling of drinking water and milk, the cessation of admission and discharge and visiting of patients, cancellation of staff leave, and the following-up of patients recently

discharged from hospital and of members of the staff who were on leave. On this date also the local doctors and the Ministry of Health were informed of the outbreak.

While the above measures were being carried out, further cases were developing the early signs of the disease. Before the epidemic ended on 23 October the diagnosis of typhoid fever had been made in 116 persons, thirty-two of whom were patients in the hospital, the remainder being members of the resident and non-resident staff. Seven of the cases were fatal. No cases were reported among the local population other than members of the non-resident staff of the hospital, twenty-one of whom were affected, and no secondary cases occurred in the homes of any of these cases.

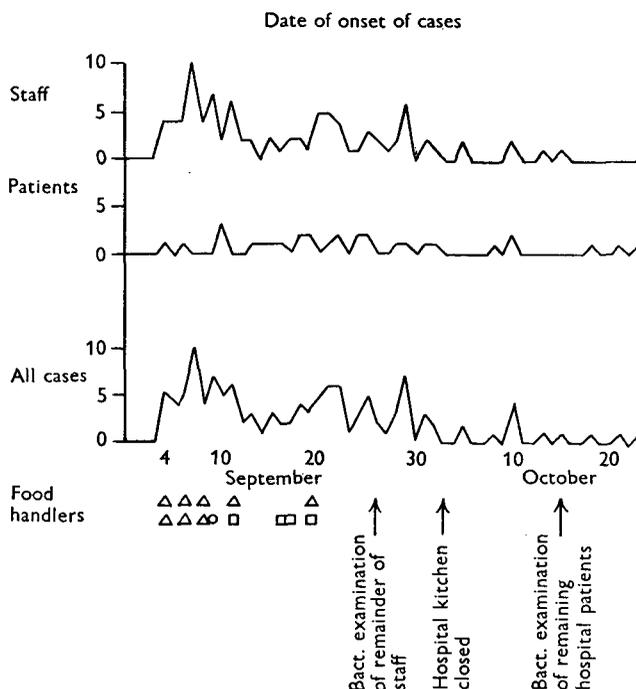


Fig. 1. Δ , indicates a person taken ill at work; \circ , indicates a symptomless excretor found at work; \square , indicates a faecal excretor found at work who later developed typhoid fever.

The epidemic affected members of the day and night nursing staff, domestics (both resident and non-resident), patients in a number of different wards, members of the male outside staff, and of the administrative and clerical staff.

The accompanying chart (Fig. 1) was made after the epidemic, when the exact date of onset of all reported cases had been ascertained.

The reduction in numbers of available nursing staff caused by the high proportion of nurses attacked during the first week of the epidemic placed a great strain on the administration of the hospital, and it was decided that from 19 September onwards such patients as were fit to go should be discharged, arrangements being made through the Medical Officer of Health for them to be kept under surveillance after leaving the hospital. Only one patient discharged after this date developed the disease.

ESTIMATED DATE OF INFECTION

In the early stages of the epidemic an estimate of the probable date of infection had to be based upon the dates of onset of cases arising in the hospital. Details of the history of cases occurring among recently discharged patients were not immediately available, and as they were obtained they necessitated modification of previous estimates of the date of infection. The earliest case sickened on 4 September, the peak of the first wave being on 7 September. Taking the incubation period as 10 days from 7 September the probable date of infection appeared to be 28 August. There was, however, evidence that infection had taken place both before 30 August and after 31 August. Thus a maid who returned from a fortnight's leave on 1 September and had her first meal in the hospital on that morning, developed typhoid fever on 13 September. Three patients who were discharged on 30 and 31 August and 1 September, subsequently developed typhoid fever on 14 and 25 September and 1 October respectively. No patients discharged before 30 August developed the disease. Further suggestive evidence regarding the date of infection is supplied by the manner in which the disease attacked certain groups of the staff.

(a) On 28 and 29 August seven of a group of eighteen physiotherapists were away on leave. The eleven who were in the hospital on these dates all became infected, while the seven who were on leave remained free from infection.

(b) On 30 August sixteen of the twenty-two members of the night nursing staff were on duty and took the midnight meal. All sixteen became infected, whereas the six who were off duty escaped infection.

Thus the evidence pointed to a continuing source of infection operating at least from 29 August to 1 September, and it was not possible to incriminate any particular meal as the sole cause of the outbreak.

DISTRIBUTION OF CASES

The infection was not spread evenly through the hospital, but showed a selective action on certain groups. At the beginning of the outbreak it appeared that there was some degree of geographical concentration of cases, the majority of the staff cases being those living and/or feeding in the nurses' home, and the earliest of the cases among patients being in the ward immediately adjacent to the nurses' home. Even at this stage there were, however, exceptions, the most striking being the hospital secretary who was attacked, although living in a house at the opposite end of the hospital grounds. Careful inquiries were made into the possibility of food having been taken on occasion from the nurses home to patients in the wards. For obvious reasons it is not surprising that no specific instances were discovered, but the possibility that such transfer of food may have taken place cannot be excluded. The sick-staff consulting room and the home sister's office are in the nurses home, and it is customary for certain members of the staff whose duties take them to these rooms to take cups of tea in the home at various times.

It will be seen from Table 1 that the highest attack rates were in the resident nursing and domestic staff, and that the patients and other grades of staff sustained

considerably lower rates. The night nurses had by far the highest attack rate of any group.

It was customary for the night and day nurses to have a meal together at 7.30 p.m. The night staff had their dinner at midnight, and the main dishes of this meal had been prepared during the day at the time of the day-staff lunch, and kept at room temperature until midnight. Vegetables were freshly cooked for the night staff, having been prepared at midday. Meat was partly cooked at noon, and re-cooked at midnight. Custards and trifles were made by the day staff and left at room temperature for the midnight meal. If infection of a dish of the day-staff lunch is postulated, this dish, which would have been consumed by the day staff at once, would have been eaten by the night staff only after infecting organisms had had the chance to multiply for 12 hours. Oddy & Clegg (1947) reported an outbreak of staphylococcal food poisoning in colliers following the consumption of pressed beef sandwiches, in which those eating the sandwiches during the afternoon or night shifts of the day of preparation escaped illness, while many members of the day shift on the following day became ill after eating sandwiches from the same batch.

Table 1

Group	Attack rate (%)
Nurses, night	83
Nurses, day	26
Domestics, resident	32
Domestics, non-resident	7
Administrative and clerical	8
Workmen	15
Patients	9
Total	16

FOOD HANDLERS

Immediately the outbreak was discovered a start was made on the bacteriological and serological examination of food handlers (Jones, 1951). On 12 September it was discovered that the head cook in the nurses home was a faecal excreter of *Salmonella typhi* of the same Vi-phage type as that responsible for the outbreak. This woman gave no history suggestive of typhoid infection either in the near or remote past. She was quite symptomless, and remained so during the whole of her period of observation. Repeated examinations failed to detect agglutinins in the blood. Her faeces were positive for 8 weeks, but excretion later became intermittent, and before being released from surveillance her faeces and urine were negative on twenty-four consecutive occasions between 11 November and 12 January. Her duties in the kitchen were such that she would have taken part in the preparation of custards, trifles, etc., for the day-staff lunch which would subsequently have been eaten by the night staff at midnight. Her first specimen of faeces, collected on 10 September, was positive, and if she had been shedding organisms prior to this date during the period 28 August to 1 September she might have caused the whole of the primary wave of cases. There was, however, no

evidence that this woman was a chronic carrier. Careful examination over a period of more than 4 months showed that she was a symptomless excreter, probably infected at the same time as the first wave of cases (Jones, 1951).

Two days later one of the food servers in the nurses' home was found to be a faecal excreter and she fell ill on the following day. Before 20 September two further food servers and one kitchen worker at the nurses' home were found to be excreting typhoid bacilli, all of them subsequently developing typhoid fever.

During the early days of the epidemic studies of the Vi-antigen content of the infecting strain carried out at the Central Enteric Reference Laboratory suggested that the epidemic strain was of relatively low virulence, and led to the prediction by Dr A. Felix that the proportion of symptomless excreters and ambulant cases would be greater than would be expected in an epidemic due to an organism of average virulence, the situation in this respect being thought to be comparable to what usually happens during an outbreak of paratyphoid B fever (Felix & Anderson, 1951). This prediction was fulfilled when bacteriological examinations were extended to include all members of the nursing and domestic staff and all the remaining patients in the hospital. These examinations brought to light nineteen symptomless excreters, and fifteen faecal excreters who later developed symptoms of the disease (Jones, 1951). Thus thirty-four of the total of 135 persons involved in the outbreak were detected by isolation of the bacilli before the onset of symptoms. In view of the large secondary wave and the alarming number of symptomless excreters brought to light, it was decided to remove all existing food handlers from the kitchens. By the courtesy of the Adjutant-General's Department, army personnel were drafted into the hospital to work in the main kitchen and cook food for both patients and staff.

WATER SUPPLY

There were two separate main water supplies to the hospital. Supply A was derived from the nearby aqueduct of the water supply to a large city. A 6 in. branch from the aqueduct divided into two 4 in. mains, one of which supplied a village and the other the hospital. This latter main was known to be badly encrusted. It terminated at the main entrance to the hospital where a 3 in. supply pipe entered the site to form a ring main. This supply was treated by sand filtration about $2\frac{1}{2}$ miles above the point of connexion with the aqueduct, and then chlorinated with a dose of 0.4 p.p.m. Residual chlorine tests showed 0.1 p.p.m. at the village, but no perceptible residual at the hospital, this latter finding being attributed to the encrusted main. From this supply an average of 32,000 gallons a day was delivered to the hospital. The mains pressure was found to vary between 30 and 80 lb./sq.in. at maximum and minimum draw-off periods.

Supply B provided a small additional supply of 4000 gallons a month from the 3 in. main of the local Rural District Council supply. This served a few sinks and water closets and wash hand-basins in the older part of the nurses home. The supply, which came untreated from a borehole, served a population of 6000 persons between the source and the hospital.

In 1940, as an emergency measure, the supplies A and B had been connected

by means of a 30 ft. length of 3 in. piping fitted with an isolation valve at each end, and situated in a duct under the nurses home. Normally both valves were kept closed, but investigation showed that they were not absolutely drop-tight. The intercommunication had been last used during the fire in January 1948.

At the beginning of the outbreak it was thought advisable to discontinue supply B, and instal a chlorinator on the main of supply A as it entered the hospital.

When the possibility of water-borne typhoid fever was first considered in this instance, it was at once apparent that as no cases had been reported from the very large population receiving both supplies outside the hospital the theory of contamination at the source was untenable. There remained, however, the possibility of contamination within the hospital. Reconstruction operations within past years had resulted in some of the foul sewers running under buildings, and it is possible that these sewers had been considerably disturbed during the building of foundations. There was, in fact, some evidence of leakage in the drainage system, but tests carried out by charging the drains with common salt failed to produce evidence of leakage from sewers into the water system, and similar negative results were obtained with fluorescin. It was not possible to carry out a pressure test, but meter tests showed no sign of leak in the water-distribution system.

An event which gave rise to considerable discussion at the time of the epidemic was the fact that the main of supply B had been tapped in the road just outside the hospital on 31 August 1948 in order to connect up two cottages on the other side of the road. If infection had occurred at this time it was thought that infected water might have leaked through the two valves mentioned above and reached the main of supply A. If this had happened, the first parts of the hospital to be affected would have been the nurses' home and the wards in which the original patient cases occurred. This idea had to be abandoned for a number of reasons. First, the workmen engaged on the tapping were examined with negative results. Secondly, it was established that the tapping had been carried out under pressure. Thirdly, it was considered that the pressure maintained in the main of supply A would render it most unlikely that any leak would occur in the direction suggested, but rather the reverse. Fourthly, a patient discharged from the hospital on the day before the incident occurred developed typhoid fever.

MILK SUPPLY

Although the course of the outbreak suggested strongly that a large part of the later phase of the epidemic was due to secondary spread, there still remained considerable doubt as to the origin of the primary wave. No evidence was discovered that would incriminate water, and attention was turned to the milk supply. In presenting the following account of the origin of the milk in some detail it must be stated that although it shows the possibility that the epidemic might well have been caused by infected milk, no conclusive evidence was found that it was in fact so caused.

The principal milk supply came from a T.T. herd of eighty cows (sixty in milk) at farm X, and was not pasteurized. This farm supplied 80 gallons daily to the hospital and 30 gallons to the neighbouring town. Hygienic conditions at the

farm were of a satisfactory standard. The fact that 30 gallons a day was being distributed untreated to the population in the town without giving rise to a single case of typhoid fever suggested strongly that this milk was not the cause of the epidemic at the hospital.

When the supply of milk from farm *X* was insufficient for the needs of the hospital an accommodation supply of 12 gallons was obtained from farm *Y*, which had an attested herd and was licensed for T.T. milk. Such additional supplies had been delivered to the hospital on 19, 20, 21, 22, 29 and 30 August and on 2 September. Farm *Y* had a herd of forty-two cows (twenty-eight in milk). Apart from the accommodation milk to the hospital and that consumed by workers at the farm the whole of its milk supply, averaging 44 gallons daily, was sent to a large wholesale firm for pasteurization. Further inquiry revealed that it was more than likely that a churn of milk from farm *Y* would find its way to the nurses' home at the hospital, as the milk from this farm was loaded on to the van of the principal milk supplier on its way to the hospital, and it was customary for a churn to be delivered at the nurses' home before the main supply was unloaded at the main hospital kitchen. The last churn to be loaded (that from farm *Y*) would be the first to be unloaded, and would thus go to the nurses' home. It is worthy of note that whereas milk delivered to the main hospital kitchen was kept in a refrigerator that delivered to the nurses' home was kept at room temperature.

Unpasteurized milk from farm *Y* was also consumed by the farm workmen and their families, none of whom were affected. Bacteriological and serological examination of those living and working at the farm did not disclose any signs of infection.

The water supply at farm *Y* was obtained principally from a well in the farmyard, the water from which was pumped into an overhead tank in the dairy. This supply was supplemented by a gravity supply from a concrete tank in a field which was filled by a nearby 'spring'. These waters were used for all purposes including cooling the milk, cleaning, and general domestic use. The presumptive coliform count on both waters indicated that they were liable to pollution. The 'spring' mentioned above was open to contamination from field drains, and while investigations were being carried out in this direction a third water supply to the farm was discovered. This consisted of a small supplementary supply to taps in the cowshed, and did not form part of the recognized water supply to the farm. It originated in pool *P* (Fig. 2), which was fed by a stream originating in a ditch *Q* running for about 300 yd. along the side of the road. This was a quiet road and the ditch was quite accessible to human pollution from farm workers and other passers-by. The water in the ditch came from pool *O*, and the connecting channel between this pool and the top of the ditch also received surface drainage from a farmyard at *N*. Rather less than a quarter of a mile from farm *Y* was a large mansion *L* which had recently been divided into three flats. Sewage from this building passed to a septic tank close to the house, and from there to an effluent tank buried in a field 150 yards away. The connecting drain between the septic tank and the effluent tank was found to be broken and blocked. The overflow from the effluent tank passed to a pipe to discharge into a small ditch *S* at the edge of a field, and this ditch ran under the road to join the ditch *Q* which fed pool *P*.

Round about the point where the sewage effluent emptied into ditch *S* the ground was boggy, and it is a point of some interest that this ground formed part of a field used as pasture by cows from farm *Y*.

The cottage *M* shown in the diagram had a bucket-closet and no water-closet, but gullies outside the cottage drained into an underground road drain *R* which joined the lower part of ditch *S* and subsequently passed to pool *P*.

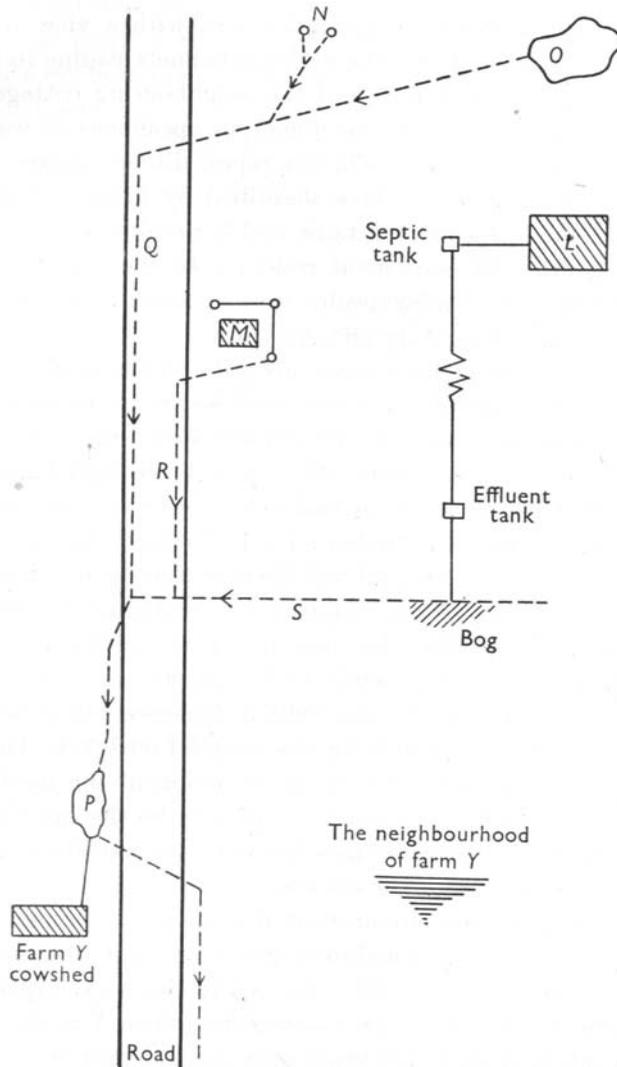


Fig. 2.

Fluorescin put into the septic tank emerged in the boggy ground above the site of the fracture in the exit pipe. Fluorescin put into the effluent tank passed through to the water in ditch *Q*.

Water from pool *P* was used in the cowshed for cleaning. No evidence was discovered that it was used for cleaning utensils and churns, which would normally

be dealt with in the dairy, where the water came from other sources. Nevertheless, these investigations had shown:

(1) That water being used in the cowshed could be contaminated by: (a) sewage from mansion *L*; (b) possible human contamination from the open ditch *Q*; (c) sullage from cottage *M*; (d) surface drainage from farmyard *N*.

(2) That cows' udders could be contaminated by human sewage in the boggy ground beside ditch *S*.

Bacteriological examinations were undertaken with a view to demonstrating specific contamination of water in the various channels leading to pool *P*, and in a search for a carrier in mansion *L* and the neighbouring cottages. No typhoid bacilli were discovered in any of the numerous specimens of water and sewage taken from a number of situations in the pipes, ditches, gullies and tanks concerned. Gauze pads, similar to those described by Moore (1948), were left for 48 hr. in the septic tank, the effluent tank, and in running water in other situations with negative results. All permanent residents in the mansion were examined with negative results, and similar results were obtained from a number of persons who had stayed there during July and August.

Cottage *M* was inhabited by a man, his wife and his son. The wife and son proved negative, but examination of the blood serum of the man himself, carried out by Felix's technique at the Central Enteric Reference Laboratory, gave the following results: TVi agglutination, 1/15 standard; TO agglutination, 1/200 + \pm ; TH agglutination, 1/200 +. Typhoid bacilli were grown from a number of faecal specimens from this man commencing on 1 December. He gave a history of an attack of typhoid fever in 1940, and had lived at the cottage since January 1948. The sanitary arrangements of the cottage have been indicated above, but it may be mentioned, in addition, that this man also used a urinal situated close to the mansion and draining into the septic tank. Rather surprisingly the organism isolated from this carrier was found by Felix & Anderson (1951) to be not identical with the typhoid strain responsible for the hospital outbreak. There can be little doubt that 10 years ago, prior to the introduction of the method of Vi-phage typing, this carrier would have been considered to be the cause of the outbreak. Using the modern laboratory techniques, however, the conclusion had to be reached that he had no connexion with the epidemic.

To sum up, therefore, the circumstantial evidence in favour of a milk-borne infection is very strong, but conclusive proof was not obtained. The affected persons could all have acquired their infection in this way. Apart from the milk consumed by farm workers the milk supplied from farm *Y* to the hospital was the only milk from this farm that was being consumed untreated. It was supplied to the hospital at the material time. A churn from the farm would probably have reached the nurses' home, and would have been kept there at room temperature, and assuming that it was infected an explanation is provided for the heavy attack rate in the nursing staff. Conditions at the farm were such that the milk could have become infected, but exhaustive tests failed to reveal the manner in which such specific infection might have occurred. Whether or not milk played any part in the epidemic remains, therefore, an open question.

OTHER FOOD

The possibility of infected food being consumed in the hospital was examined, but the foods considered were excluded as possible sources by virtue of their inability to account for the high attack rate in the nursing staff and the complete absence of typhoid fever among other consumers in the neighbourhood. During the 3 weeks preceding the onset of the epidemic the menu had sometimes included cabbages grown on the ground over which hospital sewage had been pumped during the early months of the year. The cabbages had been consumed by staff and patients alike, and had always been boiled. No evidence was found that infection had been caused in this manner.

SECONDARY SPREAD

The epidemic had an almost explosive onset, with a large secondary wave 2 or 3 weeks later. The nature of this secondary wave suggests at first sight that it was mostly composed of true secondary cases. It was not possible, however, to state for certain which cases were primary and which secondary, as there was evidence of extreme variation in the incubation period between different cases in the epidemic. Although it was possible that infection was operative before 28 August the most likely dates of infection were between 28 August and 1 September. Five cases sickened on 4 September, four on 5 September, five on 6 September, and ten on 7 September. If the infecting date were 28 August, these groups of cases had incubation periods of 7, 8, 9 and 10 days respectively. At the other end of the scale were cases who were discharged from the hospital on 30 and 31 August and 1 September, who sickened with typhoid fever on 14 and 25 September and 1 October respectively, giving minimum incubation periods of 15, 25 and 30 days. It is thus possible that a number of cases in the hospital in what appears to be the secondary wave were primary cases of long incubation. Nevertheless, the general appearance of the chart of incidence strongly suggests that there was an initial heavy infection of an article of food or drink at the end of August producing the main wave, with secondary cases occurring during the second half of September. It is a tribute to the sanitary technique of the staff that so few secondary cases occurred among the hospital patients.

SUMMARY

1. An epidemic of typhoid fever involving 135 persons in a semi-closed community of 742 persons is described.
2. There were nineteen symptomless excretors (14.1 %).
3. Among the 116 clinical cases there were seven deaths, giving a case fatality of 6.03 %. If the case fatality were calculated on the total persons involved (including symptomless excretors), the figure would be 5.2 %.
4. The origin of the outbreak was not discovered, but there was strong circumstantial evidence in favour of a milk-borne infection.

Our thanks are due to Dr A. C. Jones who carried out the vast amount of bacteriological work involved in the epidemic, to Dr A. Felix, F.R.S., for Vi-phage

typing the organisms, and to Mr J. V. Meredith for assistance in the investigations at the farm.

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