J. Hyg., Camb (1962), **60**, 1
Printed in Great Britain

Q fever in South Australia: an outbreak in a meat-works

By MARGARET D. BEECH, A. E. DUXBURY and PETER WARNER*
The Institute of Medical and Veterinary Science, Adelaide, Australia

(*Received* 5 *July* 1961)

INTRODUCTION

In May 1957 it became apparent to us that an unusually large number of people in Adelaide had contracted Q fever. Our awareness of this was because this laboratory is the only one in the State of South Australia that performs complement-fixation tests for the diagnosis of the disease. Preliminary inquiries showed that all but one of the cases of the disease occurred among workers at the Metropolitan Abattoirs or at a closely associated sausage-skin factory. It was no surprise to learn that most cases were from the local meat-works, for, since the time when the disease was first recognized in South Australia (Stokes, 1950) up to 1957, it had never been known to occur elsewhere in the city of Adelaide although we discovered an exception to this. Outside the city of Adelaide in country districts Q fever had been detected on two occasions involving seven people (Dane & Beech, 1954–55).

Owing to the unusual number of cases and the occurrence of Q fever in one not a meat-worker we decided to investigate the outbreak. We started our inquiry at the beginning of what was to be the largest known outbreak of Q fever in Adelaide. There were a total of fifty-two diagnosed cases of the disease by the end of the outbreak which occurred in December 1957.

Review of Q fever in South Australia before 1957

The first record of the recognition of Q fever in South Australia are papers by Stokes (1950, 1953) in which she describes an outbreak occurring in the Adelaide Metropolitan Abattoirs during the summer of 1947–48. The patients she mentions were diagnosed clinically by Dr J. M. Dwyer (Stokes, 1953) and were confirmed by laboratory methods. Including the outbreak described by Stokes and excluding laboratory infections there was, up to 1957, a total of 60 recognized cases of Q fever of which 50 were confirmed serologically. The monthly distribution of these is shown in Fig. 1 and Table 1 lists these cases where it will be seen that the outbreak in 1957 is considerably larger than any in the ten years preceding it.

It is also clear that the vast majority of cases of Q fever occur in meat-works and of these most come from the Adelaide Metropolitan Abattoirs and an associated casing company. The only cases occurring at other meat-works were two described by Stokes (1953) from the Noarlunga Abattoirs in 1950 situated some 20 miles south of Adelaide. These abattoirs are small, now employing only sixteen men, and

* Present address: Departments of Bacteriology, Medical College, University of Manitoba and the Winnipeg General Hospital.

a recent inquiry elicited the statement that no cases of Q fever had occurred since 1950. Stokes (1953) states that she found no antibodies in the sera of 11 volunteers from the Noarlunga Abattoirs. Up to and including 1956, only six cases of Q fever had been recorded in the casing company—two in 1947–48 and one in each of 1951, 1953, 1954 and 1955. However, it is not recorded whether these men were working at the casing company's factory or on the offal floor of the Metropolitan Abattoirs.

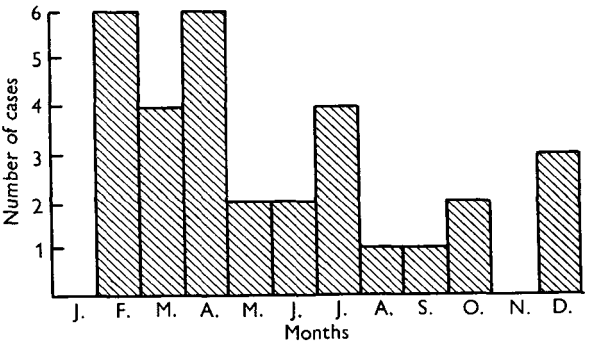

Fig. 1. Monthly incidence of cases of Q fever occurring in the Adelaide abattoirs combined for the years 1948–56 inclusive.

Table 1. *Q fever in Adelaide: number of recorded cases by years since* 1947 *occurring in meat-works and elsewhere*

Year	Meat-works	Others
1947–48	14*	0
1948	1*	0
1949	0	0
1950	6* (2)†	0
1951	9 (2)	0
1952	2	0
1953	3 (1)	0
1954	11 (3)	7‡
1955	1	0
1956	5 (2)	1§
1957	51	1‖
Total	103 (10)	9

* From Stokes (1953).
† Number of cases included in preceding figure that were not serologically proven.
‡ From Dane & Beech (1954–55)—the cases were all sheep farmers.
§ A kangaroo dealer.
‖ A dairy farmer described in the text of this paper.

Until 1954 no case of Q fever had been detected in anyone other than meat-workers in South Australia. In that year Dane & Beech (1954–55) detected seven cases of Q fever in country districts. Three of the patients were sheep farmers in the Hamley Bridge district some 40 miles north of Adelaide. The other four cases were discovered in one sheep station near Hawker some 250 miles north of Adelaide. *Coxiella burneti* was isolated from wool clippings of sheep from the latter station.

The sera of four of 20 ewes, crutched at about the same time as the men were probably infected, contained complement-fixing antibodies for the organism. These cases are remarkable in that they are the only ones to have been detected in country districts in South Australia and, with two exceptions, are the only ones detected outside meat-works in or near metropolitan Adelaide.

One of the exceptions to the statement that Q fever only occurs in meat-works in South Australia was a man who contracted the disease in 1956 in Adelaide. This man was employed by a firm of animal exporters. He denied any contact with domestic animals but had been engaged in capturing and caring for kangaroos. This is of interest as in 1955, Dane & Beech (1954–55) found high titre Q fever complement-fixing antibodies in two out of five adult kangaroos tested and Derrick & Pope (personal communication) have found antibodies in 22% of 120 kangaroos tested in Queensland. Thus, it is possible that this man contracted his disease from the animals with which he had been in close contact. The other exception to the occurrence of Q fever in meat-works in metropolitan Adelaide is the dairy farmer, described in this paper, but, as stated, he had visited the abattoirs.

The fact that Q fever occurred only among abattoir workers in Adelaide was supported by Dane & Beech (1954–55) who found no antibodies in sera from 84 healthy volunteers and from 125 patients with pyrexia of unknown origin. These findings are in accord with the experience of one (M.D.B.) of us in these laboratories over several years.

Turning now to the annual incidence of Q fever in meat-workers, it shows considerable yearly variation, averaging 5·2 cases per year up to and including 1956. The variation ranging from zero to 14 cases a year suggests that the source of infection is not continuous but sporadic. This is borne out by a study of the dates of onset of 30 out of the 31 cases occurring from 1951 to 1956 inclusive on which we have adequate information, which shows that the majority (21/30) of cases occur in association with other cases in groups of two, three or five and only nine occurred singly. Of these 30 patients, 16 were working on beef and two on calves. Six were associated with mutton, four of these comprised an outbreak and are described in the Annual Report (1953–54). Two were meat inspectors and four had some miscellaneous duties not directly connected with the early stages of slaughtering and disembowellment of animals. One of these, referred to by Stokes (1953), was the proprietor of a canteen situated within the abattoirs. Thus, the risk of infection among beef-workers appears to be relatively high especially in view of the fact that there are, on the average, 160 workers in the mutton hall as compared with 95 in the beef hall. The predominance of infection among beef-workers is supported by Stokes's (1953) findings in the 1947–48 outbreak which was apparently confined to beef-workers.

It seems that the disease when it appeared in metropolitan Adelaide occurred, to all intents and purposes, only in meat-workers. The disease appears to occur sporadically without special seasonal incidence (Figs. 1 and 3). It was concluded that probably this situation was provided by a low proportion of infected animals. Stokes (1953) found no antibodies in the sera of 793 beef cattle slaughtered in the Adelaide abattoirs but obtained from the northern part of South Australia, New

South Wales, Queensland and the Northern Territory. Similarly, no antibodies were found in 644 sera from dairy herds in coastal districts and close to Adelaide. On the other hand, Dane & Beech (1954–55) found antibodies in two out of 100 sera from cattle resident in the Northern Territory. Dane & Beech (1954–55) found complement-fixing antibodies in only one of 270 ewes slaughtered at the Adelaide abattoirs in 1954. They found antibodies in none of 180 sheep resident on stations in northern South Australia but, as mentioned above, four of 20 sheep on a station, where clinical cases of Q fever occurred, had antibodies.

These results are not altogether surprising when it is realized that approximately 100,000 cattle, 1,500,000 sheep and lambs, 85,000 calves and pigs are slaughtered each year in the abattoirs. An incidence of infected cattle of as little as 0·1% would provide 100 infected animals a year. A far smaller number than this would be sufficient to account for the cases of Q fever in the abattoirs. Similar arguments may be applied to sheep except that we need only postulate a very much smaller incidence of infection.

Description of the abattoirs and the killing process

The 'Metropolitan Abattoirs' is the only slaughterhouse in Adelaide and is situated on the periphery of the metropolitan area extending over some 800 acres. Cattle, sheep, pigs and goats are brought in by rail from as far as the northern part of South Australia, the Northern Territory, Queensland and northern New South Wales.

Table 2. *Q fever in Adelaide: distribution of people with the disease or with high titre* ($\geqslant$ 1:160) *antibodies against* Coxiella burneti *in the abattoir departments*

Department	No. employed	Cases	Attack rate %	High titres in survey	No. tested in survey
Beef	95	14	15	2	70
Inspectors	35	7	20	1	28
Offal	90	14*	16	5	59
Mutton	160	6	4	2	84
Pig and calf	24	0	0	0	8
Miscellaneous meat-workers	43	1	2	1	37
By-products	60	1	2	0	37
Chillers and freezers	110	1	1	0	35
Transport	70	1	1	0	25
Markets	65	1	2	0	55
Non-meat-workers	124	4	3	0	78
Totals	876	50†	6	11	516

* Two of these were employed at the casing company only.

† Note that two cases are not included in this total: the first was the dairy farmer referred to in the text and the other was a man who apparently worked half-time in the beef hall and half-time in the mutton hall during the relevant period.

The abattoirs are broken up into a number of departments which employ between 800 and 900 workers (Table 2). The figures given in Table 2 for the number of employees in each department are an estimate of the average employed on a

https://doi.org/10.1017/S0022172400039255 Published online by Cambridge University Press

full working day. The exact numbers, of course, are variable from day to day. First, there are the holding paddocks and the markets where the animals are kept and bought and sold before slaughter—all the employees working in these areas are designated 'markets' in Table 2, and include drovers and others likely to be exposed to dust from the hides and excreta of animals. There are then the three killing halls near one another—one for beef, one for mutton, and one for pigs and calves. Working in the killing halls are the inspectors whose ubiquity and close contact with internal organs of the animals demand that they be considered separately. Two other departments are fairly closely associated with the killing halls. The first is the 'offal floor' which is situated below the killing halls and receives the offal from all three of them. Among the offal floor workers we included those engaged in beef heads. The other departments closely associated with the killing halls are the chilling and freezing rooms—the men in these departments handle carcases that have been completely cleaned and that are ready for despatch to butchers' shops and elsewhere.

In addition to the above-mentioned departments there was a group of workers difficult to classify since many of them handled meat or were thought by us to be exposed to some risk of contracting Q fever and yet were not intimately associated with the killing process. We have somewhat arbitrarily grouped these together as 'miscellaneous' meat-workers in Table 2. This group includes brander boys, graders, tripe scalders, boners, tripe chillers and those working with small goods, calf hides, beef hides, and sheep skins and those working in the boiling-down works.

Somewhat separated from the killing halls was the by-products department where all the parts of the animals not used for human consumption are made into various types of animal foodstuffs, fertilizers, glue and tallow—even the horns are used by the manufacturers of so-called 'pearl' buttons.

The transport department consists of those men who receive carcases from the chilling and freezing rooms and distribute them to butchers and other places requiring meat for human consumption. Hence the members of the transport department handle clean meat in the same way as those working in the chilling and freezing rooms.

There is a relatively large group of workers placed under the heading of 'non-meat-workers' in Table 2. This group consists of tradesmen (bricklayers, electricians plumbers, etc.), laundry workers, garage hands, engine-room staff, dining-room staff, and office staff. None of these workers handle meat or its products but some of them may work temporarily among the meat-workers while carrying out repairs and maintenance.

Finally, associated with the abattoirs and working in close conjunction, is a sausage-skin (or casing) factory situated some three miles distant from the abattoirs. The casing company has about 100 employees and some of these actually work full time on the offal floor of the abattoir itself cleaning the intestines of animals which are subsequently transported to the casing factory. The casing company employees working full time at the abattoirs have been included with the offal floor department in Table 2. Two of the men working only in the casing company's factory contracted Q fever.

The killing process is more or less the same for all animals—they are driven from the holding paddocks along narrow passageways, and pigs and cows are stunned electrically or otherwise, while sheep have their throats cut. The pigs have the hair removed from their bodies by immersion in hot water, scraping with mechanical beaters and finally shaving with knives. Subsequently, the animals are suspended by their hind legs from an overhead rail along which they travel in a constant chain among the slaughterers. Each slaughterer performs his own particular task as the animals pass him and thus the skin, head, hooves, and entrails are removed and slid along the floor, which is kept constantly wet, into chutes leading down to the offal floor below. During the killing process inspectors examine each animal, looking closely into them and cutting into selected organs and lymph nodes. While killing is in progress there is a great deal of activity and hoses are constantly playing producing some splashing. The greater part of cows' udders are cut off when the animal is first suspended and are thrown down into the offal floor. A clean dissection of anything that remains is performed when the carcase is finally trimmed, before being transferred to the chilling rooms. The foetuses of cows are sent down a chute to the offal floor while enveloped in foetal membranes. If they are large enough they are dissected on the offal floor and the skin is removed for the manufacture of fine leather. Foetuses of the smaller animals (sheep and pigs) are discarded for processing in the by-products division. The intestines of all animals are cleaned preliminarily on the offal floor and then sent to the casing factory while the inedible remainder of the offal passes to the by-products division. Beef tripes are cleaned and treated and placed in special chilling rooms.

MATERIALS AND METHODS

Detection of Q fever patients

Since about 1950 Q fever became a compensatable disease among meat-workers in South Australia. All meat-workers are, of course, aware of this and all those with suspected Q fever are examined by a doctor appointed by the insurance company responsible for paying compensation. Hence it is unlikely that many cases of Q fever among meat-workers go unrecognized and, furthermore, all suspects have complement-fixation tests carried out in these laboratories. Initially, we depended on the results of the tests on blood samples submitted to us for our information on the occurrence of cases. Subsequently, we received additional confirmation of all cases occurring among meat-workers from the records of the Metropolitan Abattoirs, the casing company and from the doctor appointed by the insurance company. In many instances, we consulted the private doctors and a few of the patients were actually seen by us.

As far as cases occurring in people other than meat-workers were concerned, only one of these appeared. The only method we had of detecting such cases was by the results of complement-fixation tests on blood samples submitted to us by local practitioners.

The definition of our cases of Q fever was that a patient should have had a recent febrile illness compatible with the diagnosis of Q fever together with appropriate

serological findings. Unequivocal serological evidence of recent infection with *C. burneti* was taken to be a fourfold or greater increase in titre in the complement-fixation test or a titre of 1:160 or greater. In our series there was one case which did not meet these criteria—this man, a meat-worker, had a febrile illness which, clinically, was clearly Q fever. A single specimen of serum taken 5 weeks following the onset of his disease fixed complement in a dilution of 1:80 in the presence of *C. burneti* antigen.

Population of serological survey

The serological survey was carried out by calling for volunteers from all departments of the Metropolitan Abattoirs. The abattoirs were visited for the purpose of removing blood from the volunteers on five separate occasions, on 9, 15, 22 and 29 July and on 6 August 1957. Approximately equal numbers of people were bled on each occasion.

Epidemiological information

Information concerning the cases of Q fever occurring at the abattoirs and at the casing factory was obtained largely from the records of those two organizations, as was the epidemiological information concerning our serological survey population. The bulk of this information came from the employment department of the abattoirs. The department in which individuals worked was given as that where they were for most of the time—however, often such people would work for odd days in other departments. The number of years worked in a department was an estimate obtained from the employment department. Such information as we obtained about other groups (such as the movements of tradesmen) were obtained from written records. For all other information written records were consulted and these included those in the Institute of Medical and Veterinary Science and the Royal Adelaide Hospital. Details of the dairy farmer's activities were obtained from personal interviews with him, his doctor, and from the records of the Northfield Infectious Diseases Hospital and the abattoir records.

Treatment of blood samples

The blood samples obtained in the serological survey were conveyed to the laboratory within 6 hr. of having been withdrawn. The serum was either separated on the same day or the whole blood sample was refrigerated at 4–8° C. overnight and the serum removed the following day. All serum samples were kept frozen at −15 to −20° C.

The complement-fixation test

The complement-fixation test was carried out with a yolk-sac antigen using the 'Higgins' strain of *C. burneti*, which was originally isolated by Stokes (1950) from one of the patients in the outbreak in South Australia described by her. The complement-fixation test is described in detail by Beech & Dane (1954–55).

The test itself was carried out by overnight fixation at 4–8° C. and on the

following day incubated with the haemolytic system at 37° C. in a water-bath for 30 min. The tubes were then centrifuged and 50 % haemolysis read as the end-point.

Tests were ordinarily carried out using twofold serum dilutions ranging from 1:5 to 1:160. If fixation occurred in the highest dilution sometimes the test was repeated using twofold serum dilutions starting at 1:160.

Screening tests were carried out on the 516 volunteers in the survey group at a dilution of 1:10. Those sera reacting at this dilution were retested at dilutions of 1:40 and 1:160 and those reacting at the latter dilution were titrated to obtain end-points.

The examination of milk for rickettsiae

Samples of milk examined for rickettsiae were inoculated in 5 ml. quantities intraperitoneally into a guinea-pig. After inoculation the rectal temperatures of the animals were taken twice daily for fourteen days. One month after inoculation, the guinea-pigs were bled from the heart and each serum was tested by the complement-fixation test described above. This test was carried out in parallel with serum obtained from each animal before inoculation.

RESULTS

The temporal distribution of cases

The incidence of all Q fever cases, which totalled 52, is shown in Figs. 2 and 3. There were no deaths. The cases tended to occur in groups. At the beginning there occurred two large groups, the first of these consisted of 14 cases and was spread over 8 weeks from the end of April to the middle of June and the second consisted of 23 cases and was spread over 7 weeks from the end of June to the beginning of August. The remaining four groups were very much smaller, the third group containing eight cases occurred mainly in September, then a single case occurred late in October followed by two groups of three cases, one group occurring in November and the other in December. Before this 1957 outbreak the last case was detected in June 1956 and no more cases occurred during the 6 months following the outbreak.

In the first group, the first 11 cases occurred between 27 April and 29 May. The onset of four cases in this group occurred on 8 May and seven cases occurred within the space of 9 days. The seventh patient in this group was a dairy farmer living a few miles from the abattoirs. He and his brother-in-law visited the abattoirs on 1 April and on 3 April the two of them went into the beef hall and watched the slaughter of some of their animals. The dairy farmer subsequently developed serologically proven Q fever on 8 May—35 days after his last visit to the abattoirs. His brother-in-law did not contract the disease and no antibodies were found in his serum a few months later. Sera from 10 of the remaining animals in the dairy farmer's herd failed to fix complement in the presence of the *C. burneti* antigen. Six pools of milk, each from eight cows comprising samples from his entire herd of 48 animals, failed to yield evidence of infection in guinea-pigs after intraperitoneal inoculation.

Four other patients out of the first 11 were known to be working in the beef

hall when the animals from the dairy herd were killed. Two of these fell ill on the same day as the dairy farmer, 35 days after his last visit, and one of them six days later still. The fourth case, an inspector, contracted Q fever on 28 May—55 days after the dairy farmer's last visit to the abattoirs. Three more cases may have been associated with this group. They contracted the disease 26, 35 and 47 days

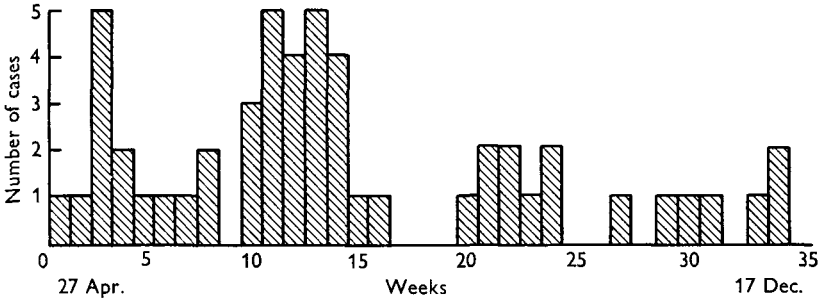

Fig. 2. Weekly incidence of cases of Q fever occurring in Adelaide during the 1957 outbreak.

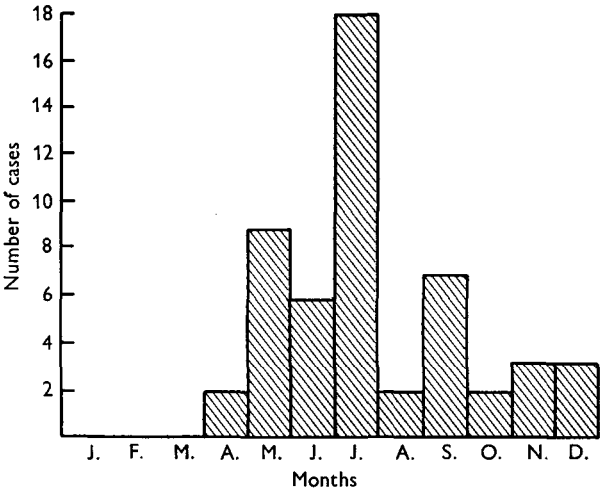

Fig. 3. Monthly incidence of cases of Q fever occurring in Adelaide during the 1957 outbreak.

after the dairy farmer's visit and may have had indirect connexion with his animals. Another case, which occurred 24 days after the dairy farmer's visit, was a bricklayer. His disease was not at first recognized as Q fever until it was realized that his place of work was the abattoirs when blood tests clinched the diagnosis and an examination of his work book showed that he had been working near the beef hall during the relevant period. The remaining two cases of the first 11 had no direct contact with the dairy farmer's animals, but like all employees were exposed to chance and indirect contact.

Thus the first 11 cases could perhaps be regarded as a group associated with the episode of the dairy farmer's visits to see his own cattle slaughtered. The remaining three cases in the first group of 14 (Fig. 2) occurred early in June and provided no

information of exceptional interest except that two were working in the beef hall at the relevant time. The third was working in the by-products division.

The second large group shown in Fig. 2, consisting of 23 cases, started at the end of June and went on throughout July and eventually petered out at the beginning of August covering a total of 7 weeks. The distribution of cases showed two relatively indistinct peaks.

During the several weeks preceding the onset of this disease in this second large group, 15 cases were associated with beef or beef offal at one time or another and occurred throughout the period. Four cases, occurring on 27 June, 1 July, 16 July and 22 July, were associated with mutton either full or part-time.

An inspector, in the second group, was on vacation from 1 July to the 21st of the same month. On 2 August he contracted Q fever. Thus the incubation period of the disease must have been 11 days or less, or 33 days or more. There is no evidence that he could have been infected outside the abattoirs.

The third group was one month later and consisted of eight cases occurring between 6 September and 1 October, a period of 26 days. Four of these were associated with beef, two were inspectors, one worked with 'small goods', and only one worked with mutton. An inspector in this group was on vacation from 2 September until he returned to work on 23 September, and subsequently contracted Q fever on 1 October—an incubation period of 8 days or less, or 30 days or more. There was no evidence that he had contracted the disease elsewhere.

The next case was an isolated one occurring at the end of October. Two weeks later was the first of three cases occurring within 13 days. The last group of three cases started 2 weeks later on 2 December and occurred within the space of 12 days.

Distribution of cases among departments

Table 3 shows the distribution of cases of Q fever according to departments. The department recorded was that in which a man was working at about the time he contracted the disease. With one exception, this designation also coincided with the department in which the man had been working for the greater part of his time during the 2 months preceding the onset of his illness. The one exception (not included in the Table) had been working about half-time in both the beef and mutton departments. In the offal department have been included two cases who worked solely at the casing factory.

It can be seen from Table 3 that the first three departments ('beef', 'inspectors', and 'offal') have a very high attack rate of 15–20 %. The last six departments (from and including 'miscellaneous meat-workers' down to 'non-meat-workers') have an average attack rate of 2 %. The incidence of Q fever in these two groups is obviously significantly different and it can be said the disease was relatively infrequent among those not on the killing floors. On the killing floors, however, although the attack rate is high among inspectors and beef and offal workers, in the mutton hall it (4 %) does not differ significantly from that (2 %) in the non-killing departments, although there is some indication that it may in fact be higher. No cases of Q fever occurred in the pig and calf hall.

Also listed in Table 3 are 'high titre' antibodies detected in the survey. These

are sera reacting with *C. burneti* antigen in a dilution of 1:160 or greater and on the assumption that such sera are indicative of recent infection we may consider them together with the cases to obtain further information about the present epidemic. In the mutton hall there were two high titre sera out of 84 tested as compared with only one out of 472 on the non-killing floors. The most striking feature, however, is the disproportionately high number of high titre sera in the offal department where it comprises 8% of the 59 sera tested. Table 2*a* shows a further breakdown of the offal department according as to whether the workers were confined to beef or mutton and whether they had Q fever, high titres or low titres. The most noticeable feature is that none of those working with 'mutton offal only' showed any evidence of being infected. Otherwise the 'beef only' and 'mixed groups' showed an attack rate of the same order, but the 'mixed group' in

Table 2*a*. *Q fever in Adelaide: distribution of people handling offal with Q fever or antibodies according to whether working with beef or mutton*

Department	No antibody nor illness	Low titre	High titre	Q fever	Total
Beef only	22	12	0	9*	43
Mutton only	11	1	0	0	12
Mixed	3	0	5	5†	13
Total	36	13	5	14‡	68

* This includes two cases occurring among men working at the casing company only.
† Three of these were said to be 'mostly on beef'.
‡ Five of these were also included in the survey.

Table 3. *Q fever in Adelaide: distribution of people with the disease or with antibodies against* Coxiella burneti *in the abattoir departments*

Department	No. tested in survey	Complement-fixation titre (reciprocal, excluding cases)					Q fever cases		No. employed
		< 10	10	40	> 40 < 160	⩾ 160	Bled in survey	Not bled in survey	
Beef	70	46	9	3	1	2	9	5	95
Inspectors	28	17	4	2	0	1	4	3	35
Offal	59	36	7	1	5	5	5	9	90
Mutton	84	71	6	2	2	2	1	5	160
Pig and calf	8	4	1	2	1	0	0	0	24
Miscellaneous meat-workers	37	31	3	0	2	1	0	1	43
By-products	37	35	0	0	2	0	0	1	60
Chillers and freezers	35	33	2	0	0	0	0	1	110
Transport	25	23	2	0	0	0	0	1	70
Markets	55	54	0	0	0	0	1	0	65
Non-meat-workers	78	73	3	0	0	0	2	2	124
Totals	516	423	37	10	13	11	22	28*	876

* Two cases not included: see footnote to Table 2.

https://doi.org/10.1017/S0022172400039255 Published online by Cambridge University Press

addition contained the disproportionately large number of five workers with high titres—three of these worked with mutton and beef, one is not recorded and the other was working with pig offal.

Serological survey

Table 3 contains the detailed results of the serological survey. 22 of those contracting Q fever volunteered to provide blood either before or after their disease. These people and those with high titre sera have been included in the total as negative results when considering the incidence of low titre antibodies. This is based on the assumption that high titres are indicative of recent infection and low titres of more remote infection. Although there may have been individual exceptions to this general rule, it was felt that on the average, the incidence of low titre antibodies reflected the incidence of past infection at the beginning of 1957. It should, however, be noted that 13 out of 22 cases bled in the survey contracted Q fever after their blood sample had been obtained. In no instance were antibodies detected.

Table 4. *Q fever in Adelaide: distribution of people in the killing departments with low titre antibodies against* Coxiella burneti

Department	Low titre*	No. tested	Percentage
Beef	13	70	19
Inspectors	6	28	21
Offal	13	59	22
Mutton	10	84	12
Pig and calf	4	8	50
Totals	46	249	18

* Low titre indicates that serum fixed complement in the presence of *C. burneti* antigen in a dilution of less than 1:160.

Table 5. *Q fever in Adelaide: distribution of people in the non-killing departments with low titre* (< 1:160) *antibodies against* Coxiella burneti

Department	Low titre*	No. tested	Percentage
Miscellaneous meat-workers	5	37	14
By-products	2	37	5
Chillers and freezers	2	35	6
Transport	2	25	8
Markets	0	55	0
Non-meat-workers	3	78	4
Totals	14	267	5

* See footnote to Table 4.

Table 4 shows the incidence of low titre (less than 1:160) sera in the killing departments. If we neglect the pig and calf hall on account of the small number tested, the remainder of the departments have incidences of low titre antibodies which do not differ significantly from one another ($\chi^2_{[3]} = 3{\cdot}52$, $P = 0{\cdot}32$).

An inquiry into the apparently high incidence in the pig and calf hall showed that

https://doi.org/10.1017/S0022172400039255 Published online by Cambridge University Press

out of eight bled in the survey four had worked only in the pig and calf hall. Of these four, three had low titre antibodies and one had been employed 30 years. Of the remaining four workers, two had been employed for more than 30 years and both had been in the mutton hall for some years before going to the pig and calf hall. One of the last two workers had no antibodies and had worked two years in each of the mutton and pig and calf halls. The remaining one had low titre antibodies and had been in the 'boning' department for 18 months before spending 2 years in the pig and calf hall.

Table 6. *Q fever in Adelaide: antibody titres in people having had Q fever one or more years before testing in* 1957

No. of years since disease	No. of cases	Reciprocal of titres
17	1	10*
15	1	10*
9	2	10;* 40
7	2	10; 10
6	3	10; 10; 40
5	2	< 10; 10
4	2	10; > 40 < 160
3	6	< 10; < 10; 10; 40; 40; > 40 < 160
1	1	> 40 < 160
Total	20	

* Only evidence of past Q fever was volunteer's own word.

Table 5 shows the incidence of low titre antibodies in the non-killing departments. There were no low titres detected in the 'markets' and their incidence among the 'non-meat-workers' is low. However, if we combine these two groups and compare them with the other combined non-killing departments, the difference is not statistically significant ($\chi^2_{[1]} = 3{\cdot}64$, $P = 0{\cdot}06$). In any event it is quite clear that the incidence (5 %) of low titre antibodies in the non-killing departments (Table 5) is significantly less than that (18 %) in the killing halls (Table 4).

While conducting the survey it came to our notice that 20 of the volunteers were alleged to have had Q fever in previous years. Most of these were traced in the records of this Institute and many were confirmed by the records of the Metropolitan Abattoirs. In only three instances was a record of Q fever unavailable and the only evidence of its occurrence was the volunteer's own word. However, all three had antibodies and these are shown in Table 6 together with the other results. The results show that antibodies may be retained for 17 years or more or they may disappear altogether within 3 years. Of course, it is possible that, in some people, repeated reinfection maintains antibody levels. Our findings are in general agreement with those of Tonge (1955) and Marmion, Stoker, Walker & Carpenter (1956), who state that the 'half-life' of Q fever antibodies is from 6 to 10 years.

Table 7 shows the results of complement-fixation tests on 101 sera removed at various intervals after an attack of Q fever from the patients described in this

outbreak. As mentioned before, all the patients in this group had a rising titre or one of 1:160 or greater with one exception, namely, a man with a titre of 1:80 and a clinically typical attack of the disease. This shows that in the first week of the disease most sera will contain no antibodies although towards the end of the week a few will have titres of 1:160 or more. In the second week 70% of sera have titres of 1:160 or more and subsequently practically all sera reach this level. It is of interest, however, to see that an occasional serum will show no antibodies for a long time after the onset of the disease. One of the patients in the present outbreak had no antibodies in a serum sample removed 32 days after the onset—subsequently, his antibody titre rose to more than 1:160. Seventeen sera were titrated to an end-point and the highest titre recorded was 1:2560 in three sera. Eight of the sera reached a titre of 1:640. These levels accorded fairly well with the records of this Institute, where the highest titre was 1:10,240.

Table 7. *Q fever in Adelaide: complement-fixation titres at intervals after onset of disease*

Time after onset (weeks)	Reciprocal of titres					
	< 5	5–10	20–40	80	⩾ 160	Total
1st	23	2	1	1	3*	30
2nd	0	1	2	2	11	16
3rd	1	0	1	1	17	20
4th	1	0	0	1	10	12
5th–8th	1	0	0	3	13	17
9th–12th	0	0	0	0	6	6
Total	26	3	4	8	60	101

* All three of these results were obtained on the 7th day.

Table 8. *Q fever in Adelaide: cases occurring in abattoir departments according to length of service*

Department	Length of service (years)		
	0–14	15–29	30+
Beef	7/25 (28)	2/31 (6)	0/14
Inspectors	3/15 (20)	1/11 (9)	0/2
Offal	4/42 (10)	0/13	0/4
Mutton	1/36 (3)	0/42	0/6
Remainder (except pig and calf)	3/186 (2)	0/65	0/16
Total	18/304 (6)	3/162 (2)	0/42

Denominator = number in group.
Numerator = number of cases of Q fever.
The figure in parentheses is the attack rate in per cent.

https://doi.org/10.1017/S0022172400039255 Published online by Cambridge University Press

Length of service and incidence of infection

Table 8 shows the results of an attempt to relate the incidence of Q fever to the length of service. Although the data were originally broken down into yearly groups for the first 4 years and 5-yearly afterwards nothing informative appeared beyond what is shown in the Table. The results show that the attack rate is always higher among those employed for 14 years or less than it is for those employed 15 years or more. This agrees with the findings of Marmion & Harvey (1956) and Marmion & Stoker (1956) that newcomers to endemic areas bear the brunt of Q fever. Among those employed 30 or more years no Q fever occurred.

Table 9. *Q fever in Adelaide: distribution of people with low titre antibodies against* Coxiella burneti *in the abattoir departments according to length of service*

	Length of service (years)		
Department	0–14	15–29	30+
Beef	2/25	6/31	5/14
Inspectors	3/15	2/11	1/2
Offal	9/42	3/13	1/4
Mutton	1/36	9/42	0/6
Pig and calf	4/5	0/0	0/3
Remainder	6/186	8/65	0/16
Total	21/309	28/162	7/45

Denominator = total in group.
Numerator = number with low titre.

Table 9 shows the incidence of low titre antibodies according to the length of service. The individual figures are too small to draw conclusions from, but if we pool those for the killing floors or the killing floors and the remainder, the incidence is higher among those employed for 15 or more years than it is for those employed 14 or fewer years.

Finally, an attempt was made to calculate the risk of being infected with *C. burneti* according to the number of years worked in a particular department. Estimates of the number of years worked in each department were available for those employed for more than one and less than 10 years in the survey group. No figures were available for the inspectors and no cases from the mutton hall fell in this group, otherwise the figures confirmed that there was a greater risk of contracting Q fever on the beef and offal floors than elsewhere.

DISCUSSION

The first point of interest about the outbreak of Q fever described in this paper is the temporal distribution of cases which was spread out over many months—the cases showing a tendency to occur in groups. Exactly the same kind of distribution is described as occurring in the Brisbane, Queensland, Abattoirs (Derrick, 1944). The situation in Australia, however, shows a marked contrast to that in North America where such outbreaks as have been described were explosive,

occurring within a matter of days. In Amarillo, Texas (Topping, Shepard & Irons, 1947) the cases occurred within 21 days, in Chicago, 9 days (Shepard, 1947) and in Princeville, Quebec, 17 days (Pavilanis, Duval, Foley & L'Heureux, 1958). Another point of differentiation is that, in both the Brisbane and the present outbreak in Adelaide described here, the attack rate was low as compared with all three North American outbreaks. These differences are probably a reflexion of the endemicity of Q fever in the two Australian meat-works, whereas it seems probable that the North American outbreaks represent first exposures to the disease as was pointed out in the description of one of them (Topping *et al.* 1947). No deaths occurred in the Adelaide outbreak, nor have any been recorded. Two occurred in the Amarillo outbreak (Topping *et al.* 1947), and Tonge & Derrick (1959) mention four deaths occurring between 1935 and 1947. No deaths occurred in the Chicago (Shepard, 1947) or the Princeville outbreaks (Pavilanis *et al.* 1958).

Our attempts to trace the sources of infection for individual cases was largely unsuccessful. Only in the first group of 11 cases did we find any coherent epidemiological information. In this group the onset of the disease in seven of the cases occurred within 9 days and for four of them it occurred on a single day. It seems unlikely that the dairy farmer's animals were the source of infection since we found no evidence of infection in the remainder of his herd. Furthermore, his wife, children and brother-in-law had not been ill. Neither had any other cases been detected in Adelaide outside the abattoirs which might have been the case had the dairy farmer's herd and milk been infected with *C. burneti*. If our conclusions are correct, the dairy farmer must have been infected while visiting the abattoirs and it follows that, in him, the disease would have had an incubation period of 35 or 37 days. To our knowledge only once has a longer incubation period of 38 days been tentatively suggested (Dekking & Zanen, 1958) although Shepard (1947) records a maximum possible incubation period of 32 days. Also, though Robbins, Gauld & Warner (1946) state that the incubation period has a mean value of 19–20 days with a possible range of 14–26 days in their Table 2 they show a maximum possible incubation period of 34 days. In addition, Derrick (personal communication) has observed an incubation period of 37 days and drew our attention to a man who became ill 60 days after working with mice infected with *C. burneti* (Siegert, Simrock & Stroder, 1950). However, the organism could have persisted in the dust of the room in which the man worked and thus could have infected him later. The man was also responsible for an epidemic in which two patients contracted Q fever, 33 and 35 days respectively after he had left their ward.

From the above it seems reasonable to accept 32 and 30 days as incubation periods for the two inspectors mentioned in the results. (The alternative possibility of unusually short incubation periods of 11 and 9 days should be borne in mind.) It seems to be more than a coincidence that the only three people about whom we have appropriate information, probably have exceptionally long incubation periods. It seems possible that in this outbreak, or in meat-works, or with the particular strain of *C. burneti* involved the incubation periods may be longer than is generally recognized. If this were so it would be of considerable interest as it might enable

us to ascribe to a common source groups of cases which occur over too long a time to achieve this with the prevalently quoted range of incubation period.

At present we have reason to suspect that the opportunity for infection may be short-lived because the killing halls are thoroughly washed down so that infection is unlikely to persist. Yet the cases occur in groups spread out in time with no evidence that the early cases occur on the killing floors and then later ones in, say, the by-products department or the casing factory following the parts of infected animals. We know also that from the results of serological surveys (Stokes, 1953; Dane & Beech, 1954–55) infected animals must be very uncommon. Thus to explain the temporal spread of groups of cases we must assume either that the incubation period has a greater range than is suspected or that the infected animals come in groups.

The distribution of cases according to departments shows that the beef, inspectors and offal departments were the most severely affected and those working on mutton much less so. Such cases as did occur among workers in the mutton hall might have been infected during occasional day's work in the beef hall, during unofficial visits there or from the soiled clothes of their colleagues during the rest periods in the canteen or elsewhere. However, outbreaks do occur in the mutton hall (Dane & Beech, 1954–55). The same workers have demonstrated unequivocally that Q fever occurs in association with sheep in South Australia. In a previous outbreak in the Adelaide abattoirs (Stokes, 1950, 1953) cattle were incriminated. Derrick (1944) considered that cattle were responsible for Q fever in the Brisbane abattoirs. In two of the North American outbreaks in Chicago (Shepard, 1947) and Princeville, Quebec (Pavilanis *et al.* 1958) it was not possible to determine whether the outbreaks were due to cattle or sheep. However, in the Amarillo outbreak (Topping *et al.* 1947) cattle were in all probability the cause.

It seems that both cattle and sheep are responsible for the Adelaide abattoir outbreaks and this is borne out by the incidence of low titre antibodies in the different killing departments not being significantly different from one another.

The contrast between high incidence of infection in the killing and the low incidence in the non-killing departments is quite clear cut and agrees with Derrick (1944). In the Chicago epidemic (Shepard, 1947) and that at Princeville (Pavilanis *et al.* 1958) the outbreaks were confined to those actually handling the meat. In the Amarillo outbreak (Topping *et al.* 1947) the train crew and stock-men were very severely affected. This last contrasts strikingly with the 1957 Adelaide outbreak. Thus it seems that dust plays a negligible part in the spread of Q fever in the Adelaide abattoirs. The most likely explanation is that infection within the abattoir takes place when an infected animal or even an infected parturient or pregnant animal is slaughtered (Luoto & Huebner, 1950; Welsh Lennette, Abinanti & Winn, 1951). The attendant splashing would provide an aerosol sufficient to infect the workers by inhalation as has been shown by Welsh, Lennette, Abinanti & Winn (1958). Even though serological studies have shown that animals are seldom infected this would be in accordance with the relative infrequence of outbreaks and explain the occurrence of cases in groups.

The explanation given above could account for most of the epidemiological

features of Q fever in the Adelaide abattoirs. One would expect a fairly regular occurrence of outbreaks of the disease if cattle from a herd infected with *C. burneti* were ordinarily sent to Adelaide but, over the years it is plain that the incidence of Q fever is irregular. This could be accounted for if infected herds were at such a distance that cattle for slaughter could equally well be sent to Adelaide or Brisbane. If the choice of one or the other place depended on some irregular phenomenon such as the rainfall or the availability of water along the stock routes then cattle in south-western Queensland ordinarily sent to Brisbane might sometimes be sent to Adelaide. This theory is upheld by the fact that Q fever has been found to be endemic in western Queensland (Derrick, Pope & Smith, 1959) in a sheep-raising district lying just to eastward of the cattle region surrounding the tributaries of the Birdsville Track cattle route leading to the railhead for the Adelaide abattoirs. The same authors remark on the dryness of the area in 1957, the year of the Adelaide outbreak, and on the recent increase in the number of cases of Q fever in Queensland.

It would be most satisfactory if Q fever in the Adelaide abattoirs could be linked in some way with an endemic area in western Queensland. It would account for the striking difference between the situation in Adelaide and that in Brisbane—namely, that very few cases of the disease occur outside the abattoirs in Adelaide whereas in Brisbane, even before 1958, Q fever was known to occur commonly outside abattoirs. It must be borne in mind, however, that Dane & Beech (1954–55) discovered Q fever in country districts of South Australia and it may be that the disease goes unrecognized, although such outbreaks may be centrifugally spread from western Queensland.

SUMMARY

This paper consists of an epidemiological study of 52 cases of Q fever occurring in metropolitan Adelaide in 1957 and also a description of the results of a survey of 516 sera obtained from abattoir workers.

The only case occurring outside the abattoirs was a dairy farmer who probably became infected while visiting the abattoirs. If this were so the incubation period (35 days) of his disease would have been exceptionally long.

The general features of the outbreak, which lasted several months, differed from those on the North American continent in that the latter occurred explosively within a few days with very high attack rates. The situation in the Adelaide abattoirs is similar to that in Brisbane, where the disease appears to be endemic. However, unlike in Adelaide, cases are commonly recognized outside the abattoirs in Brisbane.

In the abattoirs the disease affected mainly inspectors, those working on killing beef, and those working on offal. Mutton workers were not so severely affected. However, all these groups had similar incidences of low titre antibodies suggesting that in the past Q fever spread equally in all killing departments. In departments not directly associated with slaughtering the incidence both of cases in 1957 and low titre antibodies was relatively small.

It was suggested that the epidemiological features of Q fever in Adelaide could

be explained by the irregular appearance of animals from infected herds situated perhaps in Queensland—a known endemic area. Perhaps the appearance of such animals in the Adelaide abattoirs might be governed by meteorological conditions such that they were prevented from going to the ordinarily most convenient slaughterhouse.

Our thanks are due first to the Metropolitan and Export Abattoirs Board, South Australia, who willingly provided information, gave us permission to investigate the outbreak and to publish this paper.

Secondly, we are much indebted to many at the Metropolitan Abattoirs, especially Messrs Redward and Kelsey and those who gave blood for our survey. We are similarly indebted to the Australian Casing Company.

We are also indebted to Dr McQueen of the South Australian Department of Health for help and encouragement in initiating this study and to many local medical practitioners, particularly Dr Miller, for allowing us to see and obtain information about their patients.

Finally, we are indebted for advice to Drs E. H. Derrick and B. P. Marmion who are not to be held responsible for the contents of this paper.

REFERENCES

Ann. Rep. (1953–54). *Inst. Med. and Vet. Sci., Adelaide*, S. Australia, p. 45.

Beech, Margaret D. & Dane, D. M. S. (1954–55). Virus antigens. *Ann. Rep. Inst. Med. and Vet. Sci.* p. 40.

Dane, D. M. S. & Beech, Margaret D. (1954–55). Q fever. *Ann. Rep. Inst. Med. and Vet. Sci.* p. 37.

Dekking, F. & Zanen, H. C. (1958). Q fever in the Netherlands. *Topical and Geographical Medicine*, **10**, 157.

Derrick, E. H. (1937). Q fever, a new fever entity. *Med. J. Aust.* no. 2, 281.

Derrick, E. H. (1944) The epidemiology of Q fever. *J. Hyg., Camb.*, **43**, 357.

Derrick, E. H., Pope, J. H. & Smith, D. J. W. (1959). Outbreaks of Q fever in Queensland associated with sheep. *Med. J. Aust.* no. 1, 585.

Luoto, L. & Huebner, R. J. (1950). Q fever studies in California. IX. Isolation of Q fever organisms from parturient placentas of naturally infected dairy cows. *U.S. Public Hlth Rep.* **65**, 541.

Marmion, B. P. & Harvey, M. S. (1956). The varying epidemiology of Q fever in the south-east region of Great Britain. I. In an urban area. *J. Hyg., Camb.*, **54**, 533.

Marmion, B. P. & Stoker, M. G. P. (1956). The varying epidemiology of Q fever in the south-east region of Great Britain. II. In two rural areas. *J. Hyg., Camb.*, **54**, 547.

Marmion, B. P., Stoker, M. G. P., Walker, C. B. V. & Carpenter, R. G. (1956). Q fever in Great Britain. Epidemiological information from a serological survey of healthy adults in East Anglia. *J. Hyg., Camb.*, **54**, 118.

Pavilanis, V., Duval, L., Foley, A. R. & L'Heureux, M. (1958). An epidemic of Q fever at Princeville, Quebec. *Canad. J. publ. Hlth*, **48**, 520.

Robbins, F. C., Gauld, R. L. & Warner, F. B. (1946). Q fever in the Mediterranean area: report of its occurrence in allied troops. II. Epidemiology. *Amer. J. Hyg.* **44**, 23.

Shepard, C. C. (1947). An outbreak of Q fever in a Chicago packing house. *Amer. J. Hyg.* **46**, 185.

Siegert, R., Simrock, W. & Stroder, U. (1950). Über einen epidemischen Ausbruch von Q-fieber in einem Krankenhaus. *Z. Tropenmed. Parasit.* **2**, 1.

Stoker, M. G. P. & Marmion, B. P. (1955). The spread of Q fever from animals to man. *Bull. World Hlth Org.* **13**, 781.

STOKES, JOAN (1950). Q fever in South Australia. I. Isolation of causative organism from four cases and its identification as *Rickettsia Burneti*. *Med. J. Aust.* no. 2, 745.

STOKES, JOAN (1953). Q fever in South Australia. II. Surveys of human and bovine sera. *Med. J. Aust.* no. 2, 778.

TONGE, J. I. (1955). Q fever. *Ann. Rep. Hlth and Med. Services*, Queensland, 1954–55, p. 90.

TONGE, J. I. & DERRICK, E. H. (1959). A fatal case of Q fever associated with hepatic necrosis. *Med. J. Aust.* no. 1, 594.

TOPPING, N. H., SHEPARD, C. C. & IRONS, J. V. (1947). Q fever in the United States. I. Epidemiological studies of an outbreak among stockhandlers and slaughterhouse workers. *J. Amer. med. Ass.* **133**, 813.

WELSH, H. H., LENNETTE, E. H., ABINANTI, F. R. & WINN, J. F. (1951). Q fever in California. IV. Occurrence of *Coxiella Burneti* in the placenta of naturally infected sheep. *U.S. Publ. Hlth Rep.* **66**, 1473.

WELSH, H. H., LENNETTE, E. H., ABINANTI, F. R. & WINN, J. F. (1958). Air-borne transmission of Q fever: the role of parturition in the generation of infective aerosols. *Ann. N.Y. Acad. Sci.* **70**, 528.