Original article

Atypical outbreak of Q fever affecting low-risk residents of a remote rural town in New South Wales

Brett N Archer, Cathie Hallahan, Priscilla Stanley, Kathy Seward, Margaret Lesjak, Kirsty Hope, Anthony Brown

Abstract

We investigated an outbreak of Q fever in a remote rural town in New South Wales, Australia. Cases identified through active and passive case finding activities, and retrospective laboratory record review were interviewed using a standard questionnaire. Two sets of case–case analyses were completed to generate hypotheses regarding clinical, epidemiological and exposure risk factors associated with infection during the outbreak. Laboratory-confirmed outbreak cases (n=14) were compared with an excluded case group (n=16) and a group of historic Q fever cases from the region (n=106). In comparison with the historic case group, outbreak cases were significantly more likely to be female (43% vs. 18% males, $P = 0.04$) and identify as Aboriginal (29% vs. 7% non-Aboriginal, $P = 0.03$). Similarly, very few cases worked in high-risk occupations (21% vs. 84%, $P < 0.01$). Most outbreak cases (64%) reported no high-risk exposure activities in the month prior to onset. In comparison with the excluded case group, a significantly increased proportion of outbreak cases had contact with dogs (100% vs. 63%, $P = 0.02$) or sighted kangaroos on their residential property (100% vs. 60%, $P = 0.02$). High rates of tick exposure (92%) were also reported, although this was not significantly different from the excluded case group. While a source of this outbreak could not be confirmed, our findings suggest infections likely occurred via inhalation of aerosols or dust contaminated by *Coxiella burnetii,* dispersed through the town from either an unidentified animal facility or from excreta of native wildlife or feral animals. Alternatively transmission may have occurred via companion animals or tick vectors. *Commun Dis Intell* 2017;41(2):E125–E133.

Keywords: Q fever, *Coxiella burnetii,* disease outbreaks, epidemiology

Introduction

Q fever is the most commonly notified zoonosis in Australia.[1] It is caused by an obligate intracellular bacterium, *Coxiella burnetii,* which is environmentally stable and found widely across the animal kingdom.[2–4] Domestic and feral ruminants, including cattle, goats and sheep, are considered the main sources of human infections. However, a wide range of other animals including native and introduced wildlife, companion animals, and various species of ticks, birds and rodents have all been implicated in human infections.[3,5] Infected animals shed *C. burnetii* in placental tissues, birth fluids, milk, urine and faeces.[4] Transmission typically occurs via airborne dissemination of these materials, or through direct contact with infected animals or other contaminated materials (such as wool, straw or clothing).[4,6] As such, workers in animal-related industries are at increased risk of infection, and vaccination of 'at risk' individuals is routinely recommended.[7]

Disease in humans is challenging to diagnose, especially outside traditional risk settings. Subclinical seroconversion occurs in up to 60% of infected individuals.[8] In the remainder, acute Q fever usually manifests as a severe, 'influenza-like' illness lasting 2–6 weeks; although, presentations such as hepatitis or pneumonia are common, particularly in some parts of the world. Neurological manifestations, myocarditis or pericarditis, may occur in some cases, and pregnant women are at risk of various obstetric complications. A protracted chronic infection may develop in 10%–30% of acute cases, presenting as endocarditis, recrudescent granulomatous lesions or post-Q fever fatigue syndrome. Persons who are immunosuppressed, those with a pre-existing heart valve defect, and pregnant women are at highest risk for chronic infections.[4]

In February 2015, an astute infectious disease physician notified public health authorities of an increase in patients presenting with a febrile illness requiring hospitalisation, including 2 cases of Q fever, in the town of Lightning Ridge, New South Wales. An investigation was conducted with the aim of characterising the epidemiological and clinical profile of cases and identifying the source of infections to guide implementation of control measures.

Methods

Setting

Lightning Ridge is a remote rural town in north-western New South Wales within the Walgett Shire Local Government Area (LGA), near the southern border of Queensland. The town has a culturally-diverse population of approximately 2,400 people (2014 estimate), of which 29.7% are aboriginal people (census 2011 LGA estimate).[9] The area is classified as remote (ASGC-RA4), is semi-arid with long-term annual mean rainfalls of <470 mm, and is subject to regular droughts.[10] The town's industries include tourism, agriculture and opal-mining, with some 1,900 informal or formal mining camps located on opal mining fields in the vicinity of Lightning Ridge and Grawin/Glengarry areas.[11] Local agriculture is predominantly sheep grazing for wool and meat.[9]

Case finding and investigations

Case finding activities were both passive and active. Routine (passive) notifications were sourced from the NSW Notifiable Diseases Information Management System (NCIMS). Between 20 February and 31 May 2015, active surveillance for possible cases was undertaken through the Lightning Ridge Health Service, the sole local general practice and the local ambulance service, and by means of an alert to general practitioners in surrounding towns. Healthcare professionals in the area were asked to maintain a high index of suspicion for possible cases, undertake polymerase chain reaction (PCR) and serological testing for Q fever, and commence empiric treatment. A retrospective review was also conducted of all Q fever test requests received from Lightning Ridge and surrounding areas by the Centre for Infectious Diseases and Microbiology Laboratory Services, Institute for Clinical Pathology and Medical Research, Pathology West, Westmead, which is the primary reference laboratory for the region. Public health authorities in neighbouring jurisdictions were alerted to the outbreak and asked to report cases who travelled to the region.

A possible case was defined as any person with onset of an acute febrile illness, (which may include a temperature >38°C, severe headache, extreme fatigue, sweating/chills, myalgia/arthralgia, and/or malaise), from 1 December 2014 to 31 May 2015, who, in the 1 month prior illness onset, was resident in the vicinity of Lighting Ridge (±40 km), had a history of travel to the area, or had direct contact with livestock or wildlife from the area.

A confirmed case was defined as a possible case who met the Communicable Diseases Network Australia definition (i.e. clinically compatible illness plus laboratory-suggestive evidence, or laboratory-definitive evidence).[12] Cases with only laboratory-suggestive evidence (i.e. single detection of specific IgM), or a single negative serology test, were actively followed-up to obtain convalescent specimens to provide laboratory-definitive evidence of acute infection (i.e. demonstrated seroconversion), or exclude the case. Cases were excluded if they had consistently negative paired sera samples, had a non-compatible illness or alternate diagnosis with at least 1 negative serology test, evidence of being vaccinated against Q fever, or evidence of a previous diagnosis of Q fever or chronic/persistent infection.

Local public health unit staff interviewed all possible and confirmed outbreak cases by phone using a standardised questionnaire. Walgett Shire Council Rangers, North West Local Land Services, local veterinarians and the Department of Primary Industries were additionally consulted regarding recent animal stocking and transport activities.

A site visit to Lightning Ridge was undertaken to review environmental conditions during the outbreak, engage local stakeholders and identify potential sources of infection.

Hypotheses-generating analytic studies

Two sets of nested, case–case analyses were completed to generate hypotheses with respect to clinical, epidemiological and exposure risk factors associated with infection during the outbreak. In the first, outbreak cases were compared to an excluded case group, which was defined as all possible case notifications that tested negative for Q fever in consecutive serum samples, had no evidence of being vaccinated or laboratory evidence of past infection (i.e. otherwise susceptible to *C. burnetii* infection), and had completed an interview.

In the second, outbreak cases were compared with an historic profile of Q fever notifications (as at 25 June 2015) with a reported date of illness onset prior to the outbreak during 2010–2014 and usual place of residence within Western New South Wales and Far West Local Health Districts (LHDs) (hereafter the historic case group). Notification records were extracted NCIMS, which included a basic case history collected by local public health units as part of routine case investigations and captured within standardised, electronic surveillance templates.

Statistical analyses

Odds ratios (OR) and corresponding 95% confidence intervals (95% CI) were calculated to measure associations for categorical variables. Fisher's Exact Test was performed where the expected cell

frequency in a contingency table was ≤5. As age demonstrated significant deviations from normality using the Kolmogorov-Smirnov test, the non-parametric Mann-Whitney U Test was performed to measure significant differences between groups. The alpha level was set at 0.05 for all analyses. Year-specific incidence (notification) rates or annual average notification rates were calculated using annual mid-year population projections published by the NSW Ministry of Health.[13]

Environmental investigations

Data on daily and average monthly rainfall, and wind speed, for Lightning Ridge and Walgett for December 2014 to February 2015 were sourced from the Australian Government Bureau of Meteorology.[10]

Results

Walgett Shire LGA has historically experienced the highest annual incidence of Q fever among Far West and Western New South Wales LHDs, with an average annual incidence of 47 cases per 100,000 population in 2010–2014. In comparison, rates ranged from 0–40 cases per 100,000 over the same period within other LGAs in these LHDs. Between 1 December 2014 and 31 May 2015, the outbreak investigation revealed 44 possible cases in and around Lightning Ridge, of which 14 cases met the confirmed case definition: a diagnosis of acute Q fever infection was excluded in the remainder. These 14 confirmed outbreak cases represented a significant increase in disease incidence for the LGA to 165 per 100,000 from baseline ($P = 0.001$). In case–case analyses, confirmed outbreak cases were compared against 16 individuals and 106 historic Q fever cases in the excluded case group and historic case group, respectively.

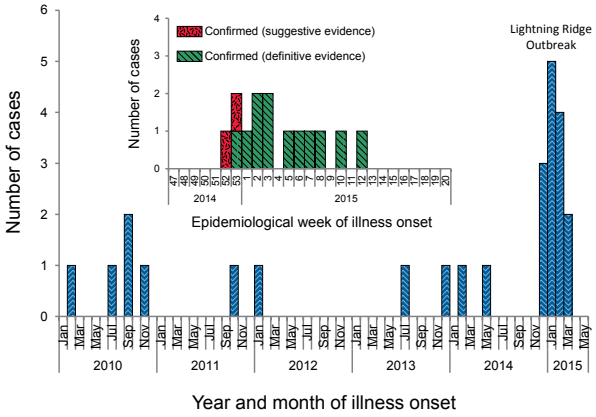
Demographics

The majority of confirmed outbreak cases were aged 40 years or over (79%), and were statistically similar in age to both the excluded case group and historic case groups (Table 1). A significantly larger proportion of outbreak cases were, however, female (43%) or identified as Aboriginal (29%) when compared with the historic Q fever cases (18% female, 7% Aboriginal people).

Clinical presentation and laboratory confirmation

Symptom onset dates for the 14 confirmed outbreak cases were clustered over a period of 12 weeks between 22 December 2014 and 26 March 2015 (Figure). Most confirmed outbreak cases reported symptoms typical of Q fever, including: high fever, rigors with profuse sweating, severe headache, malaise and extreme fatigue lasting several weeks (Table 1). With the exception of 1 case (for whom liver function tests were only performed post-recovery), all cases had abnormally elevated transaminases. Nine cases (64%) required hospitalisation; however, there were no reports of complications or deaths. When compared with the excluded case group, presenting with headache, malaise, fever, rigors, elevated transaminases and being hospitalised were all predictive of having a laboratory confirmed diagnosis.

Figure: Number of confirmed Q fever notifications, Walgett Shire Local Government Area, 1 January 2010 to 31 May 2015, by month, year and epidemiological week of illness onset



Of the 14 confirmed outbreak cases, 12 (86%) had laboratory-definitive evidence of *C. burnetii* infection: 1 by PCR only, 1 by PCR and demonstrated seroconversion, and 10 by seroconversion only. Two confirmed outbreak cases had a clinically compatible illness and laboratory-suggestive evidence of infection (detection of specific IgM in single serum sample), but did not submit convalescent samples. Of the 11 cases that demonstrated seroconversion, 6 (55%) had an initial, acute serum sample that was negative on serology tests performed, IgM negative by enzyme immunoassays, or Phase II antibody titre of less than 4 by compliment fixation testing or immunofluorescence assays.

Exposure history

The majority (79%) of confirmed outbreak cases were retired, unemployed, or worked in town-based, non-animal related occupations (Table 2). Outbreak cases were significantly less likely to work in a high-risk occupation than the historic case group (OR: 0.05, 95% CI: 0.01–0.22, $P < 0.01$), while being statistically similar to the excluded case group (OR: 1.6, 95% CI: 0.02–11.7, $P = 1.0$).

Table 1: Demographic and clinical characteristics of confirmed Q fever cases notified during an outbreak in Lightning Ridge, New South Wales compared with historic case and excluded case groups

	Outbreak cases (22 Dec 2014–26 Mar 2015)		Historic cases (1 Jan 2010 – 30 Nov 2015)		Excluded cases (3 Jan 2015–4 Mar 2015)		Outbreak vs historic cases		Outbreak vs excluded cases	
Demographics										
	Years*	IQR	Years	IQR	Years	IQR	*P* value		*P* value	
Median age	57.5	40	36.9	27	57.5	40	0.09		0.78	
	n/N*	%	n/N*	%	n/N*	%	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Female	6/14	43	19/106	18	7/16	44	3.4 (1.1–11.1)	0.04†	1.0 (0.2–4.1)	0.75
Aboriginal	4/14	29	5/70	7	2/15	13	5.2 (1.2–22.7)	0.03†	2.6 (0.4–17.2)	0.39
Clinical presentation										
Headache	13/13	100	–		9/16	56	–	–	Inf.	<0.01†
Malaise	14/14	100	–		8/16	50	–	–	Inf.	<0.01†
Lethargy	14/14	100	–		14/16	88	–	–	Inf.	0.49
Fever	13/14	93	–		7/14	50	–	–	13.0 (1.3–128.1)	0.03†
Rigors (with sweating)	13/14	93	–		7/15	47	–	–	14.9 (1.5–144.2)	0.01†
Elevated transaminases	13/14	93	–		2/9	22	–	–	45.5 (3.5–594.7)	<0.01†
Hospitalised	9/14	64	–		2/16	13	–	–	12.6 (2.0–79.4)	<0.01†

IQR: Interquartile range.

OR (95% CI): Odds ratio and corresponding 95% confidence interval.

Inf.: Odds ratio approaches infinity, cannot be calculated.

* Denominators exclude cases with missing data.

† Statistically significant difference between groups at α=0.05.

– Not applicable, or data not available/unreliably reported.

Table 2: Occupations of confirmed Q fever cases notified during an outbreak in Lightning Ridge, New South Wales compared with historic case and excluded case groups

Occupation in the month prior to illness onset*	Outbreak cases (22 Dec 2014–26 Mar 2015)		Historic cases (1 Jan 2010–30 Nov 2015)		Excluded cases (3 Jan 2015–4 Mar 2015)	
	n	%	n	%	n	%
% High-risk occupation	3	21	53†	84	2	14
Farmer or resident on farm	2	14	38	60	1	7
Shearer or wool classer	1	7	1	2	0	0
Stockyard worker or stock transporter	0	0	7	11	0	0
Abattoir and other meat industry worker	0	0	5	8	0	0
Veterinary or wildlife worker	0	0	2	3	1	7
Low-risk (non-animal related) occupation	11	79	10	16	12	86
Retired or unemployed	5	36	0	0	6	43
Construction, maintenance, mining or similar	3	21	0	0	2	14
Office, school worker or similar	3	21	0	0	4	29
Other (or undefined) low-risk occupation	0	0	10	16	0	0
Total	14	100	63	100	14	100

* Occupation not reported (or age <16 years) for n=43 historic cases and n=2 excluded cases.

† Statistically significant difference in comparison WITH the outbreak group at α=0.05.

Five confirmed outbreak cases (36%) reported participating in one or more high-risk activities including, slaughtering, shooting, transporting, and/or shearing livestock or feral animals (Table 3). For the remaining 64% of cases, interviews did not identify any apparent high-risk exposure activities. In comparison with the excluded case group, however, a significantly increased proportion of confirmed outbreak cases reported owning or having direct contact with dogs (100% of cases vs. 63% of excluded cases, $P = 0.02$), or sighting kangaroos on their residential property (100% of cases vs. 60%, $P = 0.02$). Most confirmed outbreak cases additionally reported being exposed to ticks (92%) with many noting tick bites (60%); however, statistically similar rates of tick exposure were observed in the excluded case group.

Environmental investigations

Investigations by North West Local Land Services, the Department of Primary Industries and local public health units were unable to identify any specific animal intensive enterprises, stocking, transport, birthing, animal congregations such as shearing or similar events occurring in the area within the month before the outbreak that could be considered a possible point source of infection. Lightning Ridge was not considered a regular route or stop for livestock transport trucks, and cases did not reside along major roads.

Reports of a notable increase in kangaroo activity in and around town were supported by sightings of a large number of kangaroos on residential properties during site visits. Reports of increased tick activity and exposure by cases were supported by observations by local veterinarians.

Data from the Bureau of Meteorology confirmed a drought in Lightning Ridge in the months preceding the outbreak. In 2014, the area received a total of 256 mm of rain compared with an annual mean of 468 mm (measurements available since 1997). A total of 8 mm and 25 mm of rain fell in October and November respectively. This was followed by a slight increase to 49 mm in December 2014 (the highest for the year) amidst average daily temperatures of 36°C (max 41°C). Drought conditions persisted during January to March 2015, with just 4–11 mm of rain falling each month. The town itself has artesian water available and so presents as a green oasis for local wildlife. Although wind gust data were not available for Lightning Ridge, Walgett Airport (approximately 67 km away) reported gusts of up to 65–83 km/h during December 2014 to February 2015, which supported anecdotal reports of dust storms occurring in Lightning Ridge in the month preceding the outbreak.

Table 3: Exposure history of confirmed Q fever cases notified during an outbreak in Lightning Ridge, New South Wales compared with excluded case group

Exposures in the month prior to illness onset*	Outbreak cases (22 Dec 2014–26 Mar 2015)		Excluded cases (1 Jan 2010–30 Nov 2015)		OR (95% CI)	*P* value
	n/N	%	n/N	%		
Kangaroos on residential property	14/14	100	9/15	60	Inf.	0.02†
Direct contact with dogs	14/14	100	10/16	63	Inf.	0.02†
Direct contact with ticks	12/13	92	12/15	80	3.0 (0.3–33.1)	0.60
Tick bites	6/10	60	8/13	62	0.9 (0.2–5.1)	1.00
Travel	6/14	43	4/15	27	2.1 (0.4–9.8)	0.45
Direct contact with livestock or wildlife	5/14	36	2/15	13	3.6 (0.6–22.9)	0.21
Sheep	4/14	29	1/15	7	5.6 (0.5–57.9)	0.17
Cattle	2/14	14	0/15	0	Inf.	0.22
Goats	1/14	7	0/15	0	Inf.	0.48
Pigs	1/14	7	0/15	0	Inf.	0.48
Feral pigs	2/14	14	1/15	7	2.3 (0.2–29.0)	0.60
Feral goats	2/14	14	0/15	0	Inf.	0.22
Livestock transport	2/14	14	0/15	0	Inf.	0.22
Slaughtering	2/14	14	1/15	7	2.3 (0.2–29.0)	0.60
Shearing	1/14	7	1/15	7	1.1 (0.1–19.0)	1.00
Shooting	1/14	7	2/15	13	0.5 (0.0–6.2)	1.00
Animal birthing	0/14	0	1/15	7	–	1.00
Other veterinary practices	0/14	0	0/15	0	–	1.00
Visited meat processors, zoo or saleyard	0/14	0	0/16	0	–	1.00
Wool classing	0/14	0	0/16	0	–	1.00

OR (95% CI): Odds ratio and corresponding 95% confidence interval.

Inf.: Odds ratio approaches infinity, cannot be calculated.

* Exposure events are not mutually exclusive and denominators exclude cases with missing data.

† Statistically significant difference between groups at α=0.05.

– Not applicable, or data not available/unreliably reported.

Discussion

We observed an unusual outbreak of Q fever affecting residents of a small mining town in Western New South Wales. Cases had an epidemiological and risk profile that varied from the historic profile of Q fever notifications from the region. Outbreak cases were significantly more likely to be female and identify as Aboriginal. Moreover, the majority of outbreak cases were residents in town, did not work in a high-risk occupation, and did not participate in any high-risk activities prior to their illness onset.

We additionally observed that symptoms typical of Q fever (namely headache, malaise, fever, rigors) and having elevated transaminases were predictive of infection when compared with laboratory-excluded cases. The majority of cases (71%) were only confirmed through testing of paired acute and convalescent serum specimens, highlighting the critical need for clinical and public health authorities to follow up patients to collect repeat serum, even if they have since recovered. Moreover, initial serology tests were negative in 60% of seroconverted cases, suggesting a review of laboratory records to identify cases without convalescent serology should be routinely considered as part of Q fever outbreak investigations.

The epidemiological profile and lack of definitive exposures suggests a number of hypotheses about the sources of infection in this outbreak. Most plausible is the inhalation of aerosols or dust contaminated by *C. burnetii,* dispersed through the town either from unidentified animal facilities or from excreta of native wildlife or feral animals. This hypothesis is supported by case–case analyses, which suggest an association between kangaroo activity on residential properties prior to onset

and laboratory-confirmed infection. Moreover, the observed dry conditions and dust storms, and activities which may disturb animal faeces, may have played a role in facilitating aerosol transmission. Indeed, studies elsewhere have demonstrated that while infection in individuals without apparent occupational or incidental exposures are relatively infrequent,[14] high seroprevalence rates exist in putatively low-risk communities,[15] and the largest Q fever outbreak to date was attributed to community-wide dispersal of *C. burnetii* up to 5 km from source farms.[16] Kangaroos have been shown to carry and transmit Q fever,[17,18] and a recent New South Wales case series postulated kangaroo faeces may pose a risk (especially if aerosolised by wind or lawn mowing) with 7% of patients recalling macropod contact.[19] Studies have demonstrated the infectious dose of *C. burnetii* may be as little as one aerosolised rickettsia,[20] suggesting even low levels of environmental contamination may cause an outbreak.

Transmission via tick vectors offers a strong alternate hypothesis. This is supported by the findings of high rates of exposure to ticks. Moreover, shortly after the outbreak, we were notified of a confirmed case of Q fever in a Victorian woman who camped in Lighting Ridge in the 2 weeks prior to her illness onset, receiving multiple tick bites, with no other discernible high risk exposures. *C. burnetii* was detected by PCR in multiple ticks collected from the case's body (personal communication: Dr Andrew Fuller, Infectious Diseases Physician, The Alfred Hospital). Numerous field studies show that many macropod-associated tick species (including human biting ticks) carry *C. burnetii*.[5,19] While it is largely accepted ticks play a role in transmission between wild fauna and domestic ruminants, the vector capacity of ticks to transmit *C. burnetii* to humans has been contested and such cases remain infrequently reported.[4,5] Experimental systems, however, suggest a substantial risk posed by tick excreta, with potential human infections through inhalation (e.g. during removal and disposal of ticks, or aerosol-generating procedures such as shearing), direct contact (e.g. crushing ticks with bare hands), or tick bites.[4,5]

Transmission via companion animals offers a third hypothesis. Transmission from pets has been implicated in past outbreaks; however, this was restricted to individuals exposed during aerosol generating veterinary procedures.[3,4,21]

Our findings are subject to a number of limitations. Firstly, case identification was dependent on presentation to healthcare and clinical suspicion of infection prompting laboratory testing. Studies elsewhere demonstrate each Q fever notification corresponds to up to 12 infections in the community,[22] suggesting many asymptomatic and subclinical infections were missed. Secondly, the question must be asked if the detected increase may be attributed to active case finding activities alone. While it is likely that Q fever is underreported in routine surveillance data, the majority of confirmed outbreak cases were reported following hospital admission prior to the period of active surveillance, and no new cases were detected in the 2 months following the outbreak despite ongoing surveillance; supporting the existence of a true outbreak. Thirdly, our study lacked a robust comparison group, which may have resulted in the introduction of selection biases. Although the excluded case group were demonstrated by laboratory tests to be susceptible to, yet remained uninfected by *C. burnetii*, analyses were restricted to a subset of this group with completed interviews, and these individuals were symptomatic of another illness requiring healthcare and likely affecting their activities. Therefore, this comparison group may not have had the same distribution (risk) of exposures as cases. Fourthly, historic notification data may be skewed towards certain demographic groups and persons with high risk exposures, and characteristics such as Aboriginal status may be underreported. Aboriginal status and occupation were not available for 41% and 34% of historic notifications, respectively. Therefore, the observed risk difference in demographic and occupational profile of cases in comparison to historic trends may be subject to measurement bias and over exaggerated here. More robust epidemiological studies, ideally including a seroprevalence component, are therefore needed to adequately test the hypotheses proposed here.

The National Q Fever Vaccination Program was successful in reducing the incidence of disease in high-risk settings such as abbatoirs.[23] While the majority of cases notified in Far West and Western New South Wales LHDs continue to have occupational exposures, and could be prevented through current vaccination recommendations, this outbreak represents a deviation from the norm. The documentation of infections in persons who are typically at low-risk highlights that Q fever should not be excluded from the differential diagnosis based on risk history alone, especially in regional and remote settings, or communities neighbouring animal facilities. With the decline in incidence across the State, there is an increased capacity to detect and investigate infections in low-risk populations. Sporadic cases and outbreaks in rural/regional towns, similar to that investigated here, may become more commonplace. It is critical that such events be thoroughly investigated to better inform the transmission and source dynamics of Q fever, and build an evidence-base for future interventions.

Acknowledgements

This work was completed while Brett Archer was employed as a trainee on the Public Health Training Program funded by the NSW Ministry of Health. He undertook this work whilst based at the Western New South Wales Local Health District.

We would like to acknowledge and sincerely thank James Branley (Western New South Wales and Nepean Blue Mountains Local Health Districts) who initially detected and reported the cluster.

For their participants in the outbreak advisory group, we would like to thank: Vicky Sheppeard and Sheena Adamson (Communicable Diseases Branch, Health Protection NSW); Peter Massey and Tony Merritt (Hunter New England Local Health District); Sarah Britton and Diane Ryan (Department of Primary Industries) and Shaun Slattery and Toni Jericho (North West Local Land Services).

For their valued contributions, we would also like to thank: Therese Jones, Julie Tall, David Ferrall, Carol George, Sally Doohan, Susan Turcato, Jane Connolly and Anita Hoysted (Population Health, Far West and Western NSW Local Health Districts); Dhara Patel (Pathology West, Institute for Clinical Pathology and Medical Research, Westmead); Georgina Luscombe (School of Rural Health, University of Sydney); Nectarios Rose (Health Protection NSW) and Greg Curran (Department of Primary Industries); Andrew Fuller (The Alfred Hospital); and Lucinda Franklin and Shaun Coutts (Department of Health and Human Services, Victoria).

Author details

Mr Brett N Archer[1]
Ms Cathie Hallahan[2]
Ms Priscilla Stanley[2]
Ms Kathy Seward[2]
Dr Margaret Lesjak[2]
Dr Kirsty Hope[3]
Assoc Prof Anthony Brown[4]

1. Public Health Training Program, NSW Ministry of Health, Sydney, New South Wales
2. Population Health, Far West and Western NSW Local Health Districts, Broken Hill, Bathurst and Dubbo, New South Wales
3. Communicable Diseases Branch, Health Protection NSW, Sydney, New South Wales
4. School of Rural Health, University of Sydney, Dubbo, New South Wales

Corresponding author: Mr Brett Archer, Public Health Training Program, NSW Ministry of Health, Sydney, NSW. Telephone: Email: brettarcher1@gmail.com

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