SHORT REPORT
Acute Q fever in northern Queensland: variation in incidence related to rainfall and geographical location

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SUMMARY

The aims of this study were to define the basic epidemiology of serologically confirmed acute Q fever in patients tested via the Townsville Hospital laboratory from 2000 to 2010 and to determine the impact of geographical location and seasonality on the incidence of acute cases in the Townsville region. Seven Statistical Local Areas (SLA) were identified as having an incidence higher than the average Queensland incidence over the study period. The SLA with the highest incidence was Woodstock-Ross with 24·9 cases/100 000. A clear seasonal peak was found, with the greatest number of cases observed in May, 3 months following the peak in rainfall in February. We hypothesize that an increase in wildlife numbers and drier conditions seen immediately following the wet season is the reason for the seasonal peak of human acute Q fever cases in Townsville.

Key words: Epidemiology, Q fever.

Q fever is a zoonosis caused by the obligate intracellular bacterium *Coxiella burnetii*. The northeastern state of Queensland has the highest rate of Q fever notifications in Australia, with 3·5/100 000 population in 2011 compared to the national average of 1·4 cases/100 000 [1]. In 2011, more than half of all Q fever notifications in Australia were made in Queensland [1]. There were a significant number of cases arising within the north Queensland city of Townsville and its surrounding areas. Data from 2004 to 2008 taken from the Communicable Diseases Branch demonstrated an average incidence of 5·3/100 000 during that period (range 3–8/100 000), greater still than the state-wide incidence [2]. Recently, a serum survey was performed of 1522 blood donations collected by the Australian Red Cross Blood Service in Townsville. This unpublished study by Govan *et al.* showed that 3·5% of all serum samples tested were positive for antibodies to phase II *C. burnetii* antigen [3]. It has been estimated that more than 50% of those infected with *C. burnetii* are asymptomatic or have only mild symptoms [4]. Even if symptoms are present, not all individuals will seek medical attention and the diagnosis may be overlooked by clinicians. Therefore, we are likely to significantly underestimate the true incidence of Q fever in our region of about 200 000 people [4].

Q fever is known to infect a wide variety of animals including farmed livestock, domesticated pets and

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wildlife. The association between human disease and exposure to animals or animal products is well described [5]. The organism is found in large numbers within the products of conception, urine, faeces and milk of infected animals. Transmission to humans is via inhalation of infected aerosols. However, a significant proportion of recognized human cases have no clear source of exposure. A previous survey of patients treated at Townsville Hospital showed that over 60% of acute cases had no clear history of animal or occupational exposure [6].

C. burnetii is known to survive for long periods of time in the environment, and can be infective at a very low inoculating dose. It has been postulated that exposure to *C. burnetii* in soil and dust, previously contaminated by infected animals, may provide an ongoing reservoir for human disease [4, 7]. Townsville has historically been closely associated with cattle farming, as well as live animal transport and meat processing industries. Several areas that now lie within or close to residential zones have previously been cattle grazing sites. Similarly, *C. burnetii* is well known to infect wild animals, such as wallabies and bandicoots, and can be commonly found in the local tick population, particularly the kangaroo tick *Amblyomma triguttatum* [5, 8]. The role that these potential reservoirs play in the transmission of human disease is not well understood. The aims of this study were to define the basic epidemiology of serologically confirmed acute Q fever in patients tested via the Townsville Hospital laboratory from 2000 to 2010 and to determine the impact of geographical location and seasonality on the incidence of acute cases in the Townsville region in order to focus further research into environmental and wildlife reservoirs of *C. burnetii*.

All Q fever serological tests processed through the Townsville Hospital laboratory between January 2000 and December 2010 were retrieved from the laboratory database. Ethics approval was granted by the Human Research Ethics Committee, Townsville Health Service District. All patients with a reactive IgM or IgG enzyme immunoassay (EIA) (Panbio; Alere, Australia) had confirmatory testing by immunofluorescence (IF) (Serion Virion, Germany) and were included for further analysis. Dilutions were made and ranged from <10 to $\geqslant 1280$. Demographic data were collected including age, sex and ethnicity. Residential addresses of acute cases were plotted onto a map of the local Townsville area. As this data was collected over an 11-year period, the cumulative incidence was calculated. Population data was obtained from the Australian Bureau of Statistics for each statistical local area corresponding to the nearest year of census data available [9]. Rainfall data was obtained from the Bureau of Meteorology [10].

A total of 3005 Q fever serological tests were processed through the Townsville laboratory over the study period. Once negative results were excluded, 243 patients demonstrated evidence of exposure to *C. burnetii* as confirmed by IF, including four paediatric patients. Of those patients with paired serological profiles consistent with acute or chronic Q fever, 79% were male. The average age was 48·5 years (range 18–81 years). This study includes six Indigenous patients who presented with acute Q fever and a total of 13 with evidence of past exposure. To the best of our knowledge there is only one other study in Australia that has reported Q fever in Aboriginal Australians. This was a study by Ralph *et al.* who reported four Indigenous patients with Q fever [11].

Paired serological profiles of 92 patients were diagnostic of acute Q fever. Acute disease was defined as a positive phase 2 IgM IF $\geqslant 160$ or seroconversion of phase 2 IgG EIA on convalescent sera and absence of phase 1 antibodies by IF or EIA. Four of these patients later went on to develop serological profiles consistent with chronic disease and 14 patients had profiles suggestive of chronic disease at presentation. Chronic disease was defined as a persistent phase 1 IgG IF >800. However, basing the diagnosis of chronic Q fever on serological criteria alone has recently been questioned. Reporting of IF titres varies widely between laboratories [12] and the cut-off value of phase 1 IgG >800 is based on the experience from a single European centre. This group has recently suggested that the cut-off value should be increased to $\geqslant 1600$ to improve specificity [13]. Some commercial assays may demonstrate phase 1 IgG titres >1280 several months after acute presentation without clinical evidence of chronic disease, and subsequent decline without therapy [14]. Five patients developed complications including four with endocarditis and one with haemophagocytic lymphohistiocytosis. There was one death which occurred within 24 h of valve replacement for Q fever endocarditis. There were 124 cases with serological evidence of past exposure only and in 13 patients the serological pattern was uninterpretable or there were no convalescent sera.

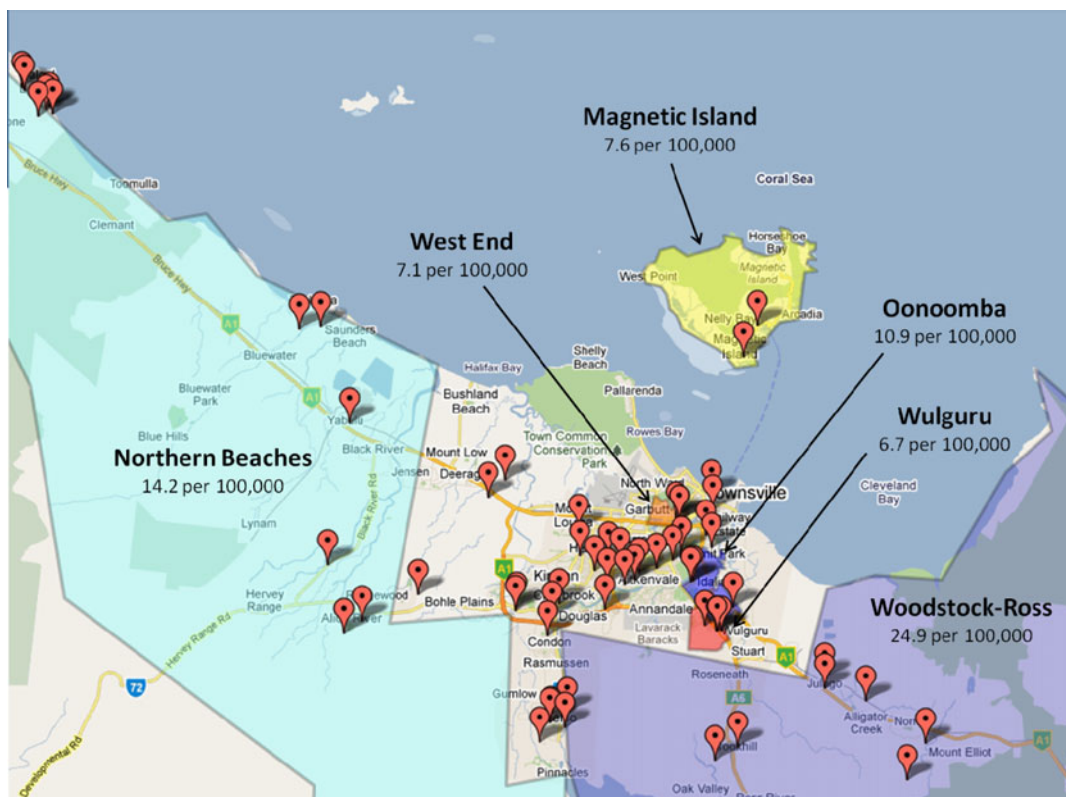All acute cases of Q fever were plotted onto a map of the local Townsville area (Fig. 1). Seven Statistical

Fig. 1 [*colour online*]. Map of acute Q fever cases in Townsville, 2000–2010. (Map data © 2011 Google.)

Local Areas (SLA) were identified as having an incidence higher than the average Queensland incidence of 5·8/100 000 over the study period [1]. The SLAs with the highest incidence were Woodstock-Ross with 24·9 cases/100 000 and Northern Beaches with 14·2 cases/100 000. These SLAs have a large land area and have historically been used as cattle grazing sites; however, there are now very few cattle properties within the Townsville area. *C. burnetii* is an organism that undergoes spore formation allowing it to survive harsh environmental conditions for extended periods of time. Small cell variants produced when cattle properties were active in the area may continue to play a potential role as a reservoir for disease. In addition, these sparsely populated areas are more likely to be heavily populated by wildlife, which may expose humans to Q fever. In some SLAs, the numbers are too small to draw firm conclusions.

Monthly rainfall data over the 11-year period was compared to the cumulative total monthly incidence of Q fever in Townsville (Fig. 2). A clear seasonal peak was found, with the greatest number of cases observed in May, 3 months following the peak in rainfall in February. As the average monthly rainfall declines from February to April, cases of acute Q fever appear to increase in these drier months and then fall again prior to the onset of the wet season in November–December. Comparison of annual rainfall and total acute Q fever cases for each year were compared (see Fig. 3). Although there was a weak positive correlation between these two variables, this was found to be non-significant [Pearson's $r = 0·48$ (range $-0·17$ to $0·84$); $P = 0·14$].

Cooper *et al.* have performed a seropositivity study of beef cattle and Australian wildlife in Queensland [15–17]. They sampled 720 beef cattle, 92 macropods, 46 bandicoots and 56 possums in northern Queensland during 2007–2010. They found that 16·8% of beef cattle were seropositive to either or both phase I and II *C. burnetii* antigens. The overall seropositivity of beef cattle was significantly less than the seropositivity of macropods (30·4%), bandicoots (23·9%), but more than possums (10·7%) [15–17].

This study confirms that the incidence of Q fever is high in northern Queensland compared to the national incidence. We have demonstrated that
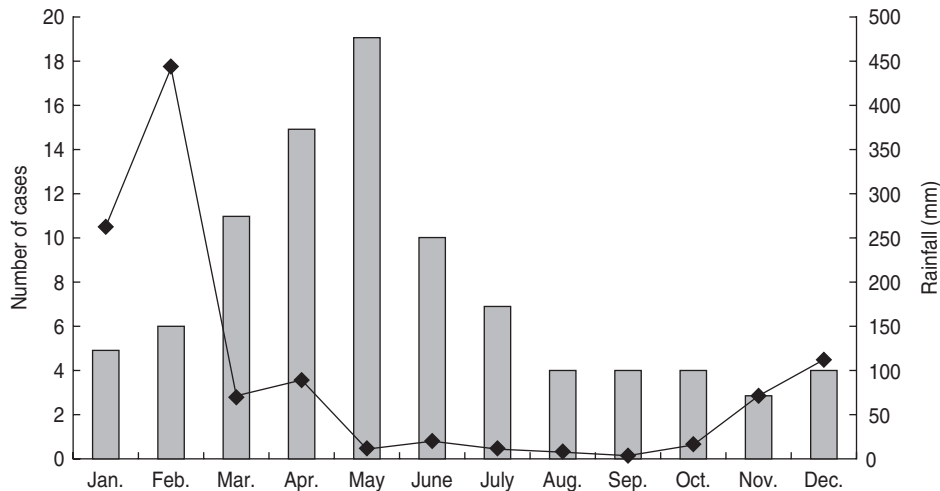
Fig. 2. Mean monthly rainfall and mean monthly acute Q fever cases, 2000–2010.
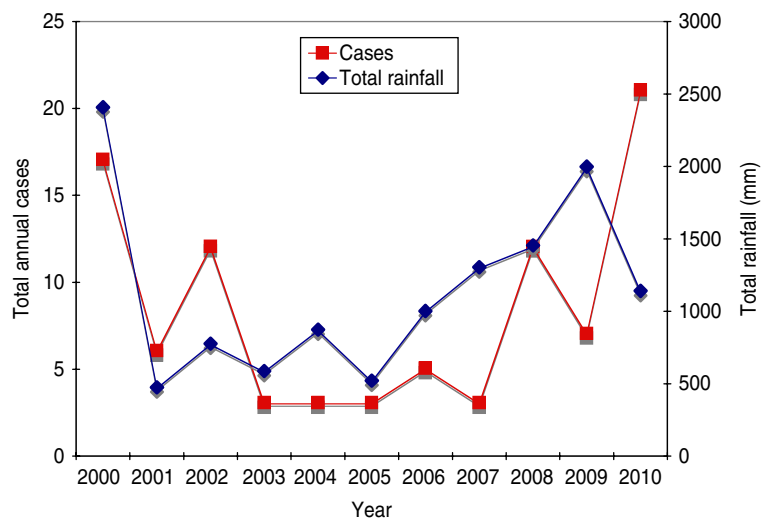

Fig. 3 [*colour online*]. Annual acute Q fever cases compared to total rainfall.

people with acute Q fever are more likely to live in sparsely populated areas on the outskirts of Townsville, where there is a denser population of wildlife.

This study was a retrospective laboratory-based analysis, with cases defined by serological criteria. Data on clinical features, timing of testing in relation to onset of symptoms and epidemiological exposures were not available for all cases. As serology alone will only roughly estimate the timing of exposure, and testing was initiated by clinicians in a non-standardized manner, temporal relationships between disease onset, exposure and environmental factors such as rainfall may be inaccurate. We acknowledge that a further limitation of this study is that it does

not take into account where people work or other environments in which they may have been exposed to Q fever. However, it is more likely that patients are infected at home or at work, where they spend the majority of their time. As part of our future research we intend to contact the patients identified as having acute Q fever for a detailed occupational and environmental exposure history.

This is the first study to our knowledge that has demonstrated a seasonal peak in acute Q fever cases in Australia. A French study by Tissot-Dupont *et al.* showed a seasonal peak of acute Q fever cases which occurred 1 month following a secondary lambing season [7]. They hypothesized that the spread of *C. burnetii*-infected aerosols was expedited by low

rainfall and high wind speeds. In Queensland, the calving season for the beef cattle industry occurs towards the end of the wet season when pasture biomass is highest. The prevailing winds in the area tend to come from the northeast (from the ocean), although may originate from the south or southeast in the mornings. However, currently there are few cattle properties immediately surrounding Townsville. As such, an aerosol route of spread of Q fever from farms carried on prevailing winds seems less probable. High rainfall seasons also contribute to increased population numbers of macropods and other wildlife. This is the main time for parturient wildlife in northern Australia. Wallabies and kangaroos are a family of marsupials referred to as macropods. The long hind limbs with the characteristic long narrow feet give them the name of 'macropod' or 'big-foot'. The wallaby is herbivorous, and being smaller than the kangaroo, relies on more densely vegetated habitats for food and shelter. The seasonal growth of vegetation attracts wallabies to settled grassy areas.

We hypothesize that an increase in wildlife numbers and drier conditions seen immediately following the wet season is the reason for the seasonal peak of human acute Q fever cases in Townsville.

Q fever has previously been recognized primarily as an occupational disease affecting those in contact with livestock, such as farmers, abattoir workers and veterinarians.

In northern Queensland, it is possible that Q fever is more likely to have been acquired from wildlife, such as wallabies, rather than via occupational exposure. We intend to focus further research into reservoirs of *C. burnetii* by using real-time PCR detection in environmental and wildlife samples.

ACKNOWLEDGEMENTS

DECLARATION OF INTEREST

None.

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