

Bug Breakfast in the *Bulletin*: Q fever

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Q fever is a zoonotic disease caused by the obligate intracellular Gram-negative bacterium *Coxiella burnetii*.¹ This Bug Breakfast discussed the disease, natural history and issues affecting population control.

Background

Q fever has been described in most countries of the world; however, it is a notifiable disease in only a few.¹ It has a low infectious dose and survives harsh environmental conditions.² As a result, it has been classified as a group B bioterrorist agent by the US Centers for Disease Control and Prevention (CDC).

Cattle, sheep and goats have been considered the primary human reservoir for *C. burnetii*; however, many other animals including Australian native species are known to be infected. Although most infected animals exhibit very few clinical signs, they shed *C. burnetii* in their milk, urine, faeces and particularly in amniotic fluid and placenta.²

Human transmission occurs through inhalation of aerosols of infected body fluids, the ingestion of unpasteurised milk or dairy products, or by inhalation of dried infectious dusts. Person-to-person transmission is rare. Those most at risk of Q fever are abattoir workers, livestock workers and veterinarians, farmers, shearers and laboratory workers. The Australian annual notification rate peaked at 4.9 per 100 000 persons in 1993 and decreased to 1.7 per 100 000 persons in 2005.³ The highest incidence of Q fever occurs in south and central-western Queensland and northern NSW areas with men aged 40–44 years having the highest age-specific rate.^{4,5}

Natural history, diagnosis and treatment

The incubation period for Q fever is usually 19–21 days (range one to six weeks). The illness presents with rapid onset of fevers, chills, profuse sweating, headaches, and muscle and joint pain, although the subclinical to clinical ratio is approximately 3:1. Occasionally hepatitis, pneumonia or neurological manifestations may occur in the acute illness. If *C. burnetii* persists, it leads to chronic,

localised Q fever infection. Endocarditis (particularly in those with underlying valvular heart disease) is the most common manifestation of chronic, localised infection. In addition, a postinfective fatigue syndrome (in the absence of ongoing infection), termed post-Q fever fatigue syndrome, is commonly reported.⁶

C. burnetii exists in two antigenic phases in laboratory culture systems, Phase I and Phase II. In nature, the organism exists in the Phase I state and is virulent and highly infectious.¹ Antibodies to both appear in a characteristic order in acute Q fever: IgM Phase II, IgM Phase I, IgG Phase II and then IgG Phase I. The appearance of IgG antibodies may be delayed up to six to eight weeks following the onset of symptoms.⁷

Diagnosis of acute Q fever requires a high index of clinical suspicion and relies on acute and convalescent serology. Although IgM antibody detection from a single serum sample is convenient, this assay is commonly associated with false positive test results.

Acute Q fever is treated with doxycycline, although rifampicin and ciprofloxacin are also effective. Chronic Q fever is characterised by ongoing illness, localised tissue injury and high titres of Phase I antibodies (IgG and IgA), and requires prolonged treatment with doxycycline, rifampicin and hydrochloroquine.

Population control of Q fever

Australia has access to the only licensed vaccine against Q fever available worldwide (QVax, CSL Ltd). Immunisation of high-risk occupational groups has been the major population control method; however, significant gaps remain in our knowledge of the optimal immunisation strategy. The vaccine is a purified suspension of formalin-inactivated *C. burnetii*. This vaccine is highly effective,⁸ but its uptake is constrained by the fact that serological and skin testing are required prior to immunisation to prevent hypersensitivity reactions in those who may have been previously exposed. The vaccine produces protection via generation of cellular immunity (rather than antibodies) and is likely to provide lifelong protection.

A Federally-sponsored National Q fever Management Program to support screening and immunisation commenced in 2001 and concluded in NSW in June 2004.⁹ The program resulted in most abattoir workers being immunised; however, many other at-risk rural populations remain unimmunised and account for the majority of notifications in many regions of Australia. Although respira-

tory precautions and personal protective equipment are likely to provide some protection in high-risk settings, their efficacy in rural working environments has not been evaluated and their practicality is often questionable.

At the time of presenting the Q fever Bug Breakfast in March 2006, the manufacturer had planned to discontinue the production of QVax. This prompted concern among rural communities and public health professionals. However, in May 2007 the Australian Government and the manufacturer committed to funding the construction of the BP Marmion Q Fever Vaccine Building in Melbourne¹⁰ to produce and test Q fever vaccine. It is anticipated to be operational by July 2009. The manufacturer has also stated that vaccine supply will continue until this facility is operational.

Q fever remains a significant public health concern, particularly for rural communities. Despite the commitment to ongoing vaccine supply and testing, further understanding of the risk factors associated with acquiring Q fever in rural communities is required.

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