Q-fever and Australian farmers: is the health system paying enough attention? A literature review

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After reading these articles, the learner should be able to:

- understand the Q-fever disease and its complications;
- understand the current vaccination program;
- understand the need for Australian farmers, farm workers and their families to be well-educated about Q-fever and included in the national vaccination program.

Competencies addressed:
1.2, 1.3, 4.1, 4.2, 7.1, 7.2, 7.3, 8.1

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Upon successful completion of the associated assessment, this activity has been accredited for 0.75 hours of Group 2 CPD (or 1.5 CPD credits) suitable for inclusion in an individual pharmacist's CPD plan.

ABSTRACT

Introduction: Q-fever is caused by *Coxiella burnetii*, a Gram-negative bacterium and Rickettsia-like organism. Transmitted from wild and domestic animals to humans, the most common route is inhalation of contaminated dust; however the oral route can be considered as a second pathway.

Aim: to understand the reasons behind not including farming workforce and their families in the national vaccinations program.

Discussion: In 1977 Q-fever became a notifiable disease nationally. Australia is the only country to have a registered Q-fever vaccine. As a result of the cost of the vaccine, Department of Health and Ageing (DoHA) supply and subsidised program arrangements are based on the active cases count per year (by occupation), rather than for occupations that expose workers to high level of possible "risk".

Conclusion: Australian farmers, farm managers, farm workers and their families need to be well educated about Q-fever and included in the national vaccination program.

OVERVIEW

Q-fever is caused by *Coxiella burnetii,* a Gram-negative bacterium and Rickettsia-like organism. Transmitted from wild and domestic animals to humans,[1] the most common route is inhalation of contaminated dust; however, the oral route can be considered as a second pathway.[2] The incubation period is 2–3 weeks, with symptoms ranging from none, to influenza-like symptoms, to more severe symptoms.

Australia is the only country that has a (licenced) registered Q-fever vaccine.[3] The DoHA-sponsored vaccination program started in 1991 at a small number of abattoirs, and then expanded to all others abattoirs from 1994. Then from 2011, it became a government-sponsored recommended program with vaccination being available to farmers, their families and all others employed in livestock-rearing industries.[1]

However, although uptake is reported to be high in the meat processing industry, uptake at primary producer sites is still more sporadic.[4] The vaccination for farmers program was ceased after 12 months; however it is still in place for abattoir workers.[4]

Patients suffering from Q-fever may experience endocarditis (the most common manifestation), vascular infection, osteoarticular infection, chronic hepatitis, chronic pulmonary infections and chronic fatigue syndrome.[5] Current pharmaceutical therapy includes doxycycline and hydroxychloroquine.[6] However some people do not recover and remain symptomatic.[7]

Farmers are the most affected group followed by abattoir workers; however cases have been identified in non-farming rural and metropolitan populations[8]. There is currently no protocol to investigate, treat or follow up persons diagnosed with acute Q-fever. Due to a lack of understanding of its importance in preventing infection, the current vaccination program is not utilised by all eligible individuals. There is also inadequate understanding of the disease complications, not only by individuals but also by health professionals.

Normally, for an infectious disease to be diagnosed and treated, signs and symptoms need to be detected early, to prevent the disease becoming chronic and to reduce long-term complications. Almost 60% of Q-fever cases are asymptomatic, 8% will have mild symptoms which do not require hospitalisation and only 2% will suffer chronic disease and typical symptoms.[9] However such a small percentage of patients with symptomatic disease no longer attracts sufficient funds for research and the disease seems to be forgotten until the next outbreak.

Q-fever is caused by *Coxiella burnetii*, a Gram-negative bacterium and Rickettsia-like organism. It is transmitted from wild and domestic animals to humans[1] from milk, urine, faeces, blood and uncooked meat.[1]

TABLE 1: **Q-fever notifications by year**

Disease	Notifications					Notifications per 100,000				
	2006	2007	2008	2009	2010	2006	2007	2008	2009	2010
Q-fever	352	409	448	361	303	1.7	2.0	2.1	1.7	1.4

Source: Australian Institute of Health and Welfare www.aihw.gov.au/australias-health-2010-data-tables/?id=6442475642 [accessed 27 August 2013]

The *C. burnetii* infection incubation period is 2–3 weeks. The most common route is inhalation of contaminated dust; however, the oral route is a second route of infection.[2]

Unpasteurised milk from domestic animals seems to allow sufficient pathogenic material via the oral route to cause infection.[2] One study[2] indicated that there may be host-related predisposing factors to explain the emergence of clinical symptoms in some individuals compared to the general population. Patients usually presenting with influenza-like symptoms (mild sore throat, headache, aches, myalgias, abdominal pain and fever of 39–40°C for 24 hours or more).

Some individuals present with more severe symptoms such as pneumonia, hepatitis, myocarditis, pericarditis, skin rash or meningoencephalitis. The severity varies from low grade to life-threatening.[9] Infection in patients with pre-existing cardiac defects, the immunocompromised or pregnant women, may result in more severe long-term complications.[10]

Vaccination during incubation period does not stop the disease development, nor does it confer life-long protection.

Patients suffering chronic Q-fever continue to experience endocarditis, the most common manifestation, vascular infection, osteoarticular infection, chronic hepatitis, chronic pulmonary infections and chronic fatigue syndrome.[9] Vaccination during incubation period does not stop the disease development,[1] nor does it confer life-long protection.

Australia is the only country to have a registered Q-fever vaccine.[3] Vaccinations started on 1991 for a few abattoirs, before expanding to all other abattoirs from 1994, from 2002, it included farmers.[1,11] The vaccination uptake increased corresponding with the increase of awareness of occupational risk and planning for risk mitigation at the work place notwithstanding the fact that in 2007 Work Cover compensations were costing Australia $3.1m annually (all causes).[10]

A comparative study undertaken by Princess Alexandra Hospital, Queensland,[10] investigated all Q-fever notifications from January 2000 to September 2006 (191 cases, including 12 older than 70 years of age mostly males), and found that 56% of cases were occupation-related versus 36% in the general community (8% were difficult to determine causality).[10] Out of the 36% community cases, only 6.3% were in urban Brisbane.[10]

The number of occupational exposure cases who were eligible but never received vaccination remained steady during the study

FIGURE 1A:
Notified cases of Q-fever, Australia, by year

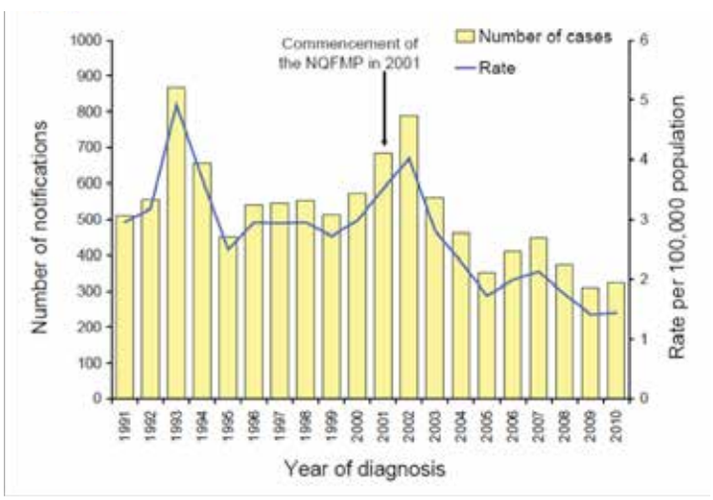
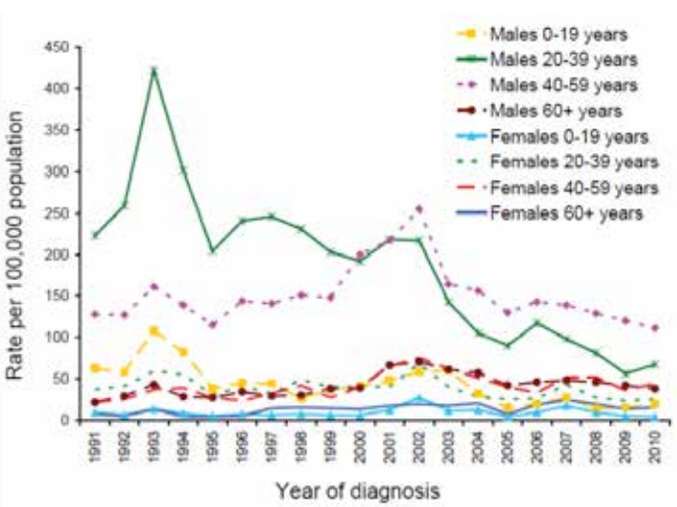


FIGURE 1B:
Notified cases of Q-fever, Australia, by year, age group and gender



Source: Department of Health and Ageing www.health.gov.au/internet/main/publishing.nsf/content/cda-cdi3601-pdf-cnt.htm/$FILE/cdi3601a.pdf [accessed 27 August 2013]

period at 71%,[10] indicating the need for better health promotion to educate the vulnerable population.

Doxycycline is the recommended first-line pharmacological agent found to be effective against the bacterium at 200mg daily for 14 days. Additionally, clarithromycin, trimethoprim/sulfamethoxazole, ciprofloxacin, rifampicin and hydroxychloroquine have also been used.[6] However some people do not recover and may remain symptomatic.[7]

In Australia, the number of notifications per year has reduced since the 2002 government-sponsored program was introduced (see Table 1).[12]

Figure 1A shows the difference between notifications and hospitalisation, which may also indicate the proportion of patients who had severe symptoms that required hospitalisation. Figure 1B shows the higher incidences in males older than 14 years of age when compared to females from the same age. Males and females younger than 14 and older than 65 years of age had lower incidences than male and females from other age groups.[13]

When a pregnant woman is infected she may remain asymptomatic, however the effect on the foetus is established. The effect of the bacterium on the foetus may vary from miscarriage, stillbirth, premature birth, or low birth weight. There may also be a residual adverse effect on subsequent pregnancies.[14] The Centre for Disease Control recommends that women who are diagnosed with Q-fever at child bearing age or who are sexually active should use contraceptives until full recovery and completion of treatment.[14]

Vomiting, diarrhoea, anorexia and skin rash are the most common symptoms in infected children. However severe acute cases may also present which may include "hepatitis, haemolytic uraemic syndrome, myocarditis, pericarditis, encephalitis, meningitis, haemophagocytosis, lymphadenitis, acalculous cholecystitis and rhabdomyolysis".[14] Infection is common after school visits to farms and wildlife parks.[15] In a study published in the *Lancet*, most children diagnosed with Q-fever who were between 2 and 14 years were admitted to hospital due to recurrent high fever for 2–21 days as the main feature, with two cases confirmed to have CNS infection (one meningoencephalitis and one encephalitis) and three with myocarditis.[15]

DISCUSSION

In 1977, Q-fever became a notifiable disease nationally.[9] Since then, the uptake of vaccination has increased, however the reduction in the number of notified cases was not as great as the increase in the vaccination uptake, or in the insurance compensation claims due to Q-fever.

A study by Tozer *et al*[3] investigated the seroprevalence of *C. burnetii* in a diverse Queensland population (metropolitan, rural, adults and paediatrics subjects). They found that the prevalence of seropositive metropolitan and paediatric subjects was higher than reported in other studies and close to that reported in people with an occupational exposure risk in livestock industries or rural living.[3] This suggests that the geographical area of residency may have the same effect as occupation.

In 2009, Massey *et al*[16] wrote a short report on the health promotion for Q-fever in place in New South Wales at the time and whether it addressed the issue of exposure to Q-fever infection and found the current program to be no longer adequate. Most current material is addressing Q-fever infections based on occupational exposure, however fewer than 50% of notifiable cases have the occupation recorded, with 16.1% farmers and 13.9% abattoir/meat workers, which is more than 5% of the reported occupational related cases.[16] Only 3% of cases were reported in children 15 years of age and under, whereas 90% of cases were recorded for people aged between 15-64 years old. Overall there were 80% males and 94.9% residents of rural NSW.[16]

Massey *et al*,[16] reported that of 75 notifications received in 2007 (January to December) in the Hunter, NSW: 61 were confirmed with acute Q-fever; 56 were interviewed by the study investigators and 42 out of the 56 were living on-farm or in a rural area; 36 were farmers, farm managers or farm workers; and 31 had contact with new livestock, with 21 lost working days.[16] Thirty eight had direct contact with animals (cattle, sheep and wild native animals) and animal tissues, and four had contact with contaminated dust. The recovery period ranged from 1–35 weeks with 34 people reporting that they were not yet recovered.[16] Despite 70% of the interviewed patients stating that they had heard about the vaccine, none of the 56 people interviewed were vaccinated and still believed themselves not to be at risk (37%). This confirmed the authors' concern regarding the need to change the health promotion strategy.[16]

Chronic Q-fever follows an acute attack. Complications in humans are not organ damage-specific and follow the same pattern as in animals. Some patients may have cardiac valve damage; while others may have damage to the liver or lungs.[13] The most common complications observed are endocarditis, hepatitis and osteomyelitis (most common in paediatric patients).[14]

In a study by Figtree *et al*, a case of acalculous cholecystitis was thought to have a possible direct connection to chronic Q-fever. This connection was confirmed in the gallbladder tissues for the first time.[17] Fifteen other cases were described in previous research (cited in Figtree *et al*, 2010) in unvaccinated subjects, however *C. burnetii* was not considered as the only causative organism.[17] The study provided the interesting conclusion that the increasing number of Q-fever incidents in people who are not in direct contact with animals is due to the drought, i.e. exposure to contaminated dust; however the link between drought (reduction in vegetation leading to bare ground and dust) and Q-fever needs to be further investigated.

Nourse *et al* studied three Q-fever osteomyelitis paediatric patients (only 11 other cases were previously reported of which six of were children).[18] The three children each had contact with animals; one lived on a cattle farm, one lived on a dairy farm (and had the symptoms for five years before being diagnosed) and one had contact with a cat at home.[18] The authors suggested that same medications (doxycycline and hydroxychloroquine) may be required but for a longer period, however longer-term research will be required to confirm the effectiveness of the treatment and that there will be no future relapse.[18]

Landais *et al* reviewed four new cases of Q-fever osteoarticular infection taking into consideration the previous research conclusion that Q-fever chronicity is related, to some extent, to host factors.[19] In this study all four cases were in adults aged 31, 56, 64 and 47 years old where the diagnosis was confirmed by serological analysis as being Q-fever complication.[19]

Unlike children, most Q-fever adult osteoarticular cases are related to prosthetics or vascular graft procedures, with two out of the four pure spondylodiscitis diagnosed in dairy farmers.[19] The authors agreed that the condition is underestimated and they recommended the use of doxycycline and hydroxychloroquine for 18 months or longer.[19] Tande *et al* recognised the importance of confirming the presence of *C. burnetii* and described an assay for detection in the bone and tissues in patients suspected to have Q-fever osteoarticular infection.[20]

Patients that present with chronic endocarditis are typically male (76%), with involvement of the aortic valve (33%), mitral valve (50%) or both valves (17%).[21]

Original research published in November 2011,[21] concluded the need for thorough cardiac examination for all patients diagnosed with Q-fever, which was not routinely conducted or recorded in patients' medical records at the time of publication. Out of 89 people hospitalised with suspected Q-fever, 75% of cases were confirmed to have Q-fever after discharge, 22% during their hospital stay and 2% were diagnosed prior to admission.[21]

According to the authors, from 2005 to 2009, only six patients had completed cardiac examination records in the Hunter and New England, NSW, districts, where patients admitted to the district hospital had even less documentation. It is alarming that in the Hess et al study in a sample of patients admitted with acute Q-fever, no patient had physical cardiac examination, no patient been categorised as at risk, or had follow up.[21] Retrospective review identified that three patients were reported to have endocarditis during their hospital stay.[21]

POTENTIAL FUTURE IMPACT OF Q-FEVER ON THE FARMING COMMUNITY

As a result of the cost of the vaccine, supply and subsidised programs from DoHA are based on the occupational active cases count per year, not on occupations that exposes workers to high level of possible "risk". This observation corresponds with the numbers of reported cases increasing in abattoir workers and decreasing for farmers in the years since the program started.

CONCLUSION

This review of the current literature and the history of Q-fever in Australia suggests that only minimal changes in farmers' awareness, program availability and vaccination uptake have occurred since the introduction of the national Q-fever vaccination program in Australia. An appropriate level of health promotion and standardised investigation and treatment protocols, with adequate risk exposure surveillance and vaccination program is required. This has the potential to reduce incidents and disease complications resulting in fewer compensation claims and loss of working days.

More importantly, standardised risk exposure surveillance and life-long follow-up plans for sufferers will help support better quality of life for Q-fever patients. Australian farmers, farm workers and their families need to be well educated about Q-fever and included in the national vaccination program. National education programs also need to be established to target people visiting farms and wildlife parks to enable prevention, early detection and treatment programs.

AUTHORS' CONTRIBUTIONS

The authorship listed is in accordance to the ICMJE definition of authorship.

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CONFLICT OF INTEREST

None declared

Key Words:
Q-fever, farmers, vaccinations, infection, risk

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