IDAHO DEPARTMENT OF HEALTH & WELFARE

Disease Bulletin

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Query About Q Fever

An increase in reported goat-associated human cases of Q fever was reported from Washington and Montana beginning in late May of 2011[1]. Beginning in August 2011, the Idaho State Department of Agriculture (ISDA) Dairy Bureau initiated a Q fever screening program for all dairy goat herds in Idaho. ISDA detected *Coxiella burnetii,* the causative agent of Q fever, in goats from one Eastern Idaho goat herd in June, 2012[2].

In response, Idaho public health officials sent a Health Alert to healthcare providers in Eastern and southeastern Idaho to enhance awareness of both Q fever laboratory detection methods and reporting requirements. ISDA quarantined the affected herd and its milk products; as of September 28, 2012, there has been no increase in reported human Q fever cases in Idaho. Both suspected and confirmed cases of Q fever must be reported within one working day to the Office of Epidemiology, Food Protection, and Immunization or your public health district.

Heightened awareness in the United States

Q fever in humans is uncommon in the United States; an average of 150 cases were reported annually during 2006–2008[3]. During 2005–September 28, 2012, five confirmed or probable human cases of Q fever were reported to the Idaho Department of Health and Welfare. Although uncommon here, large outbreaks of Q fever have occurred elsewhere. During 2007–2011, approximately 4,000 human cases, primarily associated with proximity to Q fever-positive goat herds, were reported in the Netherlands[4].

Symptoms

C. burnetii can cause acute or chronic illness in humans[3]; although, approximately half of infected persons are asymptomatic. Onset of acute Q fever occurs in persons 2–3 weeks after exposure. Symptoms experienced vary greatly, but most people have fever and some of the following symptoms:

- abdominal pain
- chest pain
- chills and/or sweats
- confusion
- diarrhea
- general malaise
- high fever
- myalgia
- nausea
- non-productive cough
- pre-term delivery or miscarriage
- severe headache
- sore throat
- vomiting

Chronic Q fever develops in <5% of patients 6 weeks to years post-infection. Endocarditis is the major form of chronic disease, comprising 60%–70% of all reported cases. Pregnant women, immunosuppressed persons, and those with pre-existing heart valve defects, arterial aneurysms, or vascular grafts are at highest risk for developing chronic disease. The estimated case fatality rate in untreated patients with endocarditis is 60%.

Diagnosis

According to Centers for Disease Control and Prevention, detectable antibody titers are typically found by 7–10 days after illness onset; therefore, a negative test during the first week of illness does not rule out Q fever[3]. The gold standard serologic test for diagnosis is the indirect immunofluorescence assay (IFA). Paired serum samples taken 2–4 weeks apart demonstrating a four-fold rise in antibody titer provide the best evidence for a correct diagnosis of acute Q fever. *C. burnetii* exists in two antigenic phases called phase I and phase II. In *acute* cases, phase II antibody levels are usually higher than those against phase I. In *chronic* cases, the reverse pattern occurs. For diagnostic purposes, testing should include Phase I and Phase II IgG and IgM serologic titers. Antibodies to phase I and

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II antigens have been known to persist for months or years after initial infection.

Treatment

Doxycycline is the treatment drug of choice for adults and children with severe illness. According to CDC[3], treatment should not be withheld until laboratory results return, if suspicion for Q fever is high. Resistance to doxycycline has not been documented; therefore, failure to respond to doxycycline suggests an alternative etiology is possible. Severely ill patients can require longer periods before their fever resolves[3]. Recommended dosages for acute and chronic Q fever in children and adults is found on the CDC website www.cdc.gov/qfever/symptoms/index.html, along with additional information on use of doxycycline in children. Treatment duration for acute disease is typically 2–3 weeks, while treatment for chronic Q fever may take up to 18 months.

Risk factors for infection

Cattle, sheep, and goats are the primary reservoirs. Although large studies of *C. burnetii* prevalence in ruminants are uncommon, a 2007 study reported an estimated 90.1% of dairy cattle herds in the western United States were infected[5] and a 1978 study found 26% of 234 California goat herds were infected[6]. *C. burnetii* is found in highest concentrations in placenta and birth fluids of affected animals. The organism can also be shed in milk, urine, and feces and survive for prolonged periods in a dried state. Transmission to humans usually occurs through inhalation of contaminated barnyard dust. The organism can become airborne on dust particles, travel on wind currents for miles, and infect individuals with no discernible animal exposure. Consumption of unpasteurized milk or milk products; the bite of a tick; and exposure to infected dogs, cats, or other animals are also risk factors for infection, but are considered rare sources of infection. Human-to-human transmission is also rare.

Prevention

Because most infections arise from occupational exposure to contaminated barnyard dust, individuals in high risk occupations or activities (veterinarians and those working in the livestock industry, particularly during calving, kidding, or lambing) should be aware of exposure risks. A Purdue University website (www.purdue.edu/rem/eh/anmluse.htm#q) provides information on Q fever risk reduction for ruminant handlers, stressing that particular attention should be paid to respiratory protection during the attendance of birthing procedures. There is currently no method for cow or goat dairies to be certified as "Q fever free". Proper pasteurization will inactivate *C. burnetii*; therefore, persons wishing to avoid infection with *C. burnetii*, and other milk-borne zoonotic pathogens, should consume only pasteurized milk and dairy products.

References

[1] Q Fever Outbreak Associated with Goat Farms – Washington and Montana, 2011. Notes from the Field, *MMWR*, October 14, 2011 60(40):1393.

[2] Personal communication.

[3] CDC. Q Fever. www.cdc.gov/qfever/index.html. Update 4/18/2011. Accessed 9/24/2012.

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[5] Veterinary Services, Centers for Epidemiology and Animal Health, APHIS, USDA. Technical Brief: Prevalence of Coxiella burnetii in bulk-tank milk on U.S. dairy operations, 2007. www.aphis.usda.gov/animal_health/nahms/dairy/downloads/dairy07/Dairy07_is_Coxiella.pdf. Published March 2011. Accessed 9/24/2012.

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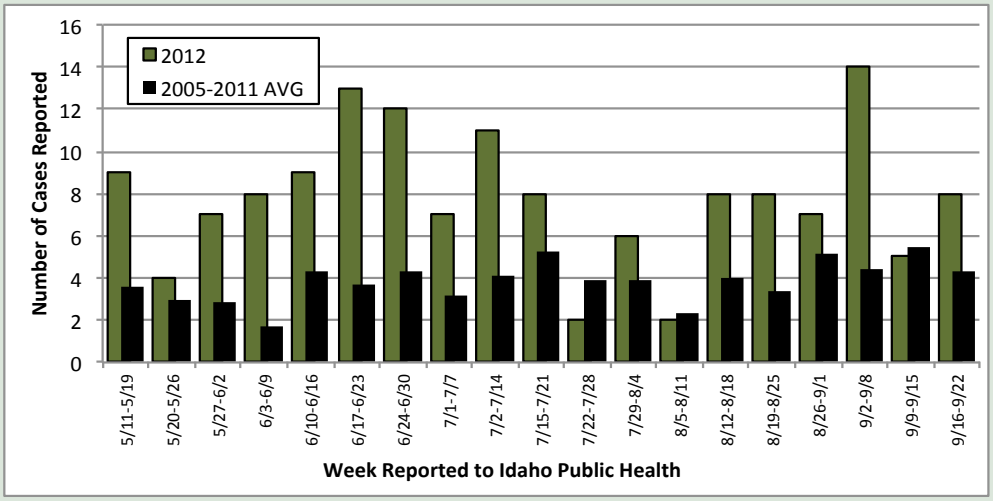
Spotlight on Pertussis: how does Idaho incidence compare?

Pertussis (whooping cough) incidence is up nationwide. Provisional data from the Centers for Disease Control and Prevention indicates the number of cases of pertussis reported in the United States during the first half of 2012 is more than double the number of cases reported during the same time in 2011[1]. Pertussis incidence typically follows a cyclical pattern, with the number of cases peaking every three to five years as a result of waning immunity in the population and increased bacterial circulation.

Incidence in western states

On April 3, 2012, Washington health authorities declared an epidemic of pertussis in the state. During January through September 22, 2012, there was a eight-fold increase in the number of reported cases of pertussis in Washington compared with the same period in 2011. Fifty-nine infants have been hospitalized; 83% of them aged less than three months. Montana has reported over five times as many cases this year as the same time last year—the highest number of cases reported since a statewide outbreak in 2005. Four of the 29 cases of pertussis were among infants that were hospitalized. In Oregon, nearly nine times as many cases have been reported as the same time last year. The Southern Nevada Health District is reporting more cases during January 1

Figure 1. 2012 weekly case counts of reported pertussis incidence in Idaho during the timeframe May 11–September 22, 2012 compared to the seven year average weekly incidence.



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PERTUSSIS CONTINUED FROM PAGE TWO

through September 22 of 2012 than in all of 2011. As recently as 2010, California had over 9,000 cases of pertussis, including 10 deaths; the highest incidence in that state in decades. While no deaths have been reported in Washington, Oregon, Montana, or Nevada this year, the death of an Idaho infant in May received local and national media attention.

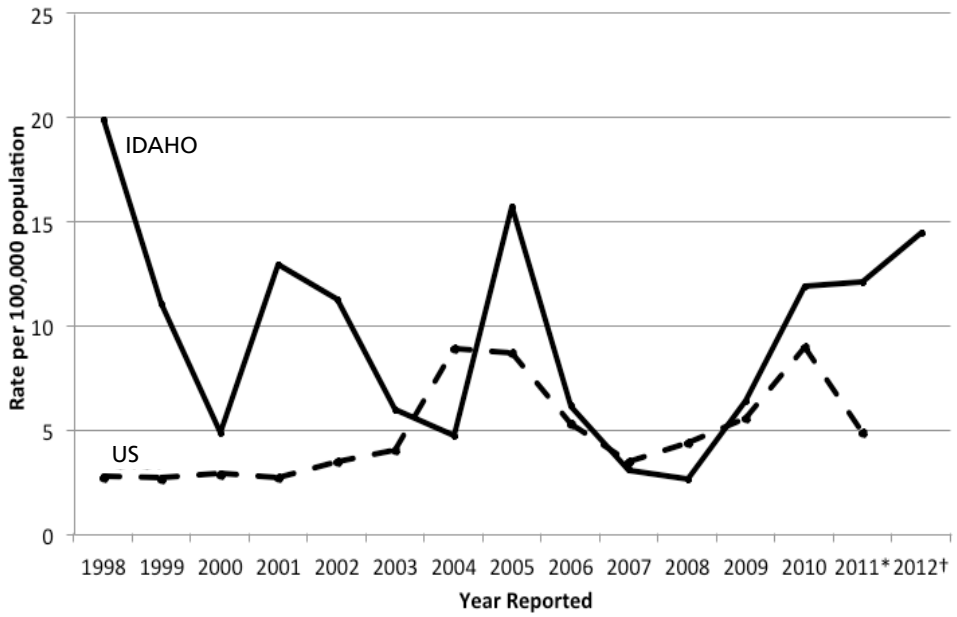
Incidence in Idaho

Fortunately, Idaho has not seen the same extreme increase in incidence as Washington. However, the number of reports of pertussis during January 1 through September 22 of this year has been higher than expected and Idaho could see the highest incidence rate of pertussis since 2005 if the trend continues. The Office of Epidemiology, Food Protection, and Immunization had received 184 reports of pertussis as of September 22, nearly double the number of cases during the same time frame in 2011. The increase in reported case counts compared with the average number of cases reported during 2005–2011 has been observed since mid-May (Figure 1). Typically, pertussis rates in Idaho have been higher compared with national rates (Figure 2). After a sharp increase in pertussis incidence in Idaho during 2005, rates of the disease gradually decreased until 2009. Since then, incidence of pertussis has been increasing in Idaho, following the cyclical pattern of incidence observed in previous years.

References

[1] CDC. ND-353-ND366 Table II. Provisional cases of selected notifiable diseases, United States, weeks ending June 30, 2012 and July 2, 2012 (26th week). MMWR. July 6, 2012 61(26). Available at www.cdc.gov/mmwr/preview/mmwrhtml/mm6126md.htm?s_cid=mm6126md_w#tab2.

Figure 2. Annual rate of pertussis per 100,000 population, Idaho and United States, 2000–2012*,†



* 2011 U.S. data are projected based on provisional case counts as of Dec. 31, 2011 published in MMWR Jan. 6, 2012 / 60(51);1762-1775 Table II "Provisional cases of selected notifiable diseases, United States, weeks ending December 31, 2011 and January 21, 2012 (52nd)"
† 2012 Idaho data are projected based on preliminary case counts as of Jun. 30, 2012.

Invasive *Neisseria meningitidis*: why should you care about serogroup?

Three cases of invasive *Neisseria meningitidis* disease with onset of illness in May and June 2012 were reported in Idaho, two from the same public health district in women aged 65–75 years. As part of the public health investigation, isolates were requested to be sent to the Idaho Bureau of Laboratories (IBL); however, only two *N. meningitidis* isolates were received from these three cases. This is the first reported case of invasive *N. meningitidis* of unknown serogroup in Idaho since 2007 (Figure). Why does this matter?

Thirteen antigenically-distinct polysaccharide capsules have been described for *N. meningitidis*; some strains have no capsule and are not classified in any serogroup. Serogroups A, B, C, W-135, and Y cause the majority of cases of invasive disease worldwide and caused the majority (24 [71%] of 34) of Idaho cases reported during 2007 through July 13, 2012 for which serogroup is known (Figure). Meningococcal vaccines MCV4 and MPSV4 contain antigens to elicit protection against serogroups A, C, W-135, and Y. Serogroup B polysaccharide capsule is poorly immunogenic due to antigenic mimicry with polysaccharide in human neurologic tissues; therefore, serogroup B antigen is not in current vaccines. Vaccines, therefore, are a very useful tool if outbreaks of meningococcal disease due to serogroups A, C, W-135, or Y are detected, but will not be helpful if serogroup B or untypeable strains are causing the outbreak. MCV4 is FDA-approved for use in persons aged 9 months through 55 years (Menactra®) or 2 years through 55 years (Menveo®); MPSV4 (Menomune®) is FDA-approved for use in persons aged 2 years and older. Although routine vaccination of persons aged >55 years against meningococcal disease is not recommended by the Advisory Committee on Immunization Practices, during an outbreak caused by *N. meningitidis* serogroups A, C, W-135, or Y, MPSV4 could be considered for use in persons at risk including those aged >55 years. A third vaccine type, MenCY-Hib (MenHibrix®) was FDA-approved in June for prevention of invasive disease caused by *N. meningitidis* serogroups C and Y and *Haemophilus influenzae* type b for children aged 6 weeks through 18 months.

As a clinician treating a patient for suspected invasive meningococcal disease, you can help inform prevention efforts by obtaining specimens for culture and requesting that your laboratory submit *N. meningitidis* isolates to IBL for serogroup determination. Serogrouping at IBL is done at no cost to the patient or provider, and

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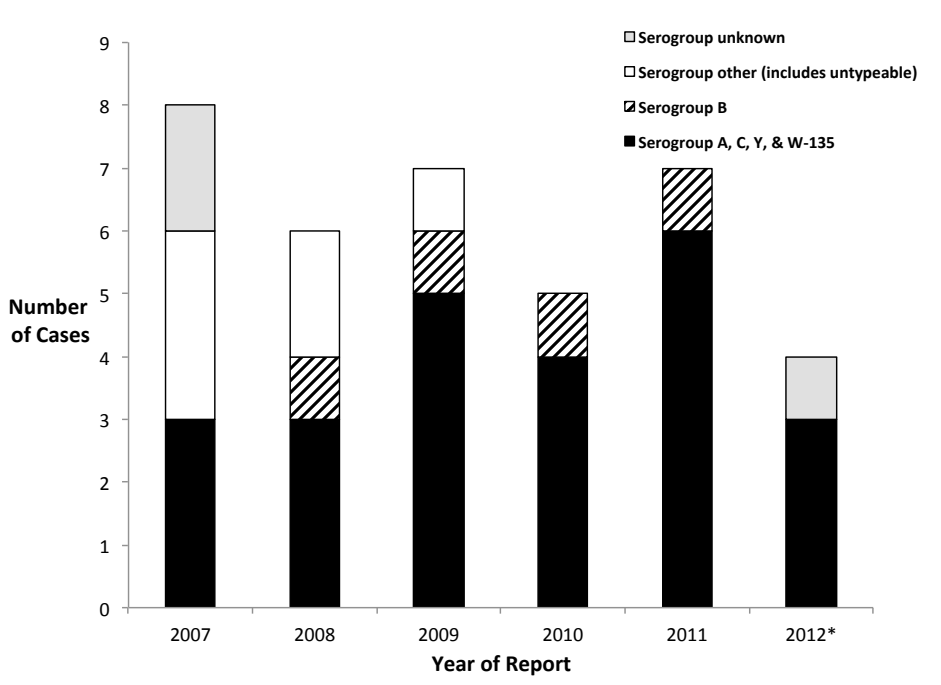
although individual serogroup results do not inform patient treatment, the results are critical to determining if an outbreak of meningococcal disease is occurring and if vaccination could be a method for controlling an outbreak. Serogroup results are also sent to the Centers for Disease Control and Prevention for national surveillance of vaccine-preventable diseases and to help guide vaccine policy and development.

Figure. Number of reported cases of *Neisseria meningitidis* invasive disease by serogroup—Idaho, 2007–2012*



* As of 7/13/2012; data are preliminary.

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