Title
Bartonella spp. in pets and effect on human health.

Permalink
https://escholarship.org/uc/item/55g462dh

Journal
Emerging infectious diseases, 12(3)

ISSN
1080-6040

Authors
Chomel, Bruno B
Boulouis, Henri-Jean
Maruyama, Soichi
et al.

Publication Date
2006-03-01

DOI
10.3201/eid1203.050931

Peer reviewed
Among the many mammals infected with *Bartonella* spp., pets represent a large reservoir for human infection because many of these bacteria can be transmitted by vectors, such as ticks and biting flies. Domestic cats are the main reservoir for *B. henselae*, *B. claridgeiae*, and *B. koehlerae*. Dogs can be infected with *B. vinsonii* subsp. *berkhoffii*, *B. henselae*, *B. claridgeiae*, *B. washoensis*, *B. elizabethae*, and *B. quintana*. The role of dogs as an important reservoir of *Bartonella* spp. is less clear than for cats, because domestic dogs are more likely to be accidental hosts, at least in nontropical regions. Nevertheless, dogs are excellent sentinels for human infections because a similar disease spectrum develops in dogs. Transmission of *B. henselae* by cat fleas is better understood, although new potential vectors (ticks and biting flies) have been identified. We review current knowledge on the etiologic agents, clinical features, and epidemiologic characteristics of these emerging zoonoses.

*Bartonella* spp. are fastidious, hemotropic, gram-negative bacteria that are mainly transmitted by vectors. Among the 11 species or subspecies known or suspected to be pathogenic for humans, 6 have been isolated from pet dogs and cats (Table 1). Domestic cats are the principal reservoir of *Bartonella* infections, as several recent review articles have been written on this subject (1,2,10).

**Feline Bartonella Species**

*B. henselae*

Since the first isolation of *B. henselae* from a domestic cat in the early 1990s, several studies have been conducted worldwide to determine the importance of cats as a reservoir of this bacterium (reviewed in [2]). Prevalence of infection varies considerably among cat populations (strays or pets) with an increasing gradient from low in cold climates (0% in Norway) to high in warm and humid climates (68% in the Philippines) (2). At least 2 genotypes have been identified and designated Houston-1 (type I) and Marseille (previously BATF) (type II) (1,2). The respective prevalence of these 2 genotypes varies considerably among cat populations from different areas. *B. henselae* type Marseille is the dominant type in cat populations in the western United States, western Europe (France, Germany, Italy, the Netherlands, United Kingdom), and Australia, whereas type Houston-1 is dominant in Asia (Japan and the Philippines) (reviewed in [2]). However, within a given country, the prevalence may also vary among cat populations. For instance, in France, Marseille type was the most common type in cats from the Nancy and Paris areas, whereas type Houston-1 was the main genotype in cats from Lyon or Marseille (references cited in [2]). However, a few studies in western Europe and Australia have reported that most human cases of CSD were caused by *B. henselae* type Houston-1, despite the fact that type Marseille was found to be the dominant type in the cat population, which suggests that type Houston-1...
strains could be more virulent to humans \((2)\). Cats are usually bacteremic for weeks to months, but some cats have been reported to be bacteremic for >1 year. Young cats (<1 year) are more likely than older cats to be bacteremic \((11)\), and stray cats are more likely to be bacteremic than pet cats \((1,2)\).

The clinical description of CSD was first reported in France by Debré et al. in 1950, but the etiologic agent was identified only in 1992 \((1,2,6)\). The annual number of cases in the United States has been estimated to be between 22,000 and 24,000, with \(\approx 2,000\) cases that require hospitalization, and thousands of cases may occur yearly in Europe. In various studies, the seroprevalence of antibodies to \(B.\) henselae in healthy persons has ranged from 3.6% to 6% (Table 2) and could be higher in some specific population groups, such as veterinarians, children, or elite orienteers (orienteering is a sport in which participants compete to find points in the landscape using a map and compass). Table 2 gives comparative \(B.\) henselae seroprevalence data for cat and healthy human populations from selected countries, which suggests that seroprevalence is low in both cats and humans at northern latitudes and increases in warmer climates \((11–24)\). Such data are informative and cannot exclude possible serologic cross-reactivity with some other \(Bartonella\) spp.

Despite the fact that \(B.\) henselae infection can cause meningitis and encephalitis, only 1 case of a fatal infection has been reported \((5)\). CSD is more frequently observed in persons <20 years of age and in persons who own a young cat (<1 year of age, especially if this cat is infested with fleas) or in persons who have been scratched or bitten by a cat \((1,2,6)\). In immunocompetent persons, CSD is mainly characterized by a benign regional lym-}

phadenopathy. Usually after a cat scratch, a papule and then a pustule develop within 7 to 12 days at the injection site, followed by a regional lymphadenopathy (usually involving a single lymph node) 1–3 weeks later that can persist for few weeks to several months. Low-grade fever, malaise, and aching are often reported; in some instances, headache, anorexia, and splenomegaly can occur. Abscessed lymph nodes are reported occasionally. In 5% to 9% of CSD patients, atypical manifestations may develop, including Parinaud oculoglandular syndrome, encephalitis, endocarditis, hemolytic anemia, hepato- splenomegaly, glomerulonephritis, pneumonia, relapsing bacteremia, and osteomyelitis.

On the basis of serologic testing or polymerase chain reaction (PCR), several recent publications have associated \(B.\) henselae with uveitis, focal retinal phlebitis, neuroretinitis, retinal and optical nerve neovascularization, and retinal artery and vein occlusions. Neurologic forms are rare, and patients usually completely recover within 1 year without sequelae. Hepatosplenomegaly and osteolytic bone lesions have been described in persons seropositive for \(B.\) henselae. Pseudotumoral lesions involving the mammary glands, the liver, or the spleen and, recently, glomerulonephritis and cases of monoclonal and biclonal gammopathy have also been associated with \(B.\) henselae antibodies. Cases of prolonged fever without adenopathy, chronic fatigue, hemolytic anemia, thrombocytopenic purpura, Henoch-Schönlein purpura syndrome, pleuritis, pneumonia, and even paronychia have been reported in patients who were seropositive for \(B.\) henselae \((1,2)\). Usually, these clinical manifestations disappear in a few weeks to a few months. Bacteremia is rarely detected in immunocompetent persons. Several cases of endocarditis
have been associated with *B. henselae* infection, most frequently in persons with preexisting valvular lesions. Besides *B. henselae*, most human cases of *Bartonella* endocarditis are caused by *B. quintana*, but a few cases of endocarditis or myocarditis have been associated with *B. elizabethae* (1 case), *B. vinsonii berkoffii* (1 case), *B. vinsonii arupensis* (1 case), *B. koehlerae* (1 case), *B. washoensis* (1 case), and *B. alsatica* (1 case) (Table 3).

In immunocompromised patients, *B. henselae* infection can cause prolonged fever, prolonged bacteremia, or both (1,2,6). Bacillary angiomatosis or peliosis is usually observed in highly immunocompromised persons (low CD4 count), who often are infected with HIV. Several severe infections have also been reported in organ transplant recipients (1,2).

The clinical spectrum of the infection in cats has not been fully investigated, but naturally infected cats primarily seem to be healthy carriers of the bacterium (1,2,6). However, cases of uveitis and rare cases of endocarditis have been molecularly associated with infection caused by *B. henselae*. Seropositive cats were more likely to have kidney disease and urinary tract infections, stomatitis, and lymphadenopathy. In experimentally infected cats, fever, lymphadenopathy, mild neurologic signs, and reproductive disorders have been reported.

### B. clarridgeiae

*B. clarridgeiae* was first isolated in the United States from the pet cat of an HIV-positive patient (25). This *Bartonella* sp. has been less frequently isolated from domestic cats than *B. henselae* because it appears to be more difficult to isolate and is unevenly distributed in cat populations worldwide. A *B. clarridgeiae* prevalence of 17% to 36% among all *Bartonella* isolates was reported in studies conducted in France, the Netherlands, the Philippines, and Thailand (2,22). However, *B. clarridgeiae* represented ≤10% of all isolates from domestic cats in the southeastern United States, Japan, or Taiwan (2) and has never been isolated in studies conducted in Europe, Australia, and North America (2). No specific pathologic features have been associated with natural infection in cats. However, in experimentally coinfected cats (*B. henselae* type II and *B. clarridgeiae*), clinical signs were minimal, and gross necropsy results were unremarkable, but histopathologic examination showed peripheral lymph node hyperplasia, splenic follicular hyperplasia, lymphocytic cholangitis/pericholangitis, lymphocytic hepatitis, lymphoplasmacytic myocarditis, and interstitial lymphocytic nephritis (26). In humans, *B. clarridgeiae* has never been isolated or detected by molecular methods. However, *B. clarridgeiae* could be a minor causative agent of CSD, as the presence of *B. clarridgeiae* antibodies were reported in a suspect case of CSD and in a patient with a chest-wall abscess (reviewed in [2]). Furthermore, anti-flagella (FlaA)–specific antibodies against *B. clarridgeiae* were detected by immunoblotting in 28 (3.9%) of 724 patients detected by immunoblotting in 28 (3.9%) of 724 patients with lymphadenopathy but in none of 100 healthy controls. However, substantial cross-reactivity between *B. henselae* and *B. clarridgeiae* detected by indirect fluorescence antibody assay was noted in human sera in a recent study from Japan (2).

### B. koehlerae

*B. koehlerae* is a *Bartonella* sp. that has rarely been isolated from domestic cats worldwide, as it is a very fastidious bacterium (2,4). Until recently, it had been isolated only from 2 cats in California and 1 cat in France (2,4,27). The first human case of *B. koehlerae* endocarditis was reported from Israel in 2004 (2). Furthermore, these authors were able to isolate *B. koehlerae* from a bacteremic stray cat from that country.

### B. quintana and B. bovis

A few suspect cases of CSD and cases of bacillary angiomatosis or endocarditis have been associated with *B. quintana*, for which the only risk factor identified was a contact with cats or cat fleas (3). Furthermore, the identification of *B. quintana* DNA in cat fleas (28) and recently in the dental pulp of a cat (3) has raised the question as to whether cats might be a possible source of human infection. However, *B. quintana* has not yet been isolated from naturally infected cats anywhere in the world where epidemiologic studies have been conducted to detect *Bartonella*-bacteremic cats. Similarly, 2 cats infected with

---

Table 2. *Bartonella henselae* seroprevalence in various cat and human populations from selected countries

| Country        | Cat seroprevalence (%) | Human seroprevalence (%) |
|----------------|------------------------|--------------------------|
|                | Stray  | Pet     | Reference | Healthy | Other | Reference |
| Sweden         | NA     | 1       | (19)      | 1       | NA    | (12)      |
| Japan          | NA     | 8.8–15.1; northern, 0–2; central 10.9–12.6; southern, 18–24 | (20)      | 4.5     | 11.0–15.0 (veterinarians) (13,14) |
| United States  | 81     | 27.9    | (11,21)   | 3.6–6   | 7.0 (veterinarians) (15) |
| Thailand       | 27.6†  | NA      | (22)      | 5.5     | NA    | (16)      |
| Italy          | 39.0   | 43.5    | (23)      | NA      | 8.5–61.6 (children) (17) |
| Jordan         | NA     | 32.0    | (24)      | NA      | NA    | (18)      |

*NA, not available.†Prevalence of bacteremic cats; no data available on seroprevalence.*
**Table 3. Clinical aspects of *Bartonella* infections in humans and dogs**

| *Bartonella* spp. | Humans | Symptoms | Dogs |
|-------------------|--------|----------|------|
| *B. claridgeiae*  | Cat-scratch disease | Endocarditis, neuroretinitis | Lethargy, anemia, weight loss |
| *B. elizabethae*  | Endocarditis, neuroretinitis | Endocarditis | Granulomatous hepatitis, peliosis hepatitis, epistaxis |
| *B. henselae*     | Cat-scratch disease, endocarditis, bacillary angiomatosis, peliosis hepatitis, granulomatous hepatitis, pseudotumoral lesions, arthritis, arthralgia, osteomyelitis, nodules, erythema, cutaneous petechiae, uveitis, neuroretinitis, purpura (Henoch-Schönlein), glomerulonephritis, perionyxis, periodontitis | Endocarditis | Not diagnosed in dogs |
| *B. grahamii*     | Neuroretinitis, bilateral retinal artery branch occlusions | Endocarditis | Not diagnosed in dogs |
| *B. koehlerae*    | Endocarditis | Endocarditis | Endocarditis, myocarditis, arrhythmia, uveitis, choroiditis, limping, splenomegaly, polyarthitis, epistaxis |
| *B. vinsonii* subsp. *anupensis* | Bacteremia, fever, arthralgia, neurologic disorders, endocarditis | Endocarditis | Not diagnosed in dogs |
| *B. vinsonii* subsp. *berkhoffii* | Endocarditis | Endocarditis | Endocarditis, myocarditis, arrhythmia, uveitis, choroiditis, limping, splenomegaly, polyarthitis, epistaxis |
| *B. washoensis*   | Fever, myocarditis | Endocarditis | Endocarditis |
| *B. quintana*     | Fever, bacteremia, endocarditis, bacillary angiomatosis | Endocarditis | Endocarditis |

*B. quintana* did not become bacteremic but seroconverted (29). Subsequently, both cats became bacteremic when challenged with *B. henselae*.

A few cases of *B. bovis* (formerly *B. weissii*) infections have been reported in cats from Illinois and Utah in the United States (1). The epidemiologic role of cats for this organism is still unknown.

**Dogs as Sentinels for Human Infections?**

Dogs can be infected with *B. v. berkoffii*, *B. henselae*, *B. claridgeiae*, *B. washoensis*, *B. elizabethae*, and *B. quintana* (2, P. Kelly et al., unpub. data). However, the role of dogs as a major reservoir of *Bartonella* spp. is not clear. Current evidence suggests that domestic dogs are more likely to be accidental hosts of various *Bartonella* spp., at least in nontropical regions. Nevertheless, domestic dogs could be one of the reservoirs for *B. v. berkoffii*, as it causes prolonged bacteremia in this species (3,6). The epidemiologic situation is quite distinct between tropical areas where several studies have shown a high prevalence of *B. v. berkoffii* antibodies, especially in stray dogs, and more northern latitudes, where very low antibody prevalence has been detected in domestic dogs, especially among pets. In sub-Saharan Africa, seroprevalence of 26% in dogs in Senegal and up to 65% in native dogs from Sudan has been reported (1). In North Africa, we found that 38% of 147 dogs from Morocco were seropositive for *B. v. berkoffii* (30). In 113 dogs from the Reunion Island, in the Indian Ocean, a seroprevalence of 18% was reported in stray dogs, whereas only 3% of dogs examined at veterinary clinics were seropositive, and no dog was bacteremic (31). In Thailand, 38% of sick dogs who exhibited fever, anemia, or thrombocytopenia were seropositive for *B. v. berkoffii* (1). On the contrary, studies in the United States and Europe reported a seroprevalence of <5% in domestic dogs; selected dog populations were at higher risk, including rural dogs and government working dogs (2). However, concerns about false-positive results in animals should be raised, as specificity and sensitivity of the tests for dogs have not been fully evaluated. In California, *B. v. berkoffii* has rarely been isolated from domestic dogs or detected by PCR, whereas coyotes (*Canis latrans*) appear to be a reservoir of this pathogen, as 35% of the coyotes tested in California were seropositive, and 28% of the coyotes tested within a highly disease-endemic region of California were bacteremic (2).

In domestic dogs, *B. v. berkoffii* is a cause of endocarditis (6) and, as in humans, the clinical spectrum of the infection attributed to this organism is expanding. *B. v. berkoffii* is now associated with cardiac arrhythmias, endocarditis and myocarditis, granulomatous lymphadenitis, granulomatous rhinitis, and epistaxis (6,32). In both humans and dogs, *Bartonella*-associated cases of endocarditis usually involve the aortic valve and are characterized by massive vegetative lesions (33). Based on serologic evidence, infection with *B. v. berkoffii* may also cause immune-mediated hemolytic anemia, neutrophilic or granulomatous meningocerebralitis, neutrophilic polyarthritis, cutaneous vasculitis, and uveitis in dogs (2).

Some other *Bartonella* spp. have infrequently been isolated from domestic dogs. *B. claridgeiae* and *B. washoensis* were isolated from cases of endocarditis (1,2), and *B. henselae* was isolated for the first time from a dog from Gabon (34). In the Gabon study, *B. claridgeiae* was isolated from 5 of 258 dogs tested (1.9%), which suggests a possible reservoir role for this *Bartonella* sp. in Africa (34). *B. henselae*, *B. elizabethae*, and *B. claridgeiae* DNA has also been detected from a few sick dogs with various clinical abnormalities (Table 3) (1,2,6). Endocarditis caused by *B. quintana* was recently diagnosed in a dog from the United States and a dog from New Zealand (P. Kelly et al., unpub. data). Two recent studies reported a


B. henselae antibody prevalence of 10% in healthy dogs in the eastern United States (35) and a prevalence of 14% of dogs in Zimbabwe (36). A much higher prevalence (27%) in sick dogs from the eastern United States was reported (35), which contrasts with the low B. henselae seroprevalence (<2%) in dogs examined at a university teaching hospital in northern California (37). A case-control study conducted on 305 dogs (102 dogs seropositive for B. henselae, B. v. berkhouii, or B. clarridgeiae and 203 seronegative dogs) suggested an association between the seropositive status and lameness, arthritis-related lameness, splenomegaly, and nasal discharge/epistaxis (37).

Unlike the domestic cat, for which clinical manifestations of natural infection is rarely documented, a wide range of clinical and pathologic abnormalities develop in dogs that are very similar to those observed in humans (32). Therefore, this species is an excellent sentinel and an important comparative model for human infections. To date, all Bartonella spp. identified in sick dogs are also pathogenic or potentially pathogenic in humans.

**Beyond the Fleas: New Emerging Vectors**

The primary mode of transmission of B. henselae to humans is through a cutaneous trauma caused mainly by the scratch of a cat. Transmission is less likely to occur by cat bite; shedding of B. henselae in cat saliva has not been clearly documented. The possibility of direct transmission of B. henselae to humans by the cat flea is something that has not been proven experimentally and is mainly hypothetical. However, the presence of cat fleas (Ctenocephalides felis) is essential for the maintenance of the infection within the cat population (6). B. henselae has been shown to multiply in the digestive system of the cat flea and survive several days in the flea feces (reviewed in [2]). Experimentally, only cats inoculated with flea feces compared to those on which fleas were deposited in retention boxes or that were fed fleas became bacteremic (38). Therefore, the main source of infection appears to be flea feces that are infected by contaminated cat claws.

Beside the cat flea, new possible vectors have been suggested. Bartonella DNA, including B. henselae, has been detected in *Ixodes ricinus* ticks collected on humans (9) and in *I. scapularis* ticks collected in households of persons coinfected with B. henselae and *Borrelia burgdorferi* (reviewed in [2]). B. quintana, B. henselae, and B. v. berkhouii DNA were also detected in questing *I. pacificus* ticks in California, and a few human cases of B. henselae infection were temporally related to a tick exposure in the United States (reviewed in [2]). Tick exposure was reported as a risk factor associated with CSD in humans (39). Similarly, tick exposure was determined to be a risk factor associated with B. v. berkhouii seropositivity in dogs (40). Additional indirect support for ticks as vectors of B. v. berkhouii in dogs relates to serologic or PCR evidence of concurrent infections with various tickborne organisms (6,33). The specific role of ticks in Bartonella transmission requires additional study, but several recent publications have reported a high prevalence of Bartonella spp. infection in ticks from various parts of the world. Finally, B. henselae type Marseille DNA was recently detected in a stable fly (8).

**Conclusion**

The number of zoonotic Bartonella species identified in the last 15 years has increased considerably. Pets have been identified as a notable reservoir of Bartonella species (i.e., cats and B. henselae or dogs and B. v. subsp. berkhouii in the tropics) and may play an important role as source for human infection. Furthermore, domestic dogs may represent excellent sentinels for Bartonella infection because of the wide diversity of the Bartonella spp. identified in canines, all of which are human pathogens. A better understanding of the modes of transmission and vectors involved in dog bartonellosis is an urgent priority to implement appropriate parasite control measures for pets.

Dr Chomel is the director of the World Health Organization/Pan American Health Organization Collaborating Center on New and Emerging Zoonoses at the University of California, Davis. His research focuses on Bartonella infections in domestic animals and wildlife and their impact on human health.

**References**

1. Chomel BB, Boulouis HJ, Breitschwerdt EB. Cat scratch disease and other zoonotic Bartonella infections. J Am Vet Med Assoc. 2004;224:1270–9.
2. Boulouis HJ, Chang CC, Henn JB, Kasten RW, Chomel BB. Factors associated with the rapid emergence of zoonotic Bartonella infections. Vet Res. 2005;36:383–410.
3. La VD, Tran-Hung L, Aboudharam G, Raout D, Drancourt M. Bartonella quintana in domestic cat. Emerg Infect Dis. 2005;11:1287–9.
4. Avidor B, Grady M, Efrat G, Leibowitz C, Shapira G, Schattner A, et al. Bartonella koehlerae, a new cat-associated agent of culture-negative human endocarditis. J Clin Microbiol. 2004;42:3462–8.
5. Kordick DL, Breitschwerdt EB. Persistent infection of pets within a household with three Bartonella species. Emerg Infect Dis. 1998;4:325–8.
6. Breitschwerdt EB, Kordick DL. Bartonella infection in animals: carriership, reservoir potential, pathogenicity, and zoonotic potential for human infection. Clin Microbiol Rev. 2000;13:428–38.
7. Chomel BB, Kasten RW, Floyd-Hawkins K, Chi B, Yamamoto K, Roberts-Wilson J, et al. Experimental transmission of Bartonella henselae by the cat flea. J Clin Microbiol. 1996;34:1952–6.
8. Chung CY, Kasten RW, Pfaff SM, Van Horn BA, Vayssier-Taussat M, Boulouis HJ, et al. Bartonella spp. DNA associated with biting flies from California. Emerg Infect Dis. 2004;10:1311–3.
SYNOPSIS

9. Sanogo YO, Zeaiter Z, Caruso G, Merola F, Shpynov S, Brouqui P, et al. *Bartonella henselae* in *Ixodes ricinus* ticks (Acari: Ixodida) removed from humans, Belluno province, Italy. Emerg Infect Dis. 2003;9:329–32.

10. Rolain JM, Brouqui P, Kocher JE, Maguina C, Dolan MJ, Raoult D. Recommendations for treatment of human infections caused by *Bartonella* species. Antimicrob Agents Chemother. 2004;48:3921–33.

11. Chomel BB, Abbott RC, Kasten RW, Floyd-Hawkins KA, Kass PH, Glaser CA, et al. *Bartonella henselae* prevalence in domestic cats in California: risk factors and association between bacteremia and antibody titers. J Clin Microbiol. 1995;33:2445–50.

12. Holmberg M, McGill S, Ehrenborg C, Wesslen L, Hjelm E, Darelid J, et al. Evaluation of human seroreactivity to *Bartonella* species in Sweden. J Clin Microbiol. 1999;37:1381–4.

13. Kikuchi E, Maruyama S, Sakai T, Tanaka S, Yamaguchi F, Hagiwara T, et al. Serological investigation of *Bartonella henselae* infections in clinically cat-scratch disease-suspected patients, patients with cardiovascular diseases, and healthy veterinary students in Japan. Microbiol Immunol. 2002;46:313–6.

14. Noah DL, Kramer CM, Verbsky MP, Rooney JA, Smith KA, Childs JD. Prevalence of antibodies to *Bartonella henselae* in domestic cats in Italy. Emerg Infect Dis. 2005;11:668–94.

15. Kikuchi E, Maruyama S, Sakai T, Tanaka S, Yamaguchi F, Hagiwara T, et al. Seroreactivity of *Bartonella henselae* and *Toxoplasma gondii* among healthy individuals in Thailand. J Vet Med Sci. 2000;62:635–7.

16. Massei F, Messina F, Gori L, Macchia P, Maggiore G. High prevalence of antibodies to *Bartonella henselae* among Italian children without evidence of cat scratch disease. Clin Infect Dis. 2004;38:145–8.

17. Al-Majali AM, Al-Qudah KM. Seroprevalence of *Bartonella henselae* and *Bartonella quintana* infections in children from Central and Northern Jordan. Saudi Med J. 2004;25:1664–9.

18. Hjelm E, McGill S, Blomqvist G. Prevalence of antibodies to *Bartonella henselae*, *B. elizabethae*, and *B. quintana* in Swedish domestic cats. Scand J Infect Dis. 2002;34:192–6.

19. Maruyama S, Boonmar S, Morita Y, Sakai T, Tanaka S, Yamaguchi F, et al. Seroprevalence of *Bartonella henselae* and *Toxoplasma gondii* in a dog population in Thailand. J Vet Med Sci. 2000;62:635–7.

20. Jameson P, Greene C, Regnery R, Dryden M, Marks A, Brown J, et al. Seroprevalence of *Bartonella henselae* antibodies in pet dogs throughout regions of North America. J Infect Dis. 1995;172:1145–9.

21. Maruyama S, Sakai T, Morita Y, Tanaka S, Kabeby H, Boonmar S, et al. Seroprevalence of *Bartonella henselae* and *B. elizabethae* in domestic cats in Japan. J Jpn Vet Med Assoc. 1997;210:342–4.

22. Regnery RL, Johnson AM, Shebly SL, Manzewitsch P, Beaver K, et al. Experimentally induced *Bartonella henselae* infections followed by challenge exposure and antimicrobial therapy in cats. Am J Vet Res. 1996;57:1714–9. Erratum in: Am J Vet Res. 1997;58:803.

23. Henn JB, VanHorn BA, Kasten RW, Kachani M, Chomel BB. *Bartonella vinsonii* subsp. *berkhoffii* antibodies in Moroccan dogs. J Trop Med Hyg. 2006; in press.

24. Muller S, Boulouis HJ, Viallard J, Beugnet F. Epidemiological survey of canine bartonellosis to *Bartonella vinsonii* subsp. *berkhoffii* and canine monocytic ehrlichiosis in dogs on the Island of Reunion. Rev Med Vet. 2004;155:377–80.

25. Breitschwerdt EB, Hegarty BC, Maggi R, Hawkins E, Dyer P. *Bartonella* species as a potential cause of epistaxis in dogs. J Clin Microbiol. 2005;43:2529–33.

26. Solano-Gallego L, Bradley J, Hegarty B, Signmon B, Breitschwerdt E. *Bartonella henselae* IgG antibodies are prevalent in dogs from southeastern USA. Vet Res. 2004;35:585–95.

27. Kelly PJ, Egophys GN, Raoult D. Antibodies reactive with *Bartonella henselae* and *Ehrlichia canis* in dogs from the communal lands of Zimbabwe. J S Afr Vet Assoc. 2004;75:116–20.

28. Henn JB, Liu CH, Kasten RW, VanHorn BA, Beckett LA, Kass PH, et al. Seroprevalence of antibodies against *Bartonella* species and evaluation of risk factors and clinical signs associated with seropositivity in dogs. Am J Vet Res. 2005;66:888–94.

29. Foil L, Andress E, Freeland RL, Roy AF, Rutledge R, Triece PC, O’Reilly KL. Experimental infection of domestic cats with *Bartonella henselae* by inoculation of *Cноnecophilades felis* (Siphonaptera: Pulicidae) feces. J Med Entomol. 1998;35:625–8.

30. Zangwill KM, Hamilton DH, Perkins BA, Regnery RL, Plikaytis BD, Hadler JL, et al. Cat scratch disease in Connecticut. Epidemiology, risk factors, and evaluation of a new diagnostic test. N Engl J Med. 1993;329:8–13.

31. Pappalardo BL, Correa MT, York CC, Peat CY, Breitschwerdt EB. Epidemiologic evaluation of the risk factors associated with exposure and seroreactivity to *Bartonella vinsonii* in dogs. J Am Vet Med Assoc. 1997;153:467–71.

Address for correspondence: Bruno B. Chomel, Department of Population Health and Reproduction, School of Veterinary Medicine, University of California, Davis, CA 95616, USA; fax: 530-752-2377; email: bchomel@ucdavis.edu

All material published in Emerging Infectious Diseases is in the public domain and may be used and reprinted without special permission; proper citation, however, is required.