# Update on canine Lyme disease

We've been aware of this tick-borne disease for more than two decades, but we still have many questions: How is it best diagnosed? Does it need to be treated in asymptomatic animals? What's the most effective form of prevention? These authors provide expert advice based on the most current research.

ASTRID NIELSSEN, DVM
ANTHONY CARR, Dr. med vet, DACVIM (internal medicine)
Department of Small Animal Clinical Sciences
Western College of Veterinary Medicine
University of Saskatchewan
Saskatoon, Saskatchewan S7N 5B4
Canada

JOHANNA HESELTINE, DVM
Department of Small Animal Clinical Sciences
Virginia-Maryland Regional College of Veterinary Medicine
Virginia Tech
Blacksburg, VA 24061-0442

ROB JOHNSON

IN 1977, a cluster of cases in children of what was initially suspected to be juvenile rheumatoid arthritis in Lyme, Conn., led to the description and identification of the causative agent of what became known as Lyme disease. Not long after the first cases in people were reported, the disease was also identified in the domestic canine population in endemic areas.

In 1982, the spirochete *Borrelia burgdorferi sensu lato* was identified by Dr. Willy Burgdorfer as the causative agent of Lyme disease. *Borrelia burgdorferi* is the largest of the *Borrelia* species. These bacteria are slow-growing, fastidious, microaerophilic organisms that require special culturing conditions.

## Transmission

*Borrelia burgdorferi* is transmitted by ticks of the *Ixodes ricinus* complex. The primary vectors are *Ixodes scapularis*, formerly called *Ixodes dammini* (deer tick, black-legged tick), in the northeastern and midwestern parts of the United States and eastern Ontario, Canada; *Ixodes pacificus* (western black-legged tick) in the western United States; and *Ixodes ricinus* (castor bean tick) in Europe. Although *B. burgdorferi* has been isolated from other arthropods, it is unlikely that these arthropods play a role in transmitting the spirochete to animal hosts.[1] Whether nonvector-mediated (through direct contact with an infected animal or its urine) and in utero transmission occur is controversial; these forms of transmission are not thought to be clinically relevant.[2]

## Prevalence

Although Lyme disease has been reported in people in 48 states and the District of Columbia, it is primarily a regional problem with established foci in the northeastern, north central, and western Pacific United States and eastern Ontario, Canada.[3] The proportion of dogs infected with *B. burgdorferi* in endemic areas can exceed three-quarters of the domestic canine population; however, it has been estimated that only 5% of infected dogs develop clinical signs attributable to Lyme disease.[4]

## Clinical manifestations

In dogs, Lyme disease is typically manifested by an acute or subacute arthritis. The first case report of *B. burgdorferi*-associated arthritis in a dog was published in 1984.[5] Although earlier attempts had failed, an experimental model was developed in 1992 that successfully fulfilled Koch's postulates for *B. burgdorferi* as the causative agent of fever, anorexia, depression, and limb or joint dysfunction in dogs.[6] Several successful models for the induction of *B. burgdorferi*-associated arthritis in dogs have since been developed.

Dogs most commonly present with a history of sudden lameness involving one or more joints. Articular

swelling, lymphadenopathy and lymph node enlargement, fever, anorexia, and lethargy may occur in association with the lameness. Experimentally, signs develop two to five months after infection, and the episodes of lameness may last only three or four days, even in the absence of therapy, and recur at intervals of several weeks or months.[7] It has not been determined whether these bouts of lameness are likely to persist or to follow a more chronic or progressive course in naturally infected dogs in the absence of therapy.

Other clinical signs less frequently attributed to Lyme disease in seropositive dogs include a protein-losing glomerulopathy resulting in renal failure,[8-10] heart block secondary to myocarditis,[11] various manifestations of neurologic disease,[12-14] and ophthalmic disease.[15] None of these disease manifestations have been noted in the experimental models of Lyme disease in dogs, although the limited numbers and breed selection in these models may have made it unlikely that unusual signs would have been noted. The protein-losing glomerulopathy postulated to be caused by *B. burgdorferi* infection (Lyme nephritis) has been associated with an acute, progressive renal failure frequently seen in conjunction with peripheral edema.[8,9] Unlike Lyme arthritis, this condition is not responsive to antibiotics or other therapies and has been fatal in all reported cases.[8,9] Labrador and golden retrievers appear to be overrepresented in the population of dogs affected with Lyme nephritis.[8,9]

## Diagnosis

Patients with Lyme disease typically have no clinically relevant complete blood count or serum chemistry profile abnormalities. Synovial fluid analysis during episodes of acute lameness usually reveals increased cellularity, with white blood cell counts ranging from 5,000 to 100,000 cells/µl.[5] Neutrophils predominate (> 95%), and protein concentrations and turbidity are increased.[5]

Attempts at culture and isolation of the spirochete from blood or urine samples are usually unrewarding. Recently, polymerase chain reaction (PCR) technology has proved useful in identifying the organism in tissues, but its sensitivity is relatively low when used to assess blood or urine.[2] It is also costly and lacks widespread accessibility.[2]

Serologic procedures such as the indirect immunofluorescent antibody test and the ELISA have typically been used to try to document exposure to *B. burgdorferi*. Since clinical signs usually do not develop for two to five months after exposure and IgG antibodies are detectable four to six weeks after exposure, antibody titers should be detectable in dogs with clinical signs of Lyme disease.[7]

The ELISA has become the most widely used test to detect anti-*B. burgdorferi* antibodies in veterinary medicine and has been refined over the years. *Borrelia burgdorferi* titer results from different laboratories are not typically comparable because of variations in the assays used. The results should be interpreted as positive, equivocal, or negative, as determined by the laboratory's own validation criteria.[16] An experimental study of an in-house commercial ELISA raised some concern about its sensitivity in identifying *B. burgdorferi* exposure.[17] The study indicated a sensitivity of 82%, revealing that cases of borreliosis could be missed if excessive diagnostic value was placed on a negative ELISA result.[17]

Vaccination against *B. burgdorferi* complicates the serodiagnosis of a dog suspected of having Lyme disease because of the presence of vaccine-induced antibody titers. The Western blot technique has been used in these animals and in others suspected of having a positive titer due to cross-reacting antibodies from other causes. This technique has been extensively investigated and has been found to effectively differentiate among naturally infected

dogs, vaccinated dogs, naturally infected dogs that have also been vaccinated, and dogs that have a positive ELISA due to cross-reacting antibodies from other causes.[16,18] It can also identify dogs suspected of having Lyme disease that have negative ELISA results.[17] It is not useful, however, in distinguishing between seropositive animals that have clinical disease and those that are asymptomatic.[16,18] A new specific ELISA recently developed and approved for use in dogs (Canine SNAP 3Dx—IDEXX) also appears to be able to differentiate among naturally infected dogs, vaccinated dogs, and dogs with cross-reacting antibodies secondary to other disease.[19] It holds promise in helping to simplify and clarify the confusing world of canine Lyme disease serodiagnosis.

A diagnosis of Lyme disease is still a diagnosis of exclusion. Other disease processes such as rheumatoid arthritis, other infectious or immune-mediated arthritides, bone disease, degenerative joint disease, Rocky Mountain spotted fever, ehrlichiosis, and bacterial endocarditis have to be ruled out. The criteria for diagnosing Lyme disease in dogs include 1) a history of exposure to *Ixodes* species ticks in an endemic area, 2) typical clinical signs, 3) a positive serologic test result from a properly validated test, and 4) a prompt response to antibiotic therapy.[2] It would be unusual for a dog with clinical Lyme disease to not fulfill all these criteria. In dogs suspected of having atypical clinical manifestations of Lyme disease, further diagnostic tests to document the role of *B. burgdorferi* in the disease may be useful (*e.g.* measuring paired cerebrospinal fluid and serum titers in a dog suspected of having central nervous system disease or obtaining a renal biopsy sample in a dog suspected of having Lyme nephritis).

## Treatment

Antimicrobial therapy is the mainstay of treatment for Lyme disease. An improvement in the animal's condition is typically expected within 48 hours after you initiate treatment. Several tetracyclines and β-lactam antibiotics are effective against the organism. Doxycycline (5 to 10 mg/kg orally every 12 to 24 hours) is frequently used because of its lipid solubility and relatively low cost. It should not be used in growing dogs. Ampicillin and amoxicillin (20 mg/kg orally every eight hours) have also been used frequently and successfully in dogs. Treatment is usually continued for 30 days. Nonsteroidal anti-inflammatory drugs may also initially be used for symptomatic treatment. Dogs typically respond well to initial antibiotic therapy, with few cases of chronic nonresponsive disease reported.[2] Dogs with recurrent infection, whether due to repeated exposure or recrudescence of the initial infection, usually respond well to the same treatment administered during the initial episode of infection.[2]

**Although antibiotic therapy is efficacious in relieving the signs of Lyme disease in dogs, it does not eliminate the organism.**

Although antibiotic therapy is clinically efficacious in relieving the signs of Lyme disease in dogs, recent investigations reveal that it does not eliminate the organism.[9,20,21] This may help explain why most animals that have been appropriately treated for Lyme disease have persistent antibody titers against *B. burgdorferi*. It appears that chronic intractable signs of Lyme disease, seen occasionally in people, are exceedingly rare in dogs.

Antibiotic therapy in asymptomatic dogs known to have been exposed to *Ixodes* species ticks or to be seropositive is likely not indicated in most instances, because the incidence of clinical disease in seropositive dogs is relatively low and such therapy is unlikely to eliminate the infection. The high rate of re-exposure in endemic areas also makes prophylactic therapy impractical for many animals.

Research in people has indicated that chronic Lyme arthritis that does not respond to appropriate antibiotic therapy may have an immunogenetic basis.[22] It is hypothesized that in genetically susceptible individuals, *B. burgdorferi* may trigger an ongoing immune-mediated response. This situation may exist in dogs as well and thereby explain the occasional affected animal that does not respond to appropriate therapy or perhaps even develops atypical signs of Lyme disease such as Lyme nephritis.

## Prevention

Vector control plays an important role in preventing infection.[23] Tick repellents marketed for veterinary use, such as products containing permethrin or amitraz, among others, can be used to control or prevent the ticks from successfully feeding. In a small experimental study, amitraz-impregnated collars were found to successfully prevent *B. burgdorferi* transmission in all four treated dogs, whereas seroconversion was noted in the four controls.[24] Daily grooming to remove

ticks may also prove helpful.

A whole-cell killed bacterin vaccine has been available to protect dogs against Lyme disease for the past 10 years. More recently, recombinant vaccines containing the outer surface protein A (OspA) antigen alone have been developed. Antibodies to this antigen have been found to reduce the number of spirochetes within the feeding tick and interfere with the movement of spirochetes into the tick's salivary glands, thereby effectively preventing transmission of the spirochete to the animal host.[25,26] The antibody response to the whole-cell bacterin also results in high concentrations of OspA antibodies and, therefore, likely prevents infection through the same mechanism as the recombinant OspA vaccines.[27] High concentrations of anti-OspA antibodies do not typically develop in natural infection, so the effect seen in an infected tick feeding on a previously naturally infected dog will not be the same as described above, though anti-OspC antibodies may have some effect within the tick. Within the past few years, recombinant OspA vaccines have been approved for use in people to prevent Lyme disease. The shift in human medicine from focusing on the development of a whole-cell vaccine to a subunit vaccine occurred because of concern that a whole-bacterin vaccine might trigger autoimmunity.[28] There are still concerns that the recombinant OspA vaccine may also trigger autoimmunity. It has recently been demonstrated that the human leukocyte function-associated antigen 1 (LFA-1) shares sequence homology with OspA.[29] There is particular concern that people with certain haplotypes, who are susceptible to treatment-resistant Lyme disease-associated chronic arthritis, may be prone to developing such an autoimmune response. Several animals identified in the literature as having demonstrated signs of Lyme arthritis or Lyme nephritis have had evidence of vaccination against Lyme disease but not natural exposure.[16,27]

In North America, the relative OspA homogeneity among *B. burgdorferi* strains means that vaccines derived from these strains should protect against challenges with most *B. burgdorferi* organisms within this region.[30] Indeed, the vaccines have demonstrated good efficacy in experimental and field tri-

als.[31-35] But as previously described, they do not prevent the development of clinical disease in previously seropositive dogs, because although the immune response mounted secondary to the vaccine effectively prevents spirochete inoculation from an infected tick into the vaccinated host, it will not eliminate the organism from a currently infected animal.

Because of the increasing concern within the veterinary community and general population regarding vaccination protocols in small-animal practice, Lyme disease vaccination in dogs has generated a lot of controversy. The arguments against vaccination are understandable, given the relatively low incidence of clinical disease in the seropositive population; a typically prompt response to appropriate antibiotic therapy; and a vaccine that is less than 100% efficacious and possibly associated with immediate, delayed, or even long-term sequelae. But the arguments for vaccination also are comprehendible, since vaccination might prevent cases of Lyme arthritis that do not respond to treatment; prevent cases of Lyme nephritis that are inevitably fatal; and protect dog populations in endemic areas against *B. burgdorferi* infection without causing long-term complications. Despite the conflicting arguments, what seems clear at this time is that universal vaccination is not indicated and should not replace appropriate vector control. In addition, dogs should be selected for vaccination based on their residence in, or travel to, an endemic area and their likely exposure to tick populations.

## Public-health aspects

Although dogs appear to be sentinel hosts for Lyme borreliosis, they are not reservoirs for human infection. The bite of an infected tick transmits the disease to both dogs and people. While dogs may carry infected ticks indoors and into more direct contact with people, the ticks usually do not detach until feeding is completed, and once they have fed they do not reattach to another host. But keep in mind that though the risk appears to be low, it is possible that improper handling of an infected tick during removal from its host, resulting in midgut rupture, could allow spirochete transmission through abraded skin or mucous membranes. So be sure to inform clients about appropriate tick handling and control measures to minimize this risk.

## Conclusion

Although much progress has been made in the past two decades, there is still much left to be learned about canine Lyme disease. We hope that, with both the development of better experimental models and the collaboration of clinical practices in endemic areas, many of the remaining questions will be answered. In the interim, it is important to remember that a positive serologic test result for Lyme disease is not a definitive diagnosis for this disease, and a negative serologic test result does not necessarily rule it out. A thorough and systematic approach is required in working up cases of suspected canine Lyme disease, and all clinical criteria, along with response to appropriate antibiotic therapy, must be considered.

### REFERENCES


**1.** Magnarelli, L.A.; Anderson, J.F.: Ticks and biting insects infected with the etiologic agent of Lyme disease, *Borrelia burgdorferi*. *J. Clin. Microbiol. 26* (8):1482-1486; 1988.

**2.** Appel, M.J.G.; Jacobson, R.H.: CVT update: Canine Lyme disease. *Kirk's Current Veterinary Therapy XII Small Animal Practice* (J.D. Bonagura, ed.). W.B. Saunders, Philadelphia, Pa., 1995; pp 303-309.

**3.** Greene, C.E. *et al.*: Lyme borreliosis. *Infectious Diseases of the Dog and Cat*, 2nd Ed. (C.E. Greene, ed.). W.B. Saunders, Philadelphia, Pa., 1998; pp 282-293.

**4.** Levy, S.A.; Magnarelli, L.A.: Relationship between development of antibodies to *Borrelia burgdorferi* in dogs and the subsequent development of limb/joint borreliosis. *JAVMA 200* (3):344-347; 1992.

**5.** Lissman, B.A. *et al.*: Spirochete-associated arthritis (Lyme disease) in a dog. *JAVMA 185* (2):219-220; 1984.

**6.** Wasmoen, T.L. *et al.*: Examination of Koch's postulates for *Borrelia burgdorferi* as the causative agent of limb/joint dysfunction in dogs with borreliosis. *JAVMA 201* (3):412-418; 1992.

**7.** Appel, M.J.G. *et al.*: Experimental Lyme disease in dogs produces arthritis and persistent infection. *J. Infect. Dis. 167* (3):651-664; 1993.

**8.** Sanders, N.A.: Canine "Lyme nephritis." *Proc. ACVIM*, ACVIM, Seattle, Wash., 2000; pp 627-628.

**9.** Dambach, D.M. *et al.*: Natural disease: Morphologic, immunohistochemical, and ultrastructural characterization of a distinctive renal lesion in dogs putatively associated with *Borrelia burgdorferi* infection: 49 cases (1987-1992). *Vet. Pathol. 34* (2):85-96; 1997.

**10.** Grauer, G.F. *et al.*: Renal lesions associated with *Borrelia burgdorferi* infection in a dog. *JAVMA 193* (2):237-239; 1988.

**11.** Levy, S.A.; Duray, P.H.: Complete heart block in a dog seropositive for *Borrelia burgdorferi*. Similarity to human Lyme carditis. *J. Vet. Intern. Med. 2* (3):138-144; 1988.

**12.** Mandel, N.S. *et al.*: Intrathecal production of *Borrelia burgdorferi* specific antibodies in a dog with central nervous system Lyme borreliosis. *Compend. Cont. Ed. 15*:581-585; 1993.

**13.** Azuma, Y. *et al.*: Neurologic abnormalities in two dogs with suspected Lyme disease. *Microbiol. Immunol. 37* (4):325-329; 1993.

**14.** Feder, B.M. *et al.*: *Borrelia burgdorferi* antibodies in canine cerebrospinal fluid. *Proc. ACVIM,* ACVIM, New Orleans, La., 1991; p 892.

**15.** Munger, R.J.: Uveitis as a manifestation of *Borrelia burgdorferi* infection in dogs. *JAVMA 197* (7):811; 1990.

**16.** Jacobson, R.H. *et al.*: Lyme disease: Laboratory diagnosis of infected and vaccinated symptomatic dogs. *Semin. Vet. Med. Surg. (Small Anim.) 11* (3):172-182; 1996.

**17.** Sheets, J.T. *et al.*: Evaluation of a commercial enzyme-linked immunosorbent assay for detection of *Borrelia burgdorferi* exposure in dogs. *JAVMA 216* (9):1418-1422; 2000.

**18.** Gauthier, D.T.; Mansfield, L.S.: Western immunoblot analysis for distinguishing vaccination and infection status with *Borrelia burgdorferi* (Lyme disease) in dogs. *J. Vet. Diagn. Invest. 11* (3):259-265; 1999.

**19.** Philipp, M.T. *et al.*: New standards for Lyme disease diagnosis. *Compend. Cont. Ed. 24* (suppl. 1A):24-27; 2002.

**20.** Straubinger, R.K. *et al.*: Status of *Borrelia burgdorferi* infection after antibiotic treatment and the effects of corticosteroids: An experimental study. *J. Infect. Dis. 181* (3):1069-1081; 2000.
**21.** Straubinger, R.K. *et al.*: Persistence of *Borrelia burgdorferi* in experimentally infected dogs after antibiotic treatment *J. Clin. Microbiol. 35* (1):111-116; 1997.
**22.** Kalish, R.A. *et al.*: Association of treatment-resistant chronic Lyme arthritis with HLA-DR4 and antibody reactivity to OspA and OspB of *Borrelia burgdorferi. Infect. Immun. 61* (7):2774-2779; 1993.
**23.** Piesman, J. *et al.*: Duration of tick attachment and *Borrelia burgdorferi* transmission. *J. Clin. Microbiol. 25* (3):557-558; 1987.
**24.** Elfassy, O.J. *et al.*: Efficacy of an amitraz-impregnated collar in preventing transmission of *Borrelia burgdorferi* by adult *Ixodes scapularis* to dogs. *JAVMA 219* (2):185-189; 2001.
**25.** Sadziene, A.; Barbour, A.G.: Experimental immunization against Lyme borreliosis with recombinant Osp proteins: An overview. *Infection 24* (2):195-202; 1996.
**26.** de Silva, A.M. *et al.*: Influence of outer surface protein A antibody on *Borrelia burgdorferi* within feeding ticks. *Infect. Immun. 67* (1):30-35; 1999.
**27.** Barthold, S.W. *et al.*: Serologic responses of dogs naturally exposed to or vaccinated against *Borrelia burgdorferi* infection. *JAVMA 207* (11):1435-1440; 1995.
**28.** Wormser, G.P.: Prospects for a vaccine to prevent Lyme disease in humans. *Clin. Infect. Dis. 21* (5):1267-1274; 1995.
**29.** Gross, D.M. *et al.*: Identification of LFA-1 as a candidate autoantigen in treatment-resistant Lyme arthritis. *Science 281*:703-706; 1998.
**30.** Recommendations for the use of Lyme disease vaccine: Recommendations of the Advisory Committee on Immunization Practices. *MMWR Recomm. Rep. 48* (RR-7):1-17; 1999.
**31.** Rice Conlon, J.A. *et al.*: Efficacy of a nonadjuvanted, outer surface protein A, recombinant vaccine in dogs after challenge by ticks naturally infected with *Borrelia burgdorferi. Vet. Ther. 1* (2):96-107; 2000.
**32.** Ma, J. *et al.*: Safety, efficacy, and immunogenicity of a recombinant Osp subunit canine Lyme disease vaccine. *Vaccine 14* (14):1366-1374; 1996.
**33.** Chang, Y.F. *et al.*: Recombinant OspA protects dogs against infection and disease caused by *Borrelia burgdorferi. Infect. Immun. 63* (9):3543-3549; 1995.
**34.** Levy, S.A. *et al.*: Performance of a *Borrelia burgdorferi* bacterin in borreliosis-endemic areas. *JAVMA 202* (11):1834-1838; 1993.
**35.** Chu, H.J. *et al.*: Immunogenicity and efficacy study of a commercial *Borrelia burgdorferi* bacterin. *JAVMA 201* (3):403-411; 1992.

## CE Questions 2 CE hours

*You can earn two hours of Continuing Education credit from Kansas State University by answering the following questions on canine Lyme disease. Circle only the best answer for each question, and transfer your answers to the form on page 624.*

### Article #1

**1.** The causative agent of Lyme disease, *B. burgdorferi*, is a bacterium that is:
a. Fast-growing

b. Anaerobic
c. Readily cultured
d. Free-living
e. None of the above

**2.** Clinically relevant transmission of *B. burgdorferi* occurs primarily via:
a. *Ixodes* species ticks
b. Ticks and nontick arthropod vectors
c. Horizontal transmission
d. Vertical transmission
e. All of the above

**3.** In some endemic areas, most dogs (> 75%) have been infected with *B. burgdorferi*. Estimates of the incidence of clinical disease in these infected dogs is:
a. 95%
b. 75%
c. 50%
d. 25%
e. 5%

**4.** Which is not a typical clinical sign in an acute case of canine Lyme disease?
a. Sudden lameness involving one or more joints
b. Fever
c. Lymphadenopathy
d. Petechiae or ecchymoses
e. Lethargy

**5.** With respect to the diagnosis of canine Lyme disease, which of the following is false?
a. Typically no clinically relevant complete blood count or serum chemistry profile abnormalities are present.
b. Synovial fluid analysis may reveal increased cellularity.
c. Attempts to culture or isolate the organism from blood or urine samples is usually unrewarding.
d. By the time dogs exhibit clinical signs of Lyme disease, antibody titers are not detectable.
e. A Western blot may be useful for determining whether a dog that has been vaccinated for Lyme disease (and therefore has a positive ELISA result) has also been naturally exposed to *B. burgdorferi.*

**6.** Which is a reasonable initial differential diagnosis for a dog with a typical clinical presentation of Lyme disease?
a. Rheumatoid arthritis
b. Idiopathic immune-mediated polyarthritis
c. Rocky Mountain spotted fever
d. Ehrlichiosis
e. All of the above

**7.** Which statement regarding treatment for canine Lyme disease is false?
a. An improvement in the animal's condition is typically seen within 48 hours after treatment is initiated.
b. Antibiotic therapy is usually continued for 30 days.
c. Dogs typically respond well to therapy, with few cases of nonresponsive disease reported.
d. Dogs with recurrent infection typically require the use of an alternative antibiotic, because of the development of resistance.
e. Although antibiotic therapy may resolve the clinical signs of canine Lyme disease, it likely does not completely eliminate the organism.

**8.** Which statement regarding prophylactic antibiotic therapy of a dog known to have tick exposure or to be seropositive for *B. burgdorferi* is most appropriate?
a. Given the high incidence of clinical disease in seropositive dogs, prophylactic treatment of these dogs is warranted.
b. Given that infection can be readily eliminated with appropriate antibiotic treatment, all tick-exposed and seropositive dogs should be prophylactically treated.
c. Given the low incidence of re-exposure in endemic areas, prophylactic therapy is practical and reasonable in exposed and seropositive animals.
d. Prophylactic therapy is not indicated in most cases of tick exposure or positive serology.
e. To eliminate the carrier status, all exposed or seropositive dogs should be treated with a course of antibiotics before vaccination for Lyme disease.

**9.** Which statement regarding the prevention of canine Lyme disease is false?
a. Vector control can play an important role in preventing infection.
b. Amitraz-impregnated collars have not been shown experimentally to prevent *B. burgdorferi* transmission.
c. Daily grooming may be helpful in preventing *B. burgdorferi* transmission.
d. Universal vaccination with either a whole-cell bacterin or OspA subunit vaccine is not indicated.
e. The decision to vaccinate individual dogs should be based on residence in or travel to an endemic area and the risk of tick exposure.

**10.** Which statement regarding the public health aspects of canine Lyme disease is true?
a. Lyme disease is transmitted to both dogs and people by the bite of an infected tick.
b. Dogs are not reservoirs for human Lyme disease infection.
c. Because the ticks do not usually detach and feed on multiple hosts, dog owners in endemic areas are not thought to have a significantly increased risk of exposure to *B. burgdorferi*, despite the fact that their pets may carry ticks indoors.
d. It is prudent to inform clients about appropriate tick handling and control measures, since although the risk is likely very low, it may be possible that midgut rupture of an infected tick during removal from its host could result in spirochete transmission through abraded skin or mucous membranes.
e. All of the above