Emerging Tick-borne Diseases

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In 1992, the Institute of Medicine (IOM) published the first treatise on ''emerging infections,'' a sobering warning of the resilience and plasticity of microbial organisms to adapt rapidly to changing environments and evolutionary pressures and to exploit newly created niches.[1] This prescient publication anticipated numerous types of public health threats, including diseases that were truly new (eg, sudden acute respiratory syndrome), were newly described (eg, hantavirus cardiopulmonary syndrome), had expanded their geographic endemicity (eg, West Nile virus), or had increased their pathogenicity (eg, methicillin-resistant *Staphylococcus aureus*). Several diseases also have ''emerged'' through facilitated transmission as a consequence of increased numbers and density of susceptible individuals (eg, opportunistic infections of HIV patients), permeation of geographic barriers (eg, H5:N1 avian influenza), or malicious intentional dissemination (eg, *Bacillus anthracis*).

Vector-borne diseases are particularly prone to the environmental pressures that contribute to changes in the ecology and the emergence of disease pathogens. These diseases are defined by and are dependent on climate and habitat that are compatible with the biologic needs of the microbiologic pathogens, their arthropod vector(s), and their mammalian reservoir(s). Ecologic changes on the macro scale (eg, global climate change) or micro scale (eg, suburban development) can alter established geographic and epidemiologic domains of vector-borne diseases.

Emerging infections are not a concern that is isolated to public health. The IOM report emphasized that ''[t]he significance of zoonoses in the emergence of human infections cannot be overstated.'' Indeed, the complex cycles of vector-borne zoonoses often include multiple mammalian and non-mammalian vertebrate and invertebrate species. Humans and domestic canids are particularly intertwined in their respective roles in and risks for diseases transmitted by ticks. In addition to being susceptible to tick-borne diseases, dogs may serve as reservoirs for human pathogens, as definitive feeding hosts for vector ticks, as mechanical transporters of ticks, and as sentinel indicators of regional disease risk. Conversely, in the absence of centralized reporting for most canine diseases, surveillance and other data collected for

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tick-borne diseases in humans can lend insight into risks for veterinary patients. Surveillance, diagnosis, treatment, and prevention of tick-borne diseases in humans and dogs can yield mutually beneficial information for public and veterinary health.

This article highlights the epidemiology of tick-borne zoonoses of concern to humans and domestic pets in North America. Because a comprehensive review of these diseases is not possible in this brief space, readers desiring detailed information on clinical signs, diagnosis, and management are directed to recently published reviews and standard texts.[2–6]

TICKS

Ticks are arthropods belonging to the order Arachnida. They are free living but require a blood meal during at least one life stage. ''Soft'' ticks (family: Argasidae) attach to the host, complete feeding within a few minutes, and promptly detach. ''Hard'' ticks (family: Ixodidae) are protracted feeders and remain attached for up to several days before reaching repletion. Successive blood meals on different hosts permit the transmission of blood-borne pathogens from one host to another. Ticks species with catholic feeding preferences can transmit microbes from evolutionarily commensal reservoir species (eg, rodents) to incidental susceptible species (eg, humans). The risk of disease transmission therefore is determined by the prevalence of infectious ticks—a function of the number and infection prevalence of the pathogen's reservoir host—and by the likelihood of an encounter between an infected tick and a susceptible host—a function of both the numbers of ticks and susceptible hosts within a fixed area and their respective behaviors.

Approximately 400 species of ixodid ticks occur worldwide, but fewer than 100 occur in North America. Only a dozen or so North American tick species parasitize humans or dogs with any frequency and are known to transmit micro-organisms of medical significance. Usually only one or two species of tick can acquire, maintain, and transmit a given pathogen. Therefore, the distribution of disease risk is restricted by the necessity for sympatric coexistence of the microbial pathogen, a competent vector tick, a reservoir host, and a susceptible host. The risk of disease parallels the geographic and seasonal distribution of ticks; therefore, veterinarians should educate themselves about which tick species are present within their practice area. Veterinarians can consult entomologists at their state universities, county or state departments of public health, or local mosquito and vector control districts for information on tick prevalence and for assistance in identifying ticks recovered from their patients.

The regions of tick-borne disease risk are not necessarily static. The spatial and temporal boundaries of risk for a given tick-borne disease may fluctuate over the short or long term as favorable conditions expand or contract. Transient meteorologic phenomena in endemic areas (eg, a wet, mild winter) can extend the number of months in which ticks are active in a given year. Protracted or permanent climatologic change can transform previously nonendemic areas to habitat favorable to ticks (eg, warmer temperatures in higher elevations or upper latitudes). Similarly, the spatial dimension of a risk area may change physically through human encroachment into or modification of existing tick habitat or change practically by susceptible individuals increasing behaviors that facilitate contact with questing ticks. Many ''emerging'' tick-borne diseases may represent micro-organism–tick–mammal disease cycles that are not truly new but have been newly discovered and described as a consequence of direct or indirect changes in the risk area and, consequently, the empiric morbidity.

LYME BORRELIOSIS

Disease caused by spirochetes of the genus *Borrelia* has been recognized in Europe since the early 1900s. Disease caused by a *Borrelia* indigenous to North America was first reported in 1977 among a localized cluster of patients diagnosed with juvenile rheumatoid arthritis.[7] The spirochete, *Borrelia burgdorferi*, described in 1982, encompasses four groups, of which Group 1, *B. burgdorferi* sensu stricto, is the principal pathogenic strain in North America.[8,9] A myriad of clinical manifestations now is recognized, including dermatologic (characteristic erythema migrans rash), neurologic (encephalitis, meningitis, radiculoneuropathy), cardiologic (atrioventricular conduction deficits), and rheumatologic (mono- or oligoarticular arthritis).

B. burgdorferi is transmitted to mammalian hosts by ixodid ticks. *Ixodes scapularis* is the principal vector in the northeastern and upper Midwestern United States; *I. pacificus* is the vector along the Pacific Coast. Distribution of favorable tick habitat (temperate, humid forests near large bodies of water), feeding hosts (deer), and reservoir hosts (rodents) determine distribution of disease. In 2006, approximately 95% of the nearly 20,000 cases of Lyme borreliosis reported in the United States were in residents of the upper midwestern (Minnesota and Wisconsin), northeastern (New York, New Hampshire, Pennsylvania, Vermont, Rhode Island, Connecticut, Massachusetts, and Maine), and mid-Atlantic (Maryland, New Jersey, and Delaware) states.[10] Expanding human populations and the resultant environmental alterations in the twentieth century probably contributed to defining these areas of endemicity. Areas that until the early 1900s were heavily wooded were converted to agrarian land that reduced habitat for deer. As populations of deer (and their ticks) plummeted, *Ixodes muris*, a one-host tick that feeds on rodents, came to dominate the acarologic landscape. In the mid-twentieth century, agriculture succumbed to suburbanization and reforestation, leading to a resurgence of deer and their attendant ectoparasites, chiefly *I. scapularis*, in areas that overlapped with human habitation. The epidemic of Lyme borreliosis apparent in the late twentieth century reflected this potentiation of transmission in the peri-residential environment and also increased recognition among health care providers and the expanded availability of often highly sensitive but poorly specific diagnostic assays.

Dogs are susceptible to infection with *B. burgdorferi*, but clinical disease generally is milder, narrower in scope, and less frequent than in humans.[11] Only about 5% to 10% of dogs exposed to infected ticks develop clinical borreliosis.[12] Clinical borreliosis manifests chiefly as polyarthritis approximately 2 to 6 months after exposure and typically is self-limited. A small percentage of dogs also develop a protein-losing glomerulopathy.[13] Serologic studies have documented immunologic evidence of borreliosis in cats, but clinical illness is rare.[14]

Aside from their shared susceptibility, dogs contribute little to the public health concerns of Lyme borreliosis. Dogs are not an efficient reservoir for the spirochete, nor are they an important or preferred feeding host for *Ixodes* ticks. It has been hypothesized that dogs may introduce ticks into the peri-domestic environment from an outdoor, distant source. Although in theory a partially fed tick may present a slightly increased risk of disease transmission (because spirochetes already have migrated from the midgut to the salivary glands), ticks generally do not re-feed if detached before repletion. Because of their frequent encounters with ticks and ready seroconversion, dogs have been proposed as sentinels for humans' risk of Lyme borreliosis.[15] Targeted research studies using domestic dogs can help sketch broad areas where *B. burgdorferi* is present, but, because of the highly focal distribution of vector ticks, a

reliable range of endemicity is delineated better by surveillance of ticks and natural rodent hosts than by serologic or clinical evidence from incidentally infected hosts.

The diagnosis of Lyme borreliosis in both humans and dogs can be challenging. Clinical signs often are nonspecific and variable. Culture of the spirochete requires special media over a lengthy incubation period and often is unrewarding. Assays for circulating antibodies remain the most common means of laboratory confirmation despite recognized shortcomings.[16] Enzyme immunoassays (EIA) and immunofluorescent assays (IFA) based on the whole cell or subunits of the spirochete generally lack specificity. The US Centers for Disease Control and Prevention recommends a two-step procedure in which specimens yielding a positive or equivocal result on a screening EIA or IFA are confirmed by Western immunoblotting using specific interpretation criteria.[17] Recently developed assays that use IR6, an antigen that is highly conserved among *Borrelia* spp and is expressed transiently only in actively infected mammalian hosts, may make more specific screening tests possible.[18,19] A commercial test that uses a recombinant form of the IR6 (C6) seems to be more specific than whole-cell sonicates.[20] Nevertheless, seropositivity may not indicate active infection and should not be used as the sole criterion for diagnosis. Interpretation of laboratory results and decisions regarding treatment should be based on the likelihood of Lyme borreliosis in the patient, including clinical, laboratory, and epidemiologic factors (eg, region of country, outdoor activities, history of tick bite). Routine treatment of seropositive asymptomatic dogs generally is unwarranted, because most dogs do not develop clinical signs, illness often is self-limited, and injudicious antimicrobial treatment may contribute to emergence of antibiotic resistance in other flora with zoonotic potential.[21,22]

RICKETTSIOSES

Obligately intracellular bacteria in the order *Rickettsiales* cause several tick-borne diseases of human and veterinary medical importance. The family Rickettsiaceae contains bacteria of the genus *Rickettsia*, including *R. rickettsii*, the agent of Rocky Mountain spotted fever (RMSF). The family Anaplasmataceae encompasses several pathogens of humans and animals in the genera *Ehrlichia* and *Anaplasma* that formerly were grouped under the broad term ''ehrlichiosis.''

Rocky Mountain Spotted Fever

R. rickettsii is one of more than a dozen species of *Rickettsia* in the spotted fever group (SFG); these rickettsiae are closely related to typhus group *Rickettsia* spp (eg, *R. typhi*) but are distinct from other rickettsiae. RMSF is the most frequently reported rickettsial illness in humans in the United States; about 2300 cases were reported in 2006.[10] RMSF in humans is characterized by high fever, myalgia, severe headache, and a petechial or maculopapular rash of the extremities, including palms and soles. Case-fatality of untreated patients is 3% to 5%. The initial clinical signs of RMSF in dogs resemble those in humans: fever, myalgias, and petechiae/ecchymoses, chiefly of the mucous membranes. Damage to the vascular endothelium leads to hypoalbuminemia and development of extremital and cerebral edema. Hypotension, shock, and renal hypoperfusion and failure also may occur.

RMSF cases are distributed throughout much of the United States because of the ranges of its two principal tick vectors: *Dermacentor variabilis* (the American dog tick) in the southeastern and south central states, where more than 80% of cases occur, and *D. andersonii* (Rocky Mountain wood tick) in the Rocky Mountains and the Northwest. Other tick species such as *Amblyomma americanum* (the lone star

tick) and *Rhipicephalus sanguineus* (the brown dog tick) also can occasionally transmit *R. rickettsii*. *Rh. sanguineus*, a one-host tick whose preferred host is canids, was implicated recently in an outbreak of RMSF among humans and domestic dogs in Arizona,[23–25] a state where RMSF is rarely reported and *Dermacentor* ticks are uncommon. Investigators hypothesized that domestic dogs contributed directly to the outbreak by transporting ticks to the peri-domestic environment, supporting large populations of ticks in close proximity to human habitation, and possibly serving as a reservoir for the *Rickettsia*. Evaluation of archived sera indicate that free-roaming canids in Arizona were exposed to *R. rickettsii* at least a decade before this outbreak.[26]

Serologic assays are the most widely available laboratory diagnostic. Because of considerable cross-reactivity between SFG rickettsiae and the variable specificity of commercial assays,[27] documentation of a fourfold change in serum antibody titer between acute and convalescent specimens—ideally, submitted simultaneously and tested in parallel—is recommended. To avoid delay in initiating treatment, a provisional diagnosis may be made based on clinical compatibility, history and species of tick infestation, and epidemiologic indicators such as region of the country and season of year (chiefly late spring to early autumn). A single elevated IgM titer in a clinically compatible patient may be sufficient for confirmation. In contrast, because canine IgG to *Rickettsia* spp may persist for up to 10 months,[27–29] detection of IgG alone may not be clinically relevant.

Ehrlichioses and Anaplasmosis

Zoonotic members of the family Anaplasmataceae are pathogens of leukocytes and usually are grouped based on their leukocytotropic propensity. Monocytotropic *Ehrlichia* spp include closely related agents of human (*E. chaffeensis*) and canine (*E. canis*) ehrlichiosis. Members of the former granulocytotropic *E. phagocytophila* group—including pathogens of humans (human granulocytic ehrlichiosis agent), ruminants (*E. phagocytophila*), equids (*E. equi*), and other mammals—recently were reclassified collectively as *Anaplasma phagocytophilum*.[30] (A closely related thrombocytotropic pathogen of dogs [*A. platys*] has not demonstrated zoonotic potential.)

E. canis was the first of the monocytic ehrlichioses to be identified, described in dogs in Algeria in 1937. Canine monocytic ehrlichiosis came to the attention of Western nations in the 1960s when several hundred military dogs died of the disease while serving in Vietnam.[31] Monocytic ehrlichiosis in humans was first recognized in the United States in the 1980s and initially was attributed to *E. canis*.[32] Subsequent investigation identified a closely related but distinct rickettsia, given the name *E. chaffeensis*.[33] Despite profound serologic cross-reactivity among patients and greater than 98% homology based on 16S rRNA, *E. canis* and *E. chaffeensis* seem to be epidemiologically distinct. *E. chaffeensis* is restricted chiefly to the southeastern and south-central United States, deer are the likely reservoir host, and *A. americanum* is the principal tick vector; whereas *E. canis* is distributed worldwide, dogs serve as the reservoir, and *Rh. sanguineus* is the vector. Although dogs may be infected incidentally with *E. chaffeensis*, they seem to have limited susceptibility and no role in its maintenance.[34] Similarly, *E. canis* infection of humans is restricted to a few reported cases in South America.[35]

Another member of the *Ehrlichia* group, *E. ewingii*, has been identified as a pathogen of both dogs and humans. *E. ewingii* shares 98% genetic homology with *E. canis* and *E. chaffeensis*[36] and seems to resemble *E. chaffeensis* in its geographic distribution (the southeastern and south-central United States), tick vector (*A. americanum*), and seasonality (spring to autumn). *E. ewingii* differs from other members of this group in that it is chiefly granulocytotropic.[37] Canine granulocytic ehrlichiosis was first

described in a dog from Arkansas in 1971,[38] and *E. ewingii* was identified as the etiologic agent in 1992.[36] The first report of *E. ewingii* infections in humans was published in 1999,[37] describing four male patients from Missouri who presented with histories of tick bites and clinical illness consistent with ehrlichiosis; three of these patients were being treated with immunosuppressive therapy. The full contribution of *E. ewingii* to human morbidity remains undetermined but seems to be low.

A. phagocytophilum is a granulocytotropic rickettsia distinct from the *E. canis/chaffeensis* group. Evidence of natural infection with *A. phagocytophilum* has been identified in humans, horses, dogs, small ruminants, and some wild mammals.[39–42] Rare cases of a mild and self-limited infection with *A. phagocytophilum* have been reported in cats from the northeastern United States.[43] Because the rodent reservoirs (*Peromyscus* mice, *Neotoma* rats) and tick vectors (*Ixodes* spp) for *A. phagocytophilum* in the United States are similar to those for Lyme borreliosis, it shares similar geographic distribution and seasonality—that is, the northeastern and upper midwestern states from spring to early summer and autumn. *Ixodes* ticks can be coinfected with both organisms,[44,45] and concurrent infections with *A. phagocytophilum* and *B. burgdorferi* have been observed in humans.[46,47] The clinical likelihood and significance of coinfection with these pathogens in other species is unknown.

The distinctive leukocytotropisms of *Ehrlichia* spp and *A. phagocytophilum* offer a means of provisionally diagnosing and differentiating infections with these rickettsiae. During the acute phase of illness, binary fission of the rickettsiae within the phagosome produces membrane-bound intracytoplasmic aggregates called ''morulae.'' Morulae in circulating leukocytes can be observed directly in Romanovsky-stained blood or buffy coat smears. Identifying the leukocytic cell line containing morulae can narrow the list of possible rickettsial pathogens, but different rickettsia species within a leukocytotropic group (eg, granulocytotropic *E. ewingii* and *A. phagocytophilum*) cannot be discriminated further based on morulae prevalence or morphology. Although observation of intraleukocytic morulae is highly specific when performed by a trained microbiologist, it offers only low-to-moderate sensitivity, depending on when the specimen was collected and the type and proportion of leukocytes infected. Typically, both the proportion of patients in whom morulae are observed (< 5%–10% for monocytic morulae[48,49] and 25% for granulocytic morulae)[50] and the proportion of leukocytes containing morulae during active infection (1%–2% for monocytes[32] and up to 80% for neutrophils)[39,51] are quite low. Therefore, serology remains the principal, but not definitive, method for diagnosis. Cross-reactivity between ehrlichiae and *A. phagocytophilum* is common in both canine and human sera.[52,53] Differentiation may be confirmed by more specific assays (Western immunoblotting or polymerase chain reaction), when available, or may be inferred through demonstration of a fourfold change in titer between acute and convalescent specimens.

TULAREMIA

''Tularemia'' is a general term for the myriad of clinical manifestations that can occur following infection with the gram-negative bacillus, *Francisella tularensis*. *F. tularensis* is distributed widely throughout North America, because of multiple mammalian reservoir species, the persistence of the bacteria in the environment, and several competent arthropod vectors. Four species of ticks—*D. andersonii*, *D. variabilis*, *D. occidentalis*, and *A. americanum*—are recognized as true biologic vectors and reservoirs for *F. tularensis* at least one of which exists in almost any given region of the United States. Other routes of transmission include handling or ingestion of tissues

from an infected mammal (principally lagomorphs), ingestion of or inoculation with contaminated water through a break in the skin or mucous membrane, inhalation of contaminated dust, and mechanical transmission by other biting arthropods such as deer flies (*Crysops* spp) and mosquitoes. Because of the potential for respiratory exposure and the low inoculum (10–50 organisms) necessary to effect infection, *F. tularensis* is considered a Category A potential bioterrorism agent.[54]

The route of infection generally determines the scope of clinical manifestations. Humans most commonly are infected through direct contact or tick bite, leading to an ulceropapular lesion at the site of inoculation and localized lymphadenomegaly. The ulceroglandular form predominates in humans, but typhoidal, glandular, oculoglandular, and pneumonic forms also occur. The spectrum of illness in domestic animals seems to be much narrower. Dogs are exposed most frequently via tick bite but seem to be relatively resistant to infection; transient mild fever and anorexia have been reported.[55] Infection in cats is more severe; because cats are likely to be infected through predation and consumption of infected rodents or rabbits,[56] lymphadenopathy and ulcerations of the oropharynx are the most frequently observed signs.[57]

Infected dogs and cats may present a low risk of transmission to humans. Bites or scratches from cats have been associated with more than 50 human cases of tularemia.[58,59] Dogs are unlikely to serve as a direct source of transmission but may facilitate exposure by bringing infectious ticks, tissues (eg, rabbit carcasses), or water (eg, a saturated coat from a contaminated lake) into the peri-domestic environment. The bacterial load in suppurative lesions is low, but because only a few organisms are necessary to cause infection, veterinary staff should use barrier protection when handling patients suspected of having tularemia. Cultures and necropsies of suspect patients should be performed only in Biosafety Level 3 facilities.

POSSIBLE EMERGING TICK-BORNE ZOONOSES

Several pathogens recently have been identified for which transmission by ticks or the zoonotic potential have yet to be established. Some members of the genus *Bartonella* have long been associated with transmission by biting arthropods; for example, *B. quintana*, the agent of trench fever in humans, is transmitted by the human body louse. *B. henselae* is a widespread commensal bacterium among healthy domestic cats but causes bacillary angiomatosis (“cat scratch disease”) in humans who are bitten or scratched. Fleas harbor the organism, and contamination of skin breaks with flea excrement, rather than the feline scratch per se, seems to be required for infection. *B. henselae* also has been identified in attached and questing ticks,[60–62] but their competence as vectors has yet to be verified.[63] Infection with *B. vinsonii* has been associated with valvular endocarditis in some dogs and humans.[64–66] Serum antibodies to *B. vinsonii* have been detected in numerous surveys of both healthy and diseased wild and domestic canids.[67–71] Often these canids had concomitant heavy tick infestations and seroreactivity to other tick-borne pathogens (eg, *E. canis*), but at present there is no direct evidence that ticks are a competent vector of *B. vinsonii.*

A skin rash resembling the erythema migrans lesion of Lyme borreliosis has been described in residents of southern and central parts of the United States where *Ixodes* ticks and *B. burgdorferi* are rare.[72,73] The disease Southern tick-associated rash illness (STARI) is associated with bites from the lone star tick, *Amblyomma americanum,*[74] and has been linked provisionally to infection with *Borrelia lonestari*.[75] Human patients who have STARI show no serologic cross-reactivity on whole-cell and C6 ELISAs for *B. burgdorferi*.[76,77] Experimentally inoculated beagles developed detectable antibodies, but *B. lonestari* could not be re-isolated from blood.[78]

White-tailed deer are the only other vertebrate in which natural infection with *B. lonestari* has been identified.[79]

Three species of *Babesia* (*B. canis*, *B. gibsoni*, and *B. conradae*), an intraerythrocytic protozoan, have been described from North American dogs.[80,81] Although *B. gibsoni* is transmitted principally by ticks, contact transmission also has been strongly suggested, particularly among fighting breeds.[82,83] Despite a close phylogenetic relationship between *B. conradae* and *B. duncani*, a human piroplasm,[84] neither *B. conradae* nor other canine *Babesia* spp seem to be zoonotic.

PREVENTION

The simple and often singular mechanism by which tick-borne diseases are transmitted (viz, by tick bite) permits a multitude of avenues for prevention. No one technique is invariably effective, however, so an integrated program of several preventive components is desirable to maximize protection from infection.

Environmental modification through landscape management (eg, removal of leaf litter) or reduction in feeding hosts (eg, culling deer) can reduce tick populations but generally is impractical over the expansive area needed to be effective. Area application of acaricides can substantially reduce tick abundance around residential property but requires frequent re-application and may pose health risks for incidentally exposed nontarget animals. In contrast, topical acaricides directed at tick feeding hosts (eg, deer feeding stations, rodent bait boxes) reduce the concentration of chemical needed, but still require frequent visits to the stations by a large proportion of the targeted mammal population.

Susceptible individuals can alter their behavior and activities to limit the opportunity for contact with ticks. Simply stated, bites from ticks can be prevented by avoiding areas where ticks are present. If traffic in tick habitats is desirable or otherwise unavoidable, owners and pets should limit contact with uncultivated grasses, bushes, and shrubs that may harbor questing ticks. Dogs should be kept on leash and maintained in the middle of roads, paths, or other routes devoid of vegetation.

Ticks can be further dissociated from potential hosts through the use of physical or chemical barriers. Long pants and long-sleeved shirts can delay or confound the tick's attachment to the skin. Chemical repellents applied to clothing (eg, permethrin) or skin (eg, N,N-diethyl-meta-toluamide [DEET]) of humans can further deter questing ticks. The use of DEET on animals is not recommended and should be avoided. Control of ticks on dogs is facilitated by the availability of collars impregnated with permethrin or amitraz and topical solutions containing fipronil, imidacloprid, permethrin, or selamectin.[85–87] Amitraz-impregnated collars seem to be more effective in interrupting the tick life cycle and to be longer acting than topical applications of fipronil.[88] Amitraz and permethrin products are contraindicated for cats. Selamectin is effective in control of *Rh. sanguineus* and *D. variabilis* on dogs and is safe to use on cats.[89,90]

Dogs residing in areas highly endemic for Lyme borreliosis and subject to heavy tick infestation may benefit from immunization against *B. burgdorferi*. Reduced incidence of serum antibodies to *B. burgdorferi* and clinical borreliosis (ie, lameness) were observed among dogs vaccinated with a whole-cell bacterin.[91,92] Newer recombinant subunit vaccines based on the Osp A antigen of *B. burgdorferi* may interrupt transmission by complement-mediated lysis of the spirochete in the tick's gut soon after it begins its blood meal.[93] Vaccination against *B. burgdorferi* does not obviate the need for other measures to prevent tick bites, because the vaccine confers no cross-protection against other tick-borne pathogens.

Individuals should examine themselves, family members, and pets thoroughly after visiting tick-infested areas. Because *Ixodes* ticks do not transmit *B. burgdorferi* spirochetes efficiently until 24 to 48 hours after attachment, transmission of spirochetes pathogens can be prevented or interrupted by prompt recognition and removal of attached ticks.[94] A single dose of doxycycline administered within 72 hours of a recognized tick bite reduced infections with *B. burgdorferi* in humans,[95] but the efficacy and necessity of this prophylactic regimen for dogs and for other tick-borne pathogens has not been evaluated.

SUMMARY

Pets and their owners share susceptibility to several tick-borne diseases depending on their geographic location, season, and activities. When presented with a pet with possible tick-borne illness, veterinarians should take the opportunity to discuss the zoonotic disease risks with the owner. A comprehensive tick control program protects both pets and their owners by interrupting feeding opportunities for the tick and breaking the maintenance cycle of the pathogen. The veterinarian should consider the regional distribution of tick-borne diseases when formulating prevention strategies, diagnostic differentials, and therapeutic decisions. Because the complex cycles of microbial pathogens, vector ticks, environment, and mammalian hosts evolve continually and can lead to the emergence of tick-borne diseases in previously nonendemic areas, veterinarians should consult their local or state departments of public health for the most current information on which tick-borne diseases are of concern in their community.

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