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CANINE BORRELIOSIS - EPIDEMIOLOGY AND DIAGNOSTICS

Bogumiła Skotarczak

Department of Genetics, Faculty of Biology, Szczecin University, Szczecin, Poland

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Abstract: *Ixodes* ticks, vectors of *B. burgdorferi*, carry the spirochaetes to a number of vertebrates; however in natural conditions only the species from outside the forest biotope display the clinical form of borreliosis, mainly humans and dogs. In dogs, *B. burgdorferi* s.l. has been implicated as a cause of various disorders often resembling those observed in human borreliosis, including polyarthropathy, anorexia, malaise, and neurological dysfunction. In areas infested with *I. ricinus*, and *I. dammini* in the USA and known to be endemic for Lyme disease, veterinarians may suspect borreliosis in dogs with limb/joint disorders. In serological diagnosis, it should be considered that the occurrence of even specific antibodies does not necessarily mean the active disease or primary exposition to the pathogen. Studies on antibody titers in the sera of dogs either naturally or experimentally infected, and on their associations with clinical symptoms, indicate the same limitations of serological tests as in the diagnostic of human Lyme disease; therefore, PCR is the most appropriate method of diagnosis.

Address for correspondence: Bogumiła Skotarczak, M.D., Professor, Department of Genetics, Faculty of Biology, University of Szczecin, Piastów 40B, 71-065 Szczecin, Poland. E-mail: Bogumila_Skotarczak@sus.univ.szczecin.pl

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INRODUCTION

Borreliosis, or Lyme disease, is caused by spirochaetes that belong to the group *Borrelia burgdorferi* sensu lato. Canine borreliosis was first described in the USA in the 1980s [22, 27, 28], and in nearly all European countries in the recent years. Reports on canine infections by *B. burgdorferi* have come from Germany [2], the Netherlands [15, 19, 20], Belgium [34], France [10, 12], the United Kingdom [32], Slovakia [48], and Spain [11].

Ticks of the genus *Ixodes*, which are vectors of *B. burgdorferi*, carry the bacteria to many vertebrates, as the spirochaete does not exhibit any particular specificity towards the host species. In natural conditions, the clinical form of borreliosis is found only in species from outside the forest biotope, i.e. humans, dogs, cats, horses, and cows; however, most often it affects dogs and humans [1, 28, 29, 30, 31, 33, 38, 47]. Lack of clinical infection symptoms in forest animals means that a balanced state in this parasite-host system has been reached as a result of evolutionary long-term relation. On the other hand, the probably young relation with humans or dogs may be fatal for the host organism.

When a tick sucks blood, the spirochaetes *B. burgdorferi* first enter the skin of the host [6] and then spread to adjacent tissues where the infection stabilizes, not being eliminated by the host immune system [39]. The processes that initiate and sustain the disease have not been well-defined; however, it is known that *B. burgdorferi* occurs in the inflamed and chronically infected tissues of the host [4, 5, 17, 51, 56]. Each year in Europe, North America, and Asia, large numbers of people and animals are being infected, not all the infected individuals however, develop clinical symptoms of the disease [49]. Berglund *et al.* [3], Levy *et al.* [26], and Steere *et al.* [46] estimate that such individuals comprise 5% to 50% of those infected. It is not clear which factors decide the outcome of the infection; however, has been observed that many *Borrelia* cells in the tissues of experimentally infected mice might have developed an inflammatory response [37].

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Although human Lyme disease has been described in numerous papers, the canine borreliosis has not been thoroughly studied, despite great similarities. In dogs, Lyme disease develops most often in arthritic form, with inflammation of limb, usually carpal or tarsal joints; one or both joints swell, and groin and prescapular lymph nodes enlarge. These symptoms are accompanied by malaise (which is manifested by fever, lack of appetite, and fatigue), and lameness after a few days. Myocarditis rarely develops in canine borreliosis; however, in older dogs, renal form and neurological dysfunctions appear [7, 14, 17, 19, 25].

Many authors have reported that incidence of *B. burgdorferi* infections in humans and dogs depends on the occurrence of ticks, depending in turn on geographical location and biotope, which represent a risk indicator for both humans and dogs [19]. Dogs that live in areas infested with ticks, where human Lyme disease cases are recorded, develop antibodies against *B. burgdorferi* s. l. [16, 22, 28, 31, 51, 52]. Therefore, this type of epidemiologic study, with delimitation of endemic *Borrelia* areas, are necessary [31].

In *I. ricinus* ticks, collected in 10 stations in North-West Poland, we detected the DNA of 3 genospecies of *B. burgdorferi* s.l. by means of PCR [58]. Later, we detected the DNA of *Babesia microti* and *B. divergens*, as well as human granulocitar ehrlichiosis (HGE) factor, at first in individual *I. ricinus* specimens [40, 41, 42], and then observed a double and triple coinfection [43, 44]. The results of our studies, as well as those by other authors, confirm the risk of coinfection spreading to humans and dogs, which to a large extent makes diagnosis difficult. In the Netherlands, dogs are often infested by ticks, yet borreliosis is rarely diagnosed [19, 21]. Hovius *et al.* [18] traced the *Borrelia* DNA in *I. ricinus* ticks collected from dogs, and reported that Dutch dogs are exposed to infections by 4 genospecies of *B. burgdorferi* s.l. (B. *burgdorfei sensu* stricto, *B. afzelii, B. garinii, B. valaisiana*), and in Thailand dogs are infected with multispecies of pathogens (*Ehrlichia, Bartonella, Babesi,*) [55].

EPIDEMIOLOGY AND DIAGNOSTICS

Should our own dog be considered as a Lyme disease risk factor? People that rest or work in tick-infested areas, i.e. remain exposed to tick bites, exhibit an increased prevalence of antibodies to *B. burgdorferi* in comparison with control groups [8, 9, 23, 24, 35, 36]. Comparing the high rate of seropositivity for antibodies to *B. burgdorferi* with intensive outdoor activity, one might expect a similar outcome if hunting dogs were compared with the control ones [14]. It has been suggested that the owners of pet dogs in the USA face an increased risk of Lyme disease. If dogs were a direct source of ticks infecting people, the risk of Lyme disease among dog owners would indeed be higher [14]. Therefore, Goossens *et al.* [14] investigated whether there is a connection between a high level of antibodies in hunting dogs and their owners. No correlation was found between the seropositivity of hunters and their dogs, which led to the conclusion that direct transfer of ticks from dog to owner is probably insignificant. However, there might be a possibility of horizontal transfer of infection, as it has been reported that *B. burgdorferi* was passed from an experimentally-infected dog to a control one, and spirochaetes were isolated from dog urine [15].

Diagnostic. In North America [20], canine borreliosis has been diagnosed based on the presence or lack of the following criteria: 1) the presence of typical clinical symptoms; 2) exclusion, or differential diagnosis; 3) explicit response to an antibiotic; 4) evident contact with a tick or remaining in an endemic area; or 5) antibodies in blood serum. The last criterion represents a serious diagnostic indicator [18]. However, even specific antibodies do not definitely indicate an active disease or primary exposition to the pathogen. The majority (86%) of seropositive dogs examined for IgG by Goossens *et al.* [14] did not show any signs typical for borreliosis. Wieler *et al.* [57], as well as many other authors, demonstrate that serological tests may be deceptive in diagnosis, as a high proportion of dogs are seropositive without showing clinical symptoms. This problem is partly a result of cross reactions that occur between the antigens of *B. burgdorferi* and related bacteria (*Treponema* spp, *Bradyspira* sp, *Leptospira* spp).

Studies on antibody titers in the sera of either naturally or experimentally infected dogs, and on their associations with clinical symptoms, indicate the same limitations of serological tests as in the diagnosis of human Lyme disease [2, 14, 18, 19, 21,54]. However, the studies by Hovius [18] suggest that it is possible to detect some cases of canine borreliosis based on the clinical criteria referred to as “bad feeling before lameness”, although most cases of the disease proceed with variable symptoms, which may result from infections by various genospecies of *B. burgdorferi* s. l. or their coinfections, which has been confirmed in dogs. Therefore, the DNA detecting test for the bacteria seems the most promising method of diagnosis of human or canine borreliosis alike [14, 18, 50, 52, 53].

Bauerfeind *et al.* [2] applied nested PCR, using the flagellin encoding gene of *B. burgdorferi* as the DNA marker, and demonstrated that the method was useful, as compared with *in vitro* culture or dark-field microscopy in canine urine samples. Straubinger *et al.* [52] demonstrated detectability of DNA in post-antibiotic treatment in tissues of dogs experimentally-infected with *B. burgdorferi* by PCR with primers specific to chromosomal 23S rRNA gene and the OspA gene on plasmids. Besides quick and sensitive diagnosis, many authors stress the importance of prophylaxis, including vaccines [50, 55]. However, due to possible infection by 3 *B. burgdorferi* genospecies, it is questionable whether European dogs can be actively immunized with an American vaccine (Lymevax), registered in Poland since 1996. Elfassy *et al.* [13] showed that amitraz-impregnated collars prevented transmission of *B. burgdorferi* in 4 out of 4 treated dogs and may be a useful management tool for preventionof borreliosis in dogs.

CONCLUSIONS

In dogs, *B. burgdorferi* s.l. has been implicated as a cause of various disorders often resembling those observed in human borreliosis including polyarthropathy, anorexia, malaise, and neurological dysfunction. In areas infested with *I. ricinus,* and *I. dammini* in the USA and known to be endemic for Lyme disease, veterinarians may suspect borreliosis in dogs with limb/joint disorders.

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