

Antibodies to Borrelia sp. in Wild Foxes and Coyotes from Wisconsin and Minnesota

Authors: Kazmierczak, James J., and Burgess, Elizabeth C.

Source: Journal of Wildlife Diseases, 25(1): 108-111

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-25.1.108

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

SHORT COMMUNICATIONS

Journal of Wildlife Diseases, 25(1), 1989, pp. 108-111 © Wildlife Disease Association 1989

Antibodies to *Borrelia* sp. in Wild Foxes and Coyotes from Wisconsin and Minnesota

James J. Kazmierczak and Elizabeth C. Burgess, Department of Medical Sciences, School of Veterinary Medicine, University of Wisconsin, Madison, Wisconsin 53706, USA

ABSTRACT: Serum samples from 93 red foxes (Vulpes vulpes) and nine gray foxes (Urocyon cinereoargenteus) trapped in Wisconsin and 23 covotes (Canis latrans) trapped in Wisconsin and Minnesota were tested for antibodies to Borrelia sp. with an indirect fluorescent antibody test which used Borrelia burgdorferi as the whole-cell antigen. Seven red foxes (8%) and two covotes (9%) had antibody titers ≥1:64. All the positive samples were from areas known to be endemic for human Lyme disease. Implications for the epizootiology of Lyme borreliosis in wild canids are not well understood, but even if these species are not actual reservoirs of B. burgdorferi they could serve to increase the range of the vector and establish new endemic foci of the spirochete.

Key words: Borrelia sp., coyote, red fox, gray fox, Canis latrans, Vulpes vulpes, Urocyon cinereoargenteus, serologic survey, Lyme disease.

Borrelia burgdorferi, the etiologic agent of Lyme disease in humans, is endemic in much of Wisconsin and Minnesota. Incidence of the disease in the upper midwestern USA is increasing, probably as a result of the expanding range of the primary vector *Ixodes dammini* (Davis et al., 1984; Osterholm et al., 1984; Godsey et al., 1987).

Borrelia burgdorferi has been shown to infect many species of wild and domestic animals. Of particular interest to this report is the susceptibility of certain Canidae, both wild and domestic (Kornblatt et al., 1985; Kazmierczak et al., 1988). Movement of domestic dogs and seasonal dispersal of wild canids have been postulated as means of spread of *I. dammini* into nonendemic areas (Godsey et al., 1987). However, the prevalence of *B. burgdorferi* infection in abundant free-living canids

like foxes and coyotes was not known. To this end, a survey was performed to obtain serologic evidence of *B. burgdorferi* infection in wild red foxes (*Vulpes vulpes*), gray foxes (*Urocyon cinereoargenteus*) and coyotes (*Canis latrans*) in Wisconsin and Minnesota.

Fox sera were obtained from the Wisconsin Department of Natural Resources (Madison, Wisconsin 53707, USA). These consisted of 93 red fox and nine gray fox samples from animals trapped in the autumn of 1984 in various parts of Wisconsin (USA). Trapping and serum collection were performed by private trappers in 19 counties (Fig. 1). Coyote sera were provided by the Departments of Natural Resources in Minnesota (St. Paul, Minnesota 55155, USA) and Wisconsin. Twenty-three coyote samples from animals trapped from 1979 through 1985 were tested. Of these, 19 were from Minnesota, obtained in the counties of Itasca (n = 16) (47°05′ to 47°40′N, 93°05′ to 93°40′W) and Aitken (n = 3)(46°48′N, 93°30′W). The four Wisconsin samples were obtained in the counties of Douglas (n = 3) (46°24′N, 92°06′W) and Dane (n = 1) (43°04′N, 89°23′W).

Antibody titers to *Borrelia* sp. in both the fox and coyote samples were determined by means of an indirect fluorescent antibody (IFA) test, using a method previously described (Steere et al., 1983). The whole-cell antigen used was the seventh culture passage of an isolate of *B. burgdorferi* cultured from a white-footed mouse (*Peromyscus leucopus*) from Wisconsin. The antiserum used was fluorescein isothiocyanate-conjugated goat anti-dog

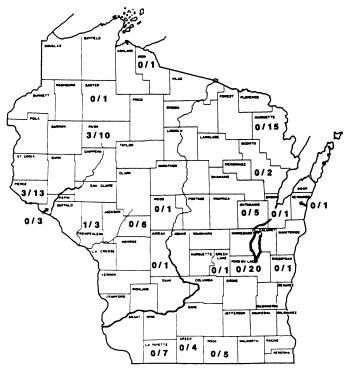


FIGURE 1. Source of fox sera from counties in Wisconsin (USA). Numerator represents the number of foxes with an antibody titer to *Borrelia* sp. ≥1:64; denominator represents total number of foxes tested in that county.

IgG (Cappell Worthington Laboratories, Malvern, Pennsylvania 19355, USA) at a dilution of 1:40. Known *B. burgdorferi*positive and -negative dog sera served as controls for each test. Sera were initially screened at a dilution of 1:8. Samples reactive at this dilution were tested at two-fold serial dilutions until an endpoint titer was determined. The antibody titer was defined to be the highest serum dilution at which definite fluorescence was observed.

Of the 93 red fox samples tested, seven were reactive in the IFA test at levels ≥1:64 (8%). These samples included three from Pierce County (titers of 1:64, 1:256, and 1:512); three from Rusk County (titers of 1:64, 1:64, and 1:256); and one from Trempealeau County (titer of 1:128) (Fig. 1). None of the grey fox sera showed significant levels of reactivity.

Two of 23 coyote samples had antibody titers of ≥ 1.64 (9%), including one animal

trapped in Itasca County, Minnesota in 1980 which had a titer of 1:64 and another trapped in Douglas County, Wisconsin in 1985 whose titer was 1:1,024.

An antibody titer of $\geq 1:64$ has been considered evidence of prior B. burgdorferi infection in dogs (Magnarelli et al., 1985; Burgess, 1986a). While the Lyme disease spirochete has some antigenic relatedness with the Leptospira interogans, the degree of cross-reactivity is minor (Magnarelli et al., 1985, 1986). Of greater concern in serologic surveys of wildlife is the considerable cross-reactivity between B. burgdorferi and some other members of the genus, notably the tick-borne relapsing fever spirochete Borrelia hermsii, and Borrelia coriaceae, the postulated agent of epizootic bovine abortion (Magnarelli et al., 1986; Lane and Burgdorfer, 1988). These two species of Borrelia in North America, however, are limited to the western United States and Canada (Barbour and Hayes,

1986). Even so, we cannot rule out the possibility that infection with some other Borrelia sp. was responsible for the reactivity seen in our IFA tests. However, the fact that significant antibody titers in our study were found only in areas where human Lyme disease is endemic (Davis et al., 1984; Osterholm et al., 1984) suggests that the antibodies were indeed produced as a result of infection with B. burgdorferi. Within the known Lyme-endemic areas of Wisconsin as described by Davis et al. (1984), seven of 39 fox samples (18%) were reactive at levels ≥ 1:64, while none of 63 fox samples obtained from outside the endemic area had significant antibody levels.

The effects of *B. burgdorferi* infections in wild Canidae are not yet known. In the domestic dog, infection can result in fever, lymphadenopathy, arthralgia and arthritis (Kornblatt et al., 1985). An experimentally infected gray wolf (*Canis lupus*) developed a lymphadenopathy, suggesting that the spirochete is pathogenic for this species (Kazmierczak et al., 1988).

Another area which requires further investigation is the reservoir competence of Canidae. There are numerous reports of foxes and coyotes being parasitized by known or suspected vectors of B. burgdorferi, including I. dammini (Spielman et al., 1979), Ixodes scapularis (Cooley and Kohls, 1945; Bloemer and Zimmerman, 1988), Amblyomma americanum, and Dermacentor variabilis (Sonenshine and Stout, 1971; Bloemer and Zimmerman, 1988). However, it is not known whether canids can develop a spirochetemia of sufficient magnitude to infect tick vectors. The fact that experimentally infected domestic dogs can infect other dogs by contact transmission of the spirochete (Burgess, 1986b) suggests that at least this species of canid could be a direct source of infection for other animals under natural conditions.

Even if they are not actual reservoirs of *B. burgdorferi*, species such as red foxes and coyotes that are widely distributed, abundant, and highly mobile could serve

to both increase the geographical range of vectors like I. dammini and to establish new endemic foci of the Lyme disease spirochete. Studies from Minnesota and Wisconsin cite juvenile dispersal distances up to 80 and 86 km for covotes and red foxes, respectively (Berg and Chesness, 1978; Pils and Martin, 1978). In addition, these canids are often in close proximity to human habitation. The average distance of fox dens to farmsteads in Wisconsin was found to be 0.5 km (Pils and Martin, 1978). Covotes are known to frequently travel long distances to associate with livestock (Danner and Smith, 1980). Such factors could easily bring humans and domestic animals in contact with coyote- and fox-borne ectoparasites.

The authors gratefully acknowledge the Wisconsin and Minnesota Departments of Natural Resources for providing serum samples for this study. We also thank Katie Short, Todd Fuller, and Susan Marcquenski for their assistance in determining sample origin. Partial funding for this study was provided by the School of Veterinary Medicine, University of Wisconsin–Madison.

LITERATURE CITED

- BARBOUR, A. G., AND S. F. HAYES. 1986. Biology of *Borrelia* species. Microbiological Reviews 50: 381-400.
- Berg, W. E., AND R. A. CHESNESS. 1978. Ecology of coyotes in northern Minnesota. *In* Coyotes: Biology, behavior, and management, M. Bekoff (ed.). Academic Press, New York, New York, pp. 229–246.
- BLOEMER, S. R., AND R. H. ZIMMERMAN. 1988. Ixodid ticks on the coyote and gray fox at Land Between the Lakes, Kentucky-Tennessee, and implications for tick dispersal. Journal of Medical Entomology 25: 5-8.
- Burgess, E. C. 1986a. Natural exposure of Wisconsin dogs to the Lyme disease spirochete (*Borrelia burgdorferi*). Laboratory Animal Science 36: 288–290.
- 1986b. Experimental infection of dogs with Borrelia burgdorferi. Zentralblatt für Bakteriologie Mikrobiologie und Hygiene, Series A 263: 49-54.
- COOLEY, R. A., AND G. M. KOHLS. 1945. The genus Ixodes in North America. National Institute of

- Health Bulletin No. 184, Washington, D.C., 246 pp.
- DANNER, D. A., AND N. S. SMITH. 1980. Coyote home range, movement, and relative abundance near a cattle feedyard. The Journal of Wildlife Management 44: 484–487.
- DAVIS, J. P., W. L. SCHELL, T. E. AMUNDSON, M. S. GODSEY, A. SPIELMAN, W. BURGDORFER, A. G. BARBOUR, M. LAVENTURE, AND R. A. KASLOW. 1984. Lyme disease in Wisconsin: Epidemiologic, clinical, and entomologic findings. Yale Journal of Biology and Medicine 57: 685–696.
- GODSEY, M. S., T. E. AMUNDSON, E. C. BURGESS, W. SCHELL, J. P. DAVIS, R. KASLOW, AND R. EDELMAN. 1987. Lyme disease ecology in Wisconsin: Distribution and host preferences of *Ixodes dammini*, and prevalence of antibody of *Borrelia burgdorferi* in small mammals. American Journal of Tropical Medicine and Hygiene 37: 180–187
- KAZMIERCZAK, J. J., E. C. BURGESS, AND T. E. AMUNDSON. 1988. Susceptibility of the gray wolf (Canis lupus) to infection with the Lyme disease agent, Borrelia burgdorferi. Journal of Wildlife Diseases 24: 522–527.
- KORNBLATT, A. N., P. H. URBAND, AND A. C. STEERE. 1985. Arthritis caused by *Borrelia burgdorferi* in dogs. Journal of the American Veterinary Medical Association 186: 960-964.
- LANE, R. S., AND W. BURGDORFER. 1988. Spirochetes in mammals and ticks (Acari: Ixodidae) from a focus of Lyme disease in California. Journal of Wildlife Diseases 24: 1-9.
- MAGNARELLI, L. A., J. F. ANDERSON, C. S. APPERSON, D. FISH, R. C. JOHNSON, AND W. A. CHAPPELL. 1986. Spirochetes in ticks and antibodies to *Borrelia burgdorferi* in white-tailed deer from Con-

- necticut, New York State, and North Carolina. Journal of Wildlife Diseases 22: 178–188.
- AND G. D. WHITNEY. 1985. Borreliosis in dogs from southern Connecticut. Journal of the American Veterinary Medical Association 186: 955-959.
- OSTERHOLM, M. T., J. C. FORFANG, K. E. WHITE, AND J. N. KURITSKY. 1984. Lyme disease in Minnesota: Epidemiologic and serologic findings. Yale Journal of Biology and Medicine 57: 677– 684.
- PILS, C. M., AND M. A. MARTIN. 1978. Population dynamics, predator-prey relationships, and management of the red fox in Wisconsin. Department of Natural Resources Technical Bulletin No. 105. Wisconsin Department of Natural Resources, Madison, Wisconsin, 56 pp.
- SONENSHINE, D. E., AND I. J. STOUT. 1971. Ticks infesting medium sized wild mammals in two forest localities in Virginia (Acarina: Ixodidae). Journal of Medical Entomology 8: 217–227.
- SPIELMAN, A., C. M. CLIFFORD, J. PIESMAN, AND M. D. CORWIN. 1979. Human babesiosis on Nantucket Island, USA: Description of the vector, *Ixodes (Ixodes) dammini*, n. sp. (Acarina: Ixodidae). Journal of Medical Entomology 15: 218–234.
- STEERE, A. C., R. L. GRODZICKI, A. N. KORNBLATT, J. E. CRAFT, A. G. BARBOUR, W. BURGDORFER, G. P. SCHMID, E. JOHNSON, AND H. E. MALA-WISTA. 1983. The spirochetal etiology of Lyme disease. New England Journal of Medicine 308: 733-740.

Received for publication 10 June 1988.