Prevalence of *Borrelia burgdorferi* and *Babesia microti* in Mice on Islands Inhabited by White-Tailed Deer

JOHN F. ANDERSON,*1 RUSSELL C. JOHNSON,2 LOUIS A. MAGNARELLI,1 FRED W. HYDE,2 AND JAMES E. MYERS3

Department of Entomology, The Connecticut Agricultural Experiment Station, New Haven, Connecticut 06504; Department of Microbiology, University of Minnesota Medical School, Minneapolis, Minnesota 55455; and Department of Environmental Management, Division of Fish and Wildlife, West Kingston, Rhode Island 02892

Received 5 May 1986/Accepted 26 December 1986

*Borrelia burgdorferi* and *Babesia microti* were isolated from 35 of 51 white-footed mice (*Peromyscus leucopus*) and meadow voles (*Microtus pennsylvanicus*) captured on two Narragansett Bay, R.I., islands inhabited by deer, the principal host for the adult stages of the vector tick, *Ixodes dammini*. Immature ticks parasitize mice from both islands. From 105 mice captured on four other islands not inhabited by deer neither pathogen was isolated, nor were *I. dammini* found.

Two recently described tick-borne diseases have been recognized in northeastern and midwestern regions of the United States. Human babesiosis, caused by *Babesia microti*, has been recorded for more than 100 patients who visited or resided on islands off shore from Massachusetts, Rhode Island, and Long Island, N.Y. (12). A more recent focus of human babesiosis has been identified in Wisconsin (26). Lyme disease (25), a far more prevalent illness, is caused by the spirochete *Borrelia burgdorferi* (10, 16). Hundreds of cases have been diagnosed in the northeastern United States, Minnesota, and Wisconsin. The tick *Ixodes dammini* (23) is the chief vector of both agents (10, 22) in northeastern and midwestern areas of the United States.

The resurgence of white-tailed deer, *Odocoileus virginianus*, may be responsible for the apparent increase of *I. dammini* (3, 19, 21, 27) and the rise in numbers of human infections. While larval and nymphal *I. dammini* are cathartic in their host selection and probably feed on most land mammals and ground-inhabiting birds, adult ticks are more discriminatory, tending to feed on and mate on deer, although they also parasitize medium-sized mammals, such as dogs, cats, raccoons (*Procyon lotor*), and Virginia opossums (*Didelphis virginiana*) (3, 5, 11, 18, 20, 23). Herein, we assess the prevalence of *B. burgdorferi* and *B. microti* in *Peromyscus leucopus* (white-footed mouse) and in *Microtus pennsylvanicus* (meadow vole) on two islands inhabited by deer and in mice on four islands not inhabited by deer in Narragansett Bay, R.I. The presence of *I. dammini* on mice was also recorded.

The six islands were Patience (87.4 ha), Prudence (1,528 ha), Gould (21.5 ha), Dyer (13 ha), Hog (78.5 ha), and Jamestown (8,159 ha). White-tailed deer browse on Prudence and Patience islands. The estimated summer populations of deer on each island is 70 deer per 1 square mile (2,590 ha). Dogs, cats, and raccoons are likewise prevalent on the islands of Prudence, Hog, and Jamestown.

Rodents were captured in Sherman box traps on Prudence, Patience, Jamestown, and Gould islands in November or December, 1984, June, 1985, and in August or September, 1985. On Dyer Island, mice were captured in June, August, and October, 1985. Traps were set on Hog Island in August, 1985. Usually, no more than 10 animals from each collection per island were kept for experimental purposes.

Rodents captured in 1985 were examined for ticks. *I. dammini* were tested for borreliae by indirect fluorescent antibody staining with monoclonal antibody H5332, which is directed against an approximately 31,000-molecular-weight protein of *B. burgdorferi* (7).

Attempts were made to isolate borreliae from the blood and from the spleen and kidney tissues of rodents captured on each of the six islands (1, 2, 15). One or two drops of blood (ca. 0.1 ml) from the heart of each animal and a 1:10 dilution of each kidney and spleen tissue sample triturated in 7 ml of Barbour-Stoenner-Kelly medium were inoculated into duplicate tubes of Barbour-Stoenner-Kelly +medium containing 0.1% agarose (SeaKem LE; FMC Corp., Marine Colloids Div., Rockland, Maine) (6, 15). Cultures were maintained at 31°C and were examined for spirochetes by dark-field microscopy 3 to 8 weeks after inoculation. Isolates were identified as *B. burgdorferi* by their reaction with monoclonal antibody H5332 (7).

For isolation of *Babesia* spp., blood was drawn from the heart of each rodent, and 0.5 ml was inoculated intraperitoneally into a Syrian hamster (13). A blood smear made from 1 drop of blood taken from the tail of the hamster was prepared weekly for 6 weeks postinjection. The slides were stained with Giemsa stain, and the erythrocytes were examined for *Babesia* spp. The parasites found were identified as *B. microti* by their morphological and staining characteristics (14).

Borreliae were isolated in Barbour-Stoenner-Kelly medium inoculated with blood or with kidney or spleen tissue from white-footed mice or meadow voles captured on deer-inhabited Prudence and Patience islands (Table 1). Cultures were grown from blood or tissues taken from one of seven meadow voles and from 18 of 23 white-footed mice from Prudence Island. Borreliae were also isolated from 10 of 23 meadow voles from Patience Island. During all three collection periods, spirochetes were most frequently obtained from spleen or kidney tissue cultured from mice captured on Prudence and Patience islands. All isolates reacted with monoclonal antibody H5332. No borreliae were isolated.

* Corresponding author.
from the 105 mice tested from Jamestown, Hog, Dyer, or Gould islands, which were not inhabited by deer.

*B. microti*-infected hamsters were inoculated with blood from white-footed mice and meadow voles captured on Prudence and Patience islands (Table 1). Isolates were obtained on all three collection dates from the blood or tissues of animals from both islands. We found that 4 of 7 meadow voles from Prudence Island and 6 of 21 from Patience Island were infected. Of the 23 white-footed mice tested from Prudence Island, 10 were parasitic. This protozoan was not isolated from 105 rodents collected on the four islands not inhabited by deer.

In our test animals captured in 1985, *B. microti* and borreliae were both present in two meadow voles from Patience Island and six white-footed mice from Prudence Island. Of the seven meadow voles and 23 white-footed mice from Prudence Island, collected in 1984 and 1985, 23 (four meadow voles and 19 white-footed mice) were infected with either one or both of the agents. On Patience Island, 12 of 21 meadow voles were similarly infected.

Immature *I. dammini* were prevalent on white-footed mice collected from Prudence Island in June (nymphs per mouse \(\pm\) standard deviation, 3.3 \(\pm\) 4) and in August (larvae per mouse \(\pm\) standard deviation, 13.0 \(\pm\) 14.2) and were less prevalent on meadow voles collected from Patience Island in June (nymphs per vole \(\pm\) standard deviation, 0.9 \(\pm\) 1.5 SD) and in August (larvae per vole \(\pm\) standard deviation, 0.1 \(\pm\) 0.3). *I. dammini* were not collected from rodents captured on the four islands not inhabited by deer. Spirochetes that reacted with monoclonal antibody H5332 in indirect fluorescent-antibody tests were detected in 33 of 129 *I. dammini* larvae and in 6 of 20 nymphs from Prudence Island.

The reactivity of the borreliae isolates from Prudence and Patience islands with monoclonal antibody H5332 established their identity as *B. burgdorferi* (7). The morphology of the parasites in erythrocytes of infected hamsters was similar to that previously described for *B. microti* (14).

*B. burgdorferi* and *B. microti* infected rodents on the two islands inhabited by deer, but were not isolated from mice captured on the four islands that were not inhabited by deer. *I. dammini* were also prevalent only on the two deer-inhabited islands, and ticks infected with *B. burgdorferi* were documented on one of these islands. The Lyme disease agent, therefore, appears to be associated with populations of deer and ticks. Our finding *B. microti* in mice that coexist with deer confirms earlier results (24). Thus, both of these pathogens, for which *I. dammini* is the vector, appear to be indirectly associated with the white-tailed deer. While borreliae and antibody to *B. burgdorferi* have been detected in white-tailed deer (4, 8, 9, 17), the epizootiological significance of deer is that they are the key hosts for adult ticks.

Although medium-sized mammals are hosts for adult *I. dammini* (3, 11, 23), our data lead us to believe that these mammals are unable to sustain sufficiently large numbers of ticks that may transmit *B. burgdorferi* and *B. microti*. Neither pathogen was isolated from rodents captured on two islands not inhabited by deer but on which dogs, raccoons, cats, or Virginia opossums were common.

The restrictive host range for adult ticks leads us to question whether the prevalence of human infection by *B. burgdorferi* and *B. microti* could be significantly suppressed by reducing populations of white-tailed deer. On Prudence and Patience islands, deer populations during 1978 to 1985 were reduced 25% annually (during the hunting season in November and December) from ca. 70 to ca. 52 deer per 2,590 ha, yet the prevalence of these two human pathogens in mice remained relatively high. About 70% of the deer were killed on Great Island, Mass., but populations of larval *I. dammini* were not substantially reduced the following year (28). On the basis of these observations, coupled with the absence of *B. burgdorferi* and *B. microti* in our rodent samples from the islands not inhabited by deer, we suspect that deer populations will have to be reduced to extremely low levels before prevalence of these two human pathogens will be noticeably reduced.

We thank Carol Lemmon, Clifford Snow III, Catherine Hammie, Carrie Kodner, and Marie Russell for technical assistance. Alan Barbour of the Rocky Mountain Laboratories, Hamilton, Mont., kindly provided the monoclonal antibody H5332.

R.C.J. was supported by Public Health Service grants AI 18153 and AM 34744 from the National Institutes of Health.

**LITERATURE CITED**


