Detection of Borreliacidal Antibodies in Dogs after Challenge with Borrelia burgdorferi-Infected Ixodes scapularis Ticks

STEVEN M. CALLISTER,1,2* DEAN A. JOBE,2 RONALD F. SCHELL,3,4 STEVEN D. LOVRICH,2 KEYSHA L. ONHEIBER,2 AND JON B. KORSHUŚ††

Section of Infectious Diseases1 and Microbiology Research Laboratory,2 Gundersen Lutheran Medical Center, La Crosse, Wisconsin 54601; Wisconsin State Laboratory of Hygiene,3 and Department of Medical Microbiology and Immunology, University of Wisconsin,4 Madison, Wisconsin 53706; and Solvay Animal Health, Inc., Mendota Heights, Minnesota 55120

Received 5 May 2000/Returned for modification 25 June 2000/Accepted 28 July 2000

Detection of borreliacidal antibodies is an accurate serodiagnostic test for confirmation of Lyme disease in dogs. In this study, 13 pathogen-free beagles, 12 to 26 weeks old, were infected with Borrelia burgdorferi by tick challenge. Dogs were monitored for clinical signs and symptoms of Lyme disease along with borreliacidal antibody production against B. burgdorferi sensu stricto isolates 297 and 50772. Ten (77%) dogs developed lameness in one or more legs within 21 days after attachment of Ixodes scapularis ticks. Eight (80%) of the lame animals had concurrent fever of ≥38°C. Spirochetes were also recovered from the skin and joints of 12 (92%) dogs, but rarely from other organs. Borreliacidal antibodies against B. burgdorferi isolate 297 were detected in only four (31%) dogs, and the levels of killing antibodies remained low for the duration of the infection. In contrast, borreliacidal antibodies against B. burgdorferi isolate 50772 were detected in 13 (100%) dogs within 21 days of infection. Furthermore, the borreliacidal antibody levels correlated with the severity of B. burgdorferi infection. Detection of borreliacidal antibodies, especially against B. burgdorferi isolate 50772, is also a reliable serodiagnostic test for detection of Lyme disease in dogs.

Lyme disease is an Ixodes sp. tick-associated zoonosis caused by Borrelia burgdorferi sensu lato. This multisystem disorder has become the most common tick-transmitted illness in the United States and causes significant morbidity in humans and animals. Common signs and symptoms of Lyme disease in humans include a virus-like syndrome with acute and chronic skin lesions, carditis, neuritis, and arthritis (21). Infection with B. burgdorferi also causes a similar illness in dogs (2), although the signs of infection can be more difficult to detect. The most common clinical features in canines are arthritis and arthralgia (12).

Infection of humans and other animals with B. burgdorferi also results in production of killing (borreliacidal) antibodies. These antibodies are directed against several B. burgdorferi proteins including outer surface protein A (OspA) (5, 13–15, 17), OspB (17), OspC (18), decorin binding protein A (DbpA) (8, 11), the periplasmic 39-kDa protein (20), and the outer membrane protein p66 (10). Borreliacidal antibodies against these proteins are readily detected during early and late Lyme disease in humans by use of specific isolates of B. burgdorferi (4, 5, 7) and flow cytometry (4, 6). Detection of borreliacidal antibodies has improved the sensitivity and specificity of the serodiagnosis of human Lyme disease (4–7). However, little information is available on the production and detection of borreliacidal antibodies in naturally infected dogs. In fact, Straubinger et al. (23) detected only minimal borreliacidal antibody levels, or none at all, in tick-infected dogs even after 30 and 60 days of infection.

Recently, we demonstrated that high titers of borreliacidal antibodies, especially OspC-specific borreliacidal antibodies, were produced shortly after infection of humans with B. burgdorferi (4, 18). Previously, the serodiagnosis of Lyme disease was limited to detection of borreliacidal antibodies to OspA, OspB, and other proteins excluding OspC. This meant that borreliacidal antibodies could be detected primarily in sera from patients with later stages of Lyme disease, when anti-OspA and anti-OspB antibodies are more commonly produced (4, 5). Detection of anti-OspC borreliacidal antibodies was dependent on use of B. burgdorferi sensu stricto isolate 50772, which does not contain ospA or ospB (1). A borreliacidal antibody test using B. burgdorferi isolate 50772 greatly increased the sensitivity and specificity of detection of early Lyme disease in humans (4–7, 18).

In this investigation, we determined the borreliacidal antibody response in dogs after challenge with B. burgdorferi-infected ticks. High borreliacidal antibody levels were detected shortly after challenge and remained detectable for the duration of the infection. Detection of borreliacidal antibodies in dog sera was dependent on the use of B. burgdorferi isolate 50772. Our findings demonstrate the validity of the borreliacidal antibody test for detection of Lyme disease in dogs.

MATERIALS AND METHODS

Dogs. Thirteen 12- to 26-week-old specific-pathogen-free beagles from the colony located at Solvay Animal Health, Inc., Charles City, Iowa, were used. All dogs were kept in P2 isolation units and fed commercial food and water ad libitum. Dogs were observed daily after challenge for clinical signs of B. burgdorferi infection including lameness, lethargy, or fever. Lameness was defined as reluctance to bear weight on a limb with or without swelling or temperature.

Ticks. Adult male and female Ixodes scapularis ticks were collected by flagging wooded areas near Ettrick, Wisconsin, during May and October. Ticks were stored at 8°C in 90% relative humidity until use. The infectivity rate of the ticks was determined by examining the midguts of 50 male I. scapularis ticks after staining with a fluorescein isothiocyanate-labeled anti-OspA monoclonal antibody. Twenty-two (44%) of the 50 ticks were infected with B. burgdorferi.

Spirochetes. B. burgdorferi sensu stricto isolates 297 and 50772 were isolated from human spinal fluid and an I. scapularis tick, respectively. B. burgdorferi isolate 50772 organisms lack ospA and ospB and consequently do not produce OspA or OspB (1). In addition, B. burgdorferi isolates 50772 spirochetes express...
high levels of OspC on their surfaces after several passages at 35°C (18). The original suspensions of these spirochetes were serially 10-fold diluted in Barbour-Stoenner-Kelly (BSK) medium capable of supporting growth from a single organism (3). The resultant population of each spirochete was then passaged 10 times in fresh BSK medium at 35°C, dispensed into 200-μl aliquots in 1.5-ml screw-cap tubes (Sarstedt, Newton, N.C.), and stored at −70°C until use.

Infection of dogs. Each dog was challenged with 10 female and 6 male adult ticks. Ticks were randomly selected and placed into two small petri dishes (five females and three males per dish). Two petri dishes were attached to a shaven area on the left dorsal-anterior region of each dog and secured for 1 week. After the ticks had fed to repletion, tick midguts were examined for B. burgdorferi to ensure exposure to Lyme disease spirochetes. B. burgdorferi organisms were detected in the midgut of at least one tick per animal on which ticks fed to repletion.

Recovery of B. burgdorferi spirochetes. To establish infection following exposure to ticks, separate skin biopsy specimens were removed from the two tick bite sites 18 days after petri dishes and ticks were removed. Skin biopsy sites were shaved, washed with disinfectant, and rinsed thoroughly with distilled water. An elliptical incision was made through the dermal and subcutaneous skin layers, and a specimen of approximately 0.5 g was collected. When the dogs were euthanized at the conclusion of the study, additional tissues, including blood, heart, spleen, kidneys, bladder, cerebrospinal fluid, and joints, were also examined. These tissues were removed from the elbow, carpus, knee, and tarsus.

Two to three milliliters of blood or cerebrospinal fluid was added directly to 9 ml of BSK medium. Skin biopsy specimens and postmortem tissues were homogenized in BSK medium with a tissue homogenizer (Stomacher; Seward Medical, London, England). Subsequently, an aliquot of the homogenized sample was diluted 10-fold into a culture tube containing fresh BSK medium and mixed thoroughly. After mixing, two 0.6-ml aliquots were removed and placed separately into additional tubes containing 5.4 ml of fresh BSK medium containing 0.15% agarose and 40 μg of rifampin/ml. All cultures were incubated at 34°C and examined weekly by dark-field microscopy for 6 weeks. After the initial 6-week incubation, a 1-ml aliquot was removed from all negative cultures and transferred to 9 ml of fresh BSK medium. These cultures were examined for an additional 6 weeks. Recovered spirochetes were identified by reactivity with a monoclonal anti-OspA antibody.

Detection of borreliacidal antibodies. Blood samples were taken before challenge and at weekly intervals for 9 weeks after infection. Sera were stored at −20°C until testing. Borreliacidal antibodies were detected by flow cytometry as previously described (4, 18). Briefly, a frozen 200-μl aliquot of B. burgdorferi isolate 50772 or 297 was thawed and inoculated into 6 ml of fresh BSK medium, and cultures were incubated for 72 h at 35°C. After incubation, the number of spirochetes was determined with a Petroff-Hauser counting chamber, and the organisms were diluted in fresh BSK medium to a concentration of 10<sup>10</sup> organisms/ml. Serum samples were initially diluted 10-fold in fresh BSK medium and sterilized by passage through a 0.2-μm-pore-size microcentrifuge filter (Costar, Cambridge, Mass.). A 100-μl aliquot was transferred to a 1.5-ml screw-cap microcentrifuge tube (Sarstedt) and serially twofold diluted (1:20 to 1:20,480). The diluted serum was heat inactivated at 56°C for 10 min. Following heat inactivation, a 100-μl aliquot of B. burgdorferi (10<sup>7</sup> organisms) and 10 μl of sterile guinea pig serum (200 50% hemolytic complement [CH<sub>50</sub>] units per ml; Sigma) were added to the diluted sera. Following 20 min at 35°C, 100 μl of the assay suspensions was diluted 1:5 with phosphate-buffered saline (0.01 molar, pH 7.2) containing 1 μg of acridine orange/ml. Borreliacidal antibodies were detected with a FACScan single-laser flow cytometer (Becton Dickinson Immunocytometry Systems, San Jose, Calif.). Events were acquired for 1 to 2 min with the flow rate set at low (12 μl/min) and were analyzed using FACSScan CellQuest research software. Side scatter and fluorescence intensity parameters were used to distinguish B. burgdorferi from BSK and complement particles. Spirochetes were gated during data acquisition, and fluorescence signals were logarithmically amplified and converted to a linear scale. A ≥13% increase in fluorescence intensity compared to that of normal serum controls was considered positive (4). All assays were performed in duplicate or triplicate.

RESULTS

Challenge and confirmation of infection with B. burgdorferi. Ten female and 6 male I. scapularis ticks were placed on each dog and allowed to feed for as long as 7 days. Generally, female ticks attached within 24 h and fed to repletion. Skin biopsies were obtained 18 days after completion of the tick challenge and cultured for Lyme disease spirochetes. B. burgdorferi organisms were recovered from the skin of 12 (92%) dogs.

Clinical signs. Dogs were examined daily for as long as 210 days of infection, 6 (46%) and 10 (77%) dogs had lameness episodes, respectively. Eight dogs were lame in one leg. The remaining two dogs developed lameness in three and four limbs. In most instances, dogs were reluctant to bear weight on the affected limb and the joints were swollen and warm. Lameness episodes generally lasted 24 to 48 h. Eight (80%) of the 10 animals with lameness had concurrent fever of ≥38°C.

Recovery of B. burgdorferi. Within 3 to 4 days after the onset of lameness, dogs were necropsied and the skin, joints, and organs were cultured. The three dogs that did not develop lameness were necropsied 210 days after tick challenge. B. burgdorferi spirochetes were recovered from the skin and joints of 12 (92%) of the dogs (Table 2). Lyme disease organisms were also recovered from the bladder, spleens, and hearts of individual animals. In addition, B. burgdorferi organisms were recovered from the bladder, heart, kidney, and spleen of one dog. No spirochetes were recovered from blood or cerebrospinal fluid.

Borreliacidal antibody response. No borreliacidal antibodies were detected in any dogs prior to challenge with I. scapularis ticks. After challenge, borreliacidal antibodies that killed B. burgdorferi isolate 50772 spirochetes were detected in four (31%) dogs (Table 3). Anti-297 borreliacidal antibodies were not detectable until 21 days after challenge and remained at low titers for the duration of the infection. In contrast, borreliacidal antibodies that killed B. burgdorferi isolate 50772 were detectable in 9 (73%) and 11 (85%) dogs within 7 or 14 days after infection, respectively. Anti-50772 borreliacidal antibodies were detected in all (100%) of the animals 21 days after challenge and remained detectable for the duration of the experiment. Borreliacidal antibody levels, however, varied widely among individual animals. The intensity of the borreliacidal antibody response correlated closely with the dissemination of B. burgdorferi organisms into internal organs. Relatively low anti-50772 borreliacidal antibody levels were detected in animals with spirochetes found only in the skin and joints. Higher borreliacidal antibody levels were detected in dogs when organisms were recovered from the skin, joints, and some internal organs. The highest anti-50772 borreliacidal antibody levels (titer, 1:20,480) were detected in an animal with spirochetes in the skin, joints, and multiple internal organs.

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Mo in which lameness occurred&lt;sup&gt;a&lt;/sup&gt;</th>
<th>No. of limbs affected</th>
<th>Fever</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ND&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>ND</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>ND</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>July</td>
<td>3</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>July</td>
<td>4</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>August</td>
<td>1</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>September</td>
<td>1</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>September</td>
<td>1</td>
<td>+</td>
</tr>
<tr>
<td>9</td>
<td>September</td>
<td>1</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>October</td>
<td>1</td>
<td>+</td>
</tr>
<tr>
<td>11</td>
<td>October</td>
<td>1</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>November</td>
<td>1</td>
<td>+</td>
</tr>
<tr>
<td>13</td>
<td>November</td>
<td>1</td>
<td>+</td>
</tr>
<tr>
<td>Total no. (%) of dogs affected</td>
<td>10 (77)</td>
<td>8 (62)</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Dogs were challenged on May 21.  
<sup>b</sup> ND, not detected.
DISCUSSION

In this investigation, we determined borreliacidal antibody responses in canines after challenge with \textit{B. burgdorferi}-infected ticks. Both clinical and bacteriological evidence of \textit{B. burgdorferi} infection was obtained to account for the rapid and sustained production of borreliacidal antibodies in dogs. \textit{B. burgdorferi} organisms were recovered from the skin and joints of all infected dogs except one. However, spirochetes were only rarely recovered from the bladder, heart, kidney, or spleen of \textit{B. burgdorferi}-infected animals. These results confirmed previous observations (9) that dogs become infected with \textit{B. burgdorferi} and the skin is the major site of infection. In addition, lameness occurred in 10 (77\%) of the dogs. Lameness was primarily observed in one limb, although two animals became lame in three and four legs. Fever was also common when dogs were lame.

This is the first report that borreliacidal antibodies can be detected consistently in tick-challenged dogs. Specifically, borreliacidal antibodies against \textit{B. burgdorferi} isolate 50772 were detected in nine (73\%) dogs 1 week after ticks were attached. By week 3 of infection, all dogs, including the animal from which no spirochetes were recovered, had borreliacidal antibody titers. Furthermore, borreliacidal antibody levels increased with the duration and severity of infection. The highest borreliacidal antibody levels were detected in dogs for which \textit{B. burgdorferi} organisms were recovered from internal organs. In addition, the borreliacidal antibody response was sustained for the duration of the study.

The development of lameness and fever correlated closely with the recovery of \textit{B. burgdorferi} from the skin and joints. However, the intensity of the borreliacidal antibody response did not correlate closely with the development of these clinical signs. For example, dog 5 developed lameness in all four limbs, but spirochetes were not recovered from internal organs and the borreliacidal antibody response remained relatively low. A more in-depth clinical evaluation of \textit{B. burgdorferi}-infected animals will be necessary to determine whether less-obvious clinical signs are present when spirochetes have infected the internal organs. Regardless, the detection of anti-\textit{B. burgdorferi} 50772 borreliacidal antibodies was an accurate indicator of infection with Lyme disease organisms, and the intensity of this

\begin{table}[h!]
\centering
\begin{tabular}{|c|c|c|c|c|c|}
\hline
Dog no. & Recovery of \textit{B. burgdorferi} from: & Skin & Joints & Bladder & Heart & Kidney & Spleen \\
\hline
1 & + & + & + & + & + & + & + \\
2 & + & + & + & + & + & + & + \\
3 & + & + & + & + & + & + & + \\
4 & + & + & + & + & + & + & + \\
5 & + & + & + & + & + & + & + \\
6 & + & + & + & + & + & + & + \\
7 & + & + & + & + & + & + & + \\
8 & + & + & + & + & + & + & + \\
9 & + & + & + & + & + & + & + \\
10 & + & + & + & + & + & + & + \\
11 & + & + & + & + & + & + & + \\
12 & + & + & + & + & + & + & + \\
13 & + & + & + & + & + & + & + \\
\hline
Total no. (% of dogs) & 12 (92) & 12 (92) & 2 (15) & 2 (15) & 1 (8) & 2 (15) & \\
\hline
\end{tabular}
\caption{Recovery of \textit{Lyme} borreliosis spirochetes from skin, joints, or organs after necropsy of dogs challenged with \textit{B. burgdorferi}-infected \textit{I. scapularis}}
\end{table}

\begin{table}[h!]
\centering
\begin{tabular}{|c|c|c|c|c|c|c|c|c|c|c|c|}
\hline
Dog no. & \textit{B. burgdorferi} isolate & Borreliacidal antibody titer\textsuperscript{a} at the following day postchallenge: & 0 & 7 & 14 & 21 & 28 & 34 & 42 & 49 & 57 & 63 \\
\hline
3 & 297 & — & 1,280 & 1,280 & 1,280 & 1,280 & 1,280 & 1,280 & 1,280 & 1,280 & 1,280 \\
8 & 297 & — & — & 40 & 80 & 160 & 320 & 320 & 320 & 640 & 320 \\
9 & 297 & — & 80 & 80 & 160 & 320 & 320 & 320 & 320 & 320 & 320 \\
10 & 297 & — & 80 & 80 & 160 & 320 & 320 & 320 & 320 & 320 & 320 \\
11 & 297 & — & 20 & 20 & 80 & 80 & 160 & 320 & 320 & 320 & 320 \\
12 & 297 & — & — & 320 & 320 & 160 & 80 & 80 & 80 & 160 & 640 \\
13 & 297 & — & 40 & 80 & 20 & 80 & 160 & 640 & 1,280 & 1,280 & 1,280 \\
\hline
\textsuperscript{a}—, not detected; *, no specimen.
\end{tabular}
\caption{Detection of borreliacidal antibodies against \textit{B. burgdorferi} 297 or 50772 after challenge of dogs with \textit{B. burgdorferi}-infected \textit{I. scapularis}}
\end{table}
response could be used to predict the dissemination of spirochetes into internal organs.

Detection of borreliacidal antibodies in dog sera was dependent on using *B. burgdorferi* isolate 50772 in the borreliacidal antibody test. Previously, we also showed that this isolate was necessary for detection of borreliacidal antibodies in human sera from patients with early Lyme disease (4). *B. burgdorferi* isolate 50772 expresses high levels of OspC (18) but does not express OspA or OspB. By use of *B. burgdorferi* isolate 50772, borreliacidal antibodies were detected in 72% of sera from humans with early Lyme borreliosis (4). A more recent study confirmed that killing of *B. burgdorferi* isolate 50772 was often due to anti-OspC borreliacidal antibodies (18). The detection of borreliacidal antibodies against OspC shortly after infection is logical, since expression of OspA and OspB is downregulated by Lyme disease spirochetes after infected ticks attach to the host (19, 22). Concomitantly, expression of OspC is upregulated. Additional studies will be necessary, to determine if the anti-50772 borreliacidal antibody in this study was OspC specific or was induced by other *B. burgdorferi* antigens.

In contrast, Straubinger et al. (23) detected only minimal borreliacidal antibody levels, or none, in dogs infected with *B. burgdorferi*. When borreliacidal antibodies were detected, the response was observed only at 30 to 60 days after infection and the titer remained low. Our results were similar when *B. burgdorferi* isolate 297 was used. Only four (31%) dogs had detectable borreliacidal antibody levels. Furthermore, the anti-297 borreliacidal antibody response did not develop consistently in these four animals until day 21 of infection. These results are also similar to our previous observations using human Lyme disease sera. The sensitivity of the borreliacidal antibody test for detection of early Lyme disease in humans was only 15% when *B. burgdorferi* isolate 297 was used (4).

We believe that there is a simple explanation for this. Early after infection with *B. burgdorferi*, borreliacidal antibodies cannot be detected in dog or human sera when OspA-expressing spirochetes, like isolate 297, are used. Even if OspA-expressing isolates contain OspC, OspA may hinder the appropriate binding of the borreliacidal antibody. Recently, Patarakul et al. (16) showed that outer membrane proteins of *B. burgdorferi* isolate 297 could prevent complement deposition at lysis susceptibility sites. When ospA- and ospB-deficient isolates, such as *B. burgdorferi* isolate 50772, are used, the interaction of borreliacidal antibodies with OspC or other surface proteins is not hindered.

Detection of borreliacidal antibodies in human sera using *B. burgdorferi* isolate 50772 is now recognized as a sensitive and highly specific serodiagnostic test for Lyme disease (4–7, 18). Our results also show that borreliacidal antibody detection can be an accurate serodiagnostic test for detecting canine Lyme disease, especially during early infection. In support, no borreliacidal antibodies were detected in dogs before they were infected with *B. burgdorferi*. However, borreliacidal antibodies were easily detected shortly after infection and remained detectable for the duration of the study. The levels of anti-50772 borreliacidal antibodies also correlated with the severity of *B. burgdorferi* infection.

There is, however, a confounding factor which occurs in serodiagnostic testing for canine Lyme disease, but does not occur in humans. Dogs are routinely immunized with killed whole *B. burgdorferi* organisms. Induction of anti-50772 borreliacidal antibodies due to vaccination could confound the ability to detect infection by the borreliacidal antibody test. To address this issue, we tested serum samples from 5 and 10 dogs collected 4 weeks after a primary and booster vaccination with the Galaxy or LymeVax whole-cell Lyme disease vaccine, respectively. All dogs developed significant borreliacidal anti-

body titers (range, 1:160 to ≥1:10,240) against *B. burgdorferi* isolate 297. More importantly, no vaccinated dogs developed detectable anti-*B. burgdorferi* 50772 borreliacidal antibody levels. Although the *B. burgdorferi* organisms contained in the vaccines express OspC, the level appears to be too low for induction of anti-50772 borreliacidal antibodies. This finding is not unexpected. *B. burgdorferi* organisms used in canine Lyme disease vaccines are likely grown at a temperature chosen to maximize production of OspA. Higher temperatures are required for spirochetes to express OspC (18). The vaccines in use, which primarily anti-OspC borreliacidal antibodies, which are readily detected with *B. burgdorferi* isolate 297. Therefore, previous vaccination may not hinder the ability to detect anti-*B. burgdorferi* isolate 50772 borreliacidal antibodies induced by infection with *B. burgdorferi*.

In this study, detection of anti-50772 borreliacidal antibodies correlated closely with infection with *B. burgdorferi*. As a preliminary evaluation of the clinical potential of the borreliacidal antibody test, we tested 29 dog sera submitted for Lyme disease testing. All dogs had lameness in one or more legs. Sera from 6 (32%) of 19 dogs not previously vaccinated, 1 (17%) of 6 dogs vaccinated with LymeVax, and 1 (25%) of 4 dogs vaccinated with rLyme, a recombinant OspA vaccine, had high titers (≥1:1,280) of anti-50772 borreliacidal antibodies. Based on our results, it is likely that these animals were infected with *B. burgdorferi*. Additional studies are needed to confirm these findings. However, these results provide evidence that detection of borreliacidal antibodies with *B. burgdorferi* isolate 50772 can be used to reliably detect canine Lyme disease regardless of vaccination status.

In summary, detection of borreliacidal antibodies, especially against OspC or other proteins, can be used for the serodiagnosis of Lyme disease in dogs. A similar conclusion has been reported for the use of anti-OspC borreliacidal antibodies in humans. These parallel findings suggest that results obtained with the canine model of Lyme borreliosis can be applied to humans. Additional studies with dogs are needed to determine the course of borreliacidal antibodies and whether they can be used as a prognostic indicator of successful therapy.

ACKNOWLEDGMENTS

This study was supported by the Gundersen Lutheran Medical Foundation. We thank Kurt Reed of the Marshfield Medical Research Foundation for providing sera from dogs being evaluated for Lyme disease.

REFERENCES


