Clinicopathologic findings in dogs seroreactive to *Bartonella henselae* antigens

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**Objective**—To assess the potential clinical relevance of seroreactivity to *Bartonella henselae* antigens in dogs.

**Animals**—40 dogs seroreactive to *B henselae* and 45 dogs that did not seroreact to *B henselae*.

**Procedure**—A case-control study was conducted. Clinical and clinicopathologic findings were extracted from medical records of each dog.

**Results**—Statistical differences were not detected between dogs seroreactive or nonseroreactive to *B henselae* when analyzed on the basis of disease category or results of hematologic, biochemical, urine, or cytologic analysis. However, seroreactivity to *B henselae* antigens was detected in 2 of 4 dogs with a clinical diagnosis of granulomatous meningencephalitis, 3 of 4 dogs with immune-mediated hemolytic anemia, 3 of 4 dogs with infective endocarditis, 2 of 3 dogs with lymphoid neoplasia, and 5 of 10 dogs with polyarthritis. Additionally, seroreactivity to *B henselae* antigens was detected in 18 of 34 thrombocytopenic dogs and 14 of 27 dogs with neutropenia.

**Conclusions and Clinical Relevance**—Significant associations were not detected between seroreactivity to *B henselae* and various diseases. Prospective epidemiologic studies investigating specific diseases, such as meningoencephalitis or polyarthritis, and specific hematologic abnormalities, such as immune-mediated hemolytic anemia or thrombocytopenia, should be conducted to further define the potential clinical relevance of antibodies against *B henselae* in dogs.


Members of the genus *Bartonella* are fastidious, pleomorphic, gram-negative rods. These vector-borne, hematropic, intracellular pathogens are increasingly associated with a wide spectrum of disease manifestations in humans and other animals. In humans, infection with *Bartonella henselae* is most commonly associated with an acute, febrile lymphadenopathy, which is commonly referred to as cat-scratch disease. In a minority of patients with cat-scratch disease, infection results in atypical manifestations, including endocarditis, encephalitis, granulomatous hepatitis and splenitis, peliosis hepatitis, neuretinitis, and bacillary angiomatosis.

Cats are considered the primary reservoir for *B henselae*. Experimentally induced and naturally developing infections in cats result in prolonged bacteremia with intermittent neurologic dysfunction that is accompanied by histologic evidence of chronic inflammatory lesions in multiple organs. Epidemiologic studies have identified an increased incidence of stomatitis and renal disease in cats seroreactive to *B henselae*. In contrast to cats, dogs are believed to be more commonly infected with *Bartonella vinsonii* subsp. *berkhoffii*, an organism isolated initially in 1993 that causes persistent bacteremia, immunosuppression, granulomatous inflammation, endocarditis, and hemoglobinuria. *Bartonella henselae* DNA has been detected by our research group by the use of polymerase chain reaction (PCR) amplification and sequencing in dogs with peliosis hepatitis, granulomatous hepatitis, fever, thrombocytopenia, and neurologic dysfunction. Detection of *B henselae* DNA in the blood or tissues of these dogs was most probably facilitated by severe protracted illness or the administration of immunosuppressive drugs. Although infrequent, dogs have also been implicated in the transmission of *B henselae* to humans. Results of seroepidemiologic surveys of *B henselae* in dogs have varied by study population and geographic location. The percentage of seropositive dogs was 3% in Britain, 6.5% in Hawaii, and 7.7% in Japan. We detected a significant increase in the prevalence of antibodies against *B henselae* among sick dogs (27.2%), compared with the prevalence in a healthy population of dogs (10.1%) from the southeastern United States.

Those data, in conjunction with PCR detection of *B henselae* DNA in the aforementioned studies, induced us to begin testing sick dogs examined at our veterinary medical facility for antibodies against *B henselae*. In June 2002, the Vector Borne Disease Diagnostic Laboratory at North Carolina State University began routine testing of serum samples to determine exposure to established tick-transmitted pathogens and for seroreactivity against *B henselae* antigens. All samples were obtained from dogs examined at our university veterinary medical teaching hospital. The purpose of the study reported here was to assess the clinical relevance of *B henselae* seroreactivity in sick dogs by comparing clinical, hematologic, biochemical, and cytologic data for dogs seroreactive to *B henselae* with data for non-seroreactive dogs.
Materials and Methods

Sample population—Dogs selected for inclusion in the study were derived from a population that had test results for antibodies against *B henselae* (seroreactive or nonseroreactive) for samples submitted for testing at the discretion of the attending clinician between June 2002 and January 2003. Samples for all dogs included in the study were tested against the same panel of vector-borne pathogens, including *Babesia canis*, *B henselae*, *B vinsonii subsp berkhoffii*, *Borrelia burgdorferi*, *Ehrlichia chiae*, and *Rickettsia rickettsii*. Testing was conducted by personnel at the Vector Borne Disease Diagnostic Laboratory at North Carolina State University. To avoid bias of the study population, we did not attempt to recruit dogs with specific disease manifestations or hematologic abnormalities.

Case dogs—Dogs were included when a serum sample was reactive against *B henselae* antigens at a reciprocal titer > 64 and adequate data were available in the medical record for use in statistical analysis. Data in the medical record were considered adequate when a final diagnosis was recorded, results of physical examinations were recorded, and results of requested diagnostic testing was available for review, summarization, and statistical analyses.

Control dogs—The control group was selected from dogs nonseroreactive to *B henselae* whose serum was submitted for testing against the same panel of vector-borne pathogens. A control dog was matched with the temporarily closest case dog for which a complete medical record was available. No attempt was made to match control dogs on the basis of age, sex, or disease process.

Serologic testing—Serologic testing was performed as described elsewhere. Briefly, *B henselae* organisms were harvested from Vero cell cultures and fixed onto 30-well oxide-coated slides. Twofold dilutions of serum were applied to the slides; slides were then incubated and washed. Fluorescein-isothiocyanate–conjugated goat anti-dog IgG was added to each slide. Slides were washed and examined at 40x magnification by use of a fluorescent microscope. A reciprocal titer of 64 or greater was considered seroreactive.

Review of medical records—Medical records for each case dog and each control dog were collected. Data collected included signalment; final diagnosis; duration of clinical signs; treatment history; results of physical examinations (including vital signs), lymph node palpation, ophthalmoscopic examination, orthopedic and neurologic examinations, hematologic and biochemical analysis, urinalysis, CSF analysis, analysis of fluid obtained by arthrocentesis, cytologic evaluation of aspirates obtained from lymph nodes, and abdominal ultrasonographic examination; and any other abnormal findings.

Statistical analysis—Dogs were grouped on the basis of involvement of major organ systems for statistical analysis. When > 1 organ system was involved, that dog was included in multiple groups.

Statistical analysis was conducted by use of odds ratios, descriptive statistics, the Student *t* test, *χ*² tests, and the Wilcoxon rank sum test. Odds ratios were used to compare proportions of dogs seroreactive and nonseroreactive to *B henselae* within general disease categories, including cardiac, neurologic, ophthalmologic, hematologic, lymphoid, muscular, renal, hepatic, neoplastic, endocrine, gastrointestinal, nasal, orthopedic, and vascular. The Wilcoxon rank sum test was used to compare values for clinicopathologic variables obtained from dogs seroreactive and nonseroreactive to *B henselae*. Several dogs were tested multiple times to detect antibodies against *B henselae*. Hematologic data obtained during the examination conducted closest in time to the initial serologic test result were used for comparison, and the final diagnosis relating to the clinical signs prompting serologic testing was used for categoric analysis.

Results

Animals—Between June 2002 and January 2003, 272 samples were tested to detect antibodies against *B henselae*, of which 68 were seroreactive. There was adequate information in the medical records of 40 dogs seroreactive to *B henselae* and 45 nonseroreactive control dogs.

Seroreactive dogs ranged from 1 to 15 years of age (mean, 7 years; median, 7 years). There were 15 neutered males, 10 sexually intact males, 13 spayed females, and 2 sexually intact females. Twenty-three breeds were represented, with Bassett Hound (n = 3), Labrador Retriever (3), Golden Retriever (2), Beagle (2), Boxer (2), Chihuahua (2), Cocker Spaniel (2), and Welsh Corgi (2) represented more than once.

Nonseroreactive control dogs ranged from 1 to 14 years of age (mean, 7 years; median, 7 years). There were 19 neutered males, 6 sexually intact males, 17 spayed females, and 3 sexually intact females. Breed distribution of the control dogs was similar to that of the seroreactive dogs, with Labrador Retriever (n = 7), Golden Retriever (3), Beagle (2), and Greyhound (2) represented more than once.

Results of serologic testing—Serologic evidence of exposure to other vector-borne pathogens was found in the dogs seroreactive to *B henselae* and the control dogs. Among the 40 dogs seroreactive to *B henselae*, 16 were also seroreactive to *R rickettsii* antigens, 5 were seroreactive to *B vinsonii subsp berkhoffii* antigens, and 4 were seroreactive to *E canis* antigens. Among the 45 control dogs, 12 were seroreactive to *R rickettsii* antigens, 1 was seroreactive to *B vinsonii subsp berkhoffii* antigens, and 2 were seroreactive to *E canis* antigens.

Results of physical examinations—We did not detect consistent abnormalities for physical examination during evaluation of the medical records. When recorded, fever (rectal temperature > 39.2°C) was evident in 7 dogs seroreactive to *B henselae* and 16 control dogs. Lymphadenopathy (solitary, regional, or generalized) was detected in 9 dogs seroreactive to *B henselae* and 13 control dogs.

Comparison of results for dogs seroreactive to *B henselae* and nonseroreactive control dogs—When the percentage of dogs seroreactive to *B henselae* that had various hematologic or biochemical values was compared with the percentage of nonseroreactive dogs with those same hematologic or biochemical values, there were no significant differences between groups (Tables 1 and 2). For example, 15 of 34 (44%) dogs seroreactive to *B henselae*, for which platelet counts were available, were thrombocytopenic, whereas 18 of 39 (46%) nonseroreactive dogs were also thrombocytopenic. This most likely reflected clinical recognition of thrombocytopenia as a hematologic abnormality in dogs with suspected tick-borne infections. Although the odds ratio for thrombocytopenia was 1.48, the 95% confidence interval (0.62 to 3.54) contained the value 1.0.
Samples of CSF obtained from 11 dogs seroreactive to *B. henselae* and 10 control dogs were examined. For the seroreactive dogs, protein concentration in CSF varied from 17.2 to 1,209 g/dL (mean ± SD, 168.9 ± 367.9 g/dL; median, 31.25 g/dL) and WBC count ranged from 0 to 5,150 cells/µL (mean, 565 ± 1,614 cells/µL; median, 6.5 cells/µL). Values for CSF analysis of nonseroreactive dogs did not differ from those for the seroreactive dogs.

Synovial fluid was examined in 5 dogs seroreactive to *B. henselae* and 4 control dogs. No unique identifying characteristics were identified in seroreactive dogs. All seroreactive dogs had an increase in the percentage of neutrophils in synovial fluid. The quantity of synovial fluid submitted for most dogs was insufficient to enable a cell count, and cellularity estimates were not consistently reported.

The primary disease process or processes for dogs seroreactive to *B. henselae* and control dogs were broadly grouped into various clinical categories (cardiac, neurologic, ophthalmologic, hematologic, lymphoid, muscular, renal, hepatic, neoplastic, endocrine, gastrointestinal, nasal, orthopedic, and vascular). There was no difference in the frequency of clinical category between dogs seroreactive to *B. henselae* and control dogs (Table 2). Although several variables had an odds ratio > 2.0, all associated 95% confidence intervals contained a value of 1.0, and analysis revealed values of *P* > 0.05.

The final disease diagnoses identified in dogs seroreactive to *B. henselae* were highly varied. Among the 7 seroreactive dogs that had orthopedic disease, 3 had neutrophilic polyarthritis confirmed by evaluation of fluid obtained during arthrocentesis. The other 2 dogs seroreactive to *B. henselae* were examined initially because of lameness, but synovial fluid was not examined. Of the 5 seroreactive dogs with lymphoid disease, 3 had lymphoid neoplasia and 2 had reactive lymphadenitis. Of the 3 dogs seroreactive to *B. henselae* that had hepatic disease, 1 had cholecystitis, 1 had a repaired congenital portosystemic shunt, and 1 had portal hypertension with multiple extrahepatic shunts and a mixed pattern of increased activity for hepatic enzymes. Of the 6 dogs seroreactive to *B. henselae* that had cardiac disease, 3 had vegetative valvular endocarditis, 1 was in heart failure, 1 was hypertensive, and 1 had tricuspid regurgitation. Among the 11 seroreactive dogs that had neurologic disease, 3 were examined because of seizures, 3 had intervertebral disk disease, 2 had myelopathy (including 1 dog with intervertebral disk disease), 2 were presumptively considered to have granulomatous meningoencephalitis, 1 had multifocal

### Table 1—Percentage of dogs seroreactive to *Bartonella henselae* and nonseroreactive control dogs that had hematologic values below, within, or above established laboratory reference ranges.

<table>
<thead>
<tr>
<th>Dogs</th>
<th>PCV</th>
<th>Platelets</th>
<th>Neutrophils</th>
<th>Band neutrophils</th>
<th>Lymphocytes</th>
<th>Monocytes</th>
<th>Eosinophils</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seroreactive</td>
<td>Below</td>
<td>25</td>
<td>44</td>
<td>0</td>
<td>NA</td>
<td>28</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Within</td>
<td>75</td>
<td>35</td>
<td>39</td>
<td>86</td>
<td>64</td>
<td>57</td>
</tr>
<tr>
<td></td>
<td>Above</td>
<td>0</td>
<td>21</td>
<td>61</td>
<td>14</td>
<td>8</td>
<td>37</td>
</tr>
<tr>
<td>Nonseroreactive</td>
<td>Below</td>
<td>14</td>
<td>46</td>
<td>5</td>
<td>NA</td>
<td>30</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Within</td>
<td>81</td>
<td>23</td>
<td>39</td>
<td>81</td>
<td>65</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td>Above</td>
<td>5</td>
<td>31</td>
<td>60</td>
<td>19</td>
<td>5</td>
<td>40</td>
</tr>
</tbody>
</table>

NA = Not applicable because the laboratory reference range includes a value of 0.

### Table 2—Number of dogs identified on the basis of disease category or variable and the number seroreactive to *B. henselae*.

<table>
<thead>
<tr>
<th>Disease category or variable</th>
<th>Total</th>
<th>Seroreactive to <em>B. henselae</em></th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatic</td>
<td>4</td>
<td>3</td>
<td>3.57</td>
<td>0.41–31.36</td>
</tr>
<tr>
<td>Lymphoid</td>
<td>7</td>
<td>5</td>
<td>3.07</td>
<td>0.60–15.69</td>
</tr>
<tr>
<td>Increase in CK activity</td>
<td>7</td>
<td>5</td>
<td>3.15</td>
<td>0.61–18.16</td>
</tr>
<tr>
<td>Orthopedic</td>
<td>10</td>
<td>7</td>
<td>2.97</td>
<td>0.75–11.79</td>
</tr>
<tr>
<td>Nasal</td>
<td>5</td>
<td>3</td>
<td>1.74</td>
<td>0.28–10.79</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>34</td>
<td>15</td>
<td>0.92</td>
<td>0.37–2.32</td>
</tr>
<tr>
<td>Hematuria</td>
<td>31</td>
<td>16</td>
<td>1.69</td>
<td>0.62–4.62</td>
</tr>
<tr>
<td>Cardiac</td>
<td>11</td>
<td>6</td>
<td>1.41</td>
<td>0.46–5.02</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>54</td>
<td>24</td>
<td>0.80</td>
<td>0.15–4.52</td>
</tr>
<tr>
<td>Hyperglobulinemia</td>
<td>16</td>
<td>8</td>
<td>1.27</td>
<td>0.42–3.83</td>
</tr>
<tr>
<td>Ophthalmologic</td>
<td>16</td>
<td>8</td>
<td>1.14</td>
<td>0.27–4.88</td>
</tr>
<tr>
<td>Increase in ALT activity</td>
<td>27</td>
<td>13</td>
<td>1.09</td>
<td>0.43–2.78</td>
</tr>
<tr>
<td>Anemia</td>
<td>15</td>
<td>9</td>
<td>2.06</td>
<td>0.86–5.38</td>
</tr>
<tr>
<td>Hypoalbuminemia</td>
<td>29</td>
<td>13</td>
<td>0.67</td>
<td>0.35–2.15</td>
</tr>
<tr>
<td>Neutrophilic</td>
<td>27</td>
<td>11</td>
<td>0.69</td>
<td>0.27–1.73</td>
</tr>
<tr>
<td>Hematologic</td>
<td>20</td>
<td>8</td>
<td>0.69</td>
<td>0.25–1.90</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>11</td>
<td>4</td>
<td>0.66</td>
<td>0.16–2.22</td>
</tr>
<tr>
<td>Neutrophilia</td>
<td>48</td>
<td>22</td>
<td>1.03</td>
<td>0.41–2.55</td>
</tr>
</tbody>
</table>

OR = Odds ratio. CI = Confidence interval. CK = Creatine kinase. ALT = Alanine transaminase.
neurologic disease (additional evaluation was declined by the owner), and 1 was examined because of a head tilt and ventrolateral strabismus. Of the 3 dogs seroreactive to *B. henselae* that had nasal disease, 2 had nasal masses and 1 had epistaxis. Nasal biopsy and histologic examination of specimens were not performed on any of the dogs with nasal disease. Of the 8 seroreactive dogs that had ophthalmologic disease, 2 had retinal detachment (1 with hypertension and 1 with glaucoma); 1 had anterior uveitis; and 1 had anterior uveitis, chorioretinitis, and hyphema.

Two of 4 dogs with granulomatous meningoencephalitis, 6 of 10 dogs with seizures, 3 of 4 dogs with immune-mediated hemolytic anemia, 22 of 48 dogs with neutrophilia, and 15 of 34 dogs with thrombocytopenia were seroreactive to *B. henselae* antigens. Three of the 4 dogs with vegetative valvular endocarditis, 5 of 10 dogs with polyarthritis, and 2 of 3 dogs with lymphoid neoplasia were also seroreactive to *B. henselae* antigens. Statistical analysis of these proportions was not possible as a result of selection bias because not all dogs evaluated for these specific disease processes were not included in the study sample and the seroreactivity rate among the study sample was 50% as a result of the study design.

**Discussion**

The genus *Bartonella* contains numerous species that can induce chronic intravascular infections in humans and other animals. *Bartonella* organisms have an intravascular life cycle and are able to induce persistent infections in the host while evoking minimal immunologic recognition; thus, it is difficult to establish a cause-and-effect relationship between these highly adapted bacteria and a diverse spectrum of disease abnormalities. This has caused some researchers to suggest that *Bartonella* spp are an exception to Koch’s postulate. Clearly, the evolutionary adaptation of these organisms to infect vectors and mammalian reservoirs suggests that pathogenicity may be the exception rather than the rule. If this assumption is true, then the close association between *B. henselae* and mammalian hosts would support commensalism or mutualism, rather than parasitism.

To our knowledge, other studies have not addressed *B. henselae* as a pathogen in dogs. Although the study reported here failed to identify clinical, hematologic, or biochemical abnormalities that could be attributed to *B. henselae* infection in dogs, the results will allow investigators to focus on specific disease entities to further define the role of *B. henselae* as a pathogen or a cofactor in disease expression in dogs.

If *B. henselae* contributes to neurologic disease in dogs, the spectrum of neurologic abnormalities found in the population of dogs reported here should not be surprising. In humans with atypical manifestations of cat-scratch disease, encephalopathy, seizures, encephalitis, meningitis, myelitis, radiculitis, and palsy have been reported. In the context of establishing causation for *B. henselae*-induced neurologic abnormalities in people, cat-scratch disease is a useful disease entity because of the prototypic triad of a cat scratch or bite, an inoculation granuloma, and lymphadenopathy.

Transient neurologic signs have also been described in cats experimentally infected with *B. henselae* or *B. claridgeiae*. Cultured fetal feline microglial cells have been experimentally infected. Neurologic abnormalities, including seizures and encephalitis, have been reported in a dog seroreactive to *B. vinsonii* subsp *berkhoffii*. That dog was part of a descriptive study; neurologic abnormalities resolved following administration of antimicrobials, and antibodies against *B. vinsonii* subsp *berkhoffii* were no longer detectable in convalescent serum samples. Similar to the dogs in the study reported here, results for CSF obtained from the dogs in the descriptive study ranged from within the reference range to a mixed or neutrophilic pleocytosis. In most instances, neurologic abnormalities described in humans and cats appear to be self-limiting and resolve without treatment during a period ranging from days to months. Future studies that address the course of *Bartonella*-associated neurologic disease in dogs will be necessary to define the clinical relevance of seroreactivity to *Bartonella* spp. Ideally, these studies should incorporate bacterial culture and PCR detection of organisms in samples of blood and CSF.

Unfortunately, hepatic biopsy specimens were not obtained from the 3 dogs seroreactive to *B. henselae* classified as having hepatic disease. Granulomatous hepatitis and peliosis hepatis are complications of *B. henselae* infection in humans. *B. henselae* DNA has been amplified by use of PCR assays in a dog with peliosis hepatis and another dog with granulomatous hepatitis, and *Bartonella claridgeiae* DNA has been amplified from a Doberman Pinscher with hepatopathy. Whether *Bartonella* spp can be primary pathogens in dogs or whether these organisms frequently induce persistent infections that are more easily detected in diseased hepatic tissues is yet to be established.

Lymphadenopathy is the hallmark of typical cat-scratch disease in humans. However, lymphadenopathy does not appear to be a defining characteristic of bartonellosis in dogs. Lymph node enlargement has not been reported in dogs seroreactive to *B. vinsonii* subsp *berkhoffii* and was recorded in the medical records of only 9 dogs seroreactive to *B. henselae* in the study reported here. In a case report, submandibular granulomatous lymphadenitis attributable to infection with *B. vinsonii* subsp *berkhoffii* developed in temporal association with attachment of a tick to the pinna of the dog’s ear.

Seroreactivity to *B. henselae* was identified in 5 of 10 dogs with polyarthritis in our study. In dogs seroreactive to *B. vinsonii* subsp *berkhoffii* in another study, polyarthritis was documented in 3 dogs, and arthralgia or myalgia was reported in 7 dogs. In humans, arthralgia and myalgia are reported as less common manifestations of cat-scratch disease, and *B. henselae* has been associated with rheumatoid arthritis in juveniles. Further evaluation of dogs with polyarthritis for molecular and serologic evidence of *Bartonella* spp infection will be necessary to determine whether *Bartonella* organisms play a role in the pathogenesis of polyarthritis.

The control dogs for the study reported here were selected from dogs for which serum was submitted for analysis.
testing against the same panel of vector-borne organisms but that lacked seroreactivity to \textit{B henselae} antigens. When dogs infected with \textit{B henselae} develop clinical, hematologic, or biochemical abnormalities that are similar to those found in association with other vector-borne infections, such as anaplasmosis, babesiosis, ehrlichiosis, or rickettiosis, then this control population would limit our ability to identify significant disease associations. However, this control population was purposely selected to reduce selection bias among the study population because the likely abnormalities (thrombocytopenia, anemia, polyarthritides, unexplained neurologic abnormalities, vegetative lesions on heart valves, and unexplained illness) are common reasons for submission of samples for testing to detect vector-borne diseases. The control population was limited to a number of dogs that was similar to that for the case population after initial review of the data indicated a low likelihood of increasing the statistical relevance of any potential associations because of the low prevalence of any specific disease entity or hematologic abnormality within the case population.

In the study reported here, we did not detect any significant disease associations that correlated with seroreactivity to \textit{B henselae}. The inability of this study to detect significant differences may reflect the highly varied disease processes in the seroreactive and nonseroreactive control populations, the low number of dogs in each disease category, multifactorial influences on disease expression, or a true lack of a relationship between seroreactivity to \textit{B henselae} antigens and clinical, hematologic, and biochemical abnormalities found in the study population. Inclusion of serologic tests for \textit{Bartonella} spp in epidemiologic studies of dogs with epilepsy and immune-mediated hemolytic anemia appears to be justified. In addition, prospective studies that attempt to correlate antibody detection with molecular evidence of \textit{B henselae} in blood samples or affected tissues of dogs with polyarthritis, granulomatous meningoencephalitis, hepatitis, and infective endocarditis will be required to define the role of \textit{B henselae} in these diseases.

\footnotesize{a. Whole molecule immunoglobulin G, Cappel, Organon Teknika Corp, Durham, NC.}

\textbf{References}


