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Prevalence of *Bartonella henselae* antibodies in serum of cats with and without clinical signs of central nervous system disease

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Bartonella henselae is occasionally associated with neurological dysfunction in people and some experimentally infected cats. The purpose of this study was to determine whether *B henselae* seroprevalence or titer magnitude varies among cats with neurological disease, cats with non-neurological diseases, and healthy cats while controlling for age and flea exposure. There was no difference in *B henselae* seroprevalence rates between cats with seizures and cats with other neurological diseases. Cats with non-neurological disease and healthy cats were more likely than cats with neurological disease to be seropositive. While the median *B henselae* antibody titer was greater in cats with seizures than in cats with other neurological disease, the median *B henselae* antibody titer was also greater in healthy cats than cats with seizures. The results suggest that titer magnitude cannot be used alone to document clinical disease associated with *B henselae* infection and that presence of *B henselae* antibodies in serum of cats with neurological disease does not prove the clinical signs are related to *B henselae*.

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Bacterial infection caused by *Bartonella henselae* is the most common cause of a benign regional lymphadenopathy known as 'cat scratch disease' (CSD) in children and young adults (Wheeler et al 1997). Affected people have frequently been in contact with cats. For example, in one study, cat contact was documented in 74% of 600 suspect cases (Dalton et al 1995). The organism is also associated with bacillary angiomatosis and bacillary peliosis, two proliferative vascular disorders that may occur in humans infected with human immunodeficiency virus (Chomel et al 2003). *Bartonella henselae* has been isolated from the blood of subclinically ill, seropositive cats and also from cats with a variety of clinical manifestations like fever, depression, anorexia, lethargy, lymphadenopathy, myalgia, uveitis, gingivitis (Ueno et al 1996, Lappin et al 2000) and some cats have neurological dysfunction (Guptill et al 1997, O'Reilly et al 1999). While most cats infected by *B henselae* never develop detectable clinical signs of disease, the spectrum of *B henselae* associated illnesses in diseased cats are similar to those observed in human patients with moderate to severe CSD (Carithers 1985, Carithers and Margileth 1991, Chomel et al 2003). *Bartonella* species seroprevalence in cats varies by region but is as high as 93% in some geographical areas of the United States (Jameson et al 1995, Foley et al 1998, Nutter et al 2004). The organism is transmitted between cats by fleas (Chomel et al 1996) and *B henselae* DNA was amplified from 34.8% and 30.4% of cats and their fleas, respectively (Lappin et al 2006). Thus, *B henselae* infection is most common in cats exposed to fleas.

Neurological complications of *B henselae* infection of people are rare. It has been estimated that only 2–3% of the estimated 24,000 human patients in the US who contract CSD annually

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develop neurological complications (Carithers 1985, Carithers and Margileth 1991, Jackson et al 1993, Margileth 1993). However, a human study that sought to identify the cause and characterize signs of encephalitis in California over a 2 year period found *Bartonella* species to be the most common bacterial agent associated with encephalitis (Glaser et al 2003). Signs of neurological dysfunction include encephalopathy (Carithers 1985, Carithers and Margileth 1991, Margileth 1993, Noah et al 1995), transverse myelopathy (Pickerill and Milder 1981), demyelinating polyneuropathy (McNeill et al 2000), facial nerve paralysis (Walter and Eppes 1998), aseptic meningitis (Wong et al 1995), cerebral arteritis (Selby and Walker 1979), neuroretinitis (Ormerod and Dailey 1999) and radiculopathy (Marra 1995). Encephalopathy is considered one of the most serious complications of CSD (Noah et al 1995, Chomel et al 2003) and can manifest with fever, headaches, mentation changes (aggression, confusion and excitability), combative behavior, tonic-clonic seizures, status epilepticus and occasionally, coma that develops 1–8 weeks after the onset of lymphadenopathy (Carithers 1985, Carithers and Margileth 1991, Weston et al 2001). Some studies have proposed that *B henselae* may have a role in the pathogenesis of acquired immunodeficiency syndrome (AIDS) encephalopathy (Patnaik et al 1992, Schwartzman et al 1994, 1995). The prognosis associated with CSD encephalopathy in immunocompetent patients is generally considered to be good with rare long term complications (Carithers 1985, Carithers and Margileth 1991).

Several studies have associated mild neurological dysfunction to *B henselae* infection of experimentally inoculated cats (Kordick and Breitschwerdt 1997, O'Reilly et al 1999, Malgorzata et al 2000) and *B henselae* can replicate in cells from the central nervous tissues of cats (Munana et al 2001). The neurological signs included exaggerated or diminished response to stimuli, aggressive behavior, focal seizures, nystagmus, and generalized tremors (Kordick and Breitschwerdt 1997, O'Reilly et al 1999, Malgorzata et al 2000). However, the role *B henselae* plays in neurological disease in client-owned cats is largely unknown. Similar neurological manifestations are common in client-owned cats for which a definitive diagnosis is unknown. The objective of this study was to compare *B henselae* antibody test results among groups of cats with and without clinical manifestations of neurological disease.

Material and methods

Experimental design

The records database in the Specialized Infectious Diseases Laboratory at Colorado State University was searched between January 2002 and May 2004 for feline serum sample submissions that listed neurological disease as a presenting complaint. All cases with adequate serum available for further testing were accepted (group 1). The submission paperwork was retrieved and each of the cats was further characterized by one of the authors (LP); the neurological complaints divided into seizures or other neurological signs. Two control groups were also selected. Group 2 consisted of sequentially selected, clinically ill cats for which adequate sample was available for additional testing, the presenting complaint was non-neurological, and the sample period encompassed at least a 1 year period. Group 3 consisted of sequentially selected, healthy cats for which adequate sample was available for additional testing, and the sample period encompassed at least a 1 year period. Age, breed, sex, and state were recorded for each case. Each sample had been previously submitted for infectious disease testing; *Toxoplasma gondii*, FeLV, FIV, and coronavirus were most common. Each sample had been stored at −20 °C or −80 °C until thawed and assayed in an IgG ELISA for the detection of antibodies against *B henselae* (Lappin et al 2000). In this ELISA, a 1:64 dilution of positive control, negative control, and suspect sera are each assayed in quadruplicate wells and the mean absorbances calculated. The mean absorbance values were converted to %ELISA units by use of the following formula: (test sample mean absorbance minus the negative control sample mean absorbance)/(positive control sample mean absorbance minus the negative control sample mean absorbance) multiplied by 100. An individual cat was considered positive for *B henselae* antibodies if the %ELISA value was greater than the mean %ELISA value plus 3SD of the pre-inoculation samples for a group of 26 specific pathogen-free cats (10 kittens at 8 weeks of age and 16 cats at 3 years of age). Positive suspect serum sample results were converted to estimated titers ranging from 1:64 to 1:4096 by comparing to a standard curve.

Statistical evaluation

Whether or not the cats in this study had been exposed to *Ctenocephalides felis* was unknown. Thus, the state of origin of the case was used to

classify each cat as high risk or low risk of exposure to *C felis* as previously described (Jameson et al 1995). Samples were categorized as being from areas with low (Alaska, Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah, Wyoming) or high flea prevalence (all other states). Age, flea risk, clinical manifestation, and *B henselae* titer were entered into a spreadsheet and analyzed by use of a commercially available statistical software package (SAS, SAS Institute, Cary, NC, Version 9.1). Relationships between *B henselae* serological status (positive or negative) and clinical presentations were first assessed by Fisher's exact test. Additionally, logistic regression was used to assess the influence of serological status, age and risk of flea exposure on clinical presentation. Relationships between *B henselae* titer magnitude and clinical manifestations were assessed by determining median titers for each group and evaluated with Wilcoxon's rank sum test. Significance was defined as $P < 0.05$.

Results

Group 1 consisted of 145 cats; seizures were reported for 63 cats and other neurological signs were reported for 82 cats. Of the cats, 44 were from low flea risk states (Colorado and Montana) and 101 were from high flea risk states (Arkansas, California, Florida, Illinois, Iowa, Kansas, Kentucky, Louisiana, Maine, Massachusetts, Michigan, Mississippi, Missouri, Nebraska, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Texas, Virginia, Washington, and Wisconsin). There was no difference in *B henselae* seroprevalence rates ($P = 0.0662$) between cats with seizures (37 of 63 cats; 59%) and cats with other neurological diseases (35 of 82 cats; 43%). When the analysis was expanded to include age and risk of flea exposure by logistic regression, neither of these additional factors were significant ($P > 0.05$) and the effect of seropositivity was still not significant ($P = 0.0707$). However, the median titers of cats with seizures (median 1:64; titer range 0−1:1024) were significantly greater ($P = 0.0227$) than cats with other neurological signs (median 0; titer range 0−1:2048).

Group 2 (clinically ill cats without neurological disease) consisted of 163 cats. Of the cats, 67 were from low flea risk states (Colorado, Montana, and New Mexico) and 96 were from high flea risk states (California, Florida, Iowa, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Massachusetts, Michigan, Minnesota, Missouri, Nebraska, New York, North Carolina, Ohio, Oklahoma, Pennsylvania, Texas, Washington, and Wisconsin). In the first evaluation, group 2 cats had a greater seropositive rate than group 1 cats (Table 1; $P = 0.0153$). When the analysis was expanded to include age and risk of flea exposure by logistic regression, neither of these additional factors influenced the seropositive rate within group ($P > 0.05$) and the effect of group was still significant ($P = 0.0026$). Median titers of cats were not significantly different ($P = 0.5682$) between group 1 (median 0; titer range 0−1:2048) and group 2 (median 1:64; titer range 0−1:4096).

Group 3 (healthy cats) consisted of 97 cats. Of the cats, 53 were from low flea risk states (Colorado and Utah) and 44 were from high flea risk states (California, Connecticut, Florida, Georgia, Illinois, Indiana, Iowa, Louisiana, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, New Hampshire, New Jersey, New York, North Carolina, Ohio, Tennessee, Texas, and Wisconsin). When group 1 cats were compared to group 3 cats, group was included in the statistical model as the covariate instead of neurological manifestations. In the first evaluation, group 3 cats had

Table 1. Relationships between *Bartonella henselae* seroprevalence rates between cats with and without neurological disease

	Seroprevalence # positive (%)	*P* value*
Group 1 ($n = 145$)	72 (49.7%)	
Group 2 ($n = 163$)	104 (63.8%)	0.0153
Group 3 ($n = 97$)	68 (70.1%)	0.0022

Group 1 consisted of cats with neurological disease, group 2 consisted of clinically ill cats without neurological disease, and group 3 consisted of healthy cats.
*Compared to group 1 by Fisher's exact test in the initial analysis. There was no significant effect of age or flea risk on results by logistic regression.

a greater seropositive rate than group 1 cats (Table 1; $P = 0.0022$). When the analysis was expanded to include age and risk of flea exposure (logistic regression), neither of these additional factors influenced the seropositive rate within group ($P > 0.05$) and the effect of group was still significant ($P = 0.0001$). Median titers of cats were significantly different ($P = 0.0411$) between groups with group 3 titers (median 64; titer range 0−1:8192) being greater than those for group 1 (median 0; titer range 0−1:2048).

Discussion

Samples for this retrospective study were submitted by a variety of veterinarians that included general practitioners and veterinary neurologists and the complete medical record was not available for most cases. Thus, one of the major limitations to this study is the lack of a consistent neurological examination between cases. And so there is the possibility that some cats in group 1 did not have neurological disease and some cats in group 2 had neurological disease which potentially could have influenced the results. Encephalopathy, with several classical manifestations including headaches, confusion, excitability, combative behavior, tonic-clonic seizures, status epilepticus and coma (Carithers 1985, Carithers and Margileth 1991, Armengol and Hendley 1999, Weston et al 2001) is the most common neurological complication of CSD in man, occurring in 2−3% of affected patients (Carithers 1985). Because these different clinical signs of encephalopathy can be hard to categorize in cats, we felt subdivision of the cases into categories other than seizures or other neurological manifestations would be potentially inaccurate. In future prospective studies, cases should be solicited from a limited number of veterinary neurologists to allow for more accurate case classification which may aid in detection of clinical disease associations.

The *B henselae* seroprevalence rates in this study are similar to those reported in other studies (Jameson et al 1995, Foley et al 1998, Nutter et al 2004, Guptill et al 2004). Because some experimentally inoculated cats develop neurological disease after inoculation with *B henselae*, it is likely the same syndromes occur in client-owned cats as well (Kordick and Breitschwerdt 1997, O'Reilly et al 1999, Malgorzata et al 2000). However, in this study, the prevalence of *Bartonella* species antibodies in serum of cats with neurological manifestations of disease (group 1) was less than a group of clinically ill cats without neurological signs (group 2) and a group of healthy cats (group 3). In another study of sick cats in North Carolina, prevalence of *B henselae* antibodies was similar between cats with and without neurological dysfunction (Breitschwerdt et al 2005). Results of these studies emphasize how difficult it can be to diagnose clinical bartonellosis in individuals based on the result of a single serological test result.

Flea exposure and age have both been associated with risk of exposure to *B henselae* but these variables did not affect the statistical outcomes in our study (Foley et al 1998, Guptill et al 2004). Because we could not accurately determine the flea risk for individual cats of this study we had to assign flea risk by state (Jameson et al 1995). It would have been optimal to have exact flea history instead of just state history as flea prevalence can vary between and within states.

It is unknown why some cats develop clinical bartonellosis and others do not. There are different variants and strains of *B henselae* which may have different pathogenic potentials (O'Reilly et al 1999, Malgorzata et al 2000, Engbaek and Lawson 2004, Fabbi et al 2004). In addition, bartonellosis in people relates to the host response to the pathogen (Resto-Ruiz et al 2003). In the study described here, additional clinical information was not available. Future prospective studies of potential neurological bartonellosis in cats should include a more detailed clinical history and diagnostic workup.

Antibodies that bind to *B henselae* antigens used in serological assays may not indicate exposure or infection with *B henselae*, which also may affect the diagnosis of bartonellosis. For example, antigens from culture derived *B henselae* were used in the ELISA used in this study. However, in titration experiments, sera from a number of cats currently polymerase chain reaction (PCR) assay positive for *B clarridgeiae* DNA but not *B henselae* DNA in blood were positive in the ELISA described herein. To our knowledge, *B clarridgeiae* has not been associated with clinical illness in cats. It is now apparent that some humans with suspected *B henselae* associated illness were actually infected by *B koehlerae* (Avidor et al 2004). In the future, prospective studies evaluating for potential disease associations with *Bartonella* species should use techniques like PCR assay or culture with subsequent genotyping to allow for more stringent organism

identification (La Scola and Raoult 1999, Sander et al 1999, Fenollar and Raoult 2004).

Predictive value of the magnitude of serum antibody titers has been assessed in several human studies with variable results (Fournier et al 2002, Rolain et al 2003). For example, *B henselae* antibody titers of varying magnitudes were commonly detected in healthy children in Italy (Massei et al 2004), healthy veterinarians in Japan (Kumasaka et al 2001), and people with CSD (Fournier et al 2002). In the study described here, median *Bartonella* species antibody titers were greater in cats with seizures than in cats with other neurological disease. However, the median *Bartonella* species antibody titer was also greater in healthy cats than cats with seizures. These results suggest that titer magnitude in cats cannot be used alone to document clinical disease associated with *Bartonella* species infection.

In summary, we believe the results of this study document that presence of serum antibodies alone cannot be used to document clinical neurological bartonellosis in cats and that improved diagnostic tests are needed. Cerebrospinal fluid analysis findings in people with neurological CSD are variable and non-specific (Carithers 1985, Carithers and Margileth 1991, Noah et al 1995). However, detection of *Bartonella* species antibody production by ocular tissues and microbial DNA has been amplified from aqueous humor of cats with uveitis (Lappin et al 2000). Use of these techniques with cerebrospinal fluid of cats may prove to be of benefit in the diagnosis of neurological bartonellosis.

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