Does *Feline leukemia virus* pave the way for *Bartonella henselae* infection in cats?

**Running title:** *B. henselae* infection in cats is associated with latent FeLV infection

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Abstract: Domestic cats serve as the reservoir hosts of *Bartonella henselae* and develop mild or no clinical symptoms after experimental infection. In humans, *B. henselae* infection can result in self-limiting cat scratch disease. However, immunocompromised patients may suffer from more severe courses of infection or even develop the potentially lethal bacillary angiomatosis. It was reasoned that cats with immunocompromising viral infections may react similarly to *B. henselae* infection. The aim of our study was to investigate the influence of the most important viruses known to cause immunosuppression in cats, *Feline leukemia virus* (FeLV), *Feline immunodeficiency virus* (FIV) and *Feline panleukopenia virus* (FPV), on natural *B. henselae* infection in cats. Accordingly, 142 cats from animal shelters were necropsied and tested for *B. henselae* and concurrent infections with FeLV, FIV or FPV by PCR and immunohistochemistry. A significant association between *B. henselae* and FeLV infection (*P* = 0.0028) was found, but not with FIV (*P* = 1.0) or FPV infection (*P* = 0.756), age (*P* = 0.392) or gender (*P* = 0.126). The results suggest that the susceptibility to *B. henselae* infection is higher in cats with concurrent FeLV infection, regardless if this is latent or progressive. Histopathology and immunohistochemistry for *B. henselae* failed to identify lesions that could be specifically attributed to *B. henselae* infection. We conclude that the course of natural *B. henselae* infection in cats does not seem to be influenced by immunosuppressive viral infections in general, but latent FeLV infection may predispose cats for *B. henselae* infection or persistance.

(250 words)

Keywords: *Bartonella henselae*, cat, *Feline leukemia virus*, *Feline immunodeficiency virus*, *Feline parvovirus*, immunosuppressive infectious diseases, latent FeLV infection
Introduction:
The outcome of an infection with Bartonella (B.) henselae in human patients depends mostly on their immune status. In immunocompetent patients B. henselae causes the self-limiting cat scratch disease, characterized by local granulomatous to abscedating lymphadenitis (10). Immunocompromised hosts, on the contrary, are prone to suffer from disseminated cat scratch disease with bacteremia and may develop angioproliferative lesions including peliosis hepatis or the potentially lethal bacillary angiomatosis (38, 39). Domestic cats are one well-known reservoir host of B. henselae and transmit the agent to humans through scratch marks or bite wounds. The prevalence of feline infection is high in animal shelters and populations of stray cats, as fleas transmit the agent among cats (6). A recent epidemiologic study revealed a prevalence of B. henselae bacteremia of 18.7% in cats from animal shelters, whereas only 1% of pet cats in the same area were found to be bacteremic (4). The clinical course and pathology of feline bartonellosis differs from that in human infections. In domestic cats, natural infection has been associated with gingivitis, stomatitis, lymphadenopathy, uveitis and urinary tract diseases (13, 21, 42). Experimental infection, on the other hand, leads to prolonged and relapsing intraerythrocytic bacteremia with unspecific mild or no clinical symptoms (1, 6, 19, 35). Histologically, specific pathogen free cats with acute B. henselae infection had marked generalized lymphatic hyperplasia (15, 20). In chronic experimental infections, a variety of additional unspecific histological lesions including lymphocytic interstitial nephritis, cholangitis, hepatitis and lymphoplasmacytic myocarditis were observed (20).

In contrast to the available data on bartonellosis in otherwise healthy experimentally infected cats, systematic investigations on the significance and lesions of natural B. henselae infection in cats with immunosuppressive diseases are missing.
The aim of the present study was to systematically investigate the influence of immunosuppressive infectious diseases on natural *B. henselae* infection in cats. We hypothesized that similar to the situation in humans, immunosuppressive diseases in cats may alter the susceptibility to *B. henselae* and the course of infection. Accordingly, we focused on the three most important agents known to cause immunosuppression in cats, *Feline leukemia virus* (FeLV), *Feline immunodeficiency virus* (FIV) and *Feline panleukopenia virus* (FPV).

**FeLV infection**: FeLV, a gamma retrovirus, causes immunosuppression in up to 50% of cats with progressive infection, and a replication-defective strain named FeLV-FAIDS causes the fatal feline acquired immunodeficiency syndrome (FAIDS) in 100% of viremic cats within the first year of infection (17, 27, 32). According to a recent study by Torres and colleagues (41), four possible courses of FeLV infection can be discriminated by determination of proviral and antigen load in blood or lymphatic tissues. Besides self-limited (abortive) and regressive courses, latent and progressive infections occur. Latent FeLV infections can be distinguished from progressive infections by the absence of detectable antigen (41). Besides the risk of infecting other cats following reactivation, the clinical significance of latent FeLV infection remains unclear (28, 30). Recent studies stated the prevalence of progressive FeLV infection in Germany between 2.9% (14) and 9% (40), whereas the percentage of latent infections can be as high as 50% (40). Interestingly, in one of these studies latent infections were significantly more prevalent in cats from animal shelters (40).

**FIV infection**: FIV, a feline lentivirus, is associated with an AIDS-like syndrome in the terminal phase of infection (29). Its prevalence is highest among free-roaming, male cats over 4 years of age (7). Initial infection is characterized by relatively mild clinical symptoms including transient fever, anorexia and generalized lymphadenopathy,
which can last up to several months (31). A second, asymptomatic phase follows and lasts up to 6 years (16). The onset of feline AIDS is defined by secondary and opportunistic infections due to immunosuppression. The over-all prevalence of FIV infection in Germany is thought to range between 2.5% (14) and 8.4% (12).

**FPV infection**: FPV causes feline panleukopenia characterized by enteritis, severe lymphatic depletion and bone marrow necrosis primarily in young cats. Resulting panleukopenia leads to severe immunosuppression.

**Coinfections**: Only two studies have previously investigated a possible link between *B. henselae* and FeLV or FIV infection in cats, with conflicting results. A 1997 study observed that the prevalence or clinical signs of *B. henselae* infection in cats were not influenced by FeLV or FIV infection (13). In contrast, a different study found that cats coinfected with FIV and *B. henselae* had a significantly increased frequency of lymphadenopathy compared to cats infected with FIV alone. However, the prevalence of *B. henselae* infection was not affected by FeLV or FIV infection when compared to controls (42). Associations of *B. henselae* with FPV infections have not been investigated before.

This study provides a systematic investigation of natural infections with FeLV, FIV and FPV, the three most important viral pathogens known to induce immunosuppression in felines, and their possible association with *B. henselae* in naturally infected cats.

**Material and Methods**:  
**Patients**: From October 2006 to November 2008, blood and tissue specimens were collected during necropsy from 70 male and 72 female cats from animal shelters in the Berlin area. Cats were divided into two age groups: 0-12 months and >1 year. At necropsy, tissue samples of mandibular, retropharyngeal and popliteal lymph nodes,
tonsils, spleen, liver and peripheral blood were collected. All specimens were split in
half and frozen at -20°C or fixed in 10% formalin and embedded in paraffin through
graded alcohols. In addition, histopathology was performed on all major organ
systems using hematoxylin and eosin stained 4 µm sections from formalin-fixed,
paraffin-embedded tissue specimens.

**PCR testing for B. henselae:** DNA was extracted from all frozen tissue samples
using a commercial kit (QiaAmp DNA Mini Kit, Qiagen, Hilden, Germany). *B.
* henselae DNA was amplified using two separate nested PCR assays targeting
different segments of the htrA gene (GenBank No. L20127), as described previously
(5). Primer sequences are given in Table 1. Samples that tested positive in either of
the two htrA assays were confirmed by replication of both htrA assays and by a
second species-specific nested PCR targeting the citrate synthase (gltA) gene of *B.
henselae* (GenBank No. L38987) as described by Regnath *et al* (34). PCR was
performed in the same way as the htrA assays, except for an annealing temperature
of 65°C in the first amplification round and a primer concentration of 25 pmol with 1
mM MgCl₂. The detection limit of all nested PCR assays was determined to be
approximately 1 CFU by serial dilution of culture-grown, heat-deactivated *B.
henselae*. Bacterial DNA from culture-grown, hydrolyzed *B. henselae* strain Berlin-1
was used as positive controls (kindly provided by Mardjan Arvand, Rostock
University). Negative controls were included in all amplifications with no template
DNA. PCR products were visualized by ethidium bromide staining after agarose gel
electrophoresis.

**Immunohistochemical detection of B. henselae:** Tissue samples from all PCR-
positive cats underwent immunohistochemical analysis (IHC) using *Bartonella-
specific*, affinity-purified rabbit immunoglobulin G (IgG) as primary antibody, as
described previously (5). Vero cells infected with *B. henselae* were used as positive controls, whereas uninfected Vero cells served as negative controls.

**Detection of FeLV, FIV and FPV infection:** Primers and annealing temperatures for all amplifications are summarized in table 2 with otherwise identical PCR conditions as for *B. henselae*.

**FeLV infection:** Bone marrow was tested for FeLV genome by semi-nested PCR (18, 40) targeting the U3 LTR region of the FeLV provirus (GenBank No. L25632). To distinguish between progressive and latent FeLV infection, IHC was performed from bone marrow, spleen and mandibular lymph nodes for all cats with detectable proviral DNA in bone marrow. Tissue from a feline case of FeLV gp70 antigen positive malignant lymphoma served as positive control. Antigen retrieval was performed by 12 min microwave heating (560 W) in 10 mM citric acid, pH 6.0, supplemented with 0.05% Triton X-100. As primary antibody, mouse monoclonal IgG directed against the envelope protein gp70 of Feline leukemia virus was used diluted 1:200 (MCA1897, AbD Serotec, Duesseldorf, Germany). Negative controls were included for all slides using purified mouse IgG from preimmune sera diluted 1:200 (Mouse Super Sensitive™ Negative Control HK119, BioGenex Laboratories, San Ramon, CA). Biotinylated goat anti-mouse IgG was used as secondary antibody, diluted 1:200 (Vector Laboratories Inc., Burlingame, CA). Staining was effected using a streptavidin-biotin-peroxidase system (Vectastain Elite ABC-Kit, Vector Laboratories Inc.) with 3′,3″-diaminobenzidine - tetrahydrochloride as chromogen (DAB buffer tablets, Merck, Darmstadt, Germany).

**FIV infection:** Peripheral blood was tested for FIV infection by semi-nested PCR targeting the proviral gag protein gene (GenBank No. NC_001482) as described by English *et al* (11).
FPV infection: FPV infection was detected by histopathology and standard PCR from bone marrow and peripheral lymph nodes of all cats, using primers targeting the VP2 gene (GenBank No. GQ169552) of FPV (37).

Statistical evaluation: To test for associations between *B. henselae* infection and FeLV, FIV or FPV infection, sex or age, two-tailed Fisher’s exact tests were performed using the statistical software SPSS 16.0 with a specified significance level of *P* < 0.05.

Results:

The median age of the cats was 12 months with a range of 0.5 months to 18 years and a mean age of 48 months. 50% were 0-12 months and 47.2% older than 1 year. The age of four cats (2.8%) was unknown. All cats had either died spontaneously or were euthanized due to the lesions summarized in table 3.

The overall prevalence of *B. henselae* infection was found to be 7.7%. In 11 of 142 cats, *B. henselae* DNA was detected by PCR in blood and/or peripheral lymph nodes, spleen, tonsils and bone marrow, with the highest rate of detection in peripheral lymph nodes and blood (table 4). In 3 of the 11 cats, *B. henselae* was also identified immunohistochemically in lymphatic tissues, characterized by a strong cytoplasmatic signal in individual histiocytic cells, similar to the pattern seen in human cat scratch disease (22). However, histopathology and immunohistochemical analysis failed to identify any organ lesions that could be attributed to *B. henselae*. None of the lesions previously linked with experimental or natural infection were observed in increased frequencies in cats with *B. henselae* infection in this study when compared to controls.

Table 5 summarizes the prevalence of *B. henselae* infection in relation to infections with FeLV, FIV or FPV. 6 of 142 cats (4.2%) were infected with FeLV, four of which
were coinfected with *B. henselae*. Interestingly, latent FeLV infection (provirus positive, gp70 negative) was found in three of the four cats with concurrent FeLV and *B. henselae* infection, whereas only one of two cats with progressive FeLV infection (provirus positive, gp70 positive) was coinfected with *B. henselae*. Blood from four cats (2.8%) tested positive for FIV proviral DNA, but none of them had *B. henselae* infection. One of the four cats had typical lesions consistent with the lymphadenopathy stage of FIV infection. The other three cats had no histological changes, suggesting they were in the asymptomatic stage. FPV infection was detected in 62 of 142 cats (43.7%) by characteristic histopathological changes of lymphatic depletion, bone marrow necrosis and intestinal crypt epithelial necroses. All cases were confirmed by FPV-PCR. Of 62 cats with FPV infection, only four were coinfected with *B. henselae*.

Statistical evaluation revealed a significant association between *B. henselae* and FeLV infection (*P* = 0.00028), but not with FIV (*P* = 1.0) or FPV infection (*P* = 0.348), age (*P* = 0.524) or sex (*P* = 0.126).

**Discussion:**

In the present study, 142 cats from animal shelters were tested for *B. henselae* infection and concurrent viral infections known to cause immunosuppression in cats. Interestingly, the prevalence of *B. henselae* infection was found to be significantly correlated only with FeLV infection, regardless whether this was latent or progressive. There was no correlation with FIV or FPV infection or other allegedly immunosuppressive conditions including cachexia, very old or very young age. At the same time there was no evidence that the course and pathology of *B. henselae* infection are influenced either by FeLV infection or other conditions known to cause immunosuppression in cats.
There are several possible explanations for the noted association of FeLV and *B. henselae* infection. On the one hand, both *B. henselae* and FeLV are transmitted by fleas, which may account for the increased coinfection rate. On the other hand, both agents have a tropism to hematopoietic progenitor cells. FeLV subgroup A and C have been shown to infect bone marrow cells of all three lineages. In addition, FeLV-C inhibits erythroid progenitor cells at the level of colony-forming units, resulting in aplastic anemia. Maendle et al. have recently demonstrated invasion and persistence of *B. henselae* in human CD34+ hematopoietic progenitor cells, which resulted in a reduced proliferation but did not affect the cells’ erythroid differentiation. In consequence, infection was sustained even through their maturation, accounting for intracellular presence of *B. henselae* in human erythrocytes. In contrast, the agent was shown to be unable to invade human erythrocytes directly. Whether the same mechanism applies to the cat is not known. However, infection of hematopoietic progenitor cells may be an explanation for the high number of intracellular *B. henselae* previously detected in feline red blood cells by immunofluorescent techniques. Initially this seemed to be conflicting because direct invasion of *B. henselae* into feline erythrocytes is not very efficient. Based on these results, we hypothesize that the infection of hematopoietic progenitor cells by FeLV infection inadvertently prepares the ground for *B. henselae*, making the cells more susceptible to either infection by *B. henselae* or bacterial persistence. Mechanisms and functional aspects of this putative interaction will require further investigation.

There was no apparent association of *B. henselae* infection with FIV infection in the present study. This is consistent with the findings of previous studies in cats. Lack of correlation between the prevalence of FIV and *B. henselae* infection may be due to the fact that FIV, like HIV unlike *B. henselae* and FeLV, is not transmitted by
fleas. Similarly, HIV is not arthropode-borne (8). In contrast to *B. henselae* and FeLV, 
FIV does not affect hematopoietic progenitor cells (23). Similarly, although HIV may 
induce severe bone marrow depression, infection of hematopoietic progenitor cells in 
vivo is extremely rare (26).

To our knowledge a possible link between FPV and *B. henselae* infection has not 
been investigated before, although it is a common infection of young cats, leading to 
abrupt systemic lymphatic depletion and bone marrow necrosis. In the present study, 
FPV and *B. henselae* infection were not associated. This may have been due to the 
young age of the cats tested here, and the generally acute and rapid courses of 
feline panleukopenia, killing the cats before they had a chance to be coinfected with 
*B. henselae*.

Compared to a previous study in the same geographical area (4) that had observed 
*B. henselae* bacteremia in 18.7% cats from animal shelters, we found the prevalence 
of 7.7% to be markedly lower. This may be due to biological variance, or it may be 
linked to better flea control in the contributing animal shelters. Seasonal influence on 
prevalence can largely be excluded as sampling spanned 26 months. Interestingly, in 
three of the 11 infected cats (27%), the agent was detected in various organs but 
they had no bacteremia. This may suggest that *B. henselae* is sustained in internal 
organs of the reservoir host even after bacteremia has been cleared. However, we 
collected blood samples only once during necropsy, so it is possible that several of 
the cats were in a non-bacteremic phase of *B. henselae* infection which could have 
been identified by two or more subsequent blood tests in living animals.

Histopathological and immunohistochemical analysis disclosed no specific organ 
changes for *B. henselae* infection. Immunohistochemistry revealed a cytoplasmic 
signal in just a few histiocytes within lymphatic tissues, similar to the pattern 
observed in human cat scratch disease (22). None of the lesions previously
described in experimental or natural feline bartonellosis including stomatitis, lymphadenopathy, uveitis, urinary tract diseases, cholangitis, hepatitis or myocarditis were observed in an increased frequency in the *B. henselae*-infected cats compared to the uninfected controls, and in this aspect there was no difference between cats with diseases known to cause immunosuppression and controls.

In conclusion, our results suggest that the susceptibility to *B. henselae* infection may be raised in cats with FeLV infection, regardless whether this continues as latent or progressive infection. This could be due to interference at the level of the agents’ target cell, possibly the hematopoietic progenitor cell. In contrast, the course of *B. henselae* infection seems not to be influenced by concurrent infection with agents known to cause immunosuppression, including FeLV or FPV infection. However, due to the low over-all prevalences of FeLV and FIV infections in this study, controlled coinfection studies will be necessary to confirm and further characterize the effect of coinfections with these immunosuppressive viral agents on *B. henselae* infection in cats.

In addition, these findings may help to elucidate the significance of latent FeLV infection in cats which is as yet unclear.

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Cell 118:757-766.


Table 1: Primers and annealing temperatures for the detection of *B. henselae* by PCR.

<table>
<thead>
<tr>
<th>Assay / target gene</th>
<th>Primer name</th>
<th>Primer sequence (5'-3')</th>
<th>Annealing temperature</th>
<th>Amplicon size</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>htrA I (1209-1372 nt)</td>
<td>3+ (outer forward)</td>
<td>gtg cgt taa tta ccg atc ca</td>
<td>58.0 °C</td>
<td>163 bp</td>
<td>(3, 5)</td>
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<tr>
<td></td>
<td>4- (outer reverse)</td>
<td>cca aac tcc taa ggt tac tagt ttc</td>
<td>58.0 °C</td>
<td>163 bp</td>
<td>(3)</td>
</tr>
<tr>
<td></td>
<td>1+ (inner forward)</td>
<td>ctc cca tca agg cag gta cta</td>
<td>58.0 °C</td>
<td>82 bp</td>
<td>(5)</td>
</tr>
<tr>
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<td>2- (inner reverse)</td>
<td>gca ata cgc tgt gct aga tcac</td>
<td>58.0 °C</td>
<td>82 bp</td>
<td>(5)</td>
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<td>htrA II (1250-1332 nt)</td>
<td>8+ (outer forward)</td>
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<td>(5)</td>
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<td></td>
<td>5- (outer reverse)</td>
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<td></td>
<td>8- (inner reverse)</td>
<td>cag aca tca cat gat tat gtc cta</td>
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<tr>
<td>gltA (504-1178 nt)</td>
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<td>(34)</td>
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<td>r1 (outer reverse)</td>
<td>cag cgc ctc tct gct gct aat g</td>
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<td>675 bp</td>
<td>(34)</td>
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<tr>
<td></td>
<td>f2 (inner forward)</td>
<td>atg cct aaa aat gtt cgt aga</td>
<td>58.0 °C</td>
<td>354 bp</td>
<td>(34)</td>
</tr>
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<td></td>
<td>r2 (inner reverse)</td>
<td>cgt gct aat gca aaa aga ac</td>
<td>58.0 °C</td>
<td>354 bp</td>
<td>(34)</td>
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Table 2: Primers and annealing temperatures for PCR detection of FIV, FeLV and FPV

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<tr>
<th>Assay / target gene</th>
<th>Primer name</th>
<th>Primer sequence (5'-3')</th>
<th>Annealing temperature</th>
<th>Amplicon size</th>
<th>References</th>
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<td>FIV provirus / gag protein gene</td>
<td>E 1+ (forward)</td>
<td>cca caa tat gta gca ctt gac c</td>
<td>56.0°C</td>
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<tr>
<td>(1057-1639 nt)</td>
<td>E 2- (outer reverse)</td>
<td>ggg tac ttg cta gta ggt ggc</td>
<td>56.0°C</td>
<td>583 bp</td>
<td>(11)</td>
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<td></td>
<td>N 4- (inner reverse)</td>
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<td>184 bp</td>
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<td>(2107-2290)</td>
<td>r1 (reverse)</td>
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<td></td>
<td>(18, 40)</td>
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<td>f2 (inner forward)</td>
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<td>FPV / VP 2 gene</td>
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<td>55.0°C</td>
<td>172 bp</td>
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<td>55.0°C</td>
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Table 3: Lesions resulting in death or euthanasia in 142 cats included in this study

<table>
<thead>
<tr>
<th>Lesions resulting in death or euthanasia</th>
<th>Number of cats affected (n = 142)</th>
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<tr>
<td><strong>Inflammatory lesions</strong></td>
<td></td>
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<tr>
<td>Gastroenteritis due to Feline panleukopenia virus</td>
<td>62</td>
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<tr>
<td>Gastroenteritis due to other causes</td>
<td>9</td>
</tr>
<tr>
<td>Respiratory infections</td>
<td>15</td>
</tr>
<tr>
<td>Urogenital diseases</td>
<td>4</td>
</tr>
<tr>
<td>Feline infectious peritonitis</td>
<td>5</td>
</tr>
<tr>
<td>Other</td>
<td>5</td>
</tr>
<tr>
<td><strong>Hypertrophic cardiomyopathy</strong></td>
<td>14</td>
</tr>
<tr>
<td><strong>Neoplasia of various organs</strong></td>
<td>9</td>
</tr>
<tr>
<td><strong>Degenerative diseases</strong></td>
<td>8</td>
</tr>
<tr>
<td><strong>Trauma</strong></td>
<td>2</td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
<td>9</td>
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</tbody>
</table>
Table 4: Details of 11 cats which tested positive for *B. henselae* infection including data on coinfections with FeLV, FIV or FPV.

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Sex, Age</th>
<th>Detection of <em>B. henselae</em> by PCR and IHC</th>
<th>FeLV infection</th>
<th>Other viral infections</th>
<th>Cause of death</th>
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<tbody>
<tr>
<td>1</td>
<td>male, 9b</td>
<td>popliteal ln, popliteal ln</td>
<td>latent</td>
<td>none</td>
<td>hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(provirus + / gp70 -)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>male, 108</td>
<td>Blood, negative</td>
<td>latent</td>
<td>none</td>
<td>enteric leiomyoma</td>
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<td></td>
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<td>(provirus + / gp70 -)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>male, 3</td>
<td>popliteal ln, tonsils, spleen, blood</td>
<td>no infection</td>
<td>none</td>
<td>urolithiasis, hydronephrosis, emaciation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>male, n.a.</td>
<td>Tonsils, negative</td>
<td>no infection</td>
<td>FPV</td>
<td>necrotizing enteritis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Female, 3</td>
<td>spleen, bone marrow</td>
<td>no infection</td>
<td>FPV</td>
<td>necrotizing rhinitis, bone marrow necroses</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>male, 4</td>
<td>Blood, negative</td>
<td>latent</td>
<td>FPV</td>
<td>necrotizing enteritis and rhinitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(provirus + / gp70 -)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
...Table 4 continued

<table>
<thead>
<tr>
<th></th>
<th>Gender</th>
<th>Age</th>
<th>Sample Type</th>
<th>Finding</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>male, adult</td>
<td>tonsils, blood, mandibular, retropharyngeal and popliteal ln</td>
<td>negative</td>
<td>progressive (provirus + / gp70 +)</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>male, 144</td>
<td>blood</td>
<td>negative</td>
<td>no infection</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>female, 36</td>
<td>Blood</td>
<td>negative</td>
<td>no infection</td>
<td>none</td>
</tr>
<tr>
<td>10</td>
<td>female, 5</td>
<td>Blood</td>
<td>negative</td>
<td>no infection</td>
<td>FPV</td>
</tr>
<tr>
<td>11</td>
<td>male, 96</td>
<td>Blood</td>
<td>negative</td>
<td>no infection</td>
<td>none</td>
</tr>
</tbody>
</table>

Table footnote: IHC = immunohistochemical analysis, FeLV = Feline leukemia virus, FIV = Feline immunodeficiency virus, FPV = Feline panleukopenia virus, ln = lymph node
Table 5: Prevalence of *B. henselae* infection in relation to viral infections associated with immunosuppression in cats.

<table>
<thead>
<tr>
<th>Viral coinfection</th>
<th><em>B. henselae</em> positive (<em>n</em> = 11)</th>
<th><em>B. henselae</em> negative (<em>n</em> = 131)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FeLV latent infection</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Progressive infection</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>FIV positive</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>FPV positive</td>
<td>4</td>
<td>58</td>
</tr>
<tr>
<td>none of the above</td>
<td>3</td>
<td>67</td>
</tr>
</tbody>
</table>