

T-lymphotropic virus from domestic cats with an immunodeficiency-like syndrome. *Science* 1987;235:790-793.

5. Sparger EE. Feline T-lymphotropic lentivirus infection. *Feline Med* (Vet Learning Systems) 1988;4:9-14.

6. Lutz H, Pedersen NC, Dubin R, et al. Monoclonal antibodies to three epitopic regions of feline leukemia virus p27 and their use in enzyme-linked immunosorbent assay of p27. *J Immunol Methods* 1983;56:209-220.

7. O'Connor TP Jr, Tanguay S, Steinman R, et al. Development and evaluation of immunoassay for the detection of antibodies to the feline T-lymphotropic lentivirus (feline immunodeficiency virus). *J Clin Microbiol* 1989;27:474-479.

8. Yamamoto JK, Hansen H, Ho EW, et al. Epidemiologic and clinical aspects of feline immunodeficiency virus infection in cats from the continental United States and Canada and possible mode of transmission. *J Am Vet Med Assoc* 1989;194:213-220.

9. Hosie MJ, Robertson C, Jarrett O. Prevalence of feline leukemia virus and antibodies to feline immunodeficiency virus in cats in the United Kingdom. *Vet Rec* 1989;128:293-297.

10. Ishida T, Washizu T, Toriyabe K, et al. Feline immunodeficiency virus infection in cats of Japan. *J Am Vet Med Assoc* 1989;194:221-225.

11. Yamamoto JK, Sparger E, Ho EW, et al. Pathogenesis of experimentally induced feline immunodeficiency virus infection in cats. *Am J Vet Res* 1988;49:1246-1258.

12. Haase AT. Pathogenesis of lentivirus infections. *Nature* (London) 1986;322:130-136.

13. Shelton GH, Waltier RM, Connor SC, et al. Prevalence of feline immunodeficiency virus and feline leukemia virus infections in pet cats. *J Am Anim Hosp Assoc* 1989;25:7-12.

## Hematologic abnormalities in cats seropositive for feline immunodeficiency virus

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**F**eline immunodeficiency virus (FIV), a retrovirus (subfamily, Lentivirinae), is associated with an immunodeficiency syndrome in domestic cats.<sup>1</sup> After a prolonged period when clinical signs of disease are not manifested (eg, months to years), FIV infection is characterized by a variety of chronic disorders, opportunistic infections, and neoplasia.<sup>2-5</sup> Morphologic and biologic properties of FIV closely resemble those of human immunodeficiency virus (HIV), a lentivirus that is the etiologic agent of acquired immunodeficiency syndrome (AIDS) in human beings.<sup>1,2,6</sup> Hematologic abnormalities are a common feature of HIV infection.<sup>7-11</sup> Although lymphopenia attributable to depletion of CD4-positive (helper/inducer) cells is the most prominent abnormality, other cytopenias are often seen. The prevalence of hematologic disorders increases with progression of HIV-associated disease.<sup>8</sup> In addition, drug-induced neutropenia is frequent in AIDS patients treated with antimicrobial agents, including trimethoprim-sulfamethoxazole, pyrimethamine-sulfadoxine, and pentamidine.<sup>12-14</sup> This adverse reaction contrasts to that of drugs that induce bone marrow suppression in HIV-seropositive and noninfected individuals. The pathogenesis

of these HIV-associated hematologic abnormalities has yet to be elucidated.

The hematologic status of cats with naturally acquired FIV infection has been recently studied.<sup>5,15,16</sup> Cats with clinical signs of disease have blood and bone marrow abnormalities comparable to those reported in HIV-seropositive human beings.<sup>15</sup> Furthermore, FIV-infected cats appear to have increased incidence of drug (griseofulvin)-induced neutropenia.<sup>16</sup>

### Blood Abnormalities

Hematologic variables, including CBC, total and differential WBC counts, and platelet numbers, were assessed in 53 cats referred to the clinic because of spontaneous FIV infection.<sup>15</sup> Diagnosis of FIV infection was based on detection of FIV-specific antibodies in serum, using ELISA<sup>a</sup> and immunoblot analysis (Western blot).<sup>5</sup> Eighteen cats (34%) were coinfecting with FeLV as determined by either ELISA<sup>b</sup> or immunofluorescent antibody testing.<sup>c</sup>

Hematologic abnormalities were detected in 40 of 53 (75%) cats. Cytopenias were the most prevalent findings (Table 1). Overall, anemia, lymphopenia, neutropenia, and thrombocytopenia were observed in 36, 53, 34, and 8% of cats,

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Supported in part by research grants DK41934, CA21620, and HL02396 from the National Institutes of Health.

<sup>a</sup>FTLV PetChek ELISA, IDEXX Corp, Portland, Me.

<sup>b</sup>FeLV PetChek Screening/Confirmatory ELISA, IDEXX Corp, Portland, Me.

<sup>c</sup>FeLeuk test, National Veterinary Laboratory, Franklin Lakes, NJ.

Table 1—Hematologic cytopenias in feline immunodeficiency virus (FIV)-seropositive cats

Abnormality (definition)	Percent of cats		
	FeLV - (n = 35)	FeLV + (n = 18)	Total (n = 53)
Anemia (Hct < 24%)	31	44	36
Leukopenia (<5,500 wbc/ $\mu$ l)	23	56	34
Lymphopenia (<1,500 lymph/ $\mu$ l)	51	56	53
Neutropenia (<2,500 segs/ $\mu$ l)	26	50	34
Thrombocytopenia (<150,000 platelets/ $\mu$ l)	6	11	8

Hct = hematocrit; Lymphs = lymphocytes; segs = segmented neutrophils; - = negative; + = positive.  
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respectively. Multiple concurrent cytopenias were common (42% of cases), including 3 cats (6%) with pancytopenia. Prevalence of individual cytopenias among FeLV-positive or -negative cats was similar, with the exception of leukopenia, which was more frequent among coinfecting cats and was largely attributable to neutropenia (Table 1). Leukocytosis (>19,500 WBC/ $\mu$ l) was not observed; however, 2 cats (4%) had neutrophilia (>12,500 mature neutrophils/ $\mu$ l), 3 cats (6%) had lymphocytosis (>7,000 lymphocytes/ $\mu$ l), and 1 cat (2%) had eosinophilia (>1,500 eosinophils/ $\mu$ l). In a survey of FIV-seropositive cats manifesting clinical signs of disease, Yamamoto et al<sup>3</sup> reported similar frequency of cytopenias, but increased incidence of leukocytosis (15%) and neutrophilia (35%), compared with our results. These differences may reflect the diversity of disease states among cats with FIV infection.

In our study, mean ( $\pm$ SD) distribution of hematocrit and total leukocyte, lymphocyte, neutrophil, and platelet numbers in FIV-infected cats with cytopenias were  $17.5 \pm 5.5\%$ ;  $2,930 \pm 1,290$  WBC/ $\mu$ l,  $790 \pm 440$  lymphocytes/ $\mu$ l,  $1,220 \pm 630$  neutrophils/ $\mu$ l, and  $55,600 \pm 63,700$  platelets/ $\mu$ l, respectively. Significant difference was not evident in the severity of cytopenias between cats with FIV infection alone and cats coinfecting with FeLV.

The FIV-seropositive cats had a variety of clinical conditions ranging from occult infection to malignancy. To evaluate the relationship between hematologic abnormalities and severity of disease, infection in cats was clinically staged, using a modification of criteria adapted from the Centers for Disease Control classification system for HIV infection.<sup>4</sup> Using this approach, 9 cats (17%) lacked clinical signs of disease, 20 cats (38%) had "AIDS" (opportunistic infections, >20% weight loss, neurologic disease, and/or malignancy), and 24 cats (45%) had "AIDS"-related complex ("ARC"), clinical illness not meeting AIDS criteria. A similar distribution of clinical stages was seen among FeLV-

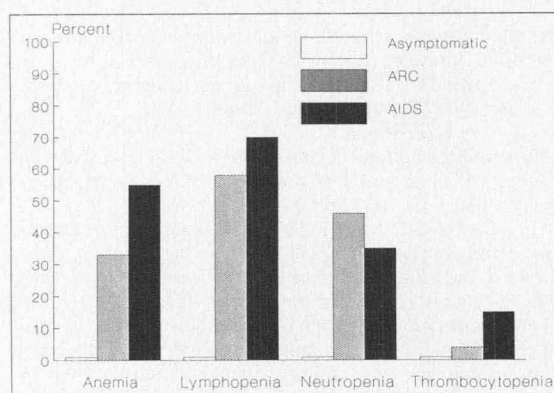


Figure 1—Distribution of cytopenias by clinical stage in FIV-seropositive cats. (Asymptomatic refers to lack of clinical signs of disease; see text for derivation of abbreviations). Reprinted with permission from *Blood* 1990;76:1107.

negative and FeLV-positive cats. Cytopenias were not seen in FIV-seropositive cats not manifesting clinical signs of disease (Fig 1). One of these (FeLV-negative) cats had lymphocytosis (9,300 lymphocytes/ $\mu$ l), but blood values were otherwise unremarkable in these cats. In contrast, cytopenias developed in 89% of cats manifesting clinical signs of disease (ie those with "ARC" or "AIDS"). In general, development of hematologic abnormalities increased with severity of signs of disease; however, these differences were not statistically significant. Likewise, severity of the cytopenias was similar among cats with "ARC" and cats with "AIDS."

To rule out the possibility that secondary pathogens contributed to the hematologic abnormalities observed in FIV-seropositive cats, sera from FIV-infected (n = 38) and clinically normal cats (n = 10) were screened for antibodies to *Toxoplasma gondii* and feline coronaviruses by use of indirect hemagglutination and indirect fluorescent antibody assays, respectively. There was no apparent correlation between titer to these agents and hematologic abnormalities. Therefore, it appears that hematologic disorders in FIV-infected cats are not simply the result of concurrent infection with known opportunistic pathogens (ie, FeLV, *T gondii*, or feline coronaviruses).

Blood abnormalities associated with FIV infection are strikingly similar to those seen in HIV-seropositive human beings. In particular, cytopenias develop with similar prevalence among cats with signs of FIV infection and HIV-infected patients with ARC or AIDS.<sup>7-11</sup> Also, in general, the prevalence of hematologic disorders increases with the severity of the disease in cats with FIV and people with HIV infections. Hematologic abnormalities in people are a consistent feature of AIDS, in that 65 to 95% of patients are anemic, 48 to 79% are lymphopenic, 20 to 46% are neutropenic, and 29 to 59% are thrombocytopenic.

### Lymphoid Malignancies

Lymphoid malignancies (ie, lymphoma or

lymphoblastic leukemia) were detected in 26% (14/53) of the FIV-seropositive cats of our study. This is a higher frequency of malignancy than has previously been reported (3% prevalence),<sup>3</sup> and undoubtedly reflects the referral pattern at our institution. As expected, these neoplasms were more frequent among cats coinfecting with FeLV than among cats with FIV infection alone (44% vs 11%). In a larger retrospective study, we found odds ratios of lymphoid cancer among FIV-positive, FeLV-positive, and coinfecting cats to be 5.6, 62.1, and 77.3, respectively, compared with noninfected control cats.<sup>5</sup> Thus, FIV infection alone appears to be an independent risk factor for development of lymphoproliferative malignancies. NonHodgkins lymphoma develops with increased frequency in HIV-infected individuals; AIDS patients coinfecting with another retrovirus, human T-cell leukemia virus-I, appear to be particularly at risk.<sup>17-19</sup> Therefore, lymphoid malignancy in FIV/FeLV coinfecting cats may represent another example of synergistic effects of retroviruses.

### Bone Marrow Abnormalities

Bone marrow abnormalities were seen in 3 of 7 (43%) cats not manifesting clinical signs of infection and in 13 of 18 (72%) cats with signs of infection ("ARC" or "AIDS"), for which aspirates or biopsy specimens were available. Abnormalities in the bone marrow from cats without clinical signs of disease were limited to increased numbers of lymphocytes, plasma cells, or eosinophils, whereas the myeloid-to-erythroid cell ratio and cell morphologic features were normal. Dysmorphic features (eg, megaloblastic erythropoiesis, karyorrhexis), hyperplasia of individual cell lineages, and neoplasia (ie, leukemia) were the most common abnormalities in bone marrow from cats with signs of disease, and were observed in FeLV-negative and -positive cats. In separate studies, myeloproliferative disorders were diagnosed in 2 cats with spontaneous FIV infection (data not shown). Myeloproliferative disease in FIV-seropositive cats has been reported, including a specific-pathogen-free kitten that developed the disorder acutely after experimental induction of FIV infection.<sup>2-4</sup> These bone marrow abnormalities are similar to those reported to be associated with HIV infection.<sup>9,10</sup> Hypercellularity, dysplasia, plasmacytosis, and lymphoid infiltrates are frequent findings in bone marrow specimens obtained from AIDS patients.

### Pathogenesis

The pathogenesis of blood and bone marrow abnormalities in FIV-seropositive cats is currently being investigated. Results of extensive bone marrow culture studies performed on 9 hematologically normal FIV-seropositive cats without clinical signs of disease indicate normal frequency of bone marrow progenitors (colony-forming units-erythroid, burst forming units-erythroid, and colony-

Table 2—Griseofulvin-induced neutropenia in FIV-seropositive cats

Cat No.	Absolute neutrophil count ( $\times 10^3/\mu\text{l}$ )		
	Sample		
	Pre-drug	End of Tx*	Post-drug†
1	5.98	1.89	7.76
2	15.74	2.31	12.18
3	2.63	2.74	7.54
4	8.12	0.39	8.10
5	5.78	0	8.98
6	6.53	0	Died
7	4.35	0.25	7.69
Mean (SD)	7.02 (4.2)	1.08 (1.2)	8.71 (1.8)
P value‡	...	<0.01	>0.1

\*Treatment (Tx) stopped on day 15, except for cat 5 (day 11). †Post-drug sample taken on days 16 (cat 5), 23 (cat 2), or 30 (cats 1, 3, 4, and 7). ‡Paired t-test.  
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forming units-granulocyte macrophage [CFU-GM]), with normal responsiveness to hematopoietic growth factors and normal cell cycle kinetics.<sup>6</sup> Sera from FIV-positive cats supported hematopoietic progenitor growth equivalent to that associated with normal cat sera, and antibodies against progenitors from normal or FIV-infected bone marrow could not be documented. Results indicate that chronic FIV infection alone is not sufficient to alter hematopoiesis and that other factors associated with progressive lentivirus infection, immune system dysfunction, and/or clinical disease are required.

A study of one FIV-seropositive cat with chronic neutropenia documented normal frequencies of bone marrow progenitors, but inhibition of CFU-GM-derived colonies was appreciable when bone marrow cells were cultured in the presence of autologous serum.<sup>20</sup> Results indicated that an inhibitory substance (possibly antibody) in the serum impaired granulocyte/macrophage differentiation. Similar findings have been reported in symptomatic, HIV-seropositive patients with neutropenia.<sup>21</sup>

### Drug-Induced Neutropenia

Recent studies indicate that FIV-infected cats are at increased risk to develop hematologic toxicosis, most notably severe neutropenia, when treated with the common anti-fungal agent, griseofulvin.<sup>16</sup> This phenomenon was discovered fortuitously during prospective evaluations of FIV-seropositive cats administered griseofulvin during an episode of dermatophytosis at our research facility. Of 7 cats, 6 (86%) developed absolute neutropenia by day 15 of griseofulvin treatment (Table 2). The neutropenia was severe ( $<400$  neutrophils/ $\mu\text{l}$ ) in 4 of the 6 affected cats. Fever, depression, and anorexia developed in 2 of the severely neutropenic cats, one of which died from sepsis. Slight decrease in lymphocyte numbers paralleled the decrease in neutrophils, and absolute lymphopenia developed in 2 of 7 (29%) treated cats.

<sup>6</sup>Linenberger ML, Persik M, Shelton GH, et al. Hematopoiesis in asymptomatic cats infected with feline immunodeficiency virus (FIV) (abstr). *Blood* 1990;76.

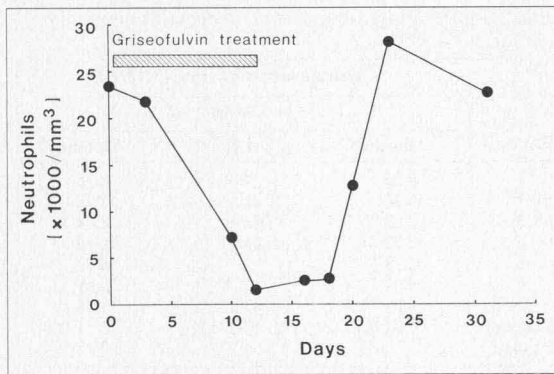


Figure 2—Neutropenia associated with rechallenge of FIV-seropositive cat 2 with griseofulvin. Reprinted with permission from *J Vet Intern Med* 1990;4:319.

Neutrophil count returned to baseline values within 15 days after drug withdrawal in all surviving cats. Clinical signs of disease or hematologic abnormalities were not observed in 4 clinically normal (FIV-seronegative) cats treated with the same lot of griseofulvin at equivalent doses, thus excluding the possibility of drug impurity. After rechallenge with griseofulvin, neutropenia recurred in 2 of 2 FIV-seropositive cats. The decline in neutrophil numbers during griseofulvin treatment 1 of one of these cats is depicted (Fig 2).

Subsequent investigations using additional cats yielded similar results (unpublished data). Griseofulvin-induced neutropenia has been observed in FIV-infected cats (with or without clinical signs of disease) after dosage ranging from 62 to 150 mg/kg of body weight administered every 24 hours over a period of 11 to 42 days. Correlation between griseofulvin dose and the severity of the neutropenia is not apparent. This dosage is higher than that recommended by the manufacturer,<sup>f</sup> but is within the range often reported in veterinary literature.<sup>22,23</sup> Griseofulvin-associated hematologic toxicosis in cats has been previously cited<sup>22-24</sup>; however, whether any of these reports involved FIV-seropositive cats is not known. Because neutropenia is a potentially fatal side effect, particularly in these immunosuppressed cats, frequent hematologic monitoring of FIV-infected cats treated with griseofulvin is indicated.

The pathogenesis of griseofulvin-associated neutropenia in FIV-seropositive cats is still unclear. The phenomenon appears analogous to the antibiotic-induced neutropenias observed in HIV-seropositive patients.<sup>12-14</sup> Although the mechanism of these toxicoses is also not yet known, immune-mediated interactions have been postulated.<sup>14</sup> Results of our preliminary studies indicate that griseofulvin enhances binding of immune complexes (or antibody) to granulocytic cells in infected cats (unpublished observations). The possibility that other antimicrobial drugs may induce neutropenia when administered to FIV-seropositive cats is being explored.

<sup>f</sup>Fulvicin U/F, Schering Corp, Kenilworth, NJ.

## Conclusion

Hematologic abnormalities are frequently associated with FIV infection, particularly in clinically ill cats. Lymphopenia is the most prevalent hematologic disorder and is probably attributable to lysis of FIV-infected T lymphocytes. Anemia, neutropenia, and marrow abnormalities are also common. The pathogenesis of these abnormalities is not known, but does not appear to involve a direct effect of FIV on hematopoietic progenitors. Likely, there are multiple mechanisms of marrow suppression that might arise during progressive FIV infection. Lymphoid malignancies develop with increased frequency in FIV-infected cats. In addition, FIV-seropositive cats are at increased risk for griseofulvin-associated neutropenia, possibly as a result of immune-mediated mechanisms. The hematologic manifestations of FIV infection bear striking resemblance to those seen in HIV-seropositive people. For this reason, cats infected with FIV represent an excellent animal model for studying the pathogenesis of blood and bone marrow abnormalities associated with HIV infection, as well as to evaluate the hematologic toxicoses associated with drug protocols. It is hoped that such studies will eventually prove beneficial to people and animals with lentivirus infections.

## References

1. Pedersen NC, Ho E, Brown ML, et al. Isolation of a T-lymphotropic virus from domestic cats with an immunodeficiency-like syndrome. *Science* 1987;235:790-793.
2. Yamamoto JK, Sparger E, Ho EW, et al. Pathogenesis of experimentally induced feline immunodeficiency virus infection in cats. *Am J Vet Res* 1988;49:1246-1258.
3. Yamamoto JK, Hansen H, Ho EW, et al. Epidemiologic and clinical aspects of feline immunodeficiency virus infection in cats from the continental United States and Canada and possible mode of transmission. *J Am Vet Med Assoc* 1989;194:213-220.
4. Ishida T, Washizu T, Toriyabe K, et al. Feline immunodeficiency virus infection in cats of Japan. *J Am Vet Med Assoc* 1989;194:221-225.
5. Shelton GH, Grant CK, Cotter SM, et al. Feline immunodeficiency virus (FIV) and feline leukemia virus (FeLV) infections and their relationships to lymphoid malignancies in cats: a retrospective study (1968-1988). *J Acquir Immune Defic Syndr* 1990;3:623-630.
6. Sparger EE, Luciw PA, Elder JH, et al. Feline immunodeficiency virus is a lentivirus associated with an AIDS-like disease in cats. *AIDS* 1989;3:43-49.
7. Spivak JL, Bender BS, Quinn TC. Hematologic abnormalities in the acquired immune deficiency syndrome. *Am J Med* 1984;77:224-228.
8. Zon LI, Arkin C, Groopman JE. Haematologic manifestations of human immunodeficiency virus (HIV) infection. *Br J Haematol* 1987;66:251-256.
9. Castella A, Croxson TS, Mildvan D, et al. The bone marrow in AIDS: a histologic, hematologic, and microbiologic study. *Am J Clin Pathol* 1985;84:425-432.
10. Frontiera M, Myers AM. Peripheral blood and bone marrow abnormalities in the acquired immunodeficiency syndrome. *West J Med* 1987;147:157-160.
11. Scadden DT, Zon LI, Groopman JE. Pathophysiology and management of HIV-associated hematologic disorders. *Blood* 1989;74:1455-1463.
12. Gordin FM, Simon GL, Wofsy CB, et al. Adverse reactions to trimethoprim-sulfamethoxazole in patients with the

acquired immunodeficiency syndrome. *Ann Intern Med* 1984;100:495-499.

13. Polsky B, Dryjanski J, Whimbey E, et al. Severe neutropenia during pentamidine treatment of *Pneumocystis carinii* pneumonia in patients with AIDS. *JAMA* 1984;251:1253-1254.

14. McPherson ML, Plaisance KI. Neutropenia associated with oral acyclovir and multiple antibacterial agents in a patient with acquired immunodeficiency syndrome. *Clin Pharm* 1988;7:398-401.

15. Shelton GH, Linenberger ML, Grant CK, et al. Hematologic manifestations of feline immunodeficiency virus infection. *Blood* 1990;76:1104-1109.

16. Shelton GH, Grant CK, Linenberger ML, et al. Severe neutropenia associated with griseofulvin therapy in cats with feline immunodeficiency virus infection. *J Vet Intern Med* 1990;4:317-319.

17. Feigal EG, Lekas P, Beckstead JH, et al. Evidence for coinfection with HTLV-I and HIV in AIDS risk group patients with high-grade non-Hodgkin's lymphoma. In: Bolognesi D, ed. *Human retroviruses, cancer, and AIDS: approaches to prevention and therapy*. New York: Alan R Liss Inc, 1988;213-228.

18. Levine AM. Non-Hodgkin's lymphomas and other malignancies in the acquired immune deficiency syndrome. *Semin Oncol* 1987;14:34-39.

19. Kaplan LD, Abrams DI, Feigal E, et al. AIDS-associated non-Hodgkin's lymphoma in San Francisco. *JAMA* 1989;261:719-724.

20. Shelton GH, Abkowitz JL, Linenberger ML, et al. Chronic leukopenia associated with feline immunodeficiency virus infection in a cat. *J Am Vet Med Assoc* 1989;194:253-255.

21. Donahue RE, Johnson MM, Zon LI, et al. Suppression of in vitro haematopoiesis following human immunodeficiency virus infection. *Nature* 1987;326:200-203.

22. Scott DW. Fungal disorders: feline dermatology 1900-1978: a monograph. *J Am Anim Hosp Assoc* 1980;16:349-356.

23. Muller GH, Kirk RW, Scott DW. *Small animal dermatology*, 4th ed. Philadelphia: WB Saunders Co, 1989;299-315.

24. Helton KA, Nesbitt GH, Caciolo PL. Griseofulvin toxicity in cats: literature review and report of seven cases. *J Am Anim Hosp Assoc* 1985;22:453-458.

## Neoplasia associated with feline immunodeficiency virus infection in cats of Southern California

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**Summary:** Between 1988 and 1991, feline immunodeficiency virus (FIV) infection status was evaluated in 1,160 cats examined at an oncology referral and general practice in Los Angeles, California. Twenty-nine (2.5%) cats were FIV positive. Neoplasia was present in 18 of the 29 (62%) cats. Sampling for neoplasia was intentionally biased in the oncology referral group. However, 33% (6/18) of FIV-infected cats with neoplasia originated from the general practice. Three neoplastic processes were observed; myeloproliferative disease (MPD; 5/18), lymphoma (LSA; 5/18), and squamous cell carcinoma (SCC; 7/18). One cat had LSA and SCC.

Extranodal sites of LSA were common (66%) in FIV-infected cats. Sites of LSA were submandibular and mesenteric lymph nodes, liver, kidneys, periorbital area, and diffuse (heart, pancreas, bladder). Sites of

SCC were sublingual ( $n = 2$ ), nasal planum ( $n = 3$ ), nasal planum and eyelids ( $n = 1$ ), and mandible ( $n = 2$ ). Feline leukemia virus co-infection was observed in 17% (5/29) of FIV-infected cats. The FIV-infected cats with MPD were young (range, 8 months to 13 years; median, 4 years) and had short survival duration (2, 6, 21, 134, 249 days) even in response to aggressive treatment. The FIV-infected cats with LSA were older (median age, 8 years; range, 4 to 14 years) and survived 60 days if untreated. Cats administered chemotherapy survived 39, 45, 217, and 243 days; the latter 2 cats had partial remission of 2 months' duration. Older FIV-infected cats had SCC (median age, 12 years; remission range, 7 to 16 years) because of more frequent association of both diseases in older cats with outdoor environment.

Lymphocytic-plasmacytic lymphadenopathy was seen in 10 necropsied FIV-infected cats (4 without neoplasia, 3 with LSA, 1 with SCC, and 2 with MPD). Lymphadenopathy associated with FIV may develop in one lymph node, and lymphoma may develop in another lymph node. Clinically, FIV-induced lymphadenopathy may be confused with progressive lymphoma.

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Supported in part by Public Health Service Grant AI25802-04.

**F**eline immunodeficiency virus (FIV) is a lentivirus that is distantly related to the human (HIV)