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Feline Viruses in Wildcats from Scotland

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ABSTRACT: Few data are available on the prevalence of feline viruses in European wildcats (*Felis silvestris*). Previous surveys have indicated that wildcats may be infected with the common viruses of domestic cats, apart from feline immunodeficiency virus (FIV). In the present study, 50 wildcats trapped throughout Scotland (UK) between August 1992 and January 1997 were tested for evidence of viral infection. All were negative for FIV by several serological or virological methods. By contrast, 10% of the cats were positive for feline leukemia virus (FeLV) antigen and infectious virus was isolated from 13% of a smaller subset. Of the wildcats tested for respiratory viruses, 25% yielded feline calicivirus (FCV) and although no feline herpesvirus was isolated, 16% of the samples had neutralizing antibodies to this virus. Antibodies to feline coronavirus (FCoV) were found in 6% of samples. Feline foamy virus (FFV) was an incidental finding in 33% of samples tested. This study confirms that wildcats in Scotland are commonly infected with the major viruses of the domestic cat, except for FIV.

Key words: Feline calicivirus, feline corona virus, feline foamy virus, feline herpesvirus, feline immunodeficiency virus, feline leukemia virus, *Felis silvestris*, survey, wildcats.

Domestic cats (*Felis silvestris catus*) and European wildcats (*Felis silvestris silvestris*) are considered subspecies and there are currently no exclusive morphological or genetic criteria for distinguishing between the two (reviewed in Daniels et al., 1998). However, there is evidence for clinal variation in morphology, genetics and ecology, such that cats living wild in northern Scotland (UK) may be considered wildcats (Daniels, 1997). A small number of wildcats was sampled in Scotland for common feline viruses (McOrist et al., 1991). Although there was evidence of infection with some viruses, none tested positive for feline immunodeficiency virus (FIV), in contrast to prevalence of up to 57% reported in feral domestic cats in England (Yamaguchi et al., 1996).

More recently an opportunity arose to extend these findings. A sample of 50 wildcats (27 females and 23 males) was trapped throughout northern Scotland between August 1992 and January 1997 and tested for FIV and five other feline viruses including feline leukemia virus (FeLV), feline coronavirus (FCoV), feline calicivirus (FCV), feline herpesvirus (FHV) and feline foamy virus (FFV). After sampling wildcats were released. Details of trapping techniques, anesthesia, sampling techniques, and the locations of wildcats trapped are described in Daniels (1997) and Daniels et al. (1998).

Blood was obtained from anesthetized cats into lithium heparin and oropharyngeal swabs were collected into transport medium (Leibovitz-15 medium (Life Technologies Ltd, Paisley, UK) with 10% fetal bovine serum and antibiotics). The samples were posted and arrived in the laboratory within 48 hr of collection. Plasma was separated from the blood and tested for FeLV p27 antigen (Indochem C Lutz, Ruedlingen, Switzerland) by enzyme immunoassay and virus isolation at (Feline Virus Unit, University of Glascow, Glascow, UK; Jarrett and Ganière, 1996). Antibodies to FCoV were assayed by indirect immunofluorescence (IF; Addie and Jarrett, 1992) at Feline Virus Unit (M. C. Golder, unpubl. data). A similar IF test was used to detect anti-FIV antibodies, using CrFK cells (from R. A. Crandell) infected with FIVGL-8 as target cells. Antibodies to FIV were also detected by western blotting (M. C. Golder, unpubl. data); (supplied by Feline Virus Unit) (Hosie and Jarrett, 1990). Isolation of FIV was attempted by the growth of virus from concanavalin A activated peripheral blood mononuclear cells (PBMC) (Hosie and Jarrett, 1990). Growth of FIV was de-

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TABLE 1. Prevalence of viruses tested in wildcats from Scotland

Disease	Assay	Number tested/positive	Prevalence (%)
Feline immunodeficiency virus	antibody IF	0/50	0
	Western blot	0/48	0
	virus isolation	0/27	0
Feline leukemia virus	antigen ELISA	5/50	10
	virus isolation	4/30	13
Feline calicivirus	virus isolation	11/43	26
Feline herpesvirus	virus isolation	0/37	0
	antibody VN	8/49	16
Feline coronavirus	antibody IF	3/49	6
Feline foamy virus	virus isolation	6/18	33

termined by an ELISA for FIV p24 (IDEXX Laboratories Buckinghamshire, UK). The presence of FFV in these cultures was detected by syncytium formation in feline embryo cells (FEA) (Jarrett et al., 1973) seeded with cultured PBMC. Fluid from sample bottles containing swabs was inoculated into confluent monolayer cultures of FEA cells for the attempted isolation of FCV and FHV (Ormerod and Jarrett, 1978).

The results are shown in Table 1. Results of IF (50 wildcats), western blotting (48) and virus isolation (27) for FIV were all negative. However, FeLV antigen was detected in the plasma of 5/50 (10%) of cats tested and virus was isolated from 4/30 (13%). FCV was isolated from the oropharyngeal swabs of 11/43 (26%) wildcats. Although no FHV was isolated from these samples anti-FHV antibodies (titer range 32-4,096) were found in the plasma of 8/49 (16%) cats tested. Antibodies were detected by a complement-enhanced neutralization test (Horimoto et al., 1989). Equal volumes of plasma dilutions, virus and fresh domestic cat serum (20%) were incubated for 5 hr and the residual infectivity in each reaction was determined by a plaque assay (M. C. Golder, unpubl. data). The antibody titer was taken as the reciprocal of the dilution of plasma that reduced the plaque count by 75%. There was evidence of exposure to FCoV since 3/49 (6%) of the wildcats had antibodies (titer range 20–1,280) Addie and Jarrett (1990). As an incidental finding FFV was isolated from 6/18 of the PBMC cultures set up to isolate FIV.

Only two previous studies have reported on the prevalence of viruses in wildcats. McOrist et al. (1991) tested 15 free roaming wildcats (and eight captive wildcats) in Scotland for FeLV antigen (2/23 positive) and FIV (0/23) and FCoV (0/23) antibodies. Artois and Redmond (1994) in France tested eight live caught wildcats for FeLV antigen (3/8 positive) and antibodies to FCV (3/5), FHV (2/8), feline parvovirus, FPV (2/3), rabies (0/3), and FIV (0/8).

Feline immunodeficiency virus and FeLV are potentially the most dangerous of the viruses tested here because of their association with fatal disease conditions in domestic wildcats (Hosie et al., 1989). However, despite the high prevalence of FIV-like lentiviruses in free ranging wild felids in Africa and North America, no immunological or pathological consequences of infection have been recorded to date (Callanan, 1995; Evermann et al., 1997). The most common route for FIV transmission is through biting and a relative lack of social contact between wildcats may explain the absence of FIV infection. However, the lack of antibodies to FIV also may indicate that the population has not yet been exposed. Other populations of wild felids presently show no evidence of exposure to FIV-like lentiviruses; these include Asiatic lions (Brown et al., 1994)

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or Florida panthers (Roelke et al., 1993). Given that it has been suggested that the seriousness of FIV infection in domestic cats is a result of a relatively recent infection rather than the proposed historical genetic host-parasite symbiosis found in pumas (Carpenter et al., 1996; Vande Woude et al., 1997), then potentially lack of exposure could pose a threat.

The prevalence of FeLV is similar to that reported for domestic cats (Yamaguchi et al., 1996). One possibility for the survival of this virus in wildcats is that the virus may be transmitted congenitally within a few families since transmission of FeLV is mainly by the oronasal route or across the placenta (Jarrett, 1994). Alternatively, transmission could be a result of contact with domestic or feral domestic cats.

The isolation rate of FCV was relatively high and may indicate that this virus was acquired by direct contact with other carriers. It is also possible that the virus was transmitted from carrier dams to their young kittens. The failure to isolate FHV is not surprising since in domestic cats this virus is carried as a latent infection and is shed only intermittently from the oropharynx (Gaskell and Dawson, 1994). However, the population had been exposed to FHV since 16% of the wildcats tested had high levels of FHV neutralizing antibodies.

Comparable prevalence of FCV and FCoV has been reported in populations of wild felids with no evidence of associated mortality (e.g., Roelke et al., 1993; Paul-Murphy et al., 1994). The low prevalence of antibodies to FCoV (3/49) was consistent with the fact that this virus is much more common in domestic cats in multicat households than in free-ranging pet cats kept singly or in feral or stray cats. Direct contact is not necessary for the transmission of FCoV which may be spread indirectly through the contamination of the environment with infected feces (Stoddart and Bennett, 1994).

The incidental finding of FFV in cultures from PBMC is the first demonstration of this virus in free-living wildcats in Scotland. The rates for the isolation of FFV were similar to those for domestic cats in the UK (Jarrett et al., 1974). While FFV is common in domestic cats, it is as yet not associated with any disease condition (Gaskell and Bennett, 1994) and unlikely to be a significant pathogen in the wild population.

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