BioOne COMPLETE

Some Viral and Protozool Diseases in the European Wildcat (Felis silvestris)

Authors: McOrist, Steven, Boid, Richard, Jones, Tudor W., Easterbee, Nigel, Hubbard, Alexandra L., et al.

Source: Journal of Wildlife Diseases, 27(4) : 693-696

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-27.4.693

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

Journal of Wildlife Diseases, 27(4), 1991, pp. 693–696
© Wildlife Disease Association 1991

Some Viral and Protozool Diseases in the European Wildcat (*Felis silvestris*)

Steven McOrist,[1] Richard Boid,[2] Tudor W. Jones,[2] Nigel Easterbee,[3] Alexandra L. Hubbard,[3] and Oswald Jarrett,[4] [1] Department of Veterinary Pathology, University of Edinburgh, Easter Bush, Midlothian, EH25 9RG, Scotland; [2] Centre for Tropical Veterinary Medicine, University of Edinburgh, Easter Bush, Midlothian, EH25 9RG, Scotland; [3] Nature Conservancy Council, 12 Hope Terrace, Edinburgh, Scotland; [4] Department of Veterinary Pathology, University of Glasgow, Bearsden, Glasgow, Scotland

ABSTRACT: Ten European wildcats (*Felis silvestris*) were examined at necropsy and an additional 23 were examined clinically for evidence of viral diseases in Scotland. Two plasma samples taken from live free-living wildcats showed positive ELISA reactions to feline leukemia antigen. A feline leukemia virus of subgroup A was isolated from one of these samples, taken from a wildcat in north-western Scotland. No antibodies to feline coronavirus or feline immunodeficiency virus were detected in any sample. Three of the live wildcats and one of the dead had chronic mucopurulent rhinotracheitis suggestive of "cat flu." One other dead wildcat had diffuse enlargement of anterior lymph nodes. The findings indicated that feline leukemia virus infection can occur in free-living *Felis silvestris*. It is possible that the disease exists as a sustained infection in some wildcat populations, although the close interaction between wildcat and the domestic cat means that the latter could act as a continual source of infection.

Key words: Feline leukemia virus, European wildcat, feline rhinotracheitis, *Felis silvestris*, survey.

Significant disease syndromes in the domestic cat are associated with viral infections. Feline leukemia virus (FeLV) is a retrovirus that is associated with several neoplastic and non-neoplastic diseases (Cotter et al., 1975). Feline infectious peritonitis (FIP) virus is a coronavirus (FCoV) and is associated with serositis and vascular lesions (Disque et al., 1968; Zook et al., 1968). Feline immunodeficiency virus (FIV) is a lentivirus which induces immunosuppression (Pedersen et al., 1987; Hopper et al., 1989). Feline rhinotracheitis (FRV) virus is a herpes virus associated with an upper respiratory tract infection (Crandell et al., 1961), and with calici viruses have been associated with the clinical syndrome "cat flu" (Gaskell and Wardell, 1977).

The European wildcat (*Felis silvestris*) occurs throughout Europe, but populations were reduced several hundred years ago from habitat loss, especially in England and southern Scotland. However, the most significant factor reducing the population was shooting and trapping, particularly during the 19th century when *F. silvestris* was lost from England and Wales (Langley and Yalden, 1977). The population minimum was reached in Scotland in the early part of the 20th century. Subsequently, recovery of the population has been due to a reduction in shooting and trapping and increased availability of favorable habitat such as newly planted forests in Scotland (Suminski, 1962; French et al., 1988).

Hybridization of the wildcat with the domestic cat probably has taken place for several centuries, since the spread of the latter across Europe in the Roman era. The extent of this hybridization is thought to have increased during the twentieth century due to the population reduction of wildcats and increased numbers of domestic cats outside urban areas. Even so, the wildcat population in Scotland is considered one of the "purest" in Europe, probably due to its remoteness (Suminski, 1962; French et al., 1988). As part of a study of genetic relationships among these cats, a survey of some viral and protozoal diseases was initiated. Between 1987 and 1989, European wildcats in three study areas of Scotland were examined (Fig. 1). These included the lowland areas of Angus and Perthshire (A), the Central Highlands (B) and the north and west (C).

Of 23 live wildcats collected from the

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use



FIGURE 1. Map of northern half of Great Britain, illustrating sites of capture of live wildcats. A, Two sites in Perthshire yielded a total of two and three sites in Fife yielded a total of three animals; B, three sites in Central Highlands that yielded a total of 10 wildcats; B3, one site on the Black Isle yielded a total of one animal; C1, four sites in Western Highlands yielded a total of six wildcats; and C2, one site in the Northern Highlands yielded a total of one animal.

three areas eight were held in open enclosures at the Scottish Highland Wildlife Park at Kingussie (B in Fig. 1). Five of these had been trapped elsewhere, but three had been bred on site from locally captured stock. A further wildcat which died in 1987 at this site and nine dead wildcats collected as road kills (three from each study area) were examined at necropsy. Fifteen other wildcats were trapped in highland and forest areas and in the lowlands amongst woodland, often adjacent to arable land. All live cats were tranquillized by a single intramuscular injection of ketamine hydrochloride (Vetalar, Parke-Davis, Pontypool, Gwent. NP4 0YH, United Kingdom) (10 mg/kg) and xylazine (Rompun, Bayer, Bury St. Edmonds, Suffolk 1P32 7AH, United Kingdom) (1.0 mg/kg). Flaccid muscle tone and moderate sedation occurred within 10 to 15 min which allowed a full clinical examination and blood collection. All wildcats recovered fully within 30 to 45 min and were released at their site of collection or into their enclosure.

Plasma samples taken after centrifugation of each blood sample were assayed for evidence of infection by feline viruses and *Toxoplasma gondii*. An indirect immunofluorescence assay which incorporated the Wellcome strain of FCoV antigen was used to detect coronavirus antibody. A commercial indirect ELISA (Petchek feline T-lymphotropic lentivirus antibody test kit, Idexx Laboratories Ltd., High Wycombe, Bucks, HP13 5DT, England) was used to detect FIV antibody. The p27 antigen of FeLV was detected by an ELISA (Idexx Laboratories Ltd.). Any plasma sample which yielded a positive FeLV antigen result was subsequently inoculated onto feline QMI cells, produced in the author's laboratory, in an attempt to isolate infectious virus. Infected cells were identified by the observation of cytopathic effects between four and 14 days. The subgroup of any viral isolate was determined by an interference assay (Russell and Jarrett, 1976). Antibodies to *T. gondii* were detected by an indirect immunofluorescence assay produced in the author's laboratory and incorporating formalinized *T. gondii* tachyzoites. All assays incorporated positive and negative controls obtained from domestic cats.

Of the nine dead cats collected as road kills, only one 3-yr-old female had diffuse enlargement two to three times normal size of the tonsils, submandibular, prescapular and parietal lymph nodes. Histological examination revealed diffuse hyperplasia of the germinal centers of the cortices of follicles in affected lymphoid tissue. All nine cats had lesions consistent with death by traumatic injury.

The cat that died at the Kingussie collection had severe chronic rhinotracheitis. Three of the live cats examined at that collection had mucopurulent ocular and nasal discharges. Other clinical findings in-

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

cluded lacerations probably due to fighting ($n = 2$) and colonies of fleas, identified as *Ctenocephalides* sp. ($n = 3$). Wildcats weighed 2.5 to 5.5 kg and were assessed by body measurements to be from 6-mo-to 3-yr-old.

All of the plasma samples were negative for antibodies to FCoV and FIV. All were positive for antibodies to *T. gondii* (titers >1:64).

FeLV antigen was detected in two samples from C1 (Fort William) and C2 (Dornoch) (Fig. 1). The sample from C1 was from a healthy, 12-mo-old male weighing 4 kg. A FeLV of subgroup A was isolated from its blood.

Concurrent genetic and molecular studies to be reported elsewhere indicated that wildcats in area C had the least relationship to domestic cats and probably formed a separate genetic group. Wildcats from the Lowland (A) and Central Highland (B) had strong and moderate hybridization, respectively.

This survey indicates that the European wildcat is susceptible to infection with feline leukemia virus and feline herpes virus or calici viruses. Lesions of rhinotracheitis occurred in wildcats which had been kept in captive groups of two or three for periods of 1 to 12 mo. The cold weather and close contact between wildcats at this site area likely to have exacerbated the disease (Johnson and Thomas, 1966). "Cat flu" is relatively common in large captive felids as well as in the domestic cat (Fowler, 1986), indicating that the inciting viruses may have broad host ranges. The death of a wildcat possibly from the disease indicates that it is a potential threat to free-living wildcats, although factors associated with captivity may have an important role in determining the severity of clinical signs in infected animals.

FeLV infection was indicated in *F. silvestris* by isolation of virus, detection of antigen by ELISA, and presence of lesions in the lymphoid system resembling those previously reported for FeLV (Hardy, 1981; Jarrett et al., 1982). Previous reports of feline leukemia in exotic felids have been either of positive ELISA results in a western cougar (*Felis concolor*) and a clouded leopard (*Neofelis nebulosa*) kept in captivity (Meric, 1984; Citino, 1986), and isolation of the virus from a leopard cell line (Rasheed and Gardner, 1981). Further investigation in the latter study did not reveal evidence of FeLV in wild felids, although European wildcats were not examined. It is likely that these occasional infections resulted from interactions with domestic cats (Citino, 1986). Also, some caution is required in interpreting positive ELISA results, due to the possibility of false positive reactions (Jarrett et al., 1982).

Therefore, the infection reported here represents the first conclusive report of FeLV in a free-living non-domestic cat, apparently from transmission occurring in the wild. The prevalence of FeLV infection in *Felis silvestris* (one of 23, 4%) is similar to that previously reported (5%) in surveys of domestic cats in Great Britain, although these were relatively small surveys (Hosie et al., 1989).

The infected wildcats were in the northern and western areas where domestic cats are common and may have come into contact with the sampled animals. Comparisons of isoenzymes, DNA hybridization and albumin heterogeneity in our wildcat samples (S. McOrist, unpubl. data) indicates that they have the least evidence of hybridization with domestic cats. Therefore, the possibility exists that FeLV occurs as a sustained infection in some populations of wildcat rather than from an occasional infection acquired from domestic cats. FeLV is transmitted readily amongst young cats via infected body fluids, such as during fighting or mating. Such interactions are probably common among wildcats and between wildcats and domestic cats in Scotland. The FeLV subgroup A is the strain most commonly isolated from domestic cats (Jarrett et al., 1982). FeLV is more commonly isolated in cats 1- to 5-yr-old and the latent period between FeLV infection and occurrence of severe clinical signs is likely to be two to four years.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

Therefore, the appearance of the latent infections in the wildcat population could be monitored prior to the appearance of clinical cases.

The absence of antibodies of FCoV or FIV titers suggests that these viruses may not actively circulate in wildcats. However, FIV antibody is more likely to be found in older, male cats (Hosie et al., 1989; Hopper et al., 1989); these comprised a small part of our survey.

The presence of antibodies to *T. gondii* in all our samples suggests that this organism actively circulates in the wild. Therefore, wildcat feces may act as a potential source of infection for domestic species and man.

Lymphadenopathy is a common finding in cats with FIV or FeLV (Hosie et al., 1989). However, the significance of lymphadenopathy in a young, female wildcat was not determined. The role of FeLV in lymphoid disease of the wildcat requires further examination of clinically affected cats.

We thank Jeremy Usher-Smith, Lucy Norval, Tony Sykes, I. Harbit, Tony Wall, A. W. Fergusson, A. Nicholson, C. MacNeil, G. Gartshaw, J. Gibb, J. Gilles, F. Law, Dr. Miranda Stevenson and George Rafferty for help in collection of samples, M. C. Golder for technical assistance and D. Buxton for helpful assistance.

LITERATURE CITED

CITINO, S. B. 1986. Transient FeLV viraemia in a clouded leopard. Journal of Zoo Animal Medicine 17: 5–7.

COTTER, S. M., W. D. HARDY, JR., AND M. ESSEX. 1975. Association of feline leukemia virus with lymphosarcoma and other disorders in the cat. Journal of the America Veterinary Medical Association 166: 449–454.

CRANDELL, R. A., J. A. REHKEMPER, W. H. NIEMANN, J. R. GANAWAY, AND F. D. MAURER. 1961. Experimental feline viral rhinotracheitis. Journal of the American Veterinary Medical Association 138: 191–196.

DISQUE, D. F., M. T. CASE, AND J. A. YOUNGREN. 1968. Feline infectious peritonitis. Journal of the American Veterinary Medical Association 152: 372–375.

FOWLER, M. E. 1986. Felidae. *In* Zoo and wild animal medicine, M. E. Fowler (ed.). W. B. Saunders, Philadelphia, Pennsylvania, 1127 pp.

FRENCH, D. D., L. K. CORBETT, AND N. EASTERBEE. 1988. Morphological discriminants of Scottish wildcats (*Felis sylvestris*), domestic cats (*Felis catus*) and their hybrids. Journal of Zoology 214: 235–259.

GASKELL, R. M., AND R. C. WARDELL. 1977. Feline viral respiratory disease: A review with particular reference to its epizootiology and control. Journal of Small Animal Practice 19: 1–16.

HARDY, W. D., JR. 1981. Feline leukemia virus nonneoplastic disease. Journal of the American Hospital Association 17: 941–949.

HOPPER, C. D., A. H. SPARKES, T. J. GRUFFYDD-JONES, S. M. CRISPIN, P. MUIR, D. A. HARBOUR, AND C. R. STOKES. 1989. Clinical and laboratory findings in cats infected with feline immunodeficiency virus. Veterinary Record 125: 341–346.

HOSIE, M. J., C. ROBERTSON, AND O. JARRETT. 1989. Prevalence of feline leukemia virus and antibodies to feline immunodeficiency virus in cats in the United Kingdom. Veterinary Record 125: 293–397.

JARRETT, O., M. C. GOLDER, AND K. WEIJER. 1982. A comparison of three methods of feline leukemia virus diagnosis. Veterinary Record 110: 325–329.

JOHNSON, R. H., AND R. G. THOMAS. 1966. Feline viral rhinotracheitis in Britain. Veterinary Record 79: 188–190.

LANGLEY, P. J. W., AND D. W. YALDEN. 1977. The decline of the rarer carnivores in Great Britain during the nineteenth century. Mammal Review 7: 95–116.

MERIC, S. M. 1984. Suspected feline leukemia virus infection and pancytopenia in a western cougar. Journal of the American Veterinary Medical Association 185: 1390–1391.

PEDERSEN, N. C., E. W. HO, M. L. BROWN, AND J. K. YAMAMOTO. 1987. Isolation of feline T-lymphotrophic lentivirus. Science 235: 790–794.

RASHEED, S., AND M. B. GARDNER. 1981. Isolation of feline leukemia virus from a leopard cat cell line and search for retrovirus in wild felidae. Journal of National Cancer Institute 67: 929–933.

RUSSELL, P. H., AND O. JARRETT. 1976. An improved assay for feline leukaemia virus pseudotypes of murine sarcoma virus. Journal of General Virology 31: 139–143.

SUMINSKI, P. 1962. Les characteres de la forme pure du chat sauvage *Felis silvestris*, Schreber. Archives des Sciences 15: 277–296.

ZOOK, B. C., N. W. KING, R. L. ROBINSON, AND H. L. MCCOMBS. 1968. Ultrastructural evidence for the viral etiology of feline infectious peritonitis. Pathologia Veterinaria 5: 91–95.

Received for publication 30 November 1990.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use