

Parasite prevalence in free-ranging farm cats, *Felis silvestris catus*

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SUMMARY

No animals tested were positive for feline leukaemia virus antigen and *Chlamydia psittaci* antibodies, but all were positive for antibodies to feline calicivirus (FCV), feline herpesvirus 1 (FHV1) and rotavirus. They had antibodies to feline parvovirus (96%), feline coronavirus (84%) and cowpox virus (2%). Antibody to feline immunodeficiency virus (FIV) was found in 53% of animals, which were less likely to be infected with *Haemobartonella felis*, and had higher FHV antibody titres than cats without FIV. FCV was isolated from 51% cats and FHV1 and feline reovirus each from 4%. *H. felis* was present in 42% of animals, and antibody to *Toxoplasma gondii* in 62%. Clinical abnormality had a significant association with FIV and feline calicivirus infections, but sex, age, social status and feeding group had no significant association with prevalence of any parasites. *Toxocara cati* and *Toxascaris leonina* eggs were found, respectively, in 91% and 82% of animals tested.

INTRODUCTION

Although parasites of domestic cats have been studied extensively [1–6], little is known of their prevalence in free-ranging cat populations. Study and assessment of the health of free-ranging populations is important because they are likely to harbour and transmit diseases more readily than pet cats, as they are subject to the stresses of living wild, and have more opportunity to interact with other cats and feed on wild rodents. As they can live away from humans, and are common worldwide, such free-ranging cats could come into contact with, and transmit diseases to, small isolated populations of endangered wild felids, such as the Florida puma, *Felis concolor coryi* [7], European wildcat [8, 9] and Iriomote wildcat, *Felis*

iriomotensis [10], and could pose a threat to their survival.

Feline leukaemia virus (FeLV), and feline immunodeficiency virus (FIV) are major non-traumatic causes of death in adult cats, and are associated with immunosuppression causing secondary infection (feline-acquired immunodeficiency syndrome, FAIDS) [5, 11, 12]. Feline herpesvirus 1 (FHV) and feline calicivirus (FCV) cause feline viral upper respiratory tract disease ('cat flu'), a major veterinary problem [13, 14]. Cowpox virus (CPoV), an *Orthopoxvirus*, and feline coronavirus (FCoV) infect big cats in zoos [15, 16].

Parasites of domestic cats also affect humans, livestock and other carnivores, and have implications for medical practice and wildlife management [3, 6–10]. As companion animals, cats have a possibility to

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infect humans with *Toxocara cati* and *Toxascaris leonina*, and more seriously, *Toxoplasma gondii* which causes toxoplasmosis and also affects livestock [1, 4, 17–19]. Rotaviruses, including feline rotavirus (FRoV), have been isolated from numerous mammals and cause acute diarrhoea in humans, cattle, sheep and pigs [20]. Feline panleucopenia virus (FPV) causes panleucopenia, which is highly infectious not only to Felidae but also to carnivores such as Mustelidae, Procyonidae and Viverridae [21].

The behaviour of free-ranging cats allows us to test the influence of age, sex and social status on parasite burden and prevalence. In areas with centralized food and nesting resources, such as a farmyard, feral farm cats live in large, socially organized colonies, based around several matrilineal feeding groups [22–4]. Large ‘Central’ feeding groups monopolize resources, while small ‘Peripheral’ groups or individuals live on the colony margins, where resources may be poor [22, 23]. Central females are reproductively more successful than Peripheral females, but this is not necessarily true for males [22, 23].

We document the effects of feeding group, social status (Central or Peripheral), sex, age and general health on the prevalence and burden of pathogenic micro- and macro-parasites in a large population of feral farm cats.

MATERIALS AND METHODS

Cat population

The cat population consisted of 50–80 feral cats at Barley Park Farm, Ducklington, Oxfordshire, UK. Milk and food were provided daily at three sites around out-buildings (Fig. 1: group 1 consists of about 20 adults, 6–8 adults for group 2 and about 5–6 adults for group 3). Cats were not given veterinary care, and were not culled.

Cats were aged as follows: adult (older than 12 months, or observed copulating); juvenile (6–12 months old, not observed copulating); and kitten (less than 6 months old). All kittens captured were 15–20 weeks old, and were assumed not to have passive maternally-derived immunity [21].

Individuals’ social status was determined by direct observation at feeding sites. At 30 min intervals during feeding times all cats present at the feeding area (*c.* 100 m²) were individually identified by coat characteristics and size. Cats present more than average were classified as Central, and those present less than

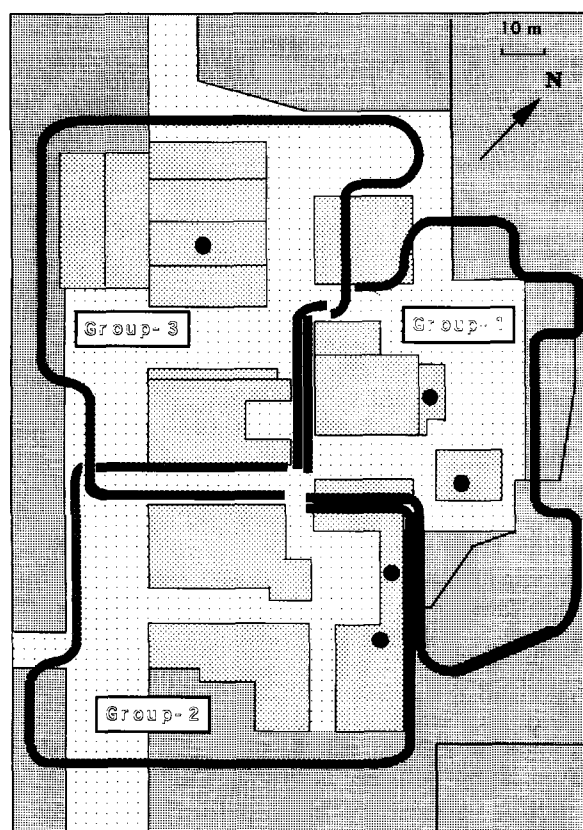


Fig. 1. Study site (Barley Park Farm). ▨, Farm yard; □, farm building; ▨, field; —, group area; ●, feeding site.

average as Peripheral [23]. Three Central feeding groups, based around three feeding sites, were identified.

Trapping and handling

Cats were trapped over 7 days in winter 1989, using box-traps baited with cat-food, placed in outbuildings and fields. Traps were set at dusk and checked at dawn. Fifty-two individuals consisting of 36 adults (15 males, 21 females), 10 juveniles (8 males, 2 females) and 6 kittens (5 males, 1 female) were caught. An adult male, an adult female and a juvenile male evaded capture.

Captured cats were immobilized by intramuscular injection with 22 mg/kg ketamine hydrochloride ('Vetalar': Parke, Davis & Co., Pontypool, Gwent, UK), weighed and measured. Under veterinary supervision, mucus samples were taken from the mouth, eyes and nose, and a blood sample from the jugular vein.

Animals were classed as clinically normal or abnormal on the basis of a thorough examination which assessed: ectoparasites; skin lesions; mucoid

discharges from the nose and eyes; ear wax; gingivitis, tartar on teeth and ulcerations on the upper palate and tongue; coat condition; and the ease with which the dorsal spinous process vertebrae could be palpated [25]. All animals were assessed by the same observer: 33 cats were classified as normal and 19 had at least one clinical abnormality [unpublished observations].

Blood and mucus samples

Blood samples were collected in an evacuated glass tube containing lithium heparin (Becton Dickinson Vacutainer Systems U.K., Oxford, UK). Immediately after sampling a smear was made, and fixed with methanol within 24 h. Within 15 min of sampling, blood was centrifuged at 2000 *g* for 10 min and the plasma transferred to small plastic tubes which were immediately frozen, and stored at -70°C within 6 h of collection.

Mucus swabs were placed into small glass bottles containing a 'virus transport medium' [900 ml of distilled water, 74.6 g of sucrose, 0.512 g of KH_2PO_4 , 1.237 g of K_2HPO_4 , 0.721 g of L-glutamic acid, and 0.015 g of phenol red, adjusted pH to 7.1–7.2 with solid KOH then autoclaved. When cooled, 100 mg of vancomycin, 100 mg of streptomycin and foetal calf serum to 10% v/v were added] [26]. These were kept cool in the field, and examined within 24 h. Faecal samples were collected where possible.

Parasitology

Commercial enzyme-linked immunosorbent assay (ELISA) kits were used to screen plasma samples for antibody to FIV ('PetChek'; Idexx GmbH), and FeLVp27 core protein ('Leukassay F II'; CVet Ltd, Bury St Edmunds, Suffolk, UK). Sera inactivated by heating for 30 min at 56°C were diluted 1:16 and screened for antibody to *T. gondii* with a commercial kit (ToxHAtest; Wellcome Diagnostics, Dartford, UK). Antibodies were scored as present or absent.

Immunofluorescence was used to detect antibodies to FRoV [27], feline coronavirus (FCoV) [28] and *C. psittaci* [29], and titres were scored as the highest dilution giving a positive result. Antibodies to FPV were detected by haemagglutination inhibition [27]. A serum neutralization test was used to detect antibodies to CPoV [30], FHV and FCV [31] using the F9 strain of FCV and the B927 strain of FHV.

Blood smears were stained with Giemsa and acridine orange, examined for the presence of *H. felis*

and scored on the basis of the percentage of cells affected (< 10%, 10–25%, 25–50%, or > 50%).

Mucus swabs were inoculated onto WFE cells for isolation and identification of FHV, FCV [32] and onto MA104 cells for the isolation of feline reovirus (FReV) [27], which were scored as present or absent.

Faecal samples were examined for *T. cati* and *T. leonina* eggs, counted by the McMaster technique.

Statistical analysis

The effects of sex, age, social status, feeding group and health category on parasitology were compared with χ^2 test, Fisher's exact test or Mann–Whitney *U*-test. Correlations were Spearman Rank correlations. All tests are two-tailed unless otherwise stated.

RESULTS

Viruses

None of the 45 animals tested was positive for FeLV antigen (Table 1).

Blood samples from all individuals tested were positive for antibodies to FCV, FHV and FRoV (Table 1), and 96% were positive for antibody to FPV, 84% for antibody to FCoV, but only 2% for antibody to CPoV (Table 1). Thirty-six animals were positive for antibodies to FCV, FHV and FCoV, 7 were for both FCV and FHV, and 2 (who were not tested for either FCV or FHV) were for only FCoV. There were positive correlations between FCV and FHV titres ($r = 0.37$, $n = 43$, $P < 0.05$), between FCV and FCoV titres ($r = 0.38$, $n = 43$, $P < 0.05$), and between FHV and FCoV titres ($r = 0.32$, $n = 43$, $P < 0.05$). Age, sex, social status, health and feeding group had no significant effects on plasma antibody titres for these viruses.

Fifty-three percent of cats had antibody to FIV ($n = 45$) (Table 2), and infected cats were significantly less likely to be infected with *H. felis* than cats without FIV antibody ($\chi^2 = 6.18$, $df = 1$, $P < 0.05$). FIV-positive cats had significantly higher FHV titres than did FIV-negative cats (Mann–Whitney *U* (24, 21) = 141.5, $P < 0.05$). Age, sex, social status, health and feeding group had no effect on FIV prevalence. Clinically abnormal females (but not males) were significantly more likely to be FIV-positive than clinically normal females (Fisher's exact test, $P < 0.05$, one-tailed).

FCV, FHV and FReV were isolated from swabs. FCV (isolated from 51% of 49 animals) was signifi-

Table 1. Distribution of plasma antibody titres against viruses, and antibody prevalence (%). The titre is the reciprocal of the highest dilution giving a positive result

Virus*	No.†	Titre index:‡								Median	% prevalence
		0	1	2	3	4	5	6	7		
FCV	43	0	7	6	3	21	6	0	0	4	100
FHV	43	0	6	22	11	4	0	0	0	2	100
FRoV	45	0	21	11	5	8	0	0	0	2	100
FPV	45	2	0	0	0	0	0	9	34	7	96
FCoV	45	7	6	15	0	6	10	1	0	2	84
CPoV	45	44	1	0	0	0	0	0	0	0	2
FIV	45	21	(24)	—	57

* FCV, feline calicivirus; FHV, feline herpesvirus 1; FRoV, feline rotavirus; FPV, feline parvovirus; FCoV, feline coronavirus; CPoV, cowpox virus; FIV, feline immunodeficiency virus.

† No. of cats tested.

‡ Titre index: 0, negative (≤ 8); 1, titre of ≤ 64 ; 2, 80–128; 3, 160–192; 4, 256–384; 5, 512–1024; 6, 2048–8197; 7, 16384.

Table 2. Presence of plasma antibodies against FIV in different age/sex classes

Age-sex classes	No.*	Negative	Positive
Adult male	15	6	9
Central	8	4	4
Peripheral	4	0	4
Adult female	20	9	11
Central	17	7	10
Peripheral	3	2	1
Juvenile male	7	5	2
Juvenile female	1	0	1
Kitten male	1	1	0
Kitten female	1	0	1

* No. of cats tested.

cantly more prevalent than FHV or FReV (each from 4% of animals), ($\chi^2 = 39.64$, $df = 2$, $P < 0.001$). Only one animal (2%) had both FCV and FReV; 21 (43%) animals were infected with none of the three viruses. FCV was isolated from clinically abnormal cats significantly more than from normal animals ($\chi^2 = 6.38$, $df = 1$, $P < 0.05$), but sex, age, social status and feeding group had no significant effects.

Other parasites

T. cati and *T. leonina* eggs were found, respectively, in 91% and 82% of a total of 11 faecal samples collected from 11 individuals. Faeces from 9 of the 11 individuals (82%) contained the eggs of both parasites.

H. felis was present in 42% of 48 animals tested, and antibody to *T. gondii* was found in 62% of 45

animals. There were no effects of social status, sex, age, health or feeding group on the prevalence of either parasite, nor on the percentage of blood cells affected by *H. felis*. Of 20 individuals infected with *H. felis*, less than 10% of blood cells were affected in 9 (19%) individuals, 10–25% of blood cells were affected in 9 (19%), and 25–50% of blood cells were affected in 2 (4%). No individuals had infections in more than 50% of blood cells.

All animals examined were negative for *C. psittaci* antibodies in serum.

DISCUSSION

Worldwide, feral cats have an FIV infection rate of 16%, compared with only 5% for cats kept indoors, and 12% for household cats with outdoor access [33]. In England and Wales, 27% of 90 feral cats tested positive for FIV antibody, but the proportion of seropositive cats varied widely (0–89%) between populations [34]. In comparison with overall rates for feral cats, our study population had a high prevalence of FIV, but the figure fell within ranges reported previously [34]. Levels of *T. gondii* in our study population were higher than, but comparable to, a British study in which 45% of 80 feral cats had antibody [35]. On Marion Island, South Antarctic, FPV was artificially introduced to control feral cats, and in 1982, 86% had antibodies to the virus [36]. Our study population had an even higher prevalence (96%) of antibodies to FPV, which appeared to have only a mild or sub-clinical effect. The lack of evidence for *C. psittaci* in our study population contrasts with previous work, which has found a relatively high

prevalence of antibodies in feral cats (69% of 36 samples) and farm cats (45% of 51 samples) in Britain [37].

There have been few other studies on parasites of feral farm cats, but surveys have been carried out using household or show cats. Among household cats, antibodies to FeLV were found in 16% of 1204 sick cats and 5% of 1007 healthy cats [5]. The lack of FeLV in our study population is surprising, given its importance in feline medicine [38]. In the USA and Europe up to 70% of cats show antibodies to reovirus [20, 39], while 28–99% of cats in the UK show evidence of previous rotavirus infection [40]. In comparison, our study population had a low level of reovirus infection, though our result is not a serological one, but a very high level (100%) of rotavirus infection.

Cats kept in colonies have more opportunity to come into contact with other individuals within the colonies than do household pets, and so may have, once the viruses enter the colonies, higher rates of viruses transmitted by direct contact. Prior to the introduction of vaccines, FCV was isolated from 8% of household pets, and 40% of colony cats [14]. Up to 95% of colony cats had antibody to FCoV [41], as did up to 20% of household cats [42]. Levels of FCV and FCoV in our study population were considerably higher than levels in household cats, and, for FCV, slightly higher than levels in colony cats.

T. cati is generally the most prevalent endoparasite of cats, found in about 10% of the adult population, while *T. leonina* is found in about 5% [43]. Considerably higher prevalences were found in our feral farm cat colony, with nearly all faecal samples providing evidence of infection.

Factors affecting parasite prevalence and burden

Parasites were highly prevalent among feral farm cats at Barley Park Farm, possibly due to severe soil and water contamination by infected excreta, and close contact between individuals. Eighteen communal latrines were found around feeding sites, and these were sometimes used by members of different groups. Thus, parasites that can be transmitted via excreta, such as FPV, FReV, FRoV, FCoV, *T. cati*, *T. leonina* and *T. gondii* [18, 20, 21] could readily be transmitted within and between groups. Rats, *Rattus norvegicus*, were present, and might maintain a permanent reservoir of *T. gondii* for cats which preyed upon them [19].

High rates of social interaction and close proximity between individuals, as is found on farms [23], would increase transmission and prevalence of parasites spread by direct contact or short distance aerosolization, such as FCV, FHV, FCoV, FeLV, FIV, FPV, *C. psittaci* and *T. gondii* [11, 13, 18, 21, 38, 39, 41]. Feral farm cats sometimes rear young cooperatively [22, 23, 44], and communally nesting kittens are licked or suckled by more than one female thus increasing the chance of spreading infectious agents that can be transmitted through contaminated saliva or direct contact. Peripheral males visit different colonies [23], are highly interactive and are frequently involved in aggressive physical contact, and so are likely to infect each other and transmit diseases not only between groups, but also between colonies. Unlike females or Central males, all Peripheral males tested were positive for FIV which is thought to be transmitted by biting [45] supporting this possibility, although there was no associated increase in clinical abnormality [unpublished observations]. In another free-ranging cat colony, cats, especially male cats, that arrived as immigrants in a study colony are likely to be FIV infected [Courchamp, Artois and Pontier: personal communication].

Relative to females, males are more susceptible to parasites, and elicit a weaker immune response [46, 47]. A quarter of our study females were clinically abnormal, compared with 46% of males, and haematological analysis suggested that males might be more vulnerable to the stress of diseases [unpublished observations]. However, the lack of sex-related differences in parasite prevalence suggests that both sexes found infection difficult to avoid.

Clinically abnormal females tended to have higher rates of infection with FIV than normal females, as found in other studies [5, 34]. FIV infection is associated with respiratory clinical signs but not with anaemia [5], and our results conform to this pattern: FIV-positive animals had lower prevalences of *H. felis*, which causes feline infectious anaemia, but higher antibody titres for FHV, which causes infectious respiratory disease, than FIV-negative cats. Strong correlations between antibody titres to FCV, FHV and FCoV infections could result from their common mode of transmission [13, 44].

Implications for endangered felids

Many endangered wild felids live in small, inbred, isolated populations, and are threatened by disease

outbreaks, which can cause high mortality among wild or captive felids [48, 49]. Iriomote wildcats, for example, number only 80–100 individuals, and have no antibodies against FPV and FIV, which are major causes of disease in domestic cats [10]. Similarly, 23 free-living European wildcats caught in Scotland were all negative for antibodies to FIV and FCoV [8], and eight wildcats caught in France were negative for FIV [9].

In order to prevent infectious disease being transmitted to endangered felids, resource centres that allow large populations of feral cats to build up and which could attract wild felids, increasing interspecific contact, should be discouraged. Where feral cats pose a risk to endangered felids, their role in disease transmission should be closely monitored. Reproductively successful Peripheral males in particular should be targeted for disease monitoring and control.

Our study shows that because of the social nature of feral farm cat colonies, infections with pathogens are highly prevalent, but are not necessarily associated with clinical signs. In particular, mature Peripheral males are probably a major vector of diseases from one area to another. It seems likely that in Britain such males could pose a significant threat to Scottish wildcats, which are already imperilled by genetic introgression from their descendant domestic cats [8, 50].

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REFERENCES

- Dubey JP. Feline toxoplasmosis and coccidiosis: a survey of domiciled and stray cats. *J Am Vet Med Assoc* 1973; **162**: 873–7.
- Nichol S, Ball SJ, Snow KR. Prevalence of intestinal parasites in domestic cats from the London area. *Vet Rec* 1981; **109**: 252–3.
- Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994.
- Parsons JC. Ascarid infections of cats and dogs. *Vet Clin North Am [Small Anim Pract]* 1987; **17**: 1307–39.
- Hosie MJ, Robertson C, Jarrett O. Prevalence of feline leukaemia virus and antibodies to feline immunodeficiency virus in cats in the United Kingdom. *Vet Rec* 1989; **128**: 293–7.
- August JR, ed. *Consultations in feline internal medicine*, vol. 2. Philadelphia: W. B. Saunders, 1994.
- Roelke ME, Forrester DJ, Jacobson ER, et al. Seroprevalence of infectious disease agents in free-ranging Florida panthers (*Felis concolor coryi*). *J Wildl Dis* 1993; **29**: 36–49.
- McOrist S, Boid R, Jones TW, Easterbee N, Hubbard AL, Jarrett O. Some viral and protozoal diseases in the European wildcat (*Felis silvestris*). *J Wildl Dis* 1991; **27**: 693–6.
- Artois M, Remond M. Viral diseases as a threat to free-living wild cats (*Felis silvestris*) in Continental Europe. *Vet Rec* 1994; **134**: 651–2.
- Mochizuki M, Akuzawa M, Nagatomo H. Serological survey of the Iriomote cat (*Felis iriomotensis*) in Japan. *J Wildl Dis* 1990; **26**: 236–45.
- Shelton GH. Management of the feline immunodeficiency virus-positive patient. In: August JR, ed. *Consultations in feline internal medicine*, vol. 2. Philadelphia: W. B. Saunders, 1994: 27–32.
- Pedersen NC, Ho EW, Brown ML, Yamamoto JK. Isolation of a T-lymphotropic virus from domestic cats with an immunodeficiency-like syndrome. *Science* 1987; **235**: 790–3.
- Gaskell RM, Dawson S. Viral-induced upper respiratory tract disease. In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 453–72.
- Wardley RC, Gaskell RM, Povey RC. Feline respiratory viruses; their prevalence in clinically healthy cats. *J Small Anim Pract* 1974; **15**: 579–86.
- Gaskell RM, Bennett M. Feline poxvirus infection. In: Appel M, ed. *Virus infections of carnivores*. Amsterdam: Elsevier Science Publishers BV, 1987: 267–86.
- Pedersen NC. Feline coronavirus infection. In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 506–14.
- Lappin MR. Diagnosis of toxoplasmosis. In: August JR, ed. *Consultations in feline internal medicine*, vol. 2. Philadelphia: W. B. Saunders, 1994: 41–6.
- Wright AI. Endoparasites. In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 595–610.

19. Webster JP. Prevalence and transmission of *Toxoplasma gondii* in wild brown rats, *Rattus norvegicus*. *Parasitology* 1994; **108**: 407–11.
20. Gaskell RM, Bennett M. Other feline virus infections. In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 535–43.
21. Gaskell RM. Feline panleucopenia. In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 445–52.
22. Macdonald DW, Apps PJ, Carr GM, Kerby G. Social dynamics, nursing coalitions and infanticide among farm cats, *Felis catus*. *Adv Ethology* 1987; **28**: 1–64.
23. Kerby G, Macdonald DW. Cat society and the consequences of colony size. In: Turner DC, Bateson P, eds. *The domestic cat: the biology of its behaviour*. Cambridge: Cambridge University Press, 1988: 67–82.
24. Liberg O, Sandell M. Spatial organisation and reproductive tactics in the domestic cat and other felids. In: Turner DC, Bateson P, eds. *The domestic cat: the biology of its behaviour*. Cambridge: Cambridge University Press, 1988: 83–98.
25. Caro TM, Holt ME, Fitzgibbon CD, Bush M, Hawkey CM, Kock RA. Health of adult free-living cheetahs. *J Zool Lond* 1987; **212**: 573–84.
26. Wills JM. Chlamydial infection in the cat. [Ph.D. thesis]. Bristol: University of Bristol, 1986: 250.
27. Muir P, Harbour DA, Gruffydd-Jones TJ, et al. A clinical and microbiological study of cats with protruding nictitating membranes and diarrhoea: isolation of a novel virus. *Vet Rec* 1990; **127**: 324–30.
28. Stoddart ME, Gaskell RM, Harbour DA, Gaskell CJ. Virus shedding and immune responses in cats inoculated with cell culture-adapted feline infectious peritonitis virus. *Vet Microbiol* 1988; **16**: 145–8.
29. O'Dair HA, Hopper CD, Gruffydd-Jones TJ, Harbour DA, Waters LA. Clinical aspects of *Chlamydia psittaci* infection in cats infected with feline immunodeficiency virus. *Vet Rec* 1994; **134**: 365–8.
30. Gaskell RM, Gaskell CJ, Evans RJ, et al. Natural and experimental poxvirus infection in the domestic cat. *Vet Rec* 1983; **112**: 164–70.
31. Scott FW. Evaluation of a feline viral rhinotracheitis-feline calicivirus disease vaccine. *Am J Vet Res* 1977; **38**: 229–34.
32. Harbour DA, Howard PE, Gaskell RM. Isolation of feline calicivirus and feline herpesvirus from domestic cats 1980 to 1989. *Vet Rec* 1991; **128**: 77–80.
33. Courchamp F, Pontier D. Feline immunodeficiency virus: an epidemiological review. *C R Acad Sci Paris* 1994; **317**: 1123–34.
34. Bennett M, McCracken H, Lutz H, et al. Prevalence of antibody to feline immunodeficiency virus in some cat population. *Vet Rec* 1989; **124**: 397–8.
35. Bennett M, Lloyd G, Jones N, et al. Prevalence of antibody to hantavirus in some cat populations in Britain. *Vet Rec* 1990; **127**: 548–9.
36. Van Rensburg PJJ, Skinner JD, Van Aarde RJ. Effects of feline panleucopaenia on the population characteristics of feral cats on Marion Island. *J Appl Ecol* 1987; **24**: 63–73.
37. Wills JM, Gaskell RM. Feline chlamydial infection (feline pneumonitis). In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 544–51.
38. Jarrett O. Feline leukaemia virus. In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 473–87.
39. Scott FW, Kahn DE, Gillespie JH. Feline viruses; isolation, characterisation and pathogenicity of a feline reovirus. *Am J Vet Res* 1970; **31**: 11–20.
40. Snodgrass DR, Angus KW, Gray EW. A rotavirus from kittens. *Vet Rec* 1979; **104**: 222–3.
41. Stoddart ME, Bennett M. Feline coronavirus infection. In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics* 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 506–14.
42. Pedersen NC. Serologic studies of naturally occurring feline infectious peritonitis. *Am J Vet Res* 1976; **37**: 1449–53.
43. Nichol S, Ball SJ, Snow KR. Prevalence of intestinal parasites in domestic cats from the London area. *Vet Rec* 1981; **109**: 252–3.
44. Kerby G. The social organisation of farm cats (*Felis catus* L.) [D.Phil. thesis]. Oxford: University of Oxford, 1987.
45. Hopper CD, Sparkes AH, Harbour DA. Feline immunodeficiency virus. In: Chandler EA, Gaskell CJ, Gaskell RM, eds. *Feline medicine and therapeutics*, 2nd ed. Oxford: Blackwell Scientific Publications, 1994: 488–505.
46. Grossman CJ. Interactions between the gonadal steroids and the immune system. *Science* 1985; **227**: 257–61.
47. Alexander J, Stimson WH. Sex hormones and the course of parasitic infection. *Parasitol Today* 1988; **4**: 189–93.
48. Morell V. Serengeti's big cats are going to the dogs. *Science* 1994; **264**: 1664.
49. Heeney JL, Evermann JF, McKeirnan AJ, et al. Prevalence and implications of feline coronavirus infections of captive and free-ranging cheetahs (*Acinonyx jubatus*). *J Virol* 1990; **64**: 1964–72.
50. French DD, Corbett LK, Easterbee N. Morphological discriminants of Scottish wildcats (*Felis silvestris*), domestic cats (*F. catus*) and their hybrids. *J Zool Lond* 1988; **214**: 235–59.