Long-term impact on a closed household of pet cats of natural infection with feline coronavirus, feline leukaemia virus and feline immunodeficiency virus

D. D. ADDIE, J. M. DENNIS, S. TOTH, J. J. CALLANAN, S. REID, O. JARRETT

A closed household of 26 cats in which feline coronavirus (FCoV), feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV) were endemic was observed for 10 years. Each cat was seropositive for FCoV on at least one occasion and the infection was maintained by reinfection. After 10 years, three of six surviving cats were still seropositive. Only one cat, which was also infected with FIV, developed feline infectious peritonitis (FIP). Rising anti-FCoV antibody titres did not indicate that the cat would develop FIP. The FeLV infection was self-limiting because all seven of the initially viraemic cats died within five years and the remainder were immune. However, FeLV had the greatest impact on mortality. Nine cats were initially FIV-positive and six more cats became infected during the course of the study, without evidence of having been bitten. The FIV infection did not adversely affect the cats' life expectancy.

THERE have been many publications describing the effects on cats of infections with either feline immunodeficiency virus (FIV), feline leukaemia virus (FeLV) or feline coronavirus (FCoV) individually, and cats have been infected with combinations of these viruses in laboratory settings. However, this is the first report of a group of cats naturally infected with all three viruses and studied over a long period.

The rate of infectivity of FIV is extremely variable with reports in the literature stating that from 0 to 100 per cent (Table 1) of cats in contact with an FIV-infected cat may become infected. In addition, it has been recognised that FIV-infected cats kept under specific pathogen-free (SPF) conditions can survive for long periods after being first diagnosed (Kohmoto and others 1998). However, it is not clear whether this phenomenon is related solely to cats kept in an artificially disease-free environment or whether it also applies to cats in the field. Both FIV and FeLV are immunosuppressive agents, but there is little information about their ability to influence each other's infectivity. In households where FCoV is endemic, 5 to 15 per cent of infected cats may develop feline infectious peritonitis (FIP), and cats which are immunocompromised by a simultaneous retrovirus infection are believed to be at greater risk (Poland and others 1996).

This paper describes a study of the transmission rates of FIV, FeLV and FCoV among 26 cats in a closed household over a period of 10 years. The viral status of the animals and their survival from first diagnosis were monitored, and their causes of death were established specifically to determine whether the viruses might have been implicated.

MATERIALS AND METHODS

Husbandry

Twenty-six pet cats kept in one household were monitored for 10 years. The cats were allowed to mix with each other but none was allowed to roam outside. Only one cat (cat Z) was introduced during the 10 years; it was the only pedigree cat, a Persian, and had been rescued in May 1989 from its previous owner who wished it to be euthanased because it had an anti-FCoV immunofluorescent antibody titre of 1280. None of the cats was vaccinated against FeLV, feline panleucopenia virus, feline calicivirus or feline herpesvirus. Virological testing was begun in March 1988, because three cats had died during 1987 after developing anaemia, diarrhoea and suspected liver failure. *Haemobartonella felis* had been identified in one of the three cats.

The cats were blood tested annually; the blood samples were taken and any postmortem examinations were performed by one of the authors (J. D.). The blood samples and selected formalin-fixed tissue samples were examined serologically and histologically at the Feline Virus Unit, University of Glasgow.

Serology and virology

The samples were initially screened for FIV antibodies by using a commercial ELISA (FIV Petcheck; Idexx) and positive results were confirmed by Western blotting (Hosie and Jarrett 1990) or by immunofluorescence (IF) (Pedersen and others 1987). Samples examined after 1991 were tested by IF. FeLV antigen was detected by using an ELISA to detect p27 (FeLV Petcheck; Idexx, or Innochem; C. Lutz) and positive results were confirmed by virus isolation (Jarrett and Ganière 1996). Virus neutralising antibody titres to FeLV were measured as described by Jarrett and Ganière (1996). Antibodies to FCoV were measured by IF (Addie and Jarrett 1992), and faeces and saliva were monitored for FCoV by the detection of FCoV RNA by reverse transcriptase-polymerase chain reaction (RT-PCR) by the method described by Herrewegh and others (1995).

Pathology

Postmortem examinations were carried out on 15 of the 19 cats that died. Sections of formalin-fixed, paraffin-embedded tissue were cut at 5 µm and stained with haematoxylin and eosin. Selected sections were also stained with Giemsa stain. To investigate the phenotype of lymphoid tumours, paraffin-embedded sections were immunostained with anti-CD79a (mb-1), anti-CD3 and MAC 387 (anti-macrophage/anti-neutrophil) antibodies by the methods of Callanan and others (1996).

Statistical analysis

To compare the survival of the cats in the different groups, classified according to their viral infection status, Kaplan-Meier product-limit survival curves were generated. The Tarone-Ware statistic was used to assess whether any observed differences were statistically significant at the 5 per cent level. Where necessary, a Cox proportional hazard model was used to assess the hazard function associated with viral status, taking into account age at testing and seroconversion; again

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D. D. Addie, PhD, BVMS, MRCVS,
S. Toth, PhD, DVM,
S. Reid, PhD, BVMS, MRCVS,
O. Jarrett, PhD, BVMS, MRCVS, FRSE,
Department of Veterinary Pathology, University of Glasgow Veterinary School, Bearsden Road, Bearsden, Glasgow G61 1QH
J. M. Dennis, BVMS, MRCVS, 1 Homefield Road, Bromley BR1 3AW
J. J. Callanan, PhD, MVB, MRCVS, MRCPath, Department of Veterinary Pathology, University College Dublin, Shelbourne Road, Dublin 4, Ireland

significance was set at the 5 per cent level. The longevity of the cats was also compared by using the Kaplan-Meier product-limit method.

RESULTS

Table 2 shows the sex and age of the cats at the end of study or at death, and the causes of death. Tables 3, 4 and 5 give the serological data for FCoV, FeLV and FIV, respectively.

FCoV antibody titres and FIP

Each cat had anti-FCoV antibodies on at least one occasion during the study (Table 3). The percentage of seropositive cats decreased from 92 per cent in July 1988 to 39 per cent in September 1989, but by March 1990, 94 per cent of the cats were again seropositive, indicating that they had been reinfected. There were two further similar cycles; the prevalence of seropositive cats decreased to 50 per cent by October 1993, increased to 88 per cent in December 1995, and decreased to 50 per cent in 1998.

The presence of FCoV-RNA in the faeces was monitored only once, in January 1996, when six of the remaining eight cats were shedding the virus. There was no absolute correlation between seropositivity and virus shedding, but all but one of the virus shedders had high antibody levels and the only seronegative cat, T, did not shed the virus.

Only cat B, which was also infected with FIV, developed FIP.

Transmission of FeLV

At the beginning of the study, seven of the cats were infected with FeLV and no more became viraemic. Cat X had initially been discordant in March 1988, was FeLV-negative in October 1988, but became persistently viraemic in March 1990. The titres of FeLV neutralising antibody were measured in 23 cats (Table 4). Four of the five FeLV-positive cats had no detectable virus neutralising antibodies and the fifth had an antibody titre of only 4. In contrast, 14 of the remaining 18 FeLV-negative cats had antibody titres of over 32 and only one was seronegative. None of these cats became FeLV-positive. Eight years after the first test and four years after the last FeLV-positive cat died, the virus neutralising antibody titres of four of the six remaining cats tested had decreased.

Of the four cats that were FeLV-positive but not also infected with FIV, cat X developed myeloid leukaemia and died at 10 years of age, 18 months after FeLV had first been isolated. Cats U and Y were euthanased at the start of the survey, within one and two months of being diagnosed, when they were six and nine years old, respectively. Cat U had an osteosarcoma involving its mandible. Cat Y was not examined postmortem. Cat M was euthanased six months after it was first diagnosed after it had developed acute respiratory distress and pallor; it was not examined postmortem. Within five years of being diagnosed, all the cats with FeLV infection had died.

Three of the seven FeLV-infected cats were also infected with FIV. Of these, cats H, R and W survived six, 24 and 58 months, respectively, after being diagnosed. Cat H was euthanased owing to intractable melaena six months after

TABLE 1: Previous studies of the transmission of feline immunodeficiency virus (FIV)

Number of cats infected	Number of cats in contact	Number of in-contact cats FIV-infected	Number of households	Reference
N	14	0	1	Yamamoto and others (1988)
16	31	0	16	Shelton and others (1989)
4	68	0	1	Shelton and others (1990)
N	34	1	N	Sparger and others (1989)
9	7	2	1	Pedersen and others (1987)
N	N	3-67%	N	Hosie and others (1989a)
N	27	14	N	Hopper and others (1989)
N	11	11	1	Hosie and others (1989b)

N Not given

TABLE 2: Sex, age at the end of the study or death, and the cause of death of the cats

Cat	Sex	Date of birth	Date of death	Age at death (years)	Age in Oct '98 (years)	†Survival (months)	FeLV/FIV status	Cause of death
A	MN	May 1981	Apr 1996	15		30	FIV	T cell alimentary lymphosarcoma*
B	MN	Aug 1983	June 1991	8		32	FIV	Feline infectious peritonitis*
C	MN	May 1982	Feb 1991	9		17	FIV	Thymoma*
D	MN	May 1981	July 1993	12		51	FIV	Chronic proliferative cholangitis*
E	MN	May 1987	June 1998	11		117	Neg	Chronic heart failure, alimentary carcinoma*
F	MN	pre 1975	June 1989	>14		8	Neg	Squamous cell carcinoma*
G	MN	June 1987	Nov 1993	6		61	Neg	B cell lymphosarcoma on face*
H	FN	June 1987	Apr 1989	2		6	FeLV, FIV	E; melaena
I	FN	1981	June 1989	8		8	Neg	Cardiomyopathy*
J	FN	1986	Jan 1991	5		27	Neg	Nephritis, cholangitis*
K	MN	Aug 1983	Nov 1992	9		49	FIV	E; heart failure, myocarditis,* glomerulonephritis, chronic bronchitis
L	FN	Apr 1980	Mar 1989	9		5	Neg	E; pale and respiratory distress
M	FN	Mar 1987	Apr 1989	2		6	FeLV	E; pale and respiratory distress
N	FN	Apr 1987			11·5	84	FIV	
O	MN	Apr 1987			11·5	48	FIV	
P	MN	Oct 1986			12	120	Neg	
Q	MN	May 1982	Nov 1989	7		13	FIV	Chronic interstitial nephritis*
R	MN	July 1982	Oct 1990	8		24	FeLV, FIV	Acute blast cell leukaemia*
S	MN	Feb 1984			14·5	84	FIV	Adenocarcinoma, glomerulonephropathy*
T	FN	Apr 1983			15·5	120	FIV	
U	MN	May 1982	Sep 1988	6		NA	FeLV	Osteosarcoma of jaw
V	FN	Sep 1981	Nov 1994	13		73	FIV	Bronchitis, cholangitis, *Haemobartonella felis**
W	MN	Apr 1983	Aug 1993	10		58	FeLV, FIV	E; intractable gingivitis, pharyngitis. Histology revealed focal interstitial nephritis, cholangitis, nodular hyperplasia of pancreas*
X	FN	May 1981	Sep 1991	10		‡18	FeLV	Myeloid leukaemia*
Y	F	1979	Mar 1988	9		NA	FeLV	E; poor body condition
Z	FN	Aug 1988			10	84	FIV	

* Histopathological diagnosis, † Survival in months since first FeLV and FIV tests, ‡ FeLV discordant in Feb 1988, negative Oct 1988 and 1989, and became FeLV-positive in March 1990
MN Male neutered, FN Female neutered, E Euthanased, NA Not applicable, diagnosed FeLV-positive at time of illness

TABLE 3: Antibody titres to feline coronavirus in the 26 cats during the study

Cat	Mar '88	Jul '88	Oct '88	Apr '89	Sep '89	Mar '90	Sep '90	Oct '91	Oct '92	Oct '93	Oct '94	Dec* '95	Oct '96	Oct '97	Oct '98
A		40	20	80	160	640	320	40	80	40	>1280	160+	Dead		
B		40	20	80	80	320	320		FIP June 91						
C		160	40	40	0	0	0	80	Dead						
D		80	40	20	0	640	640	80	0	Dead					
E		80	0	0	0	40	40	0	0	0	20	160+	160	160	Dead
F		40	0	0	Dead										
G		160	80	80	0	640	640	80	40	0	Dead				
H		160	160	80	Dead										
I		20	0	40	Dead										
J	160	10	0	0	0	320	640	Dead							
K		20	20	0	10	160	20	10	40	Dead					
L		80	20	Dead											
M	80	0	0	20	Dead										
N		160	80	160	1280	1280	1280	80	160	20	320	20+	320	80	0
O		80	80	40	320	640	1280	320	640	640	>1280	320+	640	1280	320
P		160	160	80	640		640	80	160	160	20	320+	320	160	0
Q		10	80	20	0	Dead									
R		20	0	10	20	40	0	Dead							
S		20	40	20	0	80	160	20	0	0	0	20–	0	10	80
T		0	0	0	0	80	0	0	0	0	0	0–		0	0
U		160	Dead												
V		20	10	0	0	40	160	10	0	0	0	Dead			
W		20	0	0	0	80	20	10	0	Dead					
X		80	40	10	0	320	160	Dead							
Y	160	Dead													
Z	[Acquired May '89 with IFA titre 1280]				20	160	1280	80	160	640	>1280	320+	320	320	320
SN		2	8	7	11	1	3	2	6	5	3	1	1	1	3
SP		23	15	15	7	16	15	12	7	5	6	7	5	6	3
%SP		92	65	70	39	94	83	71	54	50	66	88	83	86	50

* Virus shedding status, only cat A was shedding virus in saliva and faeces, the rest were shedding in the faeces only
SN Seronegative, IFA Immunofluorescent antibody, SP Seropositive, FIP Feline infectious peritonitis

diagnosis when it was two years of age (no histopathology available). Cat R developed acute blast cell leukaemia at eight years of age, 24 months after FeLV and FIV were first diagnosed. Cat W was euthanased 58 months after FeLV and FIV were diagnosed when it was 10 years old owing to untreatable chronic gingivitis and pharyngitis; postmortem examination revealed focal interstitial nephritis, cholangitis and nodular hyperplasia of the pancreas.

Transmission of FIV despite lack of aggression

The cats were first screened serologically for FIV in October 1988. Eight of the remaining 25 cats (cat Y, an FeLV-infected cat had died) were FIV-positive, and cat C was seropositive by ELISA but seronegative by IF (Table 3). Cat C and six more cats became seropositive over the next six years. According to the owners, the cats displayed little aggression, with the exception of occasional paw-flailing involving cats D, E and W; cat W

TABLE 4: Isolation of feline leukaemia virus (FeLV) and results of tests for virus neutralising antibody (VNA)

						FeLV status								FeLV-VNA titres		
Cat	Mar '88	Jul '88	Oct '88	Mar '90	Sep '90	Oct '91	Oct '92	Oct '93	Oct '94	Dec '95	Oct '96	Oct '97	Oct '98	Apr '89	Oct '91	Oct '97
A			–	–	–	–	–	–	–	–	Dead			>32	>32	
B			–	–	–	Dead								16		
C			–	–	–	Dead								>32		
D			–	–	–	–	–	Dead						>32	>32	
E		–	–	–	–	–	–	–	–	–	–	–	Dead	8	0	0
F			–	Dead										0		
G			–	–	–	–	–	–	Dead					>32	>32	
H			+	Dead										0		
I			–	Dead										>32		
J	–	–	–	–	–	Dead								>32		
K			–	–	–	–	–	Dead						>32	>32	
L			–	Dead												
M			+	Dead										0		
N			–	–	–	–	–	–	–	–	–	–	–	>32	>32	4
O			–	–	–	–	–	–	–	–	–	–	–	>32	>32	>32
P			–	–	–	–	–	–	–	–	–	–	–	>32	>32	8
Q			–	Dead										>32		
R			+	+	+	Dead								0		
S			–	–	–	–	–	–	–	–	–	–	–	16		8
T			–	–	–	–	–	–	–	–	ND	–	–	>32	>32	>32
U		+	Dead													
V			–	–	–	–	–	–	–	Dead				>32		
W			+	+	+		+	Dead						0		
X	+/–		–	+*	+	Dead								4		
Y	+	Dead														
Z	[Acquired May '89]			–	–	–	–	–	–	–	–	–			>32	

* Indicates first FIV seroconversion
ND Not done

TABLE 5: Results of tests for feline immunodeficiency virus (FIV) and period of survival from first diagnosis of FIV

Cat	Jul '88	Oct '88	Apr '89	Sep '89	Oct '91	Oct '92	Oct '93	Oct '94	Dec '95	Oct '96	Oct '97	Oct '98	Survival (months)
H		+	+	Dead									6*
Q		+	+	+	Dead								13
C		+/–†	–	+	Dead								17
R		+	+	+	Dead								24*
A		–	–	–	–	–	+‡	+	+	Dead			30
B		+	+	+	Dead								32
O		–	–	–	–	–	–	+‡	+	+	+	+	48
K		+	+	+	+	+	Dead						49
D		–	+‡	+	+	+	Dead						51
W		+	+	+	+	+	Dead						58*
V		+	+	+	+	+	+	+	Dead				73
N		–	–	–	+‡	+	+	+	+	+	+		84
S		–	–	–	+‡	+	+	+	+	+	+	+	84
Z	Acquired May '89			–	+‡	+	+	+	+	+	+	+	84
T		+	+	+	+	+	+	+	+	+	+	+	120
Y	Dead												
U	–	Dead											
F		–	Dead										
L		–	Dead										
M		–	–	Dead									
I		–	–	Dead									
J	–	–	–	–	Dead								
X		–	–	–	Dead								
G		–	–	–	–	–	–	Dead					
E	–	–	–	–	–	–	–	–	–	–	–	Dead	
P	–	–	–	–	–	–	–	–	–	–	–	–	

* Coinfected with FeLV, † Seropositive by ELISA but seronegative by IF, ‡ Indicates first FIV seroconversion

was infected from the start of the study, cat D became infected in its first year and cat E remained uninfected. There was no evidence of the cats biting one another.

Longevity of FIV or FeLV-infected cats after diagnosis

Seven of the 12 FIV (but not FeLV)-infected cats died at 13, 17, 30, 32, 49, 51 and 73 months after being diagnosed. The remaining five FIV-infected cats remained healthy to the end of the study, 48, 84 (three cats) and 120 months after being diagnosed. A comparison of the seronegative cats with the FIV-positive cats did not reveal any significant difference in longevity or survival from their first testing positive. There was a trend for the FIV-positive cats to survive longer in that their median survival time was 51 months compared with 17·5 months for the seronegative cats, but this difference was not statistically significant (Fig 1). The impact of FeLV was much more pronounced and the FeLV-positive cats, including those with concurrent FIV infections, survived for a significantly shorter time than the FIV-infected and seronegative cats ($P<0·01$); the median survival times of the FeLV-infected, FIV-infected and FeLV/FIV coinfected cats were six, 51 and 17·5 months, respectively. In no case was the cat's age when tested a significant covariate. FeLV infection also had a significant effect ($P<0·05$) on the cats' longevity, with median lifespan figures of 86·5 months for the FeLV/FIV coinfected cats, 150 months for the FIV-infected cats and 103 months for the retrovirus-negative cats.

Tissues from six of the seven FIV-infected cats showed a wide range of pathological changes. Cat A had an alimentary T cell lymphosarcoma and cat C had a thymoma. Other histopathological changes observed in individual cats included cholangitis, bronchitis, chronic interstitial nephritis, myocarditis, glomerulonephritis and *Haemobartonella felis*-associated haemolytic anaemia. As indicated above, cat B, which was FIV-positive and also had antibodies to FCoV, developed non-effusive FIP.

Throughout the study, seven of the 26 cats remained free of FIV and FeLV infection, but six of them died during the study. Three cats developed tumours: cat E was euthanased with severe ascites and dyspnoea, and histopathology revealed chronic heart failure and a carcinoma in the intestines. Cat F had a squamous cell carcinoma in its mouth, and cat G had a lymphosarcoma involving the buccal mucosa of its right cheek. These animals died at 11, 14 and six years of age, respectively. Cat I developed dilated cardiomyopathy, and cat J developed cholangitis and nephritis. Cat L was euthanased owing to acute respiratory stress in association with pallor, but it was not examined postmortem.

DISCUSSION

As far as the authors are aware, this is only the second longitudinal study of a household of cats naturally infected with FCoV, FeLV and FIV, a household of 73 cats having been monitored in retrospect between 1977 and 1980 (Shelton and others 1990). In the present study, the 10-year period of observation has made it possible to obtain a more definitive assessment of the relative impact of each of these persistent viral infections on the population. The pattern of transmission of FCoV and FeLV, and the diseases they produced, illustrated several important features about the diagnosis and epidemiology of the viruses, and was consistent with previous reports. The most remarkable observation was that FIV was transmitted fairly regularly throughout the period, in the absence of overt aggression between the cats, and that it had little effect on the survival of the cats.

FCoV had a modest effect on life expectancy, causing one case of FIP, although, as expected, because the virus is very

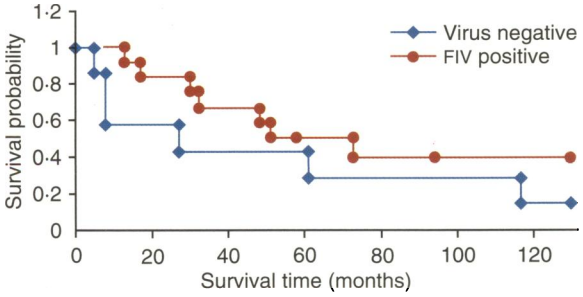

FIG 1: Survival curves of cats infected with feline immunodeficiency virus (FIV) and FIV-seronegative cats

contagious, all of the cats had at one time been infected. All but two of the cats were seropositive in the first full test in July 1988, and again in March 1990, after presumed reinfection. Since most of the cats in the household were FCoV-seropositive most of the time, it would not have been necessary to test all the cats to establish whether FCoV infection was present and a random sampling of, say, six cats would have been sufficient. However, this is only true because all the cats were mixing and were reinfecting each other; had the cats been separated into groups, FCoV would have died out in some groups and it would still have been necessary to test all the cats.

The anti-FCoV antibody titres of the cats decreased twice, in 1989 and again in 1992, indicating that in a closed household, that is, one which admits no new cats or kittens and in which the cats do not go outdoors, FCoV infection may eventually die out spontaneously. It is possible that by segregating the seropositive and seronegative cats FCoV infection could have been eliminated completely, as described by Gonon and others (1995). The percentage of seropositive cats decreased from 92 per cent in July 1988 to 39 per cent in September 1989. However, by March 1990, 94 per cent of the cats were again seropositive, indicating that they had been reinfected. The two possible sources of reinfection were the introduction of the seropositive cat Z in May 1989, or virus shed by one or more of the remaining seropositive cats. It seems unlikely that cat Z was the source of reinfection, because more cats became seronegative between the tests in April and September 1989, although cat Z was introduced in May. It is therefore more probable that the household was reinfected from within by, for example, cat O which is likely to have been a chronic FCoV shedder, because it was at no time seronegative. Alternatively, the persistence of FCoV in this household could have been due, at least in part, to the coinfection of many cats with FIV, because FIV-positive cats have been reported to shed 10 to 100 times more FCoV than FIV-negative cats (Poland and others 1996). However, the two cats which were not shedding FCoV in December 1995 were also FIV-positive. The FCoV antibody titres of cats which are not shedding the virus themselves, but are living with cats which are, can decline. However, within a few weeks, animals which have become seronegative usually begin shedding the virus again and seroconvert (D. D. Addie, unpublished observations).

Only one cat, cat B, died of FIP, 35 months after being found to be seropositive. This finding illustrates the fact that cats can be infected with FCoV quite unbeknownst to their owners because they may show no clinical signs and take a very long time before developing FIP. However, an incubation period of 35 months is very unusual because cats are most likely to die of FIP within six to 18 months of being infected (Addie and others 1995). The cat which developed FIP was also infected with FIV, so that its immunity may have been compromised by FIV, allowing FIP to develop. This explanation seems probable in view of the report of an experimental coinfection of cats with FCoV and FIV, in which two of 19 cats developed FIP within 10 weeks of becoming infected by FCoV (Poland and others 1996). That none of the FeLV-infected cats developed FIP is slightly surprising because FIP has been reported to be the third most common cause of death in FeLV-infected cats (Reinacher and Theilen 1987).

Cats A, B, K, N, O, P and Z sustained a four-fold or greater increase in antibody titre after the first reinfection, but only cat B developed FIP, confirming that a significant rise in antibody titre is not necessarily prognostic of the development of FIP. In experimental infections, seropositive cats develop FIP more rapidly than seronegative cats when they are reinfected. However, in natural infections, cats which have previously been infected have been shown to be more resistant to reinfection rather than less (Addie and others 1995).

Of the three viruses, FeLV had the greatest impact on life expectancy. Six of the seven cats that were infected when the study began died within two years, in agreement with an earlier observation that 85 per cent of viraemic cats died within 3·5 years of being naturally infected (McClelland and others 1980). The seventh infected cat died after five years. While these viraemic cats were in the household, none of the in-contact cats became viraemic, indicating that they were immune and resistant to reinfection. All but one had high titres of virus neutralising antibody indicating that they had been transiently infected, which is an excellent indicator of an immune cat (Hardy and others 1976). In this type of household, FeLV infection is therefore self-limiting, although any cats introduced would have been at great risk while the infected cats were still alive.

FIV infected an additional six cats during six years. At the last sampling in October 1998, four of the five remaining cats were infected with FIV, indicating that cats exposed to FIV do not appear to become immune and resistant to infection, as do many cats exposed to FeLV.

The present findings differ from those of Shelton and others (1990) who found no evidence of transmission of FIV in a household of 73 cats over a period of three years. A knowledge of the infectivity of FIV is important because veterinary surgeons need to know whether to advise their clients to segregate FIV-positive from FIV-negative cats. The consensus of opinion has been that FIV-infected cats are more dangerous to free-roaming cats through biting than they are to cats that live peacefully with them in the same household (Hardy 1991). The findings of other research on this subject are summarised in Table 1. The proportion of cats in contact with FIV-infected cats which became infected with FIV varied from 0 to 100 per cent. The variable transmission of the virus has been attributed to differences in the infectivity of different strains of FIV, to increased viral shedding by symptomatic cats, and to differing susceptibility to infection or different patterns of fighting among cats in the same household (Hosie and others 1989b). In the household described here there was no aggression between cats other than some paw-flailing at mealtimes. It is therefore possible that the virus may have been transmitted via saliva by mutual grooming, close contact or the sharing of food bowls. This finding contrasts with the results of a previous study which suggested that these activities were not sufficient for the transmission of FIV (Shelton and others 1989), but it is consistent with the original report of FIV in which two cats in contact with FIV-positive cats seroconverted three months after their first test (Pedersen and others 1987). On the basis of the present results it would appear to be advisable to separate FIV-infected from uninfected cats to prevent further transmission of the virus. However, since FIV-infected cats may live at least as long as their uninfected counterparts, a test and euthanasia policy to eradicate FIV from households cannot be justified, although ultimately, such decisions can only be made by the owner of the cats concerned.

The results of this study agree with other epidemiological studies of FIV which suggest that it has a long incubation period after initial primary infection and that cats may remain asymptomatic for seven years or more (Pedersen and Barlough 1991, Kohmoto and others 1998). However, in this study the FIV-infected cats appeared to survive longer after diagnosis than the cats which were FIV-negative. Although the number of cats involved was small and this difference was not statistically significant, this finding was surprising because it was expected that FIV would reduce a cat's lifespan. Although FeLV has been proposed as a potentiating co-factor in FIV infection (Pedersen and others 1990), the three cats in this household that were infected with both viruses (H, R and W) did not die any earlier than the others and cat W survived almost five years after being diagnosed. It will be important to find out whether the virus load in the four remaining cats is low, which might account for the low pathogenicity, or whether the virus is of a phenotype with a low virulence.

It was not possible in all cases to ascribe the cause of death to any of the three viruses present, so that the figures for the

longevity of the FIV-infected cats were probably reduced by cats dying of conditions other than those due to FIV. Both FeLV and FIV are directly oncogenic (Beatty and others 1998), causing T and B cell lymphomas, respectively, and they may be indirectly oncogenic by immunosuppression. Neoplastic conditions were encountered in eight of the 20 cats which died. Cat A, infected with FIV but not FeLV, had a T cell alimentary lymphosarcoma, and another FIV-infected cat (cat C) had a thymoma. Two FeLV-infected cats developed leukaemia and a third developed an osteosarcoma. Neoplasia are expected in retrovirus-infected cats but they also occurred in three of the seven FeLV- and FIV-negative cats. Transient FeLV infection has been implicated in FeLV-negative lymphosarcomas (which cat G was suffering from), but not in squamous cell carcinoma (cat F) or alimentary carcinoma (cat E).

Anorexia, pyrexia, abdominal pain and severe diarrhoea leading to death were reported in five of 10 cats experimentally coinfected with FeLV and FIV (Pedersen and others 1990). One of the cats in the present household (cat H) was euthanased because of melaena which could possibly have been attributable to the coinfection, but the diagnosis was not confirmed postmortem.

The results of this study demonstrate the dynamics of FCoV, FeLV and FIV infection in a multicat household, and the usefulness of testing cats for these viruses.

Antibody testing was useful to establish that the cats were infected with FCoV. The virus was very contagious and infected all 26 during the study; a random sampling of a few cats would therefore have revealed the infection. Repeat sampling showed that some cats eliminated FCoV infection and became seronegative, but were reinfected, possibly by a carrier cat. A significant increase in FCoV antibody titre was not a poor prognostic sign – only one of 10 cats with significantly raised antibody titres developed FIP – thus it is important not to place too much reliance on a rise in FCoV antibody titres which may not necessarily mean that the cat is going to become clinically ill. Coinfection with retroviruses did not increase a cat's chance of developing FIP.

FeLV affected fewer of the cats but was more rapidly fatal. The measurement of virus neutralising antibody titres is useful to reveal whether or not it is safe to mix FeLV-positive and negative cats. Cats with discordant FeLV results should be tested repeatedly to establish their fate. In the absence of new cats being introduced, FeLV infection died out of the household within five years.

FIV may be transmitted in households where there appears to be little or no aggression between the cats, and every attempt should therefore be made to separate FIV-infected and uninfected cats. However, FIV did not appear to affect the cats, life expectancy adversely.

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D. D. Addie, S. Toth, S. Reid, O. Jarrett, J. M. Dennis and J. J. Callanan

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Notes