J. small Anim. Pract. (1984) **25**, 159–172.

The natural history of the major feline viral diseases

ROSALIND M. GASKELL

Department of Veterinary Medicine, University of Bristol, Langford House, Langford, Bristol BS18 7DU*

ABSTRACT

This paper discusses factors that are important in the natural history of five major feline viral diseases, namely, feline panleucopenia, feline viral rhinotracheitis, feline caliciviral disease, feline leukaemia virus infection, and feline infectious peritonitis. Each disease is considered in terms of the properties and infectivity of the infecting agent, the sources of infectious virus, the mode of transmission of the disease, and the methods by which the agent persists in the cat population. Finally, each disease is discussed in terms of immunity and the role of vaccination. All these factors affect the balance of the virus–host relationship and are thus directly relevant to the epizootiology of these diseases and their control.

INTRODUCTION

This paper contains an outline of factors that are important in the natural history of five major feline viral diseases, namely feline panleucopenia, feline viral rhinotracheitis, feline caliciviral disease, feline leukaemia virus infection, and feline infectious peritonitis. All the factors that will be discussed affect the balance of the virus–host relationship and are thus directly relevant to the epizootiology of the disease. More comprehensive reviews are available elsewhere (see references: general reviews).

FELINE PANLEUCOPENIA (FP)

Agent. Historically, perhaps, FP should be considered first as it is the first of these five major feline viral diseases which was shown to be viral in origin. This was shown as far back as 1928 by Verge & Cristoforoni, but it was not until 1964 that Johnson achieved the breakthrough of being able to grow the virus in tissue culture. Subsequently, it was shown to be a small single stranded DNA virus, a

* Present address: Department of Veterinary Pathology, University of Liverpool.

0022-4510/84/0300-0159 $02.00

member of the Parvoviridae and with only one serotype of the virus known, which is also indistinguishable serologically from mink enteritis virus. The recently emerged canine parvovirus (CPV) is also closely related, but minor differences do exist, both antigenically and in the viral DNA, which mean that the possible origin of CPV from FP virus can still only be speculative (Tratschin *et al.*, 1982).

Parvoviruses are an interesting group of viruses, one characteristic of which is an affinity and requirement of the virus for actively dividing cells. Thus the pathogenesis of feline panleucopenia (Fig. 1) is immediately understandable, if this is borne in mind: the virus shows a predilection for the rapidly dividing cells of lymphoid tissue and the bone marrow, leading to panleucopenia; the crypt epithelium of the intestinal mucosa leading to enteritis; and finally the rapidly dividing cells of the neonate and the foetus, leading to cerebellar hypoplasia and possibly early foetal deaths and resorption (Gillespie & Scott, 1973).

Infectivity. Panleucopenia is a highly infectious and ubiquitous disease and affects not only the domestic cat but also other members of the Felidae (for example tigers, cheetahs and leopards), the Mustellidae (mink and skunk), Procyonidae (e.g. coati mundi, racoon) and Viverridae (binturong) (Povey, 1976). It is unlikely that the presence of the disease in these other species has any significant impact on the life cycle of the virus in the domestic cat, because the domestic cat is far more likely to be exposed to the virus from another domestic cat or its environment. The morbidity of FP in a susceptible population will rapidly approach 100 per cent, because it is so highly infectious. Although cats of most ages may be affected, it is primarily a disease of young kittens who succumb when their maternal antibody has waned. In most cases, natural exposure results in a lifelong immunity to the disease. Not all infected cats, however, will show severe clinical signs; in some the disease will only be mild or subclinical. In some areas, the disease appears to have more of a seasonal incidence, with peaks occurring in the summer and early autumn. These appear to coincide with appearance of large numbers of susceptible kittens as a result of a seasonal pattern to the birth rate (Gillespie & Scott, 1973; Reif, 1976).

Source of virus. Virus is shed in large quantities in all the excretions of an infected cat; saliva, urine, faeces and vomitus. It is also present in the blood.

Mode of transmission. Virus probably enters the body by contact with infectious discharges mainly through the nose or mouth (Csiza *et al.*, 1971a, b). Biting and flying insects, such as fleas, have also been suspected of mechanically transmitting FP, but it seems very unlikely that they play a major rôle in the epidemiology of the disease. Transplacental infection certainly occurs with FP virus, and, indeed, is not surprising in view of the affinity of the virus for actively dividing cells. As shown in Fig. 1, it has been postulated that infection early on in pregnancy will result in early foetal death and resorption, whereas later on (from the middle third of gestation to immediately post-natally), it has been shown that it will result in the cerebellar hypoplasia/ataxia syndrome with in-coordination that may be seen in young kittens (Kilham *et al.*, 1967; Csiza *et al.*, 1972).

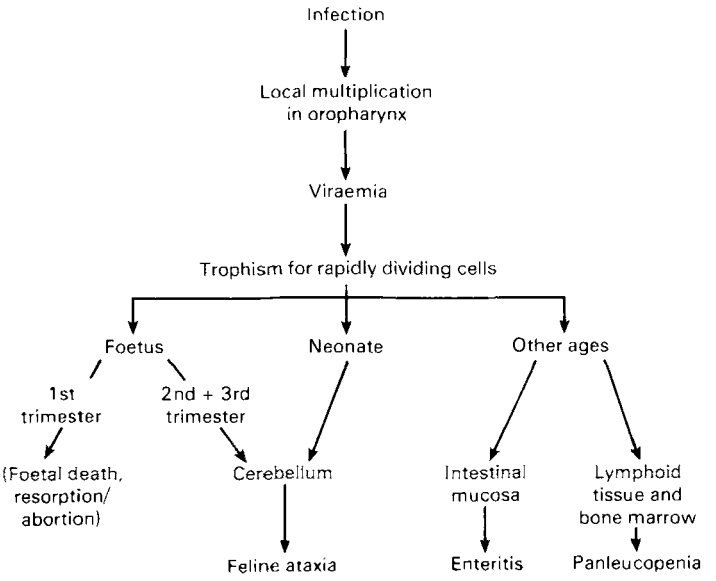

FIG. 1. Pathogenesis of feline panleucopenia.

Viral persistence. FP virus is perpetuated in the cat population in three main ways. First, by contact spread from acutely infected clinical cases to susceptible cats, given that there are a sufficient number of susceptibles in the population and sufficient opportunities for contact between them. Secondly, FP virus can persist in the recovered cat because of the existence of immune carriers. The recovered clinical case may harbour virus in its tissues for several months; indeed, in the tissues of ataxic kittens and in the faeces of mink, the virus has been shown to persist for up to a year (Bouillant & Hanson, 1965; Csiza *et al.*, 1971c). However, persistence of virus in these immune carriers is probably not particularly important —far more significant in the epidemiology of FP is the remarkable ability of the virus to survive in the environment. Most practitioners will be familiar with the difficulties of eradicating canine parvovirus from infected premises. FP virus is also extremely stable being resistant to heat and many disinfectants; it may persist in infected premises for up to a year (Johnson, 1966, 1969): Scott (1980) reporting on 27 products tested at the manufacturers' recommended concentrations found that only three solutions (Clorox, formaldehyde and glutaraldehyde) had sufficient virucidal activity against FP virus.

Immunity. FP virus is highly antigenic and immunity to the disease is high and long lived. Indeed, it has been shown that after vaccination with live attenuated virus, immunity will persist in cats kept in isolation for at least 4 years (O'Reilly & Hitchcock, 1976). In the natural situation, too, there is undoubtedly much natural boosting of immunity, except in isolated household pets. In kittens born to immune queens there is a high correlation between antibody titres in the queens and

acquired passive immunity levels in their kittens (Scott *et al.*, 1970). Most kittens, except possibly those born to queens recently recovered from the natural disease, have lost their maternally-derived antibody by 12 weeks of age.

Vaccination. Because of the high antigenicity of the virus, and the fact that there is only one serotype, vaccination against FP has been extremely successful where it has been carried out. Both modified live and inactivated systemic vaccines are available and have been reviewed by Povey (1973). Some outbreaks of disease do, however, still occur, but usually in situations where vaccination has lapsed or has not been carried out, and also in the occasional animal that for various reasons may be immunologically incompetent. Kittens tend to become infected at the stage at which they lose their passive immunity. Unfortunately, once one clinical case has occurred, unless adequate disinfection is carried out, the environment will be heavily contaminated with infected discharges, and any cat or kitten with inadequate immunity is a likely target for infection.

Generally, however, FP is a disease which man has been relatively successful in conquering, despite its highly infectious nature and despite the ability of the virus to persist for long periods in the environment and, to a lesser extent, in the cat.

FELINE VIRAL RHINOTRACHEITIS (FVR) AND FELINE CALICIVIRAL DISEASE (FCD)

These two diseases constitute the major causes of respiratory disease in the cat. Because they have many similarities with respect to their epidemiology they will be considered together.

Agent. FVR is caused by a herpesvirus, felid herpesvirus 1. As far as is known there is only one serotype of this virus and it is of reasonably uniform pathogenicity in a susceptible cat (Crandell *et al.*, 1961; Gaskell & Wardley, 1978). Although there is only one main feline calicivirus (FCV) serotype there are a number of different strains which vary slightly antigenically and which are of varying pathogenicity (Povey & Hale, 1974; Hoover & Kahn, 1975).

Infectivity. Both viruses are highly infectious to the susceptible cat. Respiratory disease tends to appear wherever cats are congregated together, infection often being introduced by the clinically normal carrier. Once present in a colony, the disease rapidly becomes endemic, its presence being noted by the existence of chronically affected animals with recurrent or persistent signs. Outbreaks of acute disease may also occur, particularly in young kittens.

There is experimental evidence for FVR that the level of the infecting dose of virus may play a role in determining the length of the incubation period and the severity of the resultant syndrome (Gaskell & Povey, 1979): Duration of contact has also been shown to be important in the transmission of this disease, particularly with shedding carriers where the discharges are not so copious (Gaskell & Povey, 1982). Similar suggestions have been made for FCD (Wardley, 1976).

Source of virus. In infected cats, both viruses are present in large amounts in the secretions of the upper respiratory tract; in the copious ocular, nasal and oral discharges. FCV is also shed occasionally in urine and faeces (Rich & Fabricant,

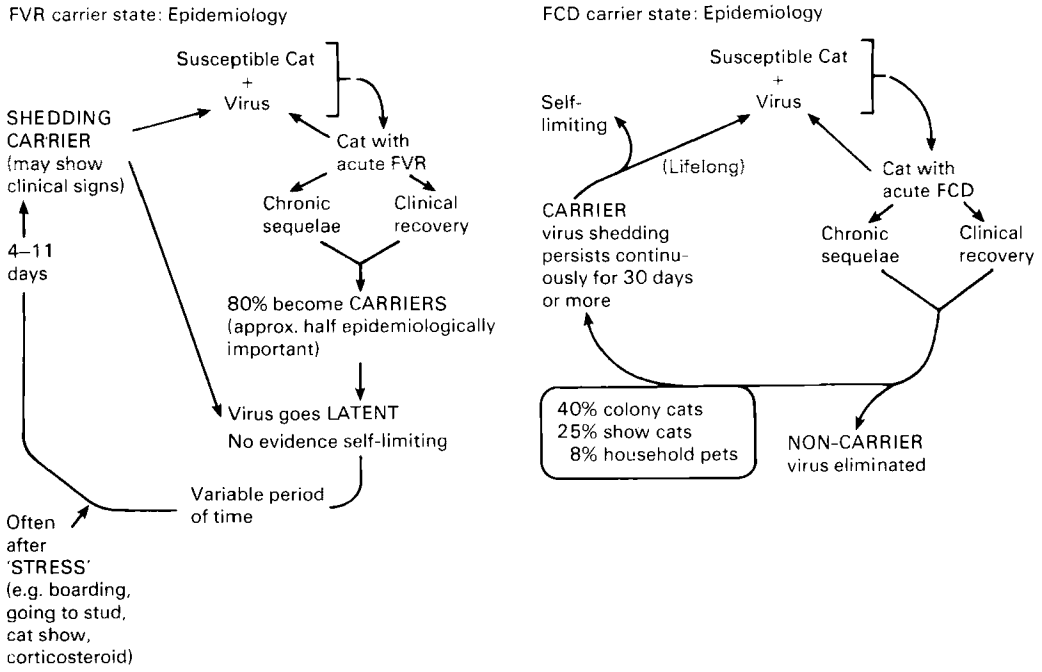

FIG. 2. Persistence of the feline respiratory viruses in the cat population.

1969; Povey & Hale, 1974), but it is probable that this is not of major epidemiological significance.

Mode of transmission. The major mode of transmission of the respiratory viruses is via the intranasal, intra-oral and conjunctival routes. Limited experimental evidence suggests that transplacental infection of FVR virus probably does not occur following natural routes of infection, although it can be induced following intravenous inoculation of virus (Hoover & Griesemer, 1971). For FCD, however, where generalization of the virus infection is far more common, it is possible that the virus may on occasions cross the placenta; occasionally, FCV has been isolated from an aborted foetus (Gaskell & Wardley, 1974; Ellis, 1981).

Viral persistence. Unlike FP, where because of the stability of the virus outside the host, a contaminated environment constitutes a major source of virus to the cat, the feline respiratory viruses are relatively fragile outside the cat (FVR virus surviving for <18 hours, FCV <8–10 days, depending on the temperature and relative humidity) and are susceptible to heat, drying and most common disinfectants (Povey & Johnson, 1970; Scott, 1980). They must therefore rely heavily for their continued survival on their ability to survive inside the cat. They do this in two ways (Fig. 2). First, by spreading from acutely infected clinical cases to susceptible cats, as outlined for FP. Secondly, they both have the ability to induce an immune carrier state in recovered animals (Gaskell & Wardley, 1978). Such animals are of great epidemiological importance, not only because they are undoubtedly infectious to in-contact cats but because the carrier state in both these diseases is a very widespread phenomenon. Studies have shown that at least 80 per

cent of FVR-recovered cats remain as viral carriers (Gaskell & Povey, 1973, 1977). In colonies where FCV is endemic, up to 40 per cent of the animals have been shown to be viral carriers (Wardley *et al.*, 1974).

The nature of these persistent infections is different in the two diseases. In FVR, the carrier state is characterized by a latent phase, with only intermittent episodes of virus shedding, often preceded by a natural 'stress', such as re-housing, or possibly kittening and lactation, or artificially, by corticosteroid treatment (Gaskell & Povey, 1973, 1977). The carrier state for FCV, however, is characterized by more-or-less continuous virus shedding and except for 'low-level' virus excretors, infectious virus can nearly always be detected (Wardley, 1976).

Immunity. The levels and duration of immunity to the two diseases are not well documented, though some data have been published to show that following vaccination, immunity to FVR and FCD may last up to a year (Bittle & Rubic, 1974, 1975). In both diseases, although the level of immunity is reasonable in the majority of cats during this time, protection may not be entirely complete in all animals (Gaskell, 1981).

There is only limited information on the duration of passive immunity in kittens born to recovered queens; in FVR it has been shown to persist from 2–10 weeks, with mean levels falling below detectable levels by 6–9 weeks of age (Edwards *et al.*, 1977; Gaskell & Povey, 1982); there is very little information about FCD, but Povey (1977) suggests that it may be more persistent than in FVR; up to, or beyond, 11 weeks.

Vaccination. Vaccines have been moderately successful in controlling feline respiratory disease in the majority of healthy previously unexposed animals. Both modified live and inactivated systemic vaccines are available and also modified live vaccines given by the intranasal route. However, there may be some problems associated with trying to control feline respiratory disease through vaccination alone. This has been reviewed in more detail elsewhere (Gaskell, 1981), but basically problems arise because the viruses are highly infectious and widespread in the population, and they persist for a long time in infected cats as a high proportion of animals remain as viral carriers. Despite vaccination it is probable that such animals can still be a source of infectious virus. Furthermore, for both FVR and FCD there is evidence that a previously unexposed cat vaccinated intramuscularly may subsequently become a virulent field virus carrier following challenge, without ever having shown any clinical signs (Orr *et al.*, 1978; Gaskell *et al.*, 1982). Such animals are clearly of epidemiological importance and could initiate outbreaks in young kittens, at the stage at which they lose their passive immunity, in colonies where the disease is endemic. Thus vaccination should be regarded as protection against disease for the individual rather than protection against infection. There is some evidence that the intranasal vaccination of previously unexposed animals may protect against the establishment of a field virus carrier state, at least in the short term, though it is not clear from these studies if such animals could withstand repeated challenge from field virus after longer periods of time (Orr *et al.*, 1980).

FELINE LEUKAEMIA VIRUS (FeLV) INFECTION

Agent. FeLV is a member of the Retrovirus family and the oncornavirus genus. Three subgroups are recognized, A, B and C (Sarma & Log, 1973; Jarrett, 1980).

Infectivity. In contrast to FP and the respiratory viruses, FeLV appears to be somewhat less infectious to a susceptible cat. However, the infectivity of the agent, or perhaps more accurately the outcome of infection, appears to be related to the infecting dose of virus and the duration of contact between the animals (Jarrett, 1979). Thus, free range cats, which are probably only transiently exposed to a low dose of infecting virus, tend to become immunized to FeLV and rarely become persistently infected or develop FeLV-related diseases. In closed colony cats, however, susceptible animals are often in prolonged, intimate contact with excreting cats and thus the dose of virus to which they are exposed is high. Although the majority of animals in such a situation do also recover from the infection, 30–40 per cent of them do not manage to eliminate the virus, become persistently viraemic, and have a high risk of developing FeLV-related diseases (Jarrett, 1979). One might speculate that if man had not interfered with the lifestyle of cats and forced them into high-risk closed colonies, only transient infections would tend to occur and we might not now see so many cats with FeLV-related diseases.

Finally, there is evidence that the infectivity of FeLV, based on ease of natural transmission and the outcome of infection may be to some extent determined by the virus subgroup (Jarrett, 1980).

Source of virus After virus has gained entry into the cat, it replicates locally in lymph nodes and in the tonsils (Rojko *et al.*, 1979). If the cat does not mount an adequate immune response at this point, virus spreads to the bone marrow and then the blood. It then spreads to various epithelial sites, such as the intestinal lamina propria, the salivary glands and the respiratory mucous membranes. Thus it is mainly shed in saliva and oropharyngeal secretions and it may also be shed, although in lower concentrations, in urine, milk and, possibly, faeces (Hardy, 1980).

Mode of transmission. The virus can be transmitted horizontally or congenitally (Hardy, 1980). Horizontal transmission may occur between related or unrelated cats, but in a group of cats, transmission will be achieved much more effectively between animals that socialize. Animals may be infected through mutual grooming, and the sharing of feeding bowls and litter trays. Infection is generally achieved via the intranasal, ocular or oral mucous membranes. Since FeLV is commonly found in peripheral blood, infection may on occasions be transmitted by biting insects, such as fleas, or by blood transfusion.

Congenital transmission across the placenta has also been reported in FeLV (Hardy, 1980; Hardy *et al.*, 1976), virus having been found in several litters of unborn foetuses, or newborn kittens, and in the uterus of viraemic pregnant queens. There is no evidence that transmission can occur genetically, i.e. via the host cell genome.

Viral persistence. Most cats undergoing FeLV infection become transiently

viraemic, mount an immune response and eliminate virus. This is usually followed by the appearance of virus neutralizing (VN) antibodies. There is recent evidence that such transiently infected animals could be part of the transmission cycle of FeLV amongst free range cats (Jarrett *et al.*, 1982). Some cats, however, become overwhelmed by the virus and develop a persistent viraemia; some of these mount an immune response (antibodies to feline oncornavirus-associated cell membrane antigen: anti-FOCMA antibodies) that protects them against the development of lymphosarcoma (though not against other FeLV-related diseases), while others do not. Both groups of persistently infected cats are of great significance in the epidemiology of the disease, for they serve as a constant source of infectious virus. There is some evidence that there may be a persistent latent infection in some FeLV recovered animals which may be activated by large doses of corticosteroids or detected *in vitro* by cultivation of explanted bone marrow cells (Post & Warren, 1980; Rojko *et al.*, 1982). It is not clear if this latent infection has any impact on the epidemiology of FeLV, but there is some recent evidence to suggest that it may not (Madewell & Jarret, 1983).

In the natural environment, FeLV is a rather labile virus, and so it does need to produce these persistent infections in cats in order to survive. Although it can survive for up to 2–3 days in a moist environment such as tissue culture fluid without cells, it loses its infectivity in only 2–3 minutes when it dries out (Hardy, 1980). It can withstand freezing but is susceptible to commonly used detergents and disinfectants, such as hypochlorite.

Immunity. The successful development of an active immunity to FeLV is a complicated phenomenon and depends on many factors, including the infecting virus dose and the virus subgroup, as discussed previously. In addition there are host factors, such as the age of the cat on exposure and possibly the genotype, that may also influence the outcome of infection.

Thus older cats appear to be inherently more capable of mounting sufficient immune response to eliminate the virus, whereas infection *in utero* or in the neonatal period always results in persistent infection and a very high risk of developing FeLV diseases (Hoover *et al.*, 1976; Jarrett, 1979). From approximately 12–16 weeks of age there is a transitional period in which some kittens become persistently infected but others become immune.

In Britain, the Abyssinian breed appears to have a greater incidence of FeLV infection on random testing, though there may be reasons other than genotype for this (Jarrett, 1979). Finally there may be other as yet unidentified factors which may influence the host's immune response and determine the response to infection.

Cats which are unsuccessful at mounting an immune response that kills the virus, stay viraemic and an unfortunate side effect of persistent infection with FeLV is immunosuppression. Hardy (1980) suggested that there are three likely mechanisms responsible for this immunosuppression: (1) FeLV-induced lymphopenia and granulocytopenia; (2) FeLV p15E inhibition of blast transformation, and (3) the formation of immunosuppressive FeLV immune complexes.

Finally there is little information on the duration of immunity in FeLV recovered cats. Although it is generally presumed to persist for an indefinite period, it has also been suggested that in some transiently infected cats immunity so produced is short lived and the cats may become susceptible to reinfection (Jarrett *et al.*, 1982). Jarrett (1979) has also observed a possible recall phenomenon for virus neutralizing antibodies from previously undetectable levels in animals with evidence of previous exposure to FeLV.

Passively transferred, maternal antibody (both anti-FOCMA and VN antibodies) to FeLV, has been shown to occur and may persist for up to two months in some cases (Essex *et al.*, 1971; Hoover *et al.*, 1977; Jarrett *et al.*, 1977).

Vaccination. Vaccines are still in the experimental stage. Several research groups, including genetic engineers attempting to synthesize polypeptides with the correct antigenic determinants, are experimenting with FeLV envelope sub-unit vaccines free of viral RNA. The problem has been, basically, that live FeLV vaccines are for a number of reasons too dangerous to be licensed, and killed FeLV vaccines are only poorly immunogenic (Olsen *et al.*, 1979; Hardy, 1980). Nevertheless, the outlook is hopeful.

FELINE INFECTIOUS PERITONITIS (FIP)

Agent. The virus causing feline infectious peritonitis (FIP) is classified as a coronavirus (Horzinek & Osterhaus (1979a). In the U.S.A. other isolates of feline coronavirus, known as feline enteric coronaviruses (FECV) have been recently identified that appear to be indistinguishable morphologically and antigenically by present tests from FIP virus. However, FECV produces an inapparent or mild enteric infection in kittens and does not appear to be the cause of either effusive or non-effusive FIP (Pedersen *et al.*, 1981a; Pedersen *et al.*, 1981b). FIP virus is also closely related to canine coronavirus, porcine TGE virus and a human coronavirus 229E (Reynolds *et al.*, 1977; Pedersen *et al.*, 1978).

Infectivity. The natural infectivity of FIP virus is difficult to assess, mainly because of the recent realization that antibodies to FIP virus cross react with those to FECV (Pedersen, 1981). In FIP problem catteries the disease incidence is usually relatively low (5–10 per cent) (Pedersen, 1976a) but until recently it was assumed that most of the animals in such colonies had been exposed to the virus because of the high proportion of cats with coronavirus antibody (Pedersen, 1976b; Loeffler *et al.*, 1978; Horzinek & Osterhaus, 1979b). However, until a serological test is developed that can distinguish antibodies to FIP and FECV, it is difficult to assess the infectivity of FIP under natural conditions.

Under experimental conditions, the infectivity of FIP virus appears to depend on a number of factors, some of which remain to be elucidated. In general, however, and as assessed experimentally, the infectivity of FIP virus is not very high. Thus a proportion of experimentally infected animals often fail to become infected, as assessed by absence of seroconversion and disease, though to some extent this depends on the route of inoculation. Thus Pedersen and his co-workers (1981a)

have noted a greater infectivity of tissue-culture propagated virus when administered intraperitoneally rather than on to a respiratory or oral mucosal surface. Other workers, however, have observed an apparently higher infectivity via the oral route (Evermann *et al.*, 1981). Such discrepancies may also be due to other factors, such as biological variation between isolates and perhaps some degree of attenuation on passage. It is possible that the infecting dose of virus may also play a role (Pedersen & Black, 1983). Finally, infectivity appears to depend to some extent on the immune status of the host; successful infection occurs more readily in animals with pre-existing antibody to coronavirus than in sero-negative animals (Pedersen & Boyle, 1980; Pedersen *et al.*, 1981a; Weiss & Scott, 1981).

Source of virus. FIP virus is present in the blood, peritoneal and pleural exudates, tissues and perhaps, on occasion, in the urine of infected cats (Pedersen, 1976a; Hardy & Hurvitz, 1971). It is not known if it is shed in the respiratory, conjunctival or oral secretions or in the faeces of infected cats. Unlike the other feline viruses discussed previously, initial isolation of FIP virus in tissue culture requires the use of sensitive coculture and explant techniques, and thus virus shedding, for example in oro-nasal secretions or faeces, would be difficult to monitor by routine swabbing techniques.

Mode of transmission. Although it is evident, from the sporadic but continuous losses in colonies with FIP, that horizontal transmission of FIP does occur, the natural route of transmission is unknown. Experimentally, virus has been transmitted by a variety of parenteral routes including subcutaneous, intramuscular, intravenous and intraperitoneal routes (Pedersen, 1976a; Evermann *et al.*, 1981) and also via the oral and respiratory routes (Pedersen *et al.*, 1981a; Evermann *et al.*, 1981; Weiss & Scott, 1981). Because the virus is present in the blood (Weiss & Scott, 1981), and is infectious by parenteral routes, transmission by blood sucking insects such as fleas may be possible (Pedersen, 1976a). It has also been suggested from epidemiological observations that transplacental transmission might perhaps occur (Pastoret & Henroteaux, 1978), but there is no definitive evidence to support this.

Viral persistence. The low incidence (1 per cent) of FIP in the general cat population (Pedersen, 1981) makes it unlikely that virus is perpetuated by successive spread from acute clinical cases to susceptible animals, unless there is a high incidence of sub-clinical infection. In some catteries, where in rare cases morbidity can approach 25 per cent or more in the first 12 months (Pedersen, 1976a), it is possible that continuous horizontal spread is responsible for perpetuating the virus.

Immune carrier cats have been postulated but not identified in FIP (Pedersen, 1981). Experimentally, most animals either fail to become infected with the virus or die of FIP (Pedersen *et al.*, 1981a). Occasional animals show seroconversion without clinical disease and these animals presumably are the likely carriers (Pedersen, 1981). Perhaps a situation exists, as in FeLV infection, whereby more persistent infections are created in susceptible animals in a pre-existing endemic

colony situation because of continuous exposure to shedding carriers. Stress may play a rôle, perhaps in activating latent infections, as has been seen for both FVR and FeLV (Gaskell & Povey, 1973, 1977; Post & Warren, 1980).

FIP virus is rather labile and thus the environment is unlikely to be a long-term source of virus to the susceptible cat. It is inactivated within one day at room temperature whether it is dried or kept moist, and it is destroyed rapidly by heat and most commonly used disinfectants, although it is surprisingly resistant to phenol (Pedersen, 1976a, b).

Immunity. As discussed previously, the development of an active immunity in FIP is in most cases self-destructive. In most experimental infections, serum antibodies appear at the same time as the clinical disease and there is evidence for the disease being immune-mediated (Jacobse-Geels *et al.*, 1980; Weiss *et al.*, 1980). However, in a small proportion (20 per cent or less) of cases antibody, which is presumably protective, develops and there is no clinical disease (Pedersen *et al.*, 1981a).

As mentioned before, pre-existing antibody to feline coronavirus appears to increase the susceptibility of the animal to FIP and this is also true for passively transferred antibody (Pedersen & Boyle, 1980; Weiss & Scott, 1981; Pedersen *et al.*, 1981b). As yet, there is no information on either the existence, duration or significance of passive immunity in kittens born to FIP immune queens.

Vaccination. Since the discovery that FIP virus could be grown in conventional tissue culture (O'Reilly *et al.*, 1979; Hitchcock *et al.*, 1981) there has been an interest in the possibility of vaccination. However, because pre-existing antibody, even that induced by vaccination, appears to enhance the disease (Pedersen & Black, 1983), it is unlikely that a successful vaccine will be developed without a more complete understanding of the pathogenesis.

REFERENCES

General reviews

GASKELL, C.J. & GASKELL, R.M. (1980) Respiratory diseases of cats. *In practice*, **2** (6), 5.

GASKELL, R.M. & WARDLEY, R.C. (1978) Feline viral respiratory disease: a review with particular reference to its epizootiology and control. *J. small Anim. Pract.* **19**, 1.

GILLESPIE, J.H. & SCOTT, F.W. (1973) Feline viral infections. *Adv. vet Sci. Comp. Med.* **17**, 163.

HARDY, W.D. (1980) (1) Feline leukemia virus diseases and (2) The virology, immunology and epidemiology of the feline leukemia virus. In: *Feline Leukemia Virus* (eds. W. D. Hardy Jr., M. Essex and A. J. McClelland), pp. 3 and 33. Elsevier North Holland Inc., New York.

HORZINEK, M.C. & OSTERHAUS, A.D.M.E. (1978) Feline infectious peritonitis: a coronavirus disease of cats. *J. small Anim. Pract.* **19**, 623.

JARRETT, O. (1979) Feline leukaemia virus. *In practice*, **1** (2), 15.

MACKEY, L. (1975) Feline leukaemia virus and its clinical effects in cats. *Vet. Rec.* **96**, 5.

PEDERSEN, N.C. (1981) *Feline Infectious Diseases*, p. 83 Proc. 48th Amer. Anim. Hosp. Ass.

POVEY, R.C. (1976) Viral diseases of cats: current concepts. *Vet Rec.* **98**, 293.

SCOTT, F.W. (1980) Virucidal disinfectants and feline viruses. *Am. J. Vet Res.* **41**, 410.

Feline panleucopenia

BOUILLANT, A. & HANSON, R.P. (1965) Epizootiology of mink enteritis III. Carrier state in mink. *Can. J. comp. Med. vet. Sci.* **29,** 183.

CSIZA, C.K., SCOTT, F.W., DELAHUNTA, A. & GILLESPIE, J.H. (1971a) Pathogenesis of feline panleukopenia virus in susceptible newborn kittens. I. Clinical signs, haematology, serology and virology. *Infect. Immun.* **3,** 833.

CSIZA, C.K., SCOTT, F.W., DELAHUNTA, A. & GILLESPIE, J.H. (1971b) Pathogenesis of feline panleukopenia virus in susceptible newborn kittens. II. Pathology and immunofluorescence. *Infect. Immun.* **3,** 838.

CSIZA, C.K., SCOTT, F.W., DELAHUNTA, A. & GILLESPIE, J.H. (1971c) Immune carrier state of feline panleukopenia virus-infected cats. *Am. J. vet. Res.* **32,** 419.

CSIZA, C.K., DELAHUNTA, A., SCOTT, F.W. & GILLESPIE, J.H. (1972) Spontaneous feline 'ataxia'. *Cornell vet.* **62,** 300.

JOHNSON, R.H. (1964) Isolation of a virus from a condition simulating feline panleucopaenia in a leopard. *Vet. Rec.* **76,** 1008.

JOHNSON, R.H. (1966) Feline panleucopaenia virus. III. Some properties compared to a feline herpes virus. *Res. vet. Sci.* **7,** 112.

JOHNSON, R.H. (1969) Feline panleucopaenia. *Vet. Rec.* **84,** 338.

KILHAM, L., MARGOLIS, G. & COLBY, E.D. (1967) Congenital infections of cats and ferrets by feline panleukopenia virus manifested by cerebellar hypoplasia. *Lab. Invest.* **17,** 465.

REIF, J.S. (1976) Seasonality, natality and herd immunity in feline panleukopenia. *Am. J. Epid.* **103,** 81.

O'REILLY, K.J. & HITCHCOCK, L.M. (1976) Persistence of antibody to feline panleucopaenia induced by a modified live virus vaccine *J. small Anim. Pract.* **17,** 549.

POVEY, R.C. (1973) Feline panleukopenia—which vaccine? *J. small Anim. Pract.* **14,** 399.

SCOTT, F.W., CSIZA, C.K. & GILLESPIE, J.H. (1970) Maternally derived immunity to feline panleukopenia. *J. Am. vet. med. Ass.* **156,** 439.

TRATSCHIN, J.D., MCMASTER, G.K., KRONAUER, G. & SIEGL, G. (1982) Canine parvovirus: relationship to wild-type and vaccine strains of feline panleukopenia virus and mink enteritis virus. *J. gen. Virol.* **61,** 33.

VERGE, J. & CRISTOFORONI, N. (1928) La gastroenterite infectieuse des chats est elle due a un virus filtrable? *C. r. Séanc. Soc. Biol. (Paris),* **99,** 312.

Feline viral rhinotracheitis and feline caliciviral disease

BITTLE, J.L. & RUBIC, W.J. (1974) Studies of feline viral rhinotracheitis vaccines. *Vet. Med. small Anim. Clin.* **69,** 1503.

BITTLE, J.L. & RUBIC, W.J. (1975) A feline calicivirus vaccine combined with feline viral rhinotracheitis and feline panleucopenia vaccine. *Feline Pract.* **5** (6), 13.

CRANDELL, R.A., REHKEMPER, J.A., NIEMANN, W.H., GANAWAY, J.R. & MAURER, F.D. (1961) Experimental feline viral rhinotracheitis. *J. Am. vet. Med. Ass.* **138,** 191.

EDWARDS, B.G., BUELL, D.J. & ACREE, W.M. (1977) Evaluation of a new feline rhinotracheitis vaccine. *Vet. med small Anim. Clin.* **72,** 205.

ELLIS, T.M. (1981) Jaundice in a Siamese cat with *in utero* feline calicivirus infection. *Aust. vet. J.* **57,** 383.

GASKELL, C.J., GASKELL, R.M., DENNIS, P.E. & WOOLDRIDGE, M.J.A. (1982) Efficacy of an inactivated feline calicivirus (FCV) vaccine against challenge with United Kingdom field strains and its interaction with the FCV carrier state. *Res. vet. Sci.* **32,** 23.

GASKELL, R.M. (1981) An assessment of the use of feline respiratory virus vaccines. In: *The Vet. Annual,* 21st issue (Eds. C.S.G. Grunsell and F.W.G. Hill), p. 267. Scientechnica, Bristol.

GASKELL, R. M. & POVEY, R.C. (1973) Re-excretion of feline viral rhinotracheitis virus following corticosteroid treatment. *Vet. Rec.* **93,** 204.

GASKELL, R.M. & POVEY, R.C. (1977). Experimental induction of feline virual rhinotracheitis virus re-excretion in FVR-recovered cats. *Vet. Rec.* **100,** 128.

GASKELL, R.M. & POVEY, R.C. (1979) The dose response of cats to experimental infection with feline viral rhinotracheitis. *J. comp. Path. Ther.* **89,** 179.

GASKELL, R.M. & POVEY, R.C. (1982) Transmission of feline viral rhinotracheitis. *Vet. Rec.* **111,** 359.

GASKELL, R.M. & WARDLEY, R.C. (1974) Unpublished observations.

HOOVER, E.A. & GRIESEMER, R.A. (1971) Experimental feline herpesvirus infection in the pregnant cat. *Am. J. Path.* **65,** 173.

HOOVER, E.A. & KAHN, D.E. (1975) Experimentally induced feline calicivirus infection: clinical signs and lesions. *J. Am. vet. med. Ass.* **166,** 463.

ORR, C.M., GASKELL, C.J. & GASKELL, R.M. (1978) Interaction of a combined feline viral rhinotracheitis–feline calicivirus vaccine and the FVR carrier state. *Vet. Rec.* **103,** 200.

ORR, C.M., GASKELL, C.J. & GASKELL, R.M. (1980) Interaction of an intranasal combined feline viral rhinotracheitis, feline calicivirus vaccine and the FVR carrier state. *Vet. Rec.* **106,** 164.

POVEY, R.C. (1977) Feline respiratory disease—which vaccine? *Feline Pract.* **7** (5), 12.

POVEY, R.C. & HALE, C.J. (1974) Experimental infections with feline caliciviruses (picornaviruses) in specific pathogen-free kittens. *J. comp. Path. Ther.* **84,** 245.

POVEY, R.C. & JOHNSON, R.H. (1970) Observations on the epidemiology and control of viral respiratory disease in cats. *J. small Anim. Pract.* **11,** 485.

RICH, L.J. & FABRICANT, C.G. (1969) Urethral obstruction in male cats: transmission studies. *Can. J. comp. Med.* **33,** 164.

WARDLEY, R.C. (1976) Feline calicivirus carrier state. A study of the host/virus relationship. *Arch. Virol.* **52,** 243.

WARDLEY, R.C., GASKELL, R.M. & POVEY, R.C. (1974) Feline respiratory viruses: their prevalence in clinically healthy cats. *J. small Anim. Pract.* **15,** 579.

Feline leukaemia

ESSEX, M., KLEIN, G., SNYDER, S.P. & HARROLD, J.B. (1971) Antibodies to feline oncornavirus-associated cell membrane antigen in neonatal cats. *Int. J. Cancer* **8,** 384.

HARDY, W.D., JR., MESS, P.W., MACEWAN, E.G., MCCLELLAND, A.J., ZUCKERMANN, E.E., ESSEX, M., COTTER, S.M. & JARRETT, O. (1976) Biology of feline leukemia virus in the natural environment. *Cancer Res.* **36,** 582.

HOOVER, E.A., SCHALLER, J.P., MATHES, L.E. & OLSEN, R.G. (1977) Passive immunity to feline leukemia: evaluation of immunity from dams naturally infected and experimentally vaccinated. *Infect. Immun.* **16,** 54.

HOOVER, E.A., OLSEN, R.G., HARDY, W.D. JR., SCHALLER, J.P. & MATHES, L.E. (1976) Feline leukemia virus infection: age-related variation in response of cats to experimental infection. *J. natn. Cancer Inst.* **57,** 365.

JARRET, O. (1980) Feline leukemia virus subgroups. In: *Feline Leukemia Virus* (Eds. W. D. Hardy, Jr., M. Essex and A. J. McClelland), p. 473. Elsevier North Holland Inc., New York.

JARRETT, O., GOLDER, M.C. & STEWART, M.F. (1982) Detection of transient and persistent feline leukaemia virus infections. *Vet. Rec.* **110,** 225.

JARRETT, O., RUSSELL, P.H. & STEWART, M.F. (1977) Protection of kittens from feline leukaemia virus infection by maternally-derived antibody. *Vet. Rec.* **101,** 304.

MADEWELL, B.R. & JARRETT, O. (1983) Recovery of feline leukaemia virus from non-viraemic cats. *Vet. Rec.* **112,** 339.

OLSEN, R.G., HOOVER, E.A., MATHES, L.E., SCHALLER, J.P. & LEWIS, M.G. (1979) Immunoprevention of feline leukemia. *Feline Prac.* **9** (4), 16.

POST, J.E. & WARREN, L. (1980) Reactivation of latent feline leukemia virus. In: *Feline Leukemia Virus* (Eds. W.D. Hardy, Jr., M. Essex and A. J. McClelland), p. 151. Elsevier North Holland Inc., New York.

ROJKO, J.L., HOOVER, E.A., QUACKENBUSH, S.L. & OLSEN, R.G. (1982) Reactivation of latent feline leukaemia virus infection. *Nature*, **298**, 385.

ROJKO, J.L., HOOVER, E.A., MATHES, L.E., OLSEN, R.G. & SCHALLER, J.P. (1979) Pathogenesis of experimental feline leukemia virus infection. *J. Natn. Cancer Inst.* **63**, 759.

SARMA, P.S. & LOG, T. (1973) Subgroup classification of feline leukemia and sarcoma viruses by viral interference and neutralization tests. *Virol.* **54**, 160.

Feline infectious peritonitis

EVERMANN, J.F., BAUMGARTENER, L., OTT, R.L., DAVIS, E.V. & MCKEIRNAN, A.J. (1981) Characterization of a feline infectious peritonitis virus isolate. *Vet. Pathol.* **18**, 256.

HARDY, W.D. & HURVITZ, A.J. (1971) Feline infectious peritonitis: experimental studies. *J. Am. vet. med. Ass.* **158**, 994.

HITCHCOCK, L.M., O'REILLY, K.J. & BEESLEY, J.E. (1981) In vitro culture of feline infectious peritonitis. *Vet. Rec.* **108**, 535.

HORZINEK, M.C. & OSTERHAUS, A.D.M.E. (1979a) The virology and pathogenesis of feline infectious peritonitis. *Arch. Virol.* **59**, 1.

HORZINEK, M.C. & OSTERHAUS, A.D.M.E. (1979b) Feline infectious peritonitis. A world wide serosurvey. *Am. J. vet. Res.* **40**, 1487.

JACOBSE-GEELS, H.E.L., DAHA, M.R. & HORZINEK, M.C. (1980) Isolation and characterisation of feline C3 and evidence for the immune complex pathogenesis of feline infectious peritonitis. *J. Immun.* **125**, 1606.

LOEFLER, D.G., OTT, R.L., EVERMANN, J.F. & ALEXANDER, J.E. (1978) The incidence of naturally occurring antibodies against feline infectious peritonitis in selected cat populations. *Feline Pract.* **8** (1), 43.

O'REILLY, K.J., FISHMAN, B. & HITCHCOCK, L.M. (1979) Feline infectious peritonitis: isolation of a coronavirus. *Vet. Rec.* **104**, 348.

PASTORET, P.-P. & HENROTEAUX, M. (1978) Epigenetic transmission of feline infectious peritonitis. *Comp. Immun. Microbiol. infect. Dis.* **1**, 67.

PEDERSEN, N.C. (1976a) Feline infectious peritonitis: something old, something new, *Feline Pract.* **6** (3), 42.

PEDERSEN, N.C. (1976b) Serologic studies of naturally occurring feline infectious peritonitis. *Am. J. vet. Res.* **37**, 1449.

PEDERSEN, N.C. (1981) *Feline Infectious Diseases*, p. 83. Proc. 48th Amer. Anim. Hosp. Ass.

PEDERSEN, N.C. & BLACK, J.W. (1983) Attempted immunisation of cats against feline infectious peritonitis using either avirulent live virus or sublethal amounts of virulent virus. *In press.*

PEDERSEN, N.C. & BOYLE, J.F. (1980) Immunologic phenomenon in the effusive form of feline infectious peritonitis. *Am. J. vet. Res.* **41**, 868.

PEDERSEN, N.C., BOYLE, J.F. & FLOYD, K. (1981a) Infection studies in kittens. using feline infectious peritonitis virus propagated in cell culture. *Am. J. vet. Res.* **42**, 363.

PEDERSEN, N.C., WARD, J. & MENGELING, W.L. (1978) Antigenic relationship of the feline infectious peritonitis virus to coronaviruses of other species. *Arch. Virol.* **58**, 45.

PEDERSEN, N.C., BOYLE, J.F., FLOYD, K., FUDGE, A. & BARKER, J. (1981b) An enteric coronavirus of cats and its relationship to feline infectious peritonitis. *Am. J. vet. Res.* **42**, 368.

REYNOLDS, D.J., GARWES, D.J. & GASKELL, C.J. (1977) Detection of transmissible gastroenteritis virus neutralising antibody in cats. *Arch. Virol.* **55**, 77.

WEISS, R.C. & SCOTT, F.W. (1981) Pathogenesis of feline infectious peritonitis: nature and development of viraemia. *Am. J. vet. Res.* **42**, 382.

WEISS, R.C., DODDS, W.J. & SCOTT, F.W. (1980) Disseminated intravascular coagulation in experimentally induced feline infectious peritonitis. *Am. J. vet. Res.* **40**, 663.