

Book 187
Montali

Chapter 44

Parvoviruses

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For the family description, Parvoviridae please see p. 61.

Of the known parvoviruses that produce disease in animals, three are known to affect non-domestic carnivores. These include feline panleukopenia virus (FPV), mink enteritis virus (MEV), and canine parvovirus Type 2 (CPV). How these viruses affect various species is not always known. Much of the information has been gleaned from outbreaks of clinical disease in groups of animals while in captivity.

The purpose of this section will be to describe what is currently known about FPV and CPV in other than domestic carnivores. Mink virus enteritis will be alluded to but has been fully described in the section on infections of mustelids.

FELINE PANLEUKOPENIA VIRUS (FPLV)

INTRODUCTION

FPV has been reported as a naturally occurring disease in many members of captive Felidae. Disease entities similar to FPV have also been recorded in certain members of Procyonidae, Mustelidae, Viverridae, and Canidae (see Figure 132).

In Felidae, FPV has been documented in leopards (*Panthera pardus*) (Johnson, 1964), lions (*Panthera leo*) (Studdert et al., 1973), and tigers (Povey and Davis, 1977) with viral isolation (Johnson, 1969). In fact, FPV was first cultured from a leopard and methods for isolation and characterization of this virus have only been available since 1967 (Johnson, 1969). The disease has been reported in many other species of large and small non-domestic cats based mainly on clinical and/or pathological findings (Wolf and Swart, 1974), and it is likely that all members of Felidae are susceptible (Povey and Devis, 1977). Feline panleukopenia does not appear to occur in free-living felids, as the reservoir seems to be domestic cats.

Diseases clinically and pathologically reminiscent of panleukopenia have been reported in the raccoon (*Procyon lotor*) coatimundi (*Nasua nasua*; Johnson and Halliwell, 1968; Waller, 1940), kinkajou (*Potos flavus*; Miller, 1961), mink (Mustelidae), Arctic fox (*Alopex lagopus*; Phillips, 1943), and maned wolf (*Chrysocyon brachyurus*; Visee et al., 1974). The latter 2 cases in canids may have been due to CPV; however, they were reported well before parvovirus had been established as an entity in dogs (Eugster et al., 1978). FPV has usually not been isolated from most of these carnivores exhibiting naturally acquired parvovirus-like illness. While the causative agent in raccoons and coatimundi

(Johnson, 1969) may indeed be FPV, there is some evidence that MVE, a virus closely antigenically and serologically related (Carmichael et al., 1980; Tratschin et al., 1982) might also be incriminated, at least in the raccoon; the agent may even be a separate virus closely related to each of the FPV and MVE agents (Barker et al., 1983; Appel and Parrish, 1982).



VIRUS PROPERTIES

Isolates from non-domestic cats appear to be identical to FPV from domestic cats in most ways; however, differences now detectable with monoclonal antibodies may demonstrate subtle antigenic differences or 'drift' in virus isolated from different species.



EPIZOOTIOLOGY

FPV has caused major outbreaks in zoos and parks exhibiting exotic cats (Cockburn, 1947; Hyslop, 1955; Vetesi and Balsai, 1971; Sedgewick, 1971; Eulenberg et al., 1974; Studdert et al., 1973; Povey and Davis, 1977; Bittle, 1981). The source of the infection has usually been traced to direct or indirect contact with domestic cats, although in one episode in a group of young lions reversion to virulence of a modified-live vaccine given prior to the epizootic could not be ruled out (O'Reilly, 1971). Reliable reports of panleukopenia among animals in their natural habitats are lacking. A disease of such contagiousness and flamboyance could hardly be missed when appearing in a wild population. Earlier cases of 'cat distemper' said to have occurred in wild American and African tiger cats (*Felis tigrina* and *Felis aurata*), caracal (*Lynx caracal*) and ocelot (*Felis pardalis*), and leopard (*Leo pardus*; Hindle and Findlay, 1932) were interpreted as probable cases of 'viral respiratory tract' infections (Hyslop, 1955) known to be associated with herpes, calici, or chlamydial agents of domestic cats and reported in some exotic species (Kane and Boever, 1976; Bush et al., 1981).

As with domestic farm cats reared in isolated conditions, felids kept in zoos, sometimes represent naive populations, highly susceptible to FPV and prone to serious losses if the virus should make its way into the collections. This might occur via direct contact of infected domestic or feral cats with the exotic cats, or by contamination of their food source or bedding. Since infected cats develop viremia early on and excrete virus in their feces, urine and saliva, the virus can be easily spread to exhibits via fleas, fomites and personnel caring for the animals.

Some species of large cats thought to be resistant to FPV in one setting have suffered heavy losses in another setting. For example, in one early outbreak in a zoo (Cockburn, 1947), lions were said to be resistant whereas 18 lions succumbed to the disease in an outbreak at another park (Studdert et al., 1973). This points out that, in facilities with a mixture of exotic cats, some species may have developed active immunity from subclinical infections as occurs commonly in 'town cats'. (Johnson, 1969). This is supported by the finding of significant FPV antibody titers in some exotic cats that had no evidence of clinical disease or history of vaccination (Kane and Boever, 1976; Bush et al., 1981). It is therefore hazardous to generalize as to resistance or susceptibility of exotic cats to FPV without knowing the immune status of the animals in question.

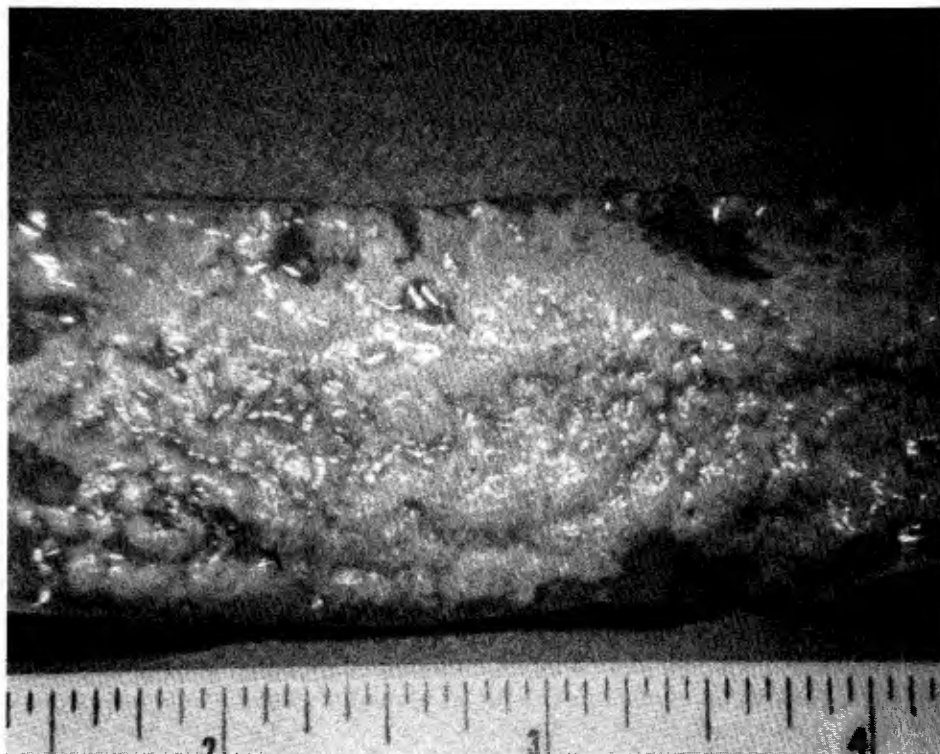


Fig. 133. Opened small intestine of a 6 month old jaguar with panleukopenia show fibrinous coating of mucosal surface.



PATHOGENESIS

Pathogenetic investigations have not been performed in non-domestic cats although it is presumed that the disease mechanisms are very similar to those in domestic cats. Usually experimental inoculation with virulent virus is not feasible in exotic cats due to difficulties with restraint and manipulability, because of their high value and out of sentimental considerations.



DISEASE SIGNS

The clinical signs described in outbreaks of FPV in exotic cats differ very little from those in domestic cats. These have included signs ranging from sudden death to acute forms characterized by anorexia, vomiting, depression and occasional diarrhea just before death. Some episodes were more protracted (> 2 days), with occasional animals recovering. A syndrome called 'star disease' has been observed in lion cubs and likened to FPV-induced cerebellar ataxia in kittens. The etiology of star disease was believed to be a viral infection in utero, but not proven to be FPV (Leclerc-cassan, 1981). Features unique to FPV infection in large cats were unsteady gait observed in affected lions (Studdert et al., 1973) and convulsions reported in some tigers (Cockburn, 1947). Total white blood counts of under 800–900/mm³ were seen in some lions.



PATHOLOGY

There are no detailed descriptions of gross and microscopic lesions of FPV in non-domestic cats, but, of those available, the changes seem comparable to

those in domestic cats. Grossly, hose-like changes in jejunum and ileum with yellow-brown fibrinous coatings of the mucosa (Fig. 133) have been described in lions (Studdert et al., 1973), with histological evidence of mucosal necrosis and lymphoid depletion in segments of the small and large intestine. Changes which characterize parvovirus infection in general, including intestinal crypt necrosis with regeneration and intranuclear inclusion bodies and bone marrow depletion have often served as supporting evidence for FPV in a variety of non-domestic felidae and other apparently susceptible carnivores mentioned above.



LABORATORY DIAGNOSIS

Since several known parvoviral agents produce similar clinical and pathological entities, with overlap in a variety of carnivore species, specific diagnosis should rely on the isolation of the causative agent and characterization using monoclonal antibodies and endonuclease patterns of the viral DNA (Parrish and Carmichael, 1983). Considerable difficulty exists because of the cross reactions of antibodies to the various parvoviruses affecting mammals; consequently, serum neutralization and hemagglutination-inhibition titers alone cannot be used to identify the virus. Hemagglutination tests (Carmichael et al., 1980) on feces from sick animals, however, are most useful in identifying parvoviruses, and along with the characteristic clinical and pathological features should serve at least to distinguish the disease from other enteropathic viral infections.



PROPHYLAXIS and CONTROL

Prevention of FPV infections in susceptible exotic species is dependent on a program that includes quarantine procedures, sanitation methods and vaccination. All new animals entering the collection should be quarantined for at least 30 d and monitored for any signs of parvovirus-like illnesses. Subgroups of carnivores known to be susceptible to any of the parvoviruses should never be mixed in quarantine or exhibit facilities. Care should be taken to prevent domestic cats, and where possible, feral animals from access to the facility grounds. Sanitation practices should include the use of 0.175% sodium hypochlorite, a recommended virucidal agent against FPV virus, for disinfection and footbath use (Scott, 1980).

Proper vaccination is of utmost importance in maintaining disease-free status in an exotic cat collection. Several regimens have been recommended (Theobald, 1978; Dinnes, 1980; Wallach and Boever, 1983); there remains some controversy as to whether modified-live vaccine can induce symptoms in certain feline species (Scott, 1979) or enhance the susceptibility of 'stressed' vaccinates to opportunistic infections (Ashton, 1980). Killed FPV vaccines have been used most successfully in exotic cats since the virus is highly antigenic and has proven to be quite efficacious in a variety of Felidae (Gray, 1972). A recent trial in 7 species of adult exotic cats employing a vaccine containing a killed FPV component prepared for domestic cats proved it to be highly efficacious and safe. Titers remained high (> 517) for 7–9 months; doubling or exceeding the 2 standard doses recommended for domestic cats did not enhance the immunity (Bush et al., 1981).

Another question is when to vaccinate young cats. Since the half-life of antibodies to FPV acquired passively in exotic neonatal Felidae is unknown and cannot necessarily be extrapolated (Dixon et al., 1952) from the half-life of

9.5 d determined for domestic kittens (Scott et al., 1970), the time to vaccinate cubs in order to avoid immune interference might be difficult to ascertain. In a group of servals (*Felis serval*; Povey and Davis, 1977) cubs from dams with FPV titers of ≥ 2048 succumbed to FPV at 14–16 weeks of age after having been vaccinated at 6–10 weeks of age.

Annual vaccination of breeding females should afford early protection of the offspring. Cubs should then be vaccinated periodically from 8 weeks, a regime that should not be completed before 16 weeks. Obtaining FPV titers from the dam and periodically from the cubs might be useful in guiding the vaccination procedure. Vaccination of species in families of carnivores other than Felidae that are prone to FPV should also be done with killed products.

CANINE PARVOVIRUS TYPE 2 (CPV)

INTRODUCTION

As soon as canine parvovirus infection emerged as a distinct entity in pet dogs in 1978 (Eugster et al., 1978; Gagnon and Povey, 1979), cases began to be reported in some non-domestic canids. Infections first occurred in 1978 in maned wolves (*Chrysocyon brachyurus*) at the San Antonio Zoo, Texas (Fletcher et al., 1979), followed by additional reports of CPV in maned wolves as well as other captive South American canids: Bush dogs (*Speothos venaticus*) and crab-eating foxes (*Cerdocyon thous*) at the National Zoo in Washington, D.C. (Mann et al., 1980; Janssen et al., 1982). Canine parvovirus was also reported in a group of captive coyotes in Washington state occurring simultaneously with coronavirus infection (Evermann et al., 1980). Outbreaks of CPV have also occurred in young raccoon dogs (*Nyctereutes procyonoides*) on fur farms in Finland (Neuvonen et al., 1982).

A parvovirus with agglutination properties different from those of CPV caused enteritis in a captive maned wolf colony in a zoo in Germany (Bieniek et al., 1981). Recently, an outbreak of a parvovirus illness in raccoons was reported to be CPV (Nettles et al., 1980); the virus was later shown to be more closely related to FPV (Appel and Parrish, 1982). Raccoons were subsequently found resistant to disease after experimental inoculation with CPV (Barker et al., 1983).



EPIZOOTIOLOGY

The pattern of CPV infections in non-domestic Canidae indicates that transmission most likely occurs from domestic dogs. The disease had not been reported in exotic Canidae in the United States prior to the dog epizootics that occurred there. All exotic cases reported so far have been in captive Canidae, and there is no evidence that CPV affects wild populations. Susceptibility of individual species of Canidae is known only for the few in which it has been documented.

In one outbreak of CPV infection in a zoo (Mann et al., 1980) the disease was lethal in maned wolves and bush dogs, whereas crab-eating foxes developed only minor symptoms. Foxes in another outbreak of CPV infection were also found resistant (Neuvonen et al., 1982); they might be inherently less susceptible. Once CPV occurs in an exotic collection, it may establish itself by its hardiness and persistence in the environment, or as a result of the periodic shedding by subclinically infected adults. This occurred in South American

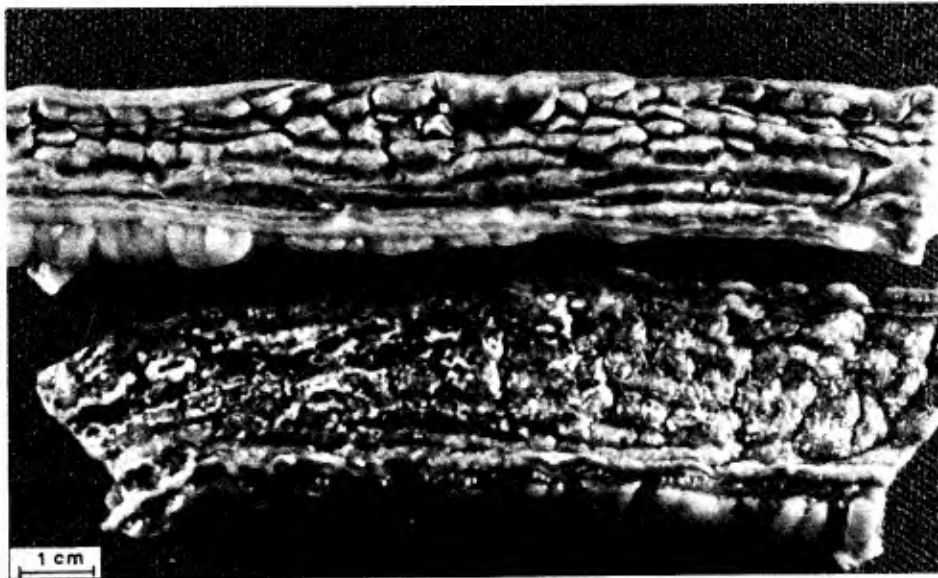


Fig. 134. Opened segments of small intestine from a bush dog with canine parvovirus infection show extensive mucosal necrosis and hemorrhages.

canids in which several outbreaks developed despite vaccination with killed CPV vaccine at various intervals after weaning (Janssen et al., 1982). Mixing wild canids with domestic dogs has led to the development of CPV infection in wild species: when wild coyote pups were housed together with mixed-bred dog pups for a parasite research project, 19 of 44 coyotes died of either CPV or a combination of CPV and canine coronavirus infection (Evermann et al., 1980). Major outbreaks of enteritis occurred in young raccoon dogs raised on fur ranches in eastern Finland in 1980. Mortality varied between 1–30% on 11 affected farms. The virus isolated from clinical cases was determined to be the canine type of parvovirus by comparative HI testing using CPV as the control antigen. Experimentally inoculated CPV obtained from a dog was infectious for adult blue foxes (*Alopex lagopus*) and raccoon dogs which seroconverted, but not for ferrets (Neuvonen et al., 1982).

Although total numbers of exotic canids reported to have acquired CPV are relatively low, the percent mortality in one study approached the 50% that has been recognized in susceptible dog populations (Evermann et al., 1980). Older animals seem less affected and good naturally acquired immunity with titers up to 5,120 has been recorded 6 weeks after recovery in several species of adult South American canids (Mann et al., 1980).

DISEASE SIGNS



Clinical signs vary from sudden death noted in coyotes and a maned wolf (Fletcher et al., 1979; Evermann et al., 1980) to anorexia, lethargy, vomiting and watery to hemorrhagic diarrhea as occurs in domestic dog. In one maned wolf, clinical signs developed 10 d after exposure (Mann et al., 1980); in multiple outbreaks affecting bush dog litters that had been vaccinated with killed vaccine, onset of illness in the pups usually occurred within 1–3 d of each other. The infection was always accompanied by severe leukopenia early in the course of the disease (Janssen et al., 1982). In one litter of bush dogs with CPV, *Campylobacter fetus* subspecies *jejuni* was isolated and this has also been found in association with parvovirus enteritis in dogs (Sandstedt and Weirup, 1981).

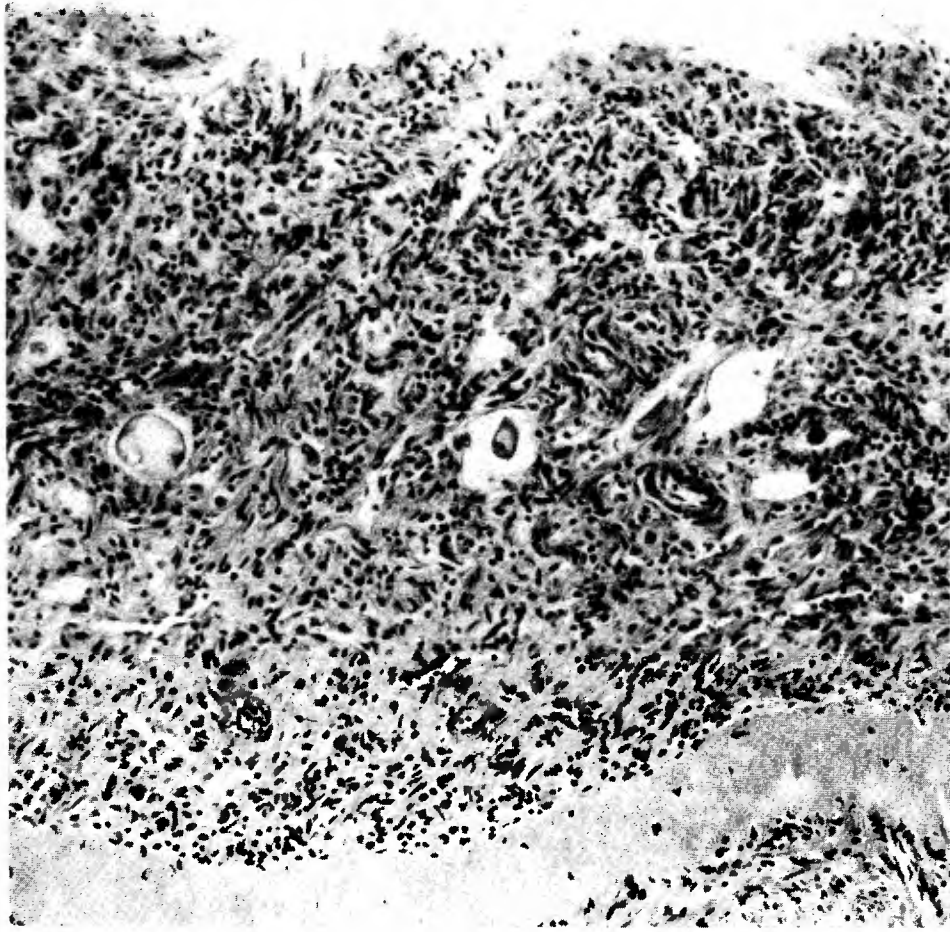


Fig. 135. Photomicrograph of small intestine of bush dog with canine parvovirus infection shows loss of villous epithelium and hyperplastic crypt enterocytes. Hematoxylin-eosin stain, X60.

In raccoon dog outbreaks, pups 3–5 weeks of age were the most affected and developed enteritis with vomiting and diarrhea of variable severity (Neuvonen et al., 1982). All in all, the clinical aspects of the disease as manifest in exotic canids and dogs do not differ very much. Sudden death associated with myocarditis, however, has not been documented in exotic carnivores.



PATHOLOGY

Gross and microscopic changes in the various canids in which CPV has occurred are similar to those previously described for the digestive tract and hematopoietic system of domestic dogs (Figs. 134, 135). The myocarditis that occurs in domestic pups up to 8 weeks of age (Bastianello, 1981) has not been described in exotic carnivores.



LABORATORY DIAGNOSIS

Methods employed for the identification of CPV in exotic Canidae include HA and HI on feces to identify viral antigen. Some investigators use 4 HA units

of CPV and pig red blood cells (Fletcher et al., 1979), whereas others prefer 8 HA units, Rhesus monkey RBC's and FPV antiserum as the control in the HI tests (Janssen et al., 1982). Comparison of titers should therefore always be based on results obtained with identical techniques. Indirect immunofluorescence to identify CPV or antibodies against the virus has been used in coyotes (Green et al., 1984).

Electron microscopy for the demonstration of parvovirus, and cultural techniques employing Crandell feline kidney cells and Walter Reed A-72 canine cells (Janssen et al., 1982) have also been used to identify CPV in exotic canids.



PROPHYLAXIS and CONTROL

As with FPV, strict quarantine and sanitization practices should be enforced since CPV is both highly resistant and contagious. The only vaccination experience has been obtained in bush dogs, maned wolves (Janssen et al., 1982) and in coyotes (Green et al., 1984). Bush dog pups vaccinated with killed vaccine (Parvocine, Dellon Lab, Omaha, Nebraska 68134) were not protected even when injected biweekly for 5–19 weeks; some went on to develop fatal disease.

Prevention of the disease with the killed vaccine in bush dog pups and maned wolf cubs 13–15 weeks old could only be achieved by isolating the animals from the colony and continually vaccinating them. Protective titers against CPV (≥ 80) were obtained in maned wolf cubs after 14–18 weeks, but in bush dog pups it required 23 weeks. This points out that immune competence may differ among species of Canidae, even though the clinical and pathologic aspects of the disease may be similar. In coyote pups the half-life of maternal antibodies was determined to be 6.7 d (Green et al., 1984) as compared to the 9.7 d reported in domestic dogs (Pollock and Carmichael, 1982); however, seronegative coyote pups vaccinated at 8 weeks with a killed CPV vaccine had not seroconverted by 11 weeks, which further stresses the differences in immune response that may exist between species. Attenuated CPV vaccines have been shown to be more effective in pups at an earlier age (Glickman and Appel, 1982). At the National Zoo (Washington D.C.) a modified-live CPV vaccine (Duramune, Fort Dodge Labs, Fort Dodge, Indiana) has afforded good protection against CPV in litters of maned wolves and bush dogs without having to isolate them from the colony. The animals were vaccinated 3–4 times at 2 week intervals from 8 weeks of age; protective titers were evident much earlier with the attenuated than with the killed vaccine in some hand-reared maned wolf cubs (Montali and Bush, unpublished).

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