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Source: Journal of Wildlife Diseases, 37(3) : 594-607

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-37.3.594

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Journal of Wildlife Diseases, 37(3), 2001, pp. 594–607
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REVIEW

Parvovirus Infections in Wild Carnivores

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ABSTRACT: Various parvoviruses infect carnivores and can cause disease. In this review article the knowledge about infections of free-ranging or captive carnivores with the feline parvoviruses, feline panleukopenia virus, and canine parvovirus, including the antigenic types CPV-2a and -2b, as well as Aleutian disease of mink virus and minute virus of canines are summarized. Particular emphasis is placed on description of the evolution of canine parvovirus which apparently involved wild carnivore hosts.

Key words: Parvovirus, carnivores, wildlife, review article.

INTRODUCTION

All known parvoviruses that infect and cause disease in carnivore species belong to the genus Parvovirus within the family *Parvoviridae* (Murphy et al., 1995). Several antigenically and genetically very closely related viruses, such as feline panleukopenia virus (FPV), canine parvovirus (CPV-2) along with its antigenic types CPV-2a and CPV-2b, mink enteritis virus (MEV), blue fox parvovirus (BFPV), raccoon parvovirus (RPV) and raccoon dog parvovirus (RDPV), are grouped informally within the feline parvovirus subgroup. Other carnivore parvoviruses are Aleutian mink disease virus (ADV) and canine minute virus (synonyms are minute virus of canines (MVC) or canine parvovirus type 1 (CPV-1)). In this review these viruses will be discussed with special emphasis on their host ranges and their clinical significance for wild carnivores.

FELINE PARVOVIRUSES

Infections with the feline parvoviruses and disease have been described in many different carnivore species with very similar clinical signs (Cooper et al., 1979). Autonomous parvoviruses can only replicate in the nucleus of dividing cells. The genome of parvoviruses does not encode a DNA polymerase, an enzyme that is necessary for the initial step of parvovirus DNA replication, the synthesis of a complementary DNA strand to the single-stranded genomic DNA of the virus. As cellular DNA polymerase is only expressed during mitosis, the first and crucial step of parvovirus replication, therefore, requires the dividing cell. The age of an infected animal is very important as fetal and newborn tissues are a rich source of mitotically active cells. In adult animals the lymphatic system and particularly the epithelium of the gut contain numerous dividing cells and are therefore main targets of parvovirus infection. Characteristic symptoms of acute infections in animals older than three weeks are hemorrhagic gastroenteritis, vomiting and lymphopenia or leukopenia. However, in fetal or neonatal infections different disease manifestations are seen. Infection of kittens with FPV can result in the so-called feline ataxia syndrome where the virus infects cells of the cerebellum, causing cerebellar hypoplasia (Johnson et al., 1967). Infection of puppies of the same age with CPV-2 resulted in myocarditis (Hayes et al., 1979) but not cerebellar lesions. It is not known whether these different manifestation (cerebellum versus myocardium) are due to properties

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of the viruses or due to specific factors of their carnivore hosts, the cat and dog. In utero infection of foxes with feline parvoviruses may cause fetal death, resorption or abortion (Vejiallainen and Smeds, 1988).

Parvovirus infections of dogs and cats are systemic infections that involve various tissues and organ systems. Animals are usually infected by the oral route, and initial virus replication takes place in the epithelium of the pharynx, including tonsils and lymph nodes. Virus spreads in a viremic phase to lymphatic organs, such as peripheral and central lymph nodes, spleen, thymus and Peyer's patches, as well as most other organ systems including bone marrow and in the pregnant animal, the fetuses (Czisa et al., 1971). After the viremic phase, virus spreads from cells of the Peyer's patches to the highly dividing cells of the Liberkühn crypts, those cells that regenerate the epithelial cells of the small intestine. Lytic infection of these cells can lead to substantial loss of gut epithelium and is responsible for the hemorrhagic gastroenteritis symptoms that are characteristic for the disease.

Infection of domestic dogs and cats with the various feline parvoviruses may result in fatal diseases, but subclinical infections with or without mild symptoms are also common. Infection induces a long lasting, possibly life-long immunity and complete elimination of the virus.

The epidemiology of feline parvovirus is characterized by an acute infection with shedding of high virus titers in the feces of diseased animals. Virus shedding ceases after a few days, but virus can stay infectious in the environment for weeks or even months. Transmission of virus is therefore not only readily achieved by contact with feces from infected animals, but also with feces-contaminated premises, fomites, inanimate objects, or personnel. Direct contact between carnivores is not required for efficient transmission.

Feline Panleukopenia virus (FPV)

Syndromes described as feline infectious enteritis, malignant panleukopenia, feline distemper or spontaneous agranulocytosis have long been recognized in domestic cats. Verge et al. (1928) showed that the cause was a filterable virus, which is now known as feline panleukopenia virus (FPV). The first outbreaks of the disease in captive felids were reported in the 1930's and 1940's (Hindle and Findlay, 1932; Goss, 1942). In 1947 an epidemic occurred in the park of the "Zoological Society of London" (Cockburn, 1947). Various feline species, such as tigers (*Panthera tigris*), leopards (*Panthera pardus*), cheetahs (*Acinonyx jubatus*), wild cats (*Felis sylvestris*), lynx (*Lynx lynx*), servals (*Leptaillurus serval*), tiger cats (*Felis tigrina; Felis aurata*) and ocelots (*Leopardus pardalis*) were infected. A possible case in a civet cat (family Viverridae) occurred. Lions (*Panthera leo*) were the only cats which were not infected in this outbreak and a possible natural resistance of this species was discussed. Hyslop et al. (1955) described cases of feline panleukopenia in captive lynx and cheetahs. After experimental inoculation of two cats with dried spleen material of these animals, the symptoms and pathological signs of an FPV disease was observed. Clinical cases associated with FPV infections in wild cats (Chappuis and Lernould, 1987) have been reported. A FPV virus was isolated from the spleen of a 9-mo-old leopard (*Panthera pardus*, Johnson 1964), and later FPV was isolated from a lion (Studdert et al., 1973), disproving the previously suggested natural resistance of lions. The agent causing feline ataxia was identified as FPV by Johnson et al. (1967). In the following years, serologic and electron microscopic detection revealed that the snow leopard (*Panthera unica*, Bieniek et al., 1968), the clouded leopard (*Neofelis nebulosa*, Zhang et al., 1988), the mountain lion (*Felis concolor*) in Florida (Roelke et al., 1993) and California (Paul-Murphy et al., 1994), the

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cheetah (Valicek et al., 1993) and the serval (Bieniek et al., 1968) were susceptible to FPV infection. More recently, FPV was diagnosed in an African wild cat (*Felis lybica*) and in several cheetahs with clinical signs of an acute enteric disease. Tissue samples were analyzed by PCR and DNA sequencing, and classical FPV sequences were obtained (Steinel et al., 2000). Based on the cases described it is generally assumed that all members of the family *Felidae* are susceptible to FPV infection and disease.

Raccoons (*Procyon lotor*) and Arctic ('Blue') foxes (*Alopex lagopus*) with clinical signs of a FPV disease were recognized in the 1940's (Waller, 1940; Phillips, 1943). The parvoviruses isolated from these species were described as FPV-like viruses and named after their respective hosts, RPV and BFPV (Appel and Parrish, 1982; Vejialainen and Smeds, 1988). A true relationship has not been established, but based on DNA sequence analysis of the gene encoding the structural protein VP2, RPV appears identical to FPV, and BFPV may represent a virus intermediate between FPV and CPV-2, as three non-coding nucleotide exchanges, typical for CPV viruses, are present in the BFPV genome (Truyen et al., 1995). A parvovirus infection of a coati mundi (*Nasua nasua*) has also been reported (Johnson and Hallowel, 1968).

Mink enteritis virus (MEV)

In 1947, a viral disease causing severe hemorrhagic enteritis was observed in farmed mink (*Mustela vison*) in Fort Williams, Ontario, Canada (Schofield, 1949). The similarities to infections of cats caused by FPV were soon realized. The disease was called mink viral enteritis and the virus, mink enteritis virus (MEV). Over several years this fatal disease has spread throughout the world, and currently occurs today wherever mink are farmed. FPV and MEV infect and replicate in mink and cats, but experimental inoculation of the heterologous host seemed to cause less severe disease than infection of the homologous host (Parrish et al., 1987). Parrish et al. (1984), described three different antigenic variants of MEV, which induce full in vivo cross-protection. Phylogenetic analysis based on nucleotide sequences of the capsid protein gene could not differentiate between FPV and MEV isolates, and based on those analyses MEV, FPV, BFPV and RPV appear to be variants of the same virus, suggesting that those viruses can infect both mink and cats (Truyen et al., 1995). Why MEV disease emerged in 1947 is unknown, but may be due to changes in the virulence of the viruses. Beside mink, other *Mustelidae* have been found to be susceptible to feline parvoviruses, such as American river otters (*Lutra canadensis*) (Hoover et al., 1985) and the skunk (*Mephitis mephitis*). Experimental inoculation of skunks with FPV, MEV or CPV-2 did not cause disease, but serologic evidence of infection could be demonstrated (Barker et al., 1983). In ferrets (*Mustela putorius furo furo*), disease has been described only after in utero infection, with a marked age resistance to infection noted (Kilham et al., 1967; Parrish et al., 1987). More recently, a free-ranging honey badger (*Mellivora capensis*) from the Kalahari Gemsbok Park (South Africa), was observed with clinical signs of diarrhea. Analysis of the fecal sample from the badger by PCR and DNA sequencing revealed characteristics of a FPV virus (Steinel et al., 2000).

Canine Parvovirus Type 2 (CPV-2)

In 1978 a new viral disease that infected canine species emerged (Appel et al., 1979). Again the clinical similarities to FPV infection in cats were noted. Subsequent antigenic and genetic analyses confirmed a very close relationship between FPV and the new canine virus, referred to as canine parvovirus type 2 (CPV-2) to distinguish it from the distantly related canine parvovirus 1 (minute virus of canines, MVC) that was isolated years before. CPV-2 is closely related to FPV with more than

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Amino Acid (VP2)									
	80	87	93	103	300	305	323	564	568
FPV	Lys	Met	Lys	Val	Ala	Asp	Asn	Asn	Ala
CPV-2	Arg	Met	Asn	Ala	Ala	Asp	Glu	Ser	Gly
CPV-2a	Arg	Leu	Asn	Ala	Gly	Tyr	Glu	Ser	Gly
CPV-2b	Arg	Leu	Asn	Ala	Gly	Tyr	Glu	Ser	Gly

FIGURE 1. Amino acid changes in the VP2 protein between FPV, CPV-2 CPV-2a/2b. Amino acid differences that are conserved among all FPV-like viruses and all CPV viruses analyzed so far are shaded in dark grey. Amino acid differences conserved in the new antigenic types are shaded in light grey. These amino acids are responsible for antigenic differences between the viruses as well as for different host ranges. Amino acids 80, 564 and 568 in FPV, as well as amino acids 87, 300 and 305 in the new antigenic types of CPV-2a and -2b appear to determine the ability to replicate in cats (Truyen et al., 1994, 1996a).

98% genome homology, and as few as six coding nucleotide differences in the VP2 protein (positions: 3025, 3065, 3094, 3753, 4477, 4498; Parrish et al., 1988; Parrish, 1991; Truyen et al., 1995; Fig. 1). The biological effects of these few genomic changes were enormous, in that CPV-2 acquired the canine host range, but lost the ability to replicate in cats (Truyen et al., 1994). The host ranges of CPV-2 and FPV are complex and differ in vitro and in vivo. FPV replicates in feline cells in vitro and in cats in vivo, but does not infect canine cells in vitro and shows only a restricted tissue spectrum in vivo. CPV-2 does replicate in canine and feline cells in vitro, but the in vivo replication is restricted to canides (Truyen et al., 1992). No feline host has ever been described to be susceptible to CPV-2, although it replicates to low titers in mink after experimental inoculation (Parrish et al., 1987).

After its emergence CPV spread to most populations of domestic and wild carnivores. The first known CPV-2 positive sera were from dogs collected in Greece during 1974 (Koptopoulos et al., 1986). In 1976, reports from Belgium (Burtonboy et al., 1979) and the Netherlands (Osterhaus et al., 1980) followed by reports that the virus had spread throughout the world (Appel et al., 1979; Kelly, 1978; Gagnon and Povey, 1979; Azetaka et al., 1981; Horner et al., 1979) infecting wild and domestic canids. Antibodies against CPV-2 were reported by 1980 in free-ranging grey wolves (*Canis lupus*) in Alaska (Zarnke et al., 1980). Three different coyote populations in the United States of America were found to be seropositive for CPV-2 by 1979 (Barker et al., 1983; Gese et al., 1991; Thomas et al., 1984). Clinical signs of parvovirus disease were observed in captive and free-ranging coyotes (Evermann et al., 1980). During 1980 and 1984 dingo pups (*Canis dingo*) became infected in the zoological garden of Stendal, Germany (Dietzmann et al., 1987).

Serologic prevalence, infection or clinical signs of disease due to FPV or CPV related viruses were found in jackals (*Canis aureus, Canis adustus, Canis mesomelas;* Alexander et al., 1994), grey foxes (*Urocyon littoralis;* Garcelon et al., 1992), the San Joaquin kit fox (*Vulpes macrotis mutica;* McCue and O'Farrel, 1988), Asiatic raccoon dogs (*Nyctereutes procyonoides*; Veijalainen, 1986), bush dogs (*Speothos venaticus*, Mann et al., 1980, Chappuis and Lernould, 1987), the crab-eating fox (*Cerdocyon thous*, Mann et al., 1980) and wild African hunting dogs (*Lycaon pictus*, Alexander et al., 1993) in the Masai Mara (Kenya). African wild dog populations of the Krüger National Park (South Africa) and of the north east of Namibia have apparently remained seronegative (van Heerden et al., 1995; Laurenson et al., 1997). In the maned wolf (*Chrysocyon brachyurus*), acute infection caused by a parvovirus was reported in the 1960's, but the virus isolated showed features (pH dependence of hemagglutination) characteristic for FPV (Bieniek et al., 1981).

Canine parvovirus infections were reported in farmed raccoon dogs (Neuvonen et al., 1982), and an isolate of an Asiatic raccoon dog parvovirus was obtained in by Vejialainen et al. (1988). DNA sequence analysis of the VP2 gene showed the virus to be CPV-2 (Truyen et al., 1995). Raccoons, in contrast, were shown to be resis-

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tant to CPV-2 infection (Appel and Parish, 1982).

With the sudden appearance of CPV-2, different hypotheses on the emergence of this new virus were developed. Single mutations of FPV, or a possible variation from a FPV modified live virus vaccine in tissue culture were proposed (Siegl, 1984). The second theory was based on two facts: first CPV-2 replicated well in feline cells in vitro and secondly restriction enzyme analysis showed more restriction sites in common between several FPV vaccine strains and CPV-2 isolates than with other FPV isolates (Tratschin et al., 1982). However, DNA sequence analysis of FPV isolates and vaccine strains showed that those were all typical FPV viruses (Truyen et al., 1998b). Truyen et al. (1995) proposed another possible reason for the emergence of CPV-2. DNA sequencing of an isolate from an Arctic fox (*Alopex lagopus*), BFPV, showed a typical FPV viral sequence in terms of all coding and most non-coding nucleotide changes, but it also contained three non-coding nucleotide changes that are the same as in CPV viruses. Another parvoviral DNA sequence from an European red fox (*Vulpes vulpes*) could be analyzed and was classified as CPV-2 virus, but with one coding nucleotide difference being specific for FPV (position: 3094; Truyen et al., 1998a). This nucleotide changes amino acid 103 of the VP2 structural protein from serine to alanine. One additional parvoviral sequence from a red fox was analyzed by the authors and that again showed intermediate features between FPV and CPV-2, further supporting this hypothesis (Steinel and Truyen, unpubl. data).

Both of these fox viruses were therefore intermediates between CPV-2 and FPV. In the phylogeny of the order *Carnivora* foxes are classified as being more closely related to the *Felidae* than wolves, coyotes or domestic dogs (Wayne et al., 1989). CPV-2 may therefore have emerged from an FPV-like virus in a wild carnivore and then adapted to the canine host.

Canine Parvovirus Type 2a and Type 2b (CPV-2a; CPV-2b)

In 1979 and around 1984, new antigenic types of CPV emerged and became widespread, and those have been designated CPV-2a and CPV-2b. The common differences of these viruses compared to the original CPV-2 are based on other coding and nucleotide changes at positions: 3045, 3685, 3699 (Fig. 1). CPV-2b had one further nucleotide substitution at position 4062. Those substitutions changed antigenic epitopes that can be detected with monoclonal antibodies, and those antigenic types are the predominant strains currently circulating in the different dog populations, and have completely replaced the original CPV-2 virus world-wide (Parrish et al., 1991; Truyen et al., 1996b; Steinel et al., 1998). There seems to be an evolutionary advantage of the new antigenic types that caused this replacement, but no such selective advantage could be defined between CPV-2a and CPV-2b. Both antigenic types coexist in different ratios in dog populations worldwide. The regaining of the feline host range by CPV-2a and CPV-2b was likely a selective advantage of the virus (Truyen et al., 1996a; Mochizuki et al., 1996). Approximately 5% of parvovirus infections in domestic cats (*Felis catus*) are caused by CPV-2a or CPV-2b (Truyen et al., 1996b). In addition large cats are also susceptible to the new antigenic types of CPV. CPV-2a and CPV-2b DNA sequences were recovered from six of nine cheetahs, as well as from one Siberian tiger, all showing clinical symptoms of parvovirus disease (Steinel et al., 2000). The very high prevalence of CPV-2a/2b infections in large cats compared to domestic cats may suggest a higher susceptibility of these species for these virus types. A similar increase in susceptibility in large cats may also be present for canine distemper virus, a morbillivirus (Roelke et al., 1996). Parvoviruses are very stable in the environment, zoos, captive holding areas, animal shelters and veterinary clinics, and direct or indirect spread from dogs may

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have resulted in increased infection of the large cat species. Since vaccination of domestic cats and dogs is very effective in preventing disease, parvovirus vaccination of all domestic and non-domestic carnivores at risk of infection is highly recommended.

New antigenic types of CPV-2 have been shown to infect species other than felids. An isolate from a stone marten (*Martes foina*) was identified as CPV-2a (Weissenboeck and Truyen, unpubl. data), while from an orphaned bat-eared fox (*Otocyon megalotis*) a CPV-2b DNA sequence was amplified (Steinel et al., 2000).

Feline parvoviruses in wildlife

Beside the infection of the Canidae, Felidae, Mustelidae and Procyonidae, little is known about the host range and the distribution of the FPV-related parvoviruses in wild carnivores. Phylogenetic analysis based on the nucleotide and amino acid sequences of the capsid protein differentiate two groups of feline parvoviruses: the FPV-like viruses from cats, raccoon, mink and foxes (BFPV, RPV and MEV), and the CPV-like viruses from dogs, raccoon dogs and cats (CPV-2/2a/2b and RDPV) (Truyen et al., 1995, 1996a) (Fig. 2). In other carnivores, most investigations so far have been based on antibody detection in selected free-ranging populations and on a few clinical cases in captive animals. Only from cheetahs, a Siberian tiger, an African wild cat, a honey badger, a stone marten and a bat-eared fox DNA sequence analysis allowed a grouping of the detected parvoviruses (Weissenboeck and Truyen, unpubl. data; Steinel et al., 2000). The variety of carnivore species and the difficulties of observing wildlife often hinders a definitive characterization of the different feline parvovirus subtypes that infect these animals.

Our current knowledge of parvovirus infections in wild carnivores is still incomplete. Virtually no data are available of possible parvovirus infections in seals (*Pinnipedia*), and many other families of the order Carnivora. Among the Ursidae and Viverridae, only serological or clinical evidence of infection was reported (*Ursus arctos*; Madic et al., 1993; *Ailuropoda melanoleuca*; Mainka et al., 1994; Cockburn, 1947). The interesting question about the mechanisms that were responsible for the emergence of canine parvovirus is also not answered and more studies are required to define the role of wild carnivores in that process.

Infections of non-carnivore species were suspected. In European hedgehogs (*Erinaceus europeaus*) with acute gastroenteritis, parvovirus antigen was demonstrated using immunohistochemistry with a polyclonal FPV antiserum. FPV-infected cats were suspected as the source of that infection (Kränzlin et al., 1993). Parvovirus infections were suspected in a beaver (*Castor canadensis*) and in a porcupine (*Erethizon dorsatum*) based on histopathological signs (Anderson et al., 1989; Frelier et al., 1984). However, in both cases a virological diagnosis was not made.

The determinants of host range in this virus group are also not well understood. From various studies, it appears that few amino acids in the capsid protein determine the ability of the viruses to replicate in the various hosts (Chang et al., 1992, Parrish, 1991, Truyen et al., 1995, 1996a). As most of these amino acids that apparently determine the feline host range (amino acids 80, 564 and 568 in FPV and 101, 300 and 305 in CPV-2a/-2b) are located at a structurally complex region of the capsid where four capsid monomers interact, a possible difference in capsid stability may be involved. The stability of the virus is crucial during the disassembly of the virus capsid to release the viral DNA. Besides that, binding to virus receptors on canine and feline cells may be different between the virus types.

Immune prophylaxis in wild carnivores

Parvoviruses can be life threatening pathogens of many carnivore species and the risk of disease can be minimized by

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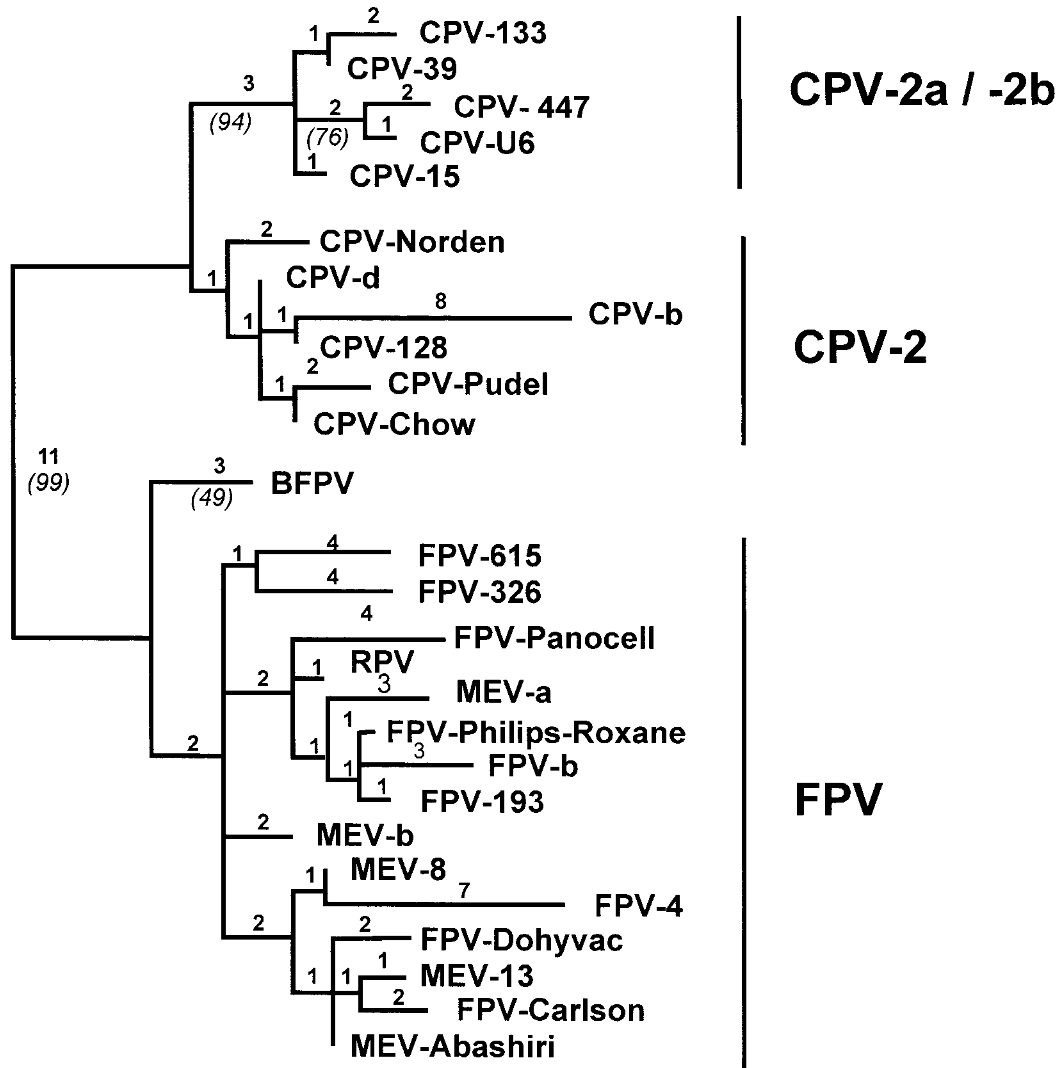

FIGURE 2. Phylogeny of the feline parvoviruses based on 90% of the DNA sequence of the structural protein VP2 (approximately 2,000 nucleotides). The phylogeny separates the FPV-like viruses from cats, mink, blue (arctic) foxes, and raccoon (FPV, MEV, BFPV, RPV, respectively) and the CPV-like viruses from dogs and raccoon dogs (CPV and RD). Both clusters are separated by 11 nucleotide changes including the six coding changes that are responsible for the different host ranges of the viruses (Parrish, 1991; Truyen et al., 1995). The CPV-like virus cluster is further subdivided in the branches represented by the original CPV isolates from 1978 (CPV-2 type) and in the branch that contains the new antigenic type viruses CPV-2a and CPV-2b. Numbers indicate nucleotide differences between the viruses and the respective branching point. Numbers in brackets indicate bootstrap values >50% which indicates the support for the respective branch. 100 bootstrap replicates were generated using the branch-and-bound algorithm of the program PAUP vs. 3.1.1. (Swofford, 1993).

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vaccination. Inactivated and modified live virus vaccines (MLV) against FPV, MEV, CPV-2, and CPV-2b are commercially available. The CPV-2b vaccine is only available in the United States currently. Live attenuated vaccines are only tested in the animal species they are licensed for, and caution should be taken if they are used in other species.

In domestic cats and dogs, inactivated vaccines were shown to be not as effective in inducing appreciable antibody titers as MLV's, and the duration of immunity appears to be much shorter. Inactivated vaccines, therefore, do not have a significant market share for domestic dogs and cats. However, the advantage of inactivated vaccines is their safety. MLV virus strains attenuated for domestic cats and dogs may not be attenuated for non-domestic species. For safety reasons the use of inactivated vaccines is recommended in exotic species as long as there are no clinical experiences with MLV for the species to be vaccinated. In South Africa, MLV's have been used in cheetahs without inducing clinical symptoms (Spencer et al., 1991). No reports have been published on the safety of MLV vaccines in pregnant animals. The feline ataxia syndrome has been described in a lion cub (Leclerc-Cassan, 1981), and vaccination before breeding should minimize the risk of that disease.

With the emergence of CPV, the use of MLV's in various carnivore species has been controversial. Possible switches of the natural host range have been suggested for MEV and initially for CPV. None of theses scenarios has been substantiated and there are no data on a role of a MLV vaccine in the emergence of a new virus type or even as the source of an epidemic in an carnivore species. Based on our current knowledge, all Felidae, Mustelidae, Procyonidae, and Canidae are natural hosts for FPV and/or CPV-2a and CPV-2b viruses. Vaccination with these virus types should therefore not alter the natural history of these viruses and their hosts.

Many different carnivore species appear to be susceptible to infections with FPV and CPV. Fowler (1986) suggested vaccination of all feline species with an inactivated FPV vaccine. Accordingly, all captive zoo Canidae should be vaccinated with an inactivated CPV-2 vaccine, as this virus type was shown to be highly effective for vaccination of domestic dogs.

In the zoological garden of Basel, Switzerland, large cats and maned wolves were vaccinated with an inactivated CPV-2 vaccine (Gutzwiller et al., 1984).

Considering the high incidence of infections with CPV-2a and CPV-2b in large cats an inactivated vaccine containing these types rather than CPV-2 would be desirable, but is not yet commercially available. This appears particularly justified as preliminary experiments revealed a 100–1,000-fold difference in the neutralizing titer of sera from cats immunized with FPV vaccines when tested against CPV-2a or CPV-2b viruses, compared to tests against the homologous FPV virus type (Truyen, 1997).

For mink, an inactivated MEV vaccine is available, but because of the close relationship and possible identity of MEV and FPV, inactivated FPV vaccines also should be effective.

In summary, vaccination of captive carnivores against feline parvovirus infections is highly recommended. The viruses are endemic in many if not all populations of wild carnivores and effective transmission is facilitated by feces of acutely infected animals, which contain high titers of virus. Based on the vaccines currently available, captive Felidae and Mustelidae should be vaccinated with FPV vaccines, and members of the Canidae with CPV-2 vaccines. For safety reasons inactivated vaccines are recommended. However, if the inoccuousness of a MLV in a particular species is defined, MLV's should be used as they are likely to induce a better immunity than inactivated vaccines.

As the new antigenic types of canine parvovirus also infect large cats and significant antigenic differences are obvious be-

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tween FPV and those viruses, the development of CPV2a/2b vaccines for both Felidae and Canidae would be highly welcome.

For free-ranging carnivores vaccination is nearly impossible. However, if there is a chance for vaccination, it should certainly be performed. Reintroduction of captive born animals should only be performed after vaccination.

ALEUTIAN MINK DISEASE

Aleutian mink disease (AD) is caused by a parvovirus which is genetically and antigenically very distinct from the feline parvovirus subgroup viruses (Murphy et al., 1985). In addition, the disease induced by ADV differs from that caused by the other agents described in this article. Classic AD is a chronic, persistent and progressive disease of adult mink (*Mustela vison*), characterized by immune complex glomerulonephritis and arteritis involving various organ systems, including liver and spleen. Typical clinical signs are polydypsia, apathy, diarrhea, melena, and hemorrhages. The fertility rate decreases and susceptibility to other diseases, as well as the death rate, increase on infected ranches (Bloom et al., 1994). Hadlow (1982) described ocular lesions in mink infected with ADV, caused by hypergammaglobulinemia and possible deposition of circulating immune complexes and a nonsuppurative meningoencephalitis has also been reported (Dyer et al., 2000).

Beside the classic chronic form of AD, an acute manifestation in kits less than 2 wk of age has been described on ranches where the dams are seronegative for ADV. Within 2 wk of infection the kits die of an acute interstitial pneumonia, characterized by parenchymal hemorrhage, extensive atelectasis and hyaline membrane formation due to a lytic infection of type II pneumocytes (Alexandersen et al., 1994). Mechanical and transplacental transmission of ADV have been described (Broll and Alexandersen, 1996), and virus is shed with urine and feces (Porter and Larson, 1990). Aerosol spread has also been proposed (de Geus et al., 1996). Repeated attempts to develop vaccines against ADV have been unsuccessful, leading to accelerated disease (Aasted et al., 1998), and the only effective means of control is diligent culling of infected animals (Cho and Greenfield, 1978).

AD was first described in mink with a light blue-gray coat color, referred to as 'Aleutian mink.' Initially, only mink of that genotype were considered to be susceptible to ADV infection, but it was eventually shown that all mink were susceptible, although in non-Aleutian mink the disease was generally less severe or inapparent. Serologic investigations demonstrated antibodies to ADV in various species of the family Mustelidae, such as mink, striped skunk (*Mephitis mephitis*), and one suspected case of clinical AD was described in an otter (*Lutra lutra*) by Wells et al. (1995). Beside mink, ferrets developed various clinical signs, including a wasting syndrome or posterior ataxia and paresis (Welchman et al., 1993), but immune complex disease is not seen. ADV infection has also been described in carnivores outside the family Mustelidae, e.g., in red foxes and raccoons (Ingram and Cho, 1974). During an outbreak of ADV on a mink farm, raccoons were suspected as the reservoir species in the transmission of ADV to mink. However, raccoons did not develop clinical signs after experimental infection and, although lysates of infected raccoon organs infected mink, raccoon-to-raccoon transmission of ADV was not demonstrated (Oie et al., 1996).

AD is a common disease of farmed mink and ferrets, but its prevalence and significance in free-ranging carnivores is largely unknown. Because of the persistent nature of the disease and the negative effects on reproductive success of infected animals, ADV may have negative impacts on wild populations of susceptible mustelids. Antibody and virus sequences were detected in free-ranging raccoons captured on mink ranches in the midst of an

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ADV outbreak in Utah (Oie et al., 1996), and the viral sequence matched that of the mink. Antibody to ADV was also identified in four of 22 free-ranging skunks from South Dakota (USA), and viral DNA was amplified from three of these (Oie et al., 1996). The sequence of the "hypervariable region" of the capsid protein gene from the skunks differed from other characterized isolates. In Spain, antibody to ADV and viral sequences have been found in free-ranging native European mink (*Mustela lutreola*), feral American mink and an Eurasian otter (*Lutra lutra*). The limited DNA sequence information suggested that these animals were not infected with isolates of ADV that have been previously identified (MaZas et al., 2001).

MINUTE VIRUS OF CANINES

In 1967 a small virus was recovered from fecal specimens from several healthy dogs and dogs with mild diarrhea (Binn et al., 1970). The virus was classified as a parvovirus and named minute virus of canines (MVC), or more recently canine minute virus. MVC infection appears to cause disease only in puppies less than about 3 wk of age or in the fetus. In puppies clinical signs are bronchitis and interstitial pneumonia, diarrhea and myocarditis (Järplid et al., 1996; Carmichael et al., 1994). Symptoms may vary in severity and most infections appear to be mild or subclinical, but fatal cases have also been reported (Järplid et al., 1996). MVC infection of pregnant bitches can lead to embryo resorption, abortion and fetal death (Carmichael et al., 1991). MVC appears antigenically unrelated to parvoviruses of other species (Macartney et al., 1988), and is genetically very distinct from FPV and CPV. In the United States and Germany, the prevalence of antibodies to MVC infection in dogs is about 50% (Carmichael et al., 1994; Truyen et al., 1996c). Confirmed MVC-induced fatal infections have been reported from the United States, Scandinavia, and Germany (Carmichael 1994; Järplid et al., 1996; Truyen et al., 1996c), and it is likely that MVC occurs worldwide. The domestic dog is the only known host, although the incidence of MVC infection of wild canides has not been reported.

ACKNOWLEDGEMENTS

The parvovirus work in the laboratories of CRP and UT is supported by grants from the National Institutes of Health (NIH), the Deutsche Forschungsgemeinschaft (DFG), the Gesellschaft zur Förderung kynologischer Forschung (GKF) and the German Academic Exchange Service (DAAD). MEB acknowledges G. Durrant and continued support from the Mink Farmers Research Foundation and the Fur Breeders Agricultural Cooperative.

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Received for publication 2 August 2000.

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