Infectious and parasitic diseases of captive carnivores, with special emphasis on the black-footed ferret (*Mustela nigripes*)

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Summary: Captive carnivores are susceptible to a wide array of infectious and parasitic diseases, which reflects the diversity of the seven families of Carnivora. Unfortunately, relatively few in-depth studies have been conducted on diseases of non-domestic carnivores, and much remains to be learned, especially regarding diseases of small carnivores (e.g. mustelids, viverrids and procyonids). The more important infectious diseases of carnivores include rabies, canine distemper, and diseases caused by paroviruses, coronaviruses and herpesviruses. Few parasitic or bacterial pathogens are significant in captive populations, and appropriate husbandry, therapy, vaccines and quarantine minimize the risk of disease. Extrapolations from one species to another regarding disease susceptibility may be incorrect. The black-footed ferret (*Mustela nigripes*) serves as an example of a carnivore significantly affected by infectious diseases, some of which were expected while others could not have been predicted from generalized knowledge of diseases of mustelids. This highlights the need to understand the natural history of each species maintained in captivity.


INTRODUCTION

The order Carnivora encompasses an extremely varied group of species which are hosts to a correspondingly diverse spectrum of infectious agents. The order is composed of seven extant families (98), namely Canidae (dogs, wolves, coyotes, jackals, foxes), Felidae (cats), Mustelidae (weasels, badgers, skunks and otters), Ursidae (bears, giant panda), Procyonidae (raccoons and relatives, lesser pandas), Viverridae (civets, genets, linsangs, mongooses, fossas) and Hyaenidae (hyenas, aardwolf). Non-domesticated carnivores are held in captivity for educational and recreational purposes, for fur production, as pets, for research, in captive breeding programmes for endangered species recovery and species conservation, and for rehabilitation. Some individuals are generations removed from the free-ranging state

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but are still considered ‘wild’ because they are not truly domesticated (33); others may have been removed from the wild only recently. The burden of infectious diseases and parasites is influenced by many factors, including the length of time since the animals were removed from the wild, degree of adjustment to captivity, quality of captive management and husbandry for the needs of the species, and proximity to other species with which they could exchange pathogens. Sources of infectious agents for captive carnivores are varied, and include contact with the following:

- individuals of the same, related or distant species
- humans
- food
- water
- fomites
- iatrogenic introduction, e.g. through attenuated vaccines.

Carnivores display a spectrum of disease susceptibility, and understanding of the diseases of many species is incomplete at present.

In this review, the disease situation of the carnivores is organized according to family, and the most significant infectious and parasitic diseases of each group are discussed. Diseases were chosen by reason of their impact on species, or their importance to the health of humans, domestic animals, and captive or free-ranging animals; these diseases are therefore of concern to those maintaining and exchanging carnivores in captivity. Following this section is a review of the recovery programme for the black-footed ferret (Mustela nigripes), as an example of a captive endangered wild species which is significantly affected by infectious diseases. General lists of infectious diseases of various families may contrast sharply with the specific disease problems of one species. This highlights the need to understand the range of disease susceptibility in specific species of interest, and emphasizes the pitfalls of extrapolating from one species to another, even between close taxonomic relatives.

**INFECTIOUS AND PARASITIC DISEASES IN THE VARIOUS FAMILIES OF CARNIVORA**

**CANIDAE**

**Viral pathogens**

Rabies is caused by a rhabdovirus of the genus *Lyssavirus*, and carnivores serve as major hosts for this important zoonotic disease. Significant wild canid reservoirs of rabies include the following:

- red foxes (*Vulpes vulpes*) in parts of North America, Asia, and Eastern and Central Europe (140)
- grey foxes (*Urocyon cinereoargenteus*) in parts of North America (74)
- Arctic (blue) foxes (*Alopex lagopus*) in North America and Asia (124)
- bat-eared foxes (*Otocyon megalotis*) in South Africa (12, 131)
- coyotes (*Canis latrans*) in parts of North America (74)
- jackals (*C. aureus, C. adustus* and *C. mesomelas*) in Africa and Asia (12, 13, 140).

Rabies is transmitted by bite, or by the contact of broken skin with virus-containing saliva from an affected animal. Diagnosis of rabies in live canids is not possible on a routine basis, thus the screening of newly-acquired canids is not possible. Recently-captured wild canids could be incubating rabies; knowledge of the epizootiology of rabies in the area of origin is helpful in assessing the risk of introducing rabies to captive wild animal populations or human caretakers. Rabies is extremely rare in canids bred in captivity; a minimum quarantine period of 180 days is recommended for any wild-caught canid (94). Rabies could be introduced into captive canids via contact with free-ranging or feral species under some types of extrinsic management. Vaccination of captive wild canids against rabies, using inactivated vaccines, is appropriate for valuable animals and animals which might be exposed to the virus (94). Vaccination of humans against rabies is good policy if they will be handling or caring for wild-caught carnivores.

Canine distemper is probably the most important infectious disease of canids. It has caused significant mortality in captive canids in zoos (23, 89) and in the fur industry (23), as well as in free-ranging canids (2, 38). Canine distemper is caused by a *Morbillivirus*. The virus is relatively labile in the environment, and is readily inactivated by solvents, disinfectants, heat and ultraviolet light; half-lives of 1-3 h at 37°C, 2 h at 21°C, and 9-11 days at 4°C have been reported (4). All species of canids appear to be susceptible to infection, with varying morbidity and mortality; detailed knowledge of canine distemper in wild canids is lacking. The virus infects a wide variety of organs and is shed in most excretions of infected animals; transmission occurs via close direct contact or aerosol. Fomite transmission is possible, but is unlikely due to the fragility of the virus. Studies in domestic dogs have shown that incubation varies from 1-3 weeks, with virus being shed as early as 4-5 days post-infection, possibly continuing for months until either death or complete recovery occurs (4). The fatality rate for wild canids is not known, but serological surveys indicate that many canids survive infection (3, 31, 136, 143). Most quarantine protocols of > 30 days will protect captive populations from introduction of the disease via canids, but recovered canids which appear clinically normal could shed the virus and expose susceptible hosts. Captive canids could also contract canine distemper via exposure to feral domestic dogs or other free-ranging species. Examination of faeces by negative-stain electron microscopy may detect virus shed in faeces (99), and serology will give an indication of exposure of captured wild canids. Attenuated canine distemper vaccines are available for domestic dogs and many have been used safely in wild canids (15, 127). Some species, however, are exquisitely susceptible to vaccine-induced disease, including grey foxes (61) and *African hunting dogs* (*Lycaon pictus*) (127). In addition, attenuated vaccines may induce immunosuppression, leading to secondary infection by opportunistic pathogens. Some attenuated vaccine viruses may be shed by vaccinated individuals, which could thus potentially transmit the virus to more susceptible species (89).

Canine parvoviral enteritis is caused by canine parvovirus type 2 (43). This virus is very hardy in the environment, remains infectious for three months at 20°C, and may survive in faeces for several years (7). A dilute solution of sodium hypochlorite
is adequate for sanitation (123). Most species of Canidae appear to be susceptible to infection, but fatality rates are not known; clinical disease and/or mortality have been observed in coyotes (44; E.S. Williams, unpublished findings), bush dogs (Speothos venaticus) (78), crab-eating foxes (Cerdocyon thous) (78), maned wolves (Chrysocyon brachyurus) (45, 78), raccoon dogs (Nyctereutes procyonoides) (97) and grey wolves (82, 83, 84). Red foxes and ranched blue foxes are susceptible to infection but appear to be resistant to disease (9, 97). Juveniles appear to be more susceptible to paroviral infection than adults (78, 83). The incubation period in domestic dogs, and probably wild canids, is 4-7 days (7). Transmission occurs via direct contact with infected animals and their faeces, which contain abundant virus. Exposure of captive wild canids might occur via contact with infected domestic dogs, fomites, or other affected individuals within the captive facility. Periodic shedding may occur in subclinically-affected adult animals (66). Quarantine procedures of > 30 days, along with more specialized tests, are probably adequate to prevent introduction of this disease into captive facilities from wild-caught or transferred captive canids. Testing faeces for the presence of virus is possible using antigen capture enzyme-linked immunosorbent assay (ELISA) techniques or negative-stain electron microscopy. Attenuated parovirus vaccines have been used in coyotes (58), maned wolves and bush dogs (88). Although inactivated vaccines require multiple boosters (66), they may be preferred (15) if attenuated vaccines have not been shown to be safe in a particular species.

Infectious canine hepatitis, caused by canine adenovirus type 1, induces infection and/or disease in foxes (59, 114), coyotes (79) and grey wolves (31). This disease is probably of little consequence in the wild, but significant mortality has occurred in ranched foxes. Transmission occurs via direct contact or by ingestion of virus-contaminated material. The virus is shed in urine, faeces and saliva, and by aerosol. Shedding may be prolonged in domestic dogs (111) and this is likely to be the case also in wild canids. The incubation period is usually a week or less (57). Attenuated vaccines are available for domestic dogs; these have been used in captive foxes (57) and are recommended for wild canids (15). Killed vaccines should be used, unless attenuated vaccines have been shown to be safe in the species to be vaccinated.

**Bacterial pathogens**

*Brucella* spp. infections -- probably via consumption of infected prey -- have been reported in wolves, coyotes, foxes, African wild dogs and jackals (39, 41, 150). The disease is probably subclinical in most carnivores, but birth of weak or stillborn pups has been observed in wolves (95) and congenital infections may occur in coyotes (39). Knowledge of the status of wild carnivore prey with regard to brucellosis should help to determine the testing required for captured wild canids. Transmission between infected canids and other species is theoretically possible but unlikely. Canids in captivity for a prolonged period are extremely unlikely to be exposed to *Brucella* spp. or to carry the organism.

Wild canids are susceptible to bovine tuberculosis, caused by *Mycobacterium bovis*, but the disease is rare in these species in the wild, unless it is common in their prey. It is likely that under situations of increased density, stresses related to captivity, contact with carrier animals, and potential exposure to contaminated feedstuff, canids could become infected; but they are considered relatively resistant to infection (134). There are few reports of bovine tuberculosis in captive and free-ranging canids (130;
J. Rhyan, personal communication). The most likely source of *M. bovis* infection for canids in captivity is through contaminated feed. This is unlikely if commercial, appropriately-processed feed products are used.

Parasites

Wild canids are afflicted by numerous endo- and ectoparasites. Most of these are of little consequence; some may serve as vectors for zoonotic diseases, however, or cause clinical disease in young or immunocompromised individuals. Treatment for endo- and ectoparasites is usually accomplished easily with topical, oral or injectable products and is recommended when moving canids from the wild to captivity and between captive facilities, or routinely with captive animals if a parasitic problem is identified.

Sarcoptic mange, caused by *Sarcoptes scabiei*, is an important disease of wolves, foxes and coyotes (91, 109, 119, 135, 137). There is a degree of host specificity of the mites, but experiments have successfully cross-transmitted scabies between wild canids (119, 128). Humans are also susceptible to infection by *S. scabiei* from wild canids (119, 129). Treatment with acaricides is successful (128).

Wild canids may serve as hosts for *Trichinella* spp., and infestation has been reported in grey wolves and red foxes from Alaska (115), and in wild carnivores from north-western North America (60). The normal mode of transmission, via contaminated feed, is unlikely to occur in captivity as long as uncontaminated or appropriately-treated diets are fed to carnivores. Capture of infected wild canids is possible, but under captive conditions they are likely to be dead-end hosts.

The canine heartworm (*Dirofilaria immitis*) is found in wild canids in areas where this parasite is present in domestic dogs. *D. immitis* has been expanding its range and is endemic in many parts of North America (139). Coyotes (36, 55), red foxes (53, 73, 122), grey foxes (85) and wolves (113) have been reported as hosts. Heartworms are transmitted via the bite of infected mosquitoes. Prophylactic anthelmintics are administered to susceptible canids in areas where the parasite is endemic, during seasons when mosquitoes are present; vector control may also be useful for sensitive species. Capture of infected wild canids is possible, but under captive conditions they are likely to be dead-end hosts.

Wild canids are important as definitive hosts of *Echinococcus granulosis* and *E. multilocularis* (1). The normal sylvatic cycle of *E. granulosis* involves coyotes and wolves, and their primary prey – i.e. deer (*Odocoileus* spp.), moose (*Alces alces*) and caribou (*Rangifer tarandus*). The cycle of *E. multilocularis* occurs most frequently in Arctic and red foxes, with microtine rodents as the primary intermediate hosts. These cestodes form hydatid cysts in intermediate hosts, or in abnormal hosts (e.g. humans). Infections in humans can be fatal, and therefore treatment of susceptible definitive hosts from areas with endemic echinococcosis should be part of captive management plans. Contact of humans and intermediate hosts with faeces from untreated foxes or other canids should be prevented.

FELIDAE

Viral pathogens

Rabies occurs in wild felids, although these species are not primary reservoirs of the virus. The above discussion of rabies in wild canids is generally applicable to felids. Incubation periods of more than two years have been experimentally produced in
domestic cats (93), although it is not known whether chronic rabies may occur in non-domestic felids. A quarantine period of six months is required for movement of domestic cats to the United Kingdom (which is free of rabies) and is recommended for wild-caught felids (94).

Feline panleukopenia, also called feline distemper or feline infectious enteritis, is caused by a parvovirus. The disease agent is closely related to canine parvovirus, mink enteritis virus and raccoon enteritis virus (104). Probably all Felidae are susceptible to feline panleukopenia (14) and the disease has occurred in many captive species under a variety of management conditions (88). Cats which recover from panleukopenia may shed the virus in faeces for weeks, and the virus may persist at low levels for more than a year (35). Quarantine periods of > 30 days are probably adequate for cats. The most frequent source of feline panleukopenia virus for captive felids is probably direct or indirect contact with domestic cats (88). The virus is very resistant to environmental conditions and may remain infective for months or years. All captive cats should be vaccinated to prevent feline panleukopenia (15). Inactivated vaccines should probably be used (28), unless attenuated vaccines have been shown to be safe in the species of interest; some non-domestic cats, including the bobcat (Felis rufus) (E.S. Williams, unpublished findings), may be susceptible to attenuated vaccines, while studies with cheetahs (Acinonyx jubatus) demonstrated the safety of an attenuated virus vaccine (126, 138).

Feline leukaemia virus, a retrovirus of felines, is common in domestic cats; but little is known about the disease in non-domesticated wild felids, except that some are susceptible to infection (19, 20, 67, 134). In domestic cats, the virus causes a variety of syndromes ranging from neoplasia to reproductive problems and immunosuppression (106, 134). Transmission occurs via direct contact with carriers; in utero transmission may occur in domestic cats. Oral transmission via predation of domestic cats has been suspected in a mountain lion (Felis concolor) (67). Measures should be taken to prevent introduction of this virus into collections of non-domestic felids, due to the significant disease problems associated with this virus in domestic cats and the lack of treatment or of proven efficacy of vaccination in non-domestic felids. Serological screening of wild cats is recommended. Routine vaccination of non-domestic felids is not recommended, although vaccination might be justified in specific circumstances (20).

Feline immunodeficiency virus is a newly-recognized retrovirus (107) which infects a variety of wild felids as well as domestic cats. Relatively little is known about the relationship between this virus and wild hosts, although some populations of wild felids show high seroprevalence (21, 22, 76, 102). In domestic cats, feline immunodeficiency virus may cause subclinical infection or immunosuppression, with resultant increased susceptibility to other pathogens and opportunistic infections. Serological surveys have demonstrated that the virus is common in some free-ranging populations of mountain lion, lion (Panthera leo), leopard (P. pardus) and cheetah, but appears to be less common in captive populations (11, 21). The implications of the presence of this virus in wild cats are not yet known; as feline retroviruses are essentially carried for life, however, and could be transmitted horizontally, serological testing and exclusion of seropositive animals may be justified.

The causative agent of feline infectious peritonitis (a feline coronavirus), or antibodies against this virus, have been reported in many wild felids (153). Much attention has focused on cheetahs, in view of the marked susceptibility of this species
to feline infectious peritonitis virus (22, 63), which is perhaps due to limited genetic variability (92, 100). Vaccines have not been approved for use in non-domestic cats. Some captive non-domestic cat facilities require serum antibody tests and electron microscopy of faecal samples prior to introduction of new animals (153).

Canine distemper is discussed in greater detail above (see ‘Canidae’). This virus has recently been recognized as a significant pathogen of captive lions, tigers (Panthera tigris) and leopards (152), and free-ranging lions (90). Susceptibility of domestic cats to experimental canine distemper has been recognized (5), but this was considered unusual. A few deaths in captive wild felids were reported as early as 1983, in a Bengal tiger (P. tigris tigris) (16) and a Siberian tiger (P. tigris altaica) (54). Possible sources of canine distemper virus include contact with free-ranging and feral canids, raccoons (Procyon lotor) (8), or any of numerous other susceptible free-ranging species. At present, little is known about the susceptibility, transmission and pathogenesis of canine distemper in wild felids. Vaccination with inactivated canine distemper vaccines may be considered if exposure is possible; attenuated vaccines have not yet been tested for safety and efficacy in wild felids.

Several respiratory viral diseases occur in captive wild cats, including feline viral rhinotracheitis caused by feline herpesvirus type 1 and feline calicivirus infection (47). Feline herpesvirus has been isolated from cheetah (121, 132) and clouded leopard (Neofelis nebulosa) (18). Transmission of these viruses usually occurs via direct contact and possibly via fomites; high densities of cats in a captive situation probably increase transmission. Cats which have recovered from feline herpesvirus infections could infect other cats. Vaccines developed for domestic cats have been tested and used successfully in non-domestic cats (28, 126, 138).

Pseudorabies (Aujeszky’s disease), caused by a herpesvirus, is a minor consideration in captive non-domestic cats. This disease has most frequently been a problem in Central and Eastern Europe (56). In species other than swine, pseudorabies is manifest as ‘mad itch’. Swine are the major reservoir, and wild felids may develop pseudorabies following direct or indirect contact with swine, particularly by ingestion of pork (50, 56). The virus may survive in muscles of carcasses for 11-36 days (56) but the disease can be effectively prevented in wild felids by providing appropriately-processed diets.

Feline spongiform encephalopathy is a recently-identified member of the spongiform encephalopathy group of infectious diseases. Cases in captive cheetah (108) and mountain lion (148) have been associated with the bovine spongiform encephalopathy epizootic in Great Britain. Epidemiological evidence suggests that the cats were exposed via contaminated feed in Great Britain, and there is no evidence to date of horizontal or vertical transmission among non-domestic cats. The occurrence of a case in a cheetah imported to Australia (108) highlights the potential for moving unrecognized disease inadvertently over long distances.

Bacterial pathogens

Plague, caused by Yersinia pestis, may infect and cause illness and mortality in felids. Wild and domestic cats serve as a link between wild rodent plague and humans in North America (10). Unlike most carnivores, cats appear to be relatively susceptible to clinical disease induced by Y. pestis. Approximately 38% of experimentally-infected domestic cats succumbed to plague; the bacteria could be cultured from the oral cavity
for ten days post-inoculation (49). An adequate quarantine period for wild felids captured from areas with sylvatic plague – based on findings from experimental infections of domestic cats – is two weeks. It is recommended that wild felids from plague endemic areas be treated for fleas, as these could potentially harbour *Y. pestis*.

Bovine tuberculosis may occur in captive wild felids; cats are considered to be more susceptible to *M. bovis* infection than canids (134). Wild felids most likely contract tuberculosis from contaminated meat and offal. Unfortunately, diagnostic tests are not considered accurate in wild felids, and usual quarantine periods may not prevent the introduction of *M. bovis* into captive populations. Establishing the origin of wild felids and the possibility of exposure to tuberculosis before introduction into captive populations is prudent.

Anthrax, caused by *Bacillus anthracis*, has caused mortality in captive wild felines (46), particularly cheetahs (65). The route of transmission to these captive cats was via contaminated food. Use of appropriately-prepared foodstuffs should prevent the introduction of anthrax to captive felids.

Parasites

Endo- and ectoparasites in captive wild felids are relatively easy to treat topically, orally or by injectable preparations (118).

Wild felids can act as hosts for *Trichinella spiralis* (40, 154); due to the oral route of transmission, however, incorporation of an infected animal into captivity is unlikely to result in establishment of the parasite in captive populations. The potential introduction of *Trichinella* spp. into captive felids via contaminated foodstuffs, however, is a greater problem. Appropriate treatment of feed will reduce the chances of introducing this parasite into captive animals.

Most cestode parasites of wild felids are innocuous, with a few exceptions. Canids and rodents are the usual hosts of *Echinococcus multilocularis* (34), but wild felines may occasionally be infected (75). The parasite does not cause significant disease in cats. Humans, however, could be infected by contact with cat faeces which contain eggs (34), and untreated infections may be fatal in humans (149). Anthelmintic treatment is appropriate for animals removed from the wild; reinfection is unlikely to occur in captivity, unless the cats have access to infected intermediate rodent hosts.

Many wild and domestic cats are infected with *Toxoplasma gondii* (118), although toxoplasmosis is rare in cats. Transmission occurs via ingestion of infective oocysts or consumption of secondary hosts (e.g. mice and rats) harbouring protozoal cysts. Infected cats may be identified by faecal examination for oocysts. Oocysts are relatively resistant to environmental conditions and could be transmitted to in-contact species, some of which may be highly susceptible to toxoplasmosis, e.g. Callitrichidae (151) or black-footed ferrets (26).

**MUSTELIDAE**

**Viral pathogens**

Skunks (*Mephitis mephitis*) are one of the primary reservoirs of rabies in North America (74), but all species of mustelids are considered susceptible. The observations about rabies in Canidae and Felidae also apply to Mustelidae. A rabies vaccine has
recently been approved for use in domestic ferrets (*Mustela putorius furo*) in the United States of America (USA) (94). Inactivated vaccines are also appropriate for use in valuable non-domestic mustelids or in those which could be exposed to the virus by virtue of their housing.

Canine distemper is the most important viral infection of mustelids, although the fatality rate in each species is not known. Some, such as the black-footed ferret, are exquisitely susceptible to fatal infection (143). Based on the presence of seropositive individuals, other species appear to be somewhat resistant and to survive infection. Features of canine distemper in mustelids are similar to those in canids (23). Attenuated canine distemper vaccines prepared in avian cell lines have been used successfully in American badgers (*Taxidea taxus*) (51) and other mustelids (15). However, attenuated vaccines are not safe in all species (29). In addition to the risk of vaccine-induced canine distemper, attenuated vaccines induce immunosuppression in mustelids (69, 77). Quarantine periods of > 30 days are probably adequate to prevent introduction of canine distemper via wild-caught mustelids. Free-ranging black-footed ferrets and American badgers have been found to be incubating canine distemper when captured (143; D. Hoff and E.S. Williams, unpublished findings).

Parvoviruses cause several significant diseases in mustelids. Mink enteritis virus is closely related to feline panleukopenia virus and has caused significant disease in ranch mink (*Mustela vison*) (25). Mink are also susceptible to feline panleukopenia virus, but skunks are resistant to both feline panleukopenia virus and mink enteritis virus (9). Vaccines are available for mink virus enteritis and are commonly used on fur farms.

Aleutian disease, also caused by a parvovirus, is an important disease in farmed mink, particularly animals of the Aleutian colour phase (105). Skunks (64), domestic ferrets (71, 72, 101, 112, 141), and possibly otter (*Lutra lutra*) (142) and other species (72) are also susceptible to strains of this virus. Transmission occurs via direct contact, fomites or vertical transmission. Transmission is suspected to have occurred via inadequately-cleaned cages after several years (37). The virus is shed in saliva, urine and faeces (52) for long periods; but it is not considered highly contagious (105), although high densities of animals increase transmission. As with all parvoviruses, Aleutian disease virus is very resistant to environmental inactivation (105). In the fur industry, where Aleutian disease has caused the greatest problems, test and slaughter programmes have been used to eradicate the disease on premises, and testing followed by isolation or removal has been suggested for commercial ferret facilities (103). This may not be possible in groups of valuable or endangered captive mustelids; in such circumstances, serological testing of animals and exclusion from the facility of those which are seropositive should be used to prevent introduction of the disease. As Aleutian disease is more severe in the Aleutian genotype than in standard mink, it is possible that endangered, possibly inbred, mustelids could be seriously affected by this virus.

Transmissible mink encephalopathy is a member of the spongiform encephalopathy group of diseases (81). This disease is rare in the mink industry, but it is of interest in view of the similarity to bovine spongiform encephalopathy, scrapie and human diseases. Transmissible mink encephalopathy is transmitted orally via consumption of contaminated feed – possibly contaminated with the agent of scrapie – (62, 80) or via cannibalism (24). The disease has been experimentally reproduced in other mustelids (81). There is no evidence of natural transmission by means other than contaminated
feed, and use of uninfected feed for mustelids should therefore prevent introduction of the disease to captive animals. While spongiform encephalopathy in wild felids occurred during the recent bovine spongiform encephalopathy epizootic in Great Britain, there was no report of spongiform encephalopathy in mustelids.

**Bacterial pathogens**

Mustelids are susceptible to bovine tuberculosis, and may serve as significant wild reservoirs of the disease. Tuberculosis has been reported in free-ranging European badgers (*Meles meles*) (32) and feral domestic ferrets in Great Britain and New Zealand, respectively. These species could become infected in captivity via contact with tuberculous individuals or contaminated feed.

**URSIDAE**

There are few reports of infectious diseases in captive Ursidae, suggesting either that bears are unusually resistant to the variety of pathogens which affect the other carnivore families or, more probably, that they have not been studied in such great detail.

**Viral pathogens**

Bears appear to be relatively susceptible to infection, and mortality due to infection with pseudorabies virus has occurred in brown bears (*Ursus arctos*) (V. Guberti, personal communication). Pseudorabies in bears is similar to that in other carnivores (see ‘Felidae’ above). Avoiding contaminated feed — particularly fresh swine products in areas where pseudorabies is endemic — should prevent introduction of the disease to captive bears.

Canine adenovirus type 1, or canine hepatitis virus, has caused mortality in captive black bears (*U. americana*) (114). Bears may be susceptible to attenuated vaccines (46). Canine adenovirus may cause significant illness and death, or may lead to subclinical infection. The source of the virus in captive bears is most likely to be contact with infective feral or captive canids.

**Parasitic pathogens**

Infection by *T. spiralis* is found in wild (154) and captive bears (118). Fowler (46) reported an infected polar bear (*U. maritimus*) after at least ten years of captivity. In captivity, these animals are dead-end hosts. *Trichinella*-infected rodents could be sources of infection for captive carnivores (118).

**PROCYONIDAE**

**Viral pathogens**

Raccoons are important reservoirs of rabies in eastern North America (74). The recent outbreak in the north-eastern USA serves as a dramatic example of the dangers of movement of wild animals without appropriate regulation and quarantine; rabid
raccoons were inadvertently introduced into West Virginia from the south-eastern USA. Free-ranging raccoons could be a source of rabies for captive wild species in endemic areas.

Canine distemper is the most important viral infection of procyonids in captivity and the wild (23). All species appear to be susceptible to infection, and some appear to be particularly susceptible; vaccine-induced canine distemper has been reported in kinkajous \((Potos flavus)\) (70) and red pandas \((Ailurus fulgens)\) (27). Attenuated canine distemper vaccines may be immunosuppressive in procyonids; a red panda died due to an opportunistic infection following vaccination (87). Sources of canine distemper in captive procyonids are contact with infected feral or other captive species. Degree of risk is dependent on the management of the animals.

Raccoons are susceptible to feline panleukopenia virus and mink enteritis virus (9), and a closely-related raccoon parvovirus (96), but do not develop clinical signs following infection with canine parvovirus (6, 9). Other procyonids (88) are probably susceptible to parvoviral enteritis, although this is seldom reported in the literature.

Experimental and natural pseudorabies infections have been reported in raccoons (110). Transmission of the virus between raccoons by direct contact (110) suggests that this species could serve as a reservoir, but most raccoons are probably dead-end hosts. Captive procyonids could contract the disease via contact with infected swine.

**Parasitic pathogens**

Internal parasites are common in procyonids, and include some \((e.g.\ Baylisascaris procyonis)\) (120) which may be pathogenic in other animal species and in humans. Anthelmintic treatment of captive procyonids should be routine in areas where parasites are a problem.

**VIVERRIDAE AND HYAENIDAE**

**Viral pathogens**

Very little information is present in the literature on diseases of viverrids and hyenas. Reviews suggest that species in these families are susceptible to the same diseases as canids and felids (42, 116). These comments highlight how little is known about diseases of these animals.

Mongooses \((Cynictis penicillata, Herpestes auropunctatus)\) are important reservoirs of rabies in Africa and the Caribbean (30, 74). Rabies also has been reported in hyenas \((Crocuta crocuta)\) in Africa (86). Concerns about the capture of wild viverrids and hyenas which are incubating rabies are similar to those observed for the other carnivore families, and lengthy quarantine periods may be appropriate (94). Species in these families are assumed to be susceptible to canine distemper (23) and parvoviruses (88), but this is based on few reports. As with other species, use of inactivated vaccines is recommended unless attenuated vaccines have been shown to be safe in a particular species. Those studying or managing viverrids and hyenas should be encouraged to publish information on disease susceptibility in these species.
DISEASE IN BLACK-FOOTED FERRETS

The history of the endangered black-footed ferret in captivity provides a good illustration of the real and potential problems with infectious and parasitic diseases which face managers of captive wild species, and of the interface between the wild and captivity. This also demonstrates the usefulness, but also the problems, of assuming that diseases of closely-related species – e.g. domestic ferrets or Siberian polecats (*M. eversmanni*) – will be the same as those of black-footed ferrets. Captive black-footed ferrets are susceptible to a number of diseases, and management practices therefore attempt to minimize the introduction of exotic pathogens into the captive population and hence into the wild via reintroduction. Understanding of the disease susceptibility of black-footed ferrets continues to increase over time.

Recovery efforts for the black-footed ferret have involved removal to captivity of members of this species from the last known wild population, in Wyoming (USA), followed by an aggressive captive breeding programme and return of animals to the wild. Management of infectious diseases has played an important and varying role in all phases (145).

**Viral pathogens**

The real threat of moving infectious diseases from the wild to captivity along with free-ranging individuals was exemplified by the experience with six black-footed ferrets captured in 1985 for captive breeding. Two wild-caught black-footed ferrets were incubating canine distemper; these animals subsequently died, as did four in-contact animals (133, 143). Canine distemper is the most important infectious disease of mustelids, especially in highly-susceptible black-footed ferrets. Although impractical in some situations, individual (rather than group) isolation for quarantine could prevent transmission of canine distemper between recently-captured animals. Extensive disease prevention precautions are in place through the Black-Footed Ferret Species Survival Plan, which requires that the animals be kept in isolation facilities, and that personnel must take a shower or change clothing, as well as wearing a face-mask and disinfecting their hands, prior to contact with black-footed ferrets (145). This is necessitated by several factors, namely the ubiquity of canine distemper virus, the relatively small population of black-footed ferrets, the 100% mortality expected from this disease in susceptible animals; and the absence of a completely effective vaccine. Inactivated canine distemper vaccines are used in black-footed ferrets (145), but in trials with black-footed ferret × Siberian polecat hybrids, morbidity and mortality occurred following challenge with virulent virus (77, 147). Some attenuated vaccines induce canine distemper in black-footed ferrets (29; E.S. Williams, unpublished findings) and may induce significant immunosuppression (77). Research is continuing for the development of a safe and effective vaccine.

Rabies is always a consideration when moving carnivores from the wild to captivity. In the case of the black-footed ferret, rabies was not considered to be a significant threat. The distribution of rabies in wild and domestic animals is well known in Wyoming, due to continuous diagnostic surveillance for this disease. The source population of captive black-footed ferrets was from an area where no cases of rabies had been reported. This exemplifies the usefulness of background information on the occurrence and distribution of animal diseases when collecting free-ranging animals for captivity.
Aleutian disease virus is considered a potential pathogen of black-footed ferrets and one which could have serious ramifications on wild and captive populations. The disease is currently absent in the captive population, and Species Survival Plan management of the animals seeks to prevent its introduction. Black-footed ferrets are isolated from other mustelids (particularly mink or other ferrets) not shown to be free of Aleutian disease. Siberian polecats – maintained for cross-fostering purposes, breeding experience (for the males) and research – were from an Aleutian disease-free population. Contact between black-footed ferrets in outside facilities and free-ranging mustelids is prevented by the use of double fencing. Serological screening for Aleutian disease antibodies is recommended in the future, if animals are moved from the wild to captivity, or within the captive population if the Species Survival Plan disease prevention protocol is changed. This is particularly true as at least one of the black-footed ferret reintroduction sites contains seropositive skunks (E.S. Williams, unpublished findings).

Human influenza has been documented as causing disease in domestic ferrets (48). Thus, when caretakers of black-footed ferrets are ill they do not handle the animals, and all handlers wear face-masks and disinfect their hands when in direct contact with these animals. To date, influenza has not been observed in black-footed ferrets.

Introduction of enteric diseases is of concern, due to the susceptibility of domestic ferret kits to rotavirus-induced morbidity and mortality (48). Rotavirus has been suspected in some cases of diarrhoea in black-footed ferrets, where virus has been demonstrated by negative-stain electron microscopy and by antigen capture ELISA (E.S. Williams, unpublished findings). It is possible that rotavirus carriers were brought in from the wild, that rotaviruses were introduced via food contaminated with the virus, or that the virus was introduced via fomites. Isolation procedures, including the use of footbaths and showers, serve to decrease the possibility that enteric viruses will be introduced.

The apparent heightened susceptibility of black-footed ferrets to a number of infectious disease agents has raised concerns about retroviral infections. Some of these viruses are known to cause significant immunosuppression in other species. The authors are currently testing black-footed ferret sera and tissues for evidence of retroviruses. These viruses could cause significant damage to captive propagation efforts for this endangered species. Introduction of retroviruses should therefore be prevented by isolation and lack of contact with other mustelids.

**Bacterial pathogens**

The most serious bacterial disease of black-footed ferrets is plague (146). The recently-recognized susceptibility of black-footed ferrets to plague exemplified the need to be cautious in extrapolating the significance of disease in closely-related species (e.g. domestic ferrets) which are resistant to parenteral challenge with *Y. pestis* (144). Risk of this disease is minimized in the captive environment by strict quarantine procedures for prairie dogs (*Cynomys* spp.), which form part of the diet of black-footed ferrets (145), and by the use of isolation facilities. Wild-caught prairie dogs are maintained in quarantine for at least ten days, which is considered to be longer than the incubation period of plague in white-tailed prairie dogs (E.S. Williams, unpublished findings), and necropsy is performed on all animals which die during quarantine to determine the cause of death. This not only serves to ensure that the
black-footed ferrets have a food source which is free of plague, but also reduces the danger to the humans who handle and process the prairie dogs and care for the captive black-footed ferrets.

Black-footed ferrets in external cages in locations with endemic plague are not protected from this disease (146). While these cages prevent the escape of black-footed ferrets and the entry of large species, small free-ranging rodents – some of which might carry plague – could gain access to the cages. Black-footed ferrets which have been in external cages are not returned to the breeding colony until they have completed a six-week quarantine period in individual isolation.

Parasites

Parasites of black-footed ferrets and their prairie dog prey have been studied (17, 68, 117, 125). Internal parasites probably constitute the greatest disease management problem in black-footed ferrets on a day-to-day basis. _Eimeria ictidea_ and _E. furonis_ are natural parasites of black-footed ferrets (68), and coccidiosis is a problem primarily in young ferrets (kits) (145). The latter problem is managed by attention to sanitation and by treatment with anticoccidial agents when clinical signs are apparent. Even with good sanitation, the method of management of these animals – with nest boxes and artificial tunnels to simulate prairie dog burrows – does not totally prevent contact of kits with faeces and hence with the parasite.

Another important parasite of captive black-footed ferret kits and a few adults is _Cryptosporidium_ sp., which causes ill thrift and mucoid diarrhoea. This parasite was not anticipated to be a significant pathogen of black-footed ferrets, on the basis of information on known diseases of mustelids. It is not known whether the parasite is natural in this species (i.e. present in free-ranging populations prior to their capture), or whether black-footed ferrets acquired this parasite in captivity. _Cryptosporidium_ is a very common parasite and could have been introduced through food, contact with human carriers, or water. There are no specific treatments for this parasite, although some antibiotics – azithromycin, in particular – have shown some efficacy in reducing clinical disease in captive black-footed ferrets (E.S. Williams _et al._, unpublished findings).

An outbreak of toxoplasmosis was documented at one captive breeding facility (26). Source of infection was not definitively determined but was felt to be associated with rabbit meat used in the diet. This outbreak appeared to be due to a single exposure to the pathogen; the disease has not recurred in this or other captive breeding facilities. In addition, there has been no clinical evidence of transmission from black-footed ferret dams to offspring. This outbreak served to highlight several points about disease and captive maintenance of endangered species: first, some of these species may have a heightened degree of susceptibility by virtue of the degree of inbreeding, the stresses of captivity, or infection by immunosuppressive agents; and second, that – even with relatively strict quarantine procedures – introduction of significant pathogens is possible and surveillance should therefore remain high.
LES MALADIES INFECTIEUSES ET PARASITAIRES DES CARNIVORES VIVANT EN CAPTIVITÉ, ET PARTICULIÈREMENT DU PUTOIS D’AMÉRIQUE (MUSTELA NIGRIPES). - E.S. Williams et E.T. Thorne.

Résumé : Les carnivores vivant en captivité peuvent souffrir d’une grande variété de maladies infectieuses et parasitaires, en raison de la diversité des sept familles de Carnivores. Malheureusement, rares sont les études approfondies portant sur les maladies des espèces non domestiques de carnivores, et beaucoup de lacunes subsistent, notamment en ce qui concerne les maladies affectant les petits carnivores (c’est-à-dire les mustélidés, les viverridés et les procyonidés). Les principales maladies des carnivores sont la rage, la maladie de Carré et les infections dus à des parvovirus, des coronavirus et des herpesvirus. Les maladies parasitaires ou bactériennes ne présentent guère de danger pour les populations élevées en captivité, et un élevage correctement mené, l’application de médicaments, la vaccination et la quarantaine suffisent à réduire le risque de maladie. S’agissant de la susceptibilité à l’égard des agents pathogènes, la connaissance que l’on a d’une espèce peut induire en erreur lorsqu’on l’applique à une autre espèce. Le putois d’Amérique (Mustela nigripes) est un exemple de carnivore particulièrement touché par des maladies infectieuses, dont certaines sont prévisibles tandis que d’autres ne peuvent l’être à partir de la seule connaissance des maladies des autres mustélidés. Ceci souligne le besoin de mieux comprendre l’histoire naturelle de chaque espèce maintenue en captivité.


ENFERMEDADES INFECCIOSAS Y PARASITARIAS DE CARNÍVOROS EN CAUTIVIDAD, CON ESPECIAL REFERENCIA AL TURÓN PATINEGRO (MUSTELA NIGRIPES). – E.S. Williams y E.T. Thorne.

Resumen: Los carnívoros en cautividad son sensibles a un amplio espectro de enfermedades infecciosas y parasitarias, hecho que refleja la diversidad existente entre las siete familias del orden Carnívora. Por desgracia, los estudios sobre las enfermedades de los carnívoros no domésticos son relativamente escasos y queda mucho por aprender, en especial sobre las enfermedades de los pequeños carnívoros (por ejemplo los mustélidos, vivérvidos y procionídos). Entre las enfermedades infecciosas más importantes de los carnívoros se encuentran la rabia, el moquetillo canino y las afecciones causadas por parvovirus, coronavirus y herpesvirus. Pocos son los patógenos bacterianos o parasitarios importantes en lo que concierne a las poblaciones cautivas, y las adecuadas prácticas de manejo, terapia, vacunación y cuarentena minimizan el riesgo de enfermedad. Las extrapolaciones de una especie a otra sobre la sensibilidad a enfermedades pueden resultar incorrectas. El turón patinegro (Mustela nigripes) constituye un ejemplo de carnívoros especialmente afectado por enfermedades infecciosas, algunas de las cuales eran previsibles, mientras que otras no podían preverse a partir del conocimiento general de las enfermedades que afectan a los mustélidos. Esto pone de relieve la necesidad de comprender la historia natural de cada una de las especies mantenidas en cautividad.

REFERENCES


