Disease and Endangered Species: The Black-footed Ferret as a Recent Example

E. TOM THORNE*
Wyoming Game and Fish Department
Research Laboratory
Box 3312 University Station
Laramie, Wyoming, U.S.A. 82071

ELIZABETH S. WILLIAMS
Department of Veterinary Science
University of Wyoming
Laramie, Wyoming, U.S.A. 82070

Abstract: Diseases may play major roles in the conservation of endangered species. Although the threat of disease received extensive consideration and influenced research and management activities governing the endangered black-footed ferret (Mustela nigripes) in Wyoming, a canine distemper epizootic in 1985 severely affected a captive breeding program and led to extirpation of the species from the wild. This recent example of the catastrophic effect of epizootic disease in an endangered species is described in an historical context. In addition, examples are given of disease further endangering other rare species, including Mauritius pink pigeon, Père David’s deer, cranes, maned wolves, native Hawaiian birds, cheetahs, and others.

Resumen: Las enfermedades pueden jugar un papel importante en la conservación de especies en peligro de extinción. Aunque la amenaza de enfermedad recibió gran consideración e influyó en la investigación y las actividades de manejo del hurón de patas negras (Mustela nigripes), especie en peligro de extinción en Wyoming, en 1985 el distemper canino epizootico impactó severamente un programa de crianza en cautiverio y condujo a la extinción de esta especie en su ambiente silvestre. Este reciente ejemplo del efecto catastrófico de una enfermedad epizootica sobre una especie en peligro de extinción es descrito en un contexto histórico. Además, se dan ejemplos de enfermedades que amenazan aún más a otras especies raras, incluyendo la paloma rosada de Mauricio, el ciervo de David, el lobo de crín, el Cheeta, diversas grullas, varias aves de las islas de Hawai, entre otros.

Introduction

Although the literature presents numerous reports of disease-related mortality among free-ranging and captive wild animals, the occurrence of disease and its effects on threatened and endangered species is only infrequently documented. Yet, it is among these vulnerable species that disease may have major consequences for conservation of the species, including extinction. There are, however, a few documented epizootics and epornitics among threatened and endangered species. Many of these have involved captive animals, compromising captive propagation programs through decreased numbers of breeders and loss of genetically valuable individuals. The intent of this
paper is to describe the events leading up to and surrounding a case in which disease played a major role in the management and conservation of an endangered species and to provide examples of other diseases that may affect endangered species conservation programs.

Canine distemper is a highly contagious disease of domestic dogs and some wild carnivores (Budd 1981), and morbidity and mortality may be quite high in susceptible populations (Appel et al. 1981). The disease, caused by a morbillivirus of the paramyxovirus group, occurs throughout the world and is ubiquitous in domestic and wild species (Budd 1981). Closely related viruses include rinderpest and pest des petite ruminants of ruminants and measles of man (Appel et al. 1981). The virus, which is fragile in the environment, is readily transmitted by aerosol and by direct contact between infected and susceptible animals. Virus occurs in nasal and ocular discharges as well as urine and feces (Appel et al. 1981). Transmission by fomites is likely when conditions are suitable for virus survival, for example, in an underground burrow where the virus would be protected from sunlight, desiccation, and temperature vacillation. Because canine distemper spreads through susceptible populations primarily by direct contact, transmission should be limited by decreased opportunity for contact caused by declining numbers as animals die or are removed from the population.

All canids, mustelids, procyonids, and at least one viverrid are susceptible to canine distemper (Budd 1981). Domestic ferrets (Mustela putorius furo) experience essentially 100 percent morbidity and mortality (Appel et al. 1981, Bernard et al. 1984, Davidson 1986). Susceptibility of the black-footed ferret was demonstrated in the 1970s, when four of six black-footed ferrets vaccinated against canine distemper with modified live virus vaccine died of canine distemper (Carpenter et al. 1976). This vaccine had been previously shown to be safe in domestic ferrets and Siberian polecats (Mustela eversmanni). Canine distemper is common in dogs in Wyoming, and wild animals known to be susceptible and present in Park County include coyote (Canis latrans), red fox (Vulpes vulpes), badger (Taxidea taxus), mink (Mustela vison), skunk (Mephitis mephistis), raccoon (Procyon lotor), and weasel (Mustela frenata) (Budd 1981, Williams 1982).

The black-footed ferret is the only North American member of the polecats group or subgenus Putorius (Anderson et al. 1986). Black-footed ferrets once ranged from the southeastern corner of Alberta and southwest corner of Saskatchewan to Texas (Henderson et al. 1974, Hall 1981). The species seemed nearly extinct until it was rediscovered in September 1981 near Meeteetse, Wyoming (Nowak & Paradiso 1983).

Black-footed ferrets are small, weasel-like animals; females weigh about 700 g and males weigh about 1100 g. Their habitat consists of prairie dog colonies, and they are largely dependent upon prairie dogs for food and shelter. Black-footed ferrets are nocturnal and fossorial. Prairie dog burrows are used for shelter and escape cover; ferrets feed primarily upon prairie dogs, most of which are probably killed underground (Nowak & Paradiso 1983).

The decline of the black-footed ferret has been attributed to conversion of prairie habitat to agriculture and systematic efforts over the past 100 years to eradicate prairie dogs. Ferrets were unintentional victims of prairie dog eradication efforts, and their numbers dwindled as prairie dog towns were plowed under and prairie dogs were poisoned (Anderson et al. 1986, Flath & Clark 1986). A small population of ferrets was discovered in 1964 in South Dakota and studied during the ensuing years. Beginning in 1971, animals from that population were captured for a captive breeding effort by the United States Fish and Wildlife Service (FWS). Only nine animals were available for captive propagation, and four of these were lost to the vaccine-induced canine distemper. Nonetheless, two litters of five each were produced. In each case, four babies were born dead and the fifth died within one or two days despite intensive medical support (Carpenter & Hillman 1979, Hillman & Carpenter 1983, Carpenter 1985). The South Dakota population disappeared in the 1970s.

A Historical Account of Black-footed Ferrets and Disease in Wyoming

A recent and most dramatic example of the importance of disease to endangered species was the 1985 canine distemper epizootic that occurred in Park County, Wyoming, in the only known colony of the black-footed ferrets (Williams et al., in press). Shortly after the black-footed ferret was rediscovered in 1981, the FWS shared with the state of Wyoming responsibility for making decisions necessary for preserving the species. This was done by allowing the Wyoming Game and Fish Department (WGFD) to become the lead agency in management of the black-footed ferret under authority granted to the Service in the Endangered Species Act of 1973. One of the first actions of the WGFD was to form an interagency Black-footed Ferret Advisory Team (BFAT) to advise the department in decision making regarding research and management of the ferret (Carr 1986, Thorne 1987). From the beginning, diseases were recognized by the WGFD and BFAT as a major threat to the ferret colony, and WGFD and BFAT, along with FWS, implemented extensive precautions designed to minimize opportunity for introduction of disease by researchers and managers (Thorne et al. 1985, Thorne 1987).

We are unaware of any free-ranging species, endangered or otherwise, that has received greater consider-
ation about disease. Domestic ferrets, which have been studied extensively, and, presumably, black-footed ferrets are susceptible to many diseases and parasites (Carpenter & Hillman 1979, Bernard et al. 1984). Unfortunately, there is little that can be done to protect a free-ranging mustelid population, no matter how small, from diseases that occur naturally in their environment.

Precautions were taken, however, to minimize the opportunity for introduction of disease by researchers and managers (Thorne et al. 1985). Biologists were not allowed to take dogs to the study site and were admonished to avoid contact with strange dogs, which might have canine distemper. Domestic ferrets are susceptible to human influenza, and, presumably, black-footed ferrets also are susceptible. Consequently, persons suffering from influenza were asked not to participate in field activities, such as trapping, that might bring them into contact with a ferret and lead to its exposure. In addition, everyone near a trapped ferret was required to wear a surgical mask to prevent accidental exposure by someone unaware he or she was infected with influenza. To reduce further the chance of exposing a ferret to influenza or canine distemper, all participants in the process of handling ferrets washed their hands with chlorhexidine scrub (Nolvosan Scrub, Aveco Co., Fort Dodge, Iowa) and rinsed them with virucidal disinfectant (Nolvosan Solution, Aveco Co.). Chemically immobilized ferrets were given a prophylactic dose of penicillin G benzathine and penicillin G procaine, and a mild antibacterial ointment was placed in their eyes. When ferrets were trapped or immobilized for research, a veterinarian was always required to be present (Thorne et al. 1985).

On the advice of a number of consultants, most black-footed ferrets captured for research purposes, which began in 1982, were vaccinated with an inactivated canine distemper vaccine (Williams et al., in press). The same consultants warned that an inactivated vaccine might not be highly immunogenic in black-footed ferrets, that frequent booster doses should be administered, and resistance, if produced, would be of short duration. No attempt was made to trap ferrets for the sole purpose of vaccination because it was not deemed practical.

Concern about the effects of disease was best demonstrated after the discovery, in the spring of 1985, of sylvatic plague in prairie dogs in the complex of towns that supported the black-footed ferrets (Thorne 1987; Forrest et al., in press). This generated a great deal of concern about the future of the prairie dog complex, because it was generally believed that epizootic sylvatic plague results in nearly complete extirpation of prairie dogs in affected colonies (Barnes 1982). Consequently, what may have been the most extensive nonpublic health, nonurban sylvatic plague control effort was enacted; its objective was to preserve the prey base of black-footed ferrets. Approximately 80,000 prairie dog burrows were individually dusted with 5 percent carbaryl to kill flea vectors of sylvatic plague.

As expected, there was no black-footed ferret mortality that could be attributed to sylvatic plague; indeed, subsequent research showed domestic ferrets and Siberian polecats to be resistant to plague (Williams, unpublished data). Although the dusting effort did not eliminate sylvatic plague from the prairie dog complex and there were localized areas where prairie dog mortalities were high, expansion of prairie dogs into other areas resulted in <20 percent loss of occupied prairie dog towns by 1986 (Thorne 1987; Forrest et al., in press; Thorne & Belitsky, in press).

In addition to the flea dusting effort, a contingency plan was developed and arrangements made to capture and remove black-footed ferrets from affected prairie dog towns if it was demonstrated that ferrets were being adversely affected by the disease. Plans were made for capture and transport of ferrets to WGFD's Sybille Wildlife Research Unit near Laramie. Caging was available for six animals and temporary caging identified for an additional 20 ferrets. Arrangements were made to move, after a preliminary quarantine period, ferrets in excess of 12 to the Smithsonian Institution's Wildlife Conservation and Research Center, Front Royal, Virginia and the Fish and Wildlife Service's Denver Wildlife Research Center. Because ferrets were not shown to be adversely affected by plague, these contingency plans were not implemented.

The first meeting for the express purpose of discussing captive breeding of black-footed ferrets was held in April 1984 at WGFD headquarters in Cheyenne (Carr 1986, Thorne 1987). That was followed by progressive planning for captive propagation that included a committee report (Anonymous 1984), discussions on captive propagation at BFAT meetings, a workshop in 1984 (Anderson & Inkley 1985), funding requests, a site analysis (Reese 1985), and a recovery meeting at FWS offices in Denver in May 1985 (WGFD 1987). During the May meeting, FWS representatives for the first time expressed optimism that money for captive breeding, according to proposals previously submitted by WGFD, would be available in 1986 or 1987. Consequently, it was decided at that meeting to capture ferrets after field biologists completed 1985 population estimates. The fact that search efforts had failed to locate additional ferret colonies was becoming ominous; the ferrets to be captured in the fall of 1985 were to serve as a hedge against a catastrophe befalling the single known colony of ferrets and also to serve as founder animals for a captive breeding effort (Thorne 1987, Thorne & Belitsky, in press).

During the summer of 1985, preparations were made to house the ferrets in temporary quarters in the existing veterinary laboratory at Sybille. Modifications, in-
including a shower-in facility, were made to protect captive ferrets from exposure to disease and unnecessary disturbance. Three males and three females were to be captured in order to provide the best founder genetic diversity, and one of the males was to be mature to provide an experienced male to enhance the likelihood of breeding. Ferrets were to be captured in pairs at 10- to 14-day intervals to allow time to be sure the first captured animals were healthy and adapting to captivity. Additional ferrets would be captured in 1986 and possibly 1987, and the number taken each year would be small enough so as not to affect the free-ranging population adversely (Thorne 1987; Thorne & Belitsky, in press).

In late September and early October 1985, a scant four years after the colony's discovery, six ferrets were captured and moved to Sybille according to plan. Two adult males were included in the group, and there was a great deal of optimism about the start of captive propagation of black-footed ferrets.

However, the catastrophe the capture of these six animals was supposed to help prevent was already occurring, and they were a part of it. Field observations and population estimates by researchers demonstrated a drastic ongoing decline (Forrest et al., in press) and a meeting was called in Cheyenne on October 22 by WGFD and FWS to discuss possible causes of the decline and courses of action. In addition, the last two ferrets captured were ill with a serious disease they undoubtedly contracted in the wild before capture. One died the day before the Cheyenne meeting and canine distemper was diagnosed (Williams et al., in press).

On October 22, the presence of canine distemper was reported and its probable consequences explained. Although a great deal of precaution had been taken to prevent introduction of disease to captive ferrets from outside the ferret colony, none had been taken to prepare for disease originating from within the colony. Consequently, it was probable that all captive ferrets had been exposed and would die; they did (Williams et al., in press). In addition, trap records showed that two ferrets unwittingly captured with the disease came from widely separated locations, thus indicating that the disease was widespread across the ferret colony. Complete extirpation of the core ferret colony could be expected. The cause of the population decline that apparently had been occurring during the summer and fall could be explained by a canine distemper epizootic.

After much discussion, a decision was made on the evening of October 22 by WGFD and FWS to capture all ferrets that could be located in the main colony; no effort would be made to capture ferrets in more outlying colonies because few had been located there by field biologists during the summer, and a great deal of time and effort would be required in those areas compared to the core colony. The rationale behind the emergency capture was to attempt to remove unexposed ferrets from the colony for future use as founder animals; if left in the colony, it was expected they would ultimately be exposed to distemper and die. If diseased ferrets were captured, their removal from the colony would reduce the opportunity for transmission to other animals. Although very few ferrets had been located in outlying towns during 1985 summer surveys, the fact that none of the 49 animals that were ear-tagged as juveniles in 1984 had been relocated or captured in the core colony in 1985 (WGFD 1987; Forrest et al., in press) suggested that a few may have dispersed into those outlying towns. Low ferret density in these areas justified a hope that a few might have escaped exposure to canine distemper and would repopulate the core area after the epizootic had run its course (Thorne 1987; Williams et al., in press; Thorne & Belitsky, in press).

Six ferrets—two juvenile males, one juvenile female, and three mature females—were captured during the emergency trapping (Thorne 1987). They were placed individually in isolation and none developed canine distemper, indicating they had not been exposed when captured.

Because the two males were apparently physiologically and psychologically immature, no breeding occurred during the 1986 breeding season (DonCarlos et al., in press). During the winter and spring of 1986, WGFD prepared and approved a contingency plan to assist in the decision making process regarding how to manage ferrets that escaped distemper and were located during 1986 summer surveys (WGFD 1986). Five or six ferrets did escape canine distemper and were located during spotlighting surveys in July and August 1986; two were females that produced litters. Although it was gratifying that a few ferrets remained after the canine distemper epizootic, it was apparent there were not sufficient ferrets present to constitute a viable population. Under guidelines of the contingency plan, a decision was made by WGFD and FWS to capture all remaining ferrets for captive propagation (Thorne 1987, WGFD 1987).

During the summer and fall of 1986 one mature and three juvenile males and two mature and five juvenile females were captured. In February 1987 a mature male was captured, and no sign of another free-ranging ferret has been reported (WGFD 1987). There are 18 wild black-footed ferrets in captivity and they apparently are all that remain of what is now probably North America's most endangered mammal. The canine distemper epizootic in the only known black-footed ferret colony had devastating effects. The free-ranging colony was essentially extirpated. The captive breeding program quickly went from a carefully planned approach with ideally selected, unrelated founder animals to a crisis situation with related animals, a poor sex ratio, and few mature, experienced...
breeder males. In addition, there likely will be no opportunity to add to the original 18 animals in the captive colony.

In retrospect, it is unfortunate that ferrets were not captured for captive propagation in 1984 when the population reached its peak size. This had been recommended; certainly WGFD and FWS received much criticism for not having done so (Carr 1986, May 1986). Although temporary quarters could have been prepared at Sybille in 1984 as was done in 1985, in 1984 there was neither promise nor even a suggestion of short- and long-term funding for a captive propagation effort from either federal, state, or private agencies and organizations. Nor had any offers been made by institutions outside of Wyoming to take ferrets and participate in captive breeding.

If black-footed ferrets had been captured in 1984, it likely would have been six to 10 animals. Capture of ferrets in 1984 would not have influenced the 1985 canine distemper outbreak, and most likely it would not have influenced the way WGFD and FWS responded to the epizootic. Consequently, initiation of captive breeding in 1984 would have resulted in 24 to 28 wild caught animals rather than 18. The genetic diversity of the captive population probably would have been better.

There also has been an equal but less articulate criticism that the black-footed ferrets were "studied to death" and their capture led to the population's demise; that they would have been better off if they were never discovered. This, of course, is even less valid. Their non-discovery would not have prevented the canine distemper epizootic, it simply would have gone unnoticed and the species might now be extinct.

Disease in Other Rare Species

There are other examples of disease severely affecting populations of both free-ranging and captive endangered species. Obviously, epizootics occurring in captivity are more carefully documented. Of the wide variety of methods by which pathogens may be transmitted (Thrusfield 1986), several means seem to pose the greatest danger to captive propagation programs by virtue of detection difficulties or requirement for elaborate management efforts to exclude them. The situation for managers is even more difficult in the field as far less environmental control is possible. Characteristics of the host, especially immunologic status, may also influence the interaction of pathogen and endangered species. A brief discussion of examples of infectious diseases in rare or endangered species follows.

Of particular danger are pathogens brought to endangered animals by the insidious method of carrier animals or reservoir hosts. Carrier animals harbor pathogens and provide sources of infection for endangered species, but they are either unaffected, subclinically affected, incubating the disease and shedding the pathogen, or recovered from the infection but transmitting the disease. The Mauritius pink pigeon (Columba mayeri) is threatened with extinction in its native island habitat of Mauritius, and captive propagation is being attempted at several zoos. At the Rio Grande Zoo, Albuquerque, New Mexico, a well-managed, presumably disease-free, flock of domestic pigeons was established because foster rearing appeared to be an ideal solution to a problem encountered when flighty and unpredictable pink pigeons abandoned eggs. However, pink pigeon chicks, hatched by foster pigeons, died suddenly at 5 to 10 days of age. Pigeon herpesvirus was isolated from the dead chicks and was found to be enzootic but undetected in the domestic flock until susceptible pink pigeons were introduced and succumbed to the infection (Snyder et al. 1985). Latently infected pigeons re-excrete herpesvirus during the reproductive period, thus serving as a source of virus for uninfected young (Vindevogel et al. 1985). This case demonstrated the potential hazard from exposure of related but geographically isolated species that differ markedly in their susceptibility to a disease that may be innocuous in one species and fatal in another.

The disease malignant catarrhal fever is transmitted by reservoir hosts to other susceptible species, including endangered mammals. Père David's deer (Elaphurus davidianus) exist only in captivity. In 1959 an outbreak of malignant catarrhal fever occurred in the captive herd at Whipsnade Park, United Kingdom, and eight animals in a group of 14 were lost (Huck et al. 1961, Senior et al. 1962). Père David's deer also died in a more recent outbreak in Whipsnade Park (Ashton 1982) and at other zoos (Heuschele 1982). Additional rare and endangered ungulates, including Indian gaur (Bos gaurus) (Hatkin 1980, Castro et al. 1982, Heuschele 1982), bongo (Taurotragus eurycerus isaaci) (Heuschele 1982), banteng (Bos javanicus) (Hatkin 1980, Heuschele 1982), and Arabian oryx (Oryx leucoryx) (Heuschele 1982), have also died of malignant catarrhal fever in several zoos. The African malignant catarrhal fever virus (Alcelaphine herpesvirus, bovine herpesvirus 3) is well adapted to wildebeest (Connochaetes sp.), which carry the virus without apparent disease (Plowright et al. 1960). Virus is shed during parturition and by neonatal wildebeest calves. A similar virus may cause “sheep-associated” malignant catarrhal fever, but the agent has not yet been isolated (Hamdy et al. 1978, Rossiter 1981). Malignant catarrhal fever continues to threaten endangered ruminants held in facilities with reproducing wildebeest and possibly sheep.

In 1978 an outbreak of inclusion body disease of cranes, also caused by a herpesvirus, occurred among captive cranes at the International Crane Foundation, Baraboo, Wisconsin. Some affected cranes belonged to rare species: Stanley (Anthropoides paradisea), Man-
churian (Grus aponensis), and hooded cranes (G. monacha) (Docherty & Henning 1980, Docherty & Romaine 1983, Schuh et al. 1986). Thirty-three percent mortality occurred. Carrier birds were probably involved as important sources of the virus. Based on serologic evidence, the virus apparently first occurred in the colony as early as 1975 and remained undetected until overcrowding and environmental conditions in 1978 eventually led to clinical disease.

Canine parvoviral enteritis is a highly contagious infectious disease of canids. Recovered animals may shed the virus for weeks following infection, and the virus is quite resistant to environmental inactivation (Gillespie & Timoney 1981, Kurstak & Tijssen 1981). Deaths among maned wolves (Chrysocyon brachyurus) due to parvovirus-induced enteritis occurred at the San Antonio Zoological Gardens (Fletcher et al. 1979) and at the National Zoological Park (Mann et al. 1980); carrier wolves or other canids may have been the source of virus for the affected animals.

Infectious diseases transmitted by vectors can be difficult to control in captivity and in the wild without intensive management techniques. In 1984, eastern equine encephalitis, caused by an arbovirus, occurred in a captive flock of endangered whooping cranes (Grus americana) at the FWS Patuxent Wildlife Research Center, Maryland; seven of 39 birds died (Dein et al. 1986). This widespread mosquito-transmitted viral disease is known to occur naturally or experimentally in many native avian species, but morbidity and mortality is unusual. Sandhill cranes (G. canadensis) held at the center were clinically unaffected.

Avian malaria apparently played a major role in the mass extinction of many native Hawaiian birds (Warner 1968, Van Riper III et al. 1986). Introduction of Culex quinquefasciatus mosquito vectors in 1826 and Plasmodium relictum capistranode protozoans via exotic birds, particularly those from Asia, in the early 1900s was probably required for malaria to have such an impact. The exotic malarian parasite remains and continues to modify distribution and behavior of surviving native avian species (Van Riper III et al. 1986).

Anthropod-borne disease may have unexpected influences on the management of endangered species in ways other than by inducing illness and mortality. Re-introduction of a group of captive raised Arabian oryx into a new national park in Oman was halted due to the presence of serum antibodies to bluetongue virus, an orbivirus, in animals scheduled for release to the wild (Jones 1982). Bluetongue can be an important disease in domestic livestock and is a barrier to movement of animals from bluetongue endemic regions, such as much of North America, to other parts of the world.

Compromise of the immune system would obviously have a major influence on susceptibility of endangered species to infectious diseases. Cheetahs (Acinonyx jubatus) are considered threatened. They have been shown to represent an extreme in genetic monomorphism at isozyme and major histocompatibility complex loci that is almost unmatched by other wild species (O’Brien et al. 1983, O’Brien et al. 1985); it has been speculated this genetic uniformity resulted from inbreeding preceded by a major population bottleneck. One of the most successful cheetah captive breeding programs has been at Wildlife Safari, Winston, Oregon, where in 1982 and 1983, 18 cheetahs were lost to feline infectious peritonitis (Pfeifer et al. 1983, Evermann et al. 1983). Rarely does this feline coronavirus-induced disease cause high mortality and morbidity rates in any species that are comparable to those that occurred among Wildlife Safari cheetahs. It has been speculated that the epizootic was so extreme because of the monomorphism of the major histocompatibility complex and lack of effective antiviral immune response (O’Brien et al. 1985). Absence of genetic variability in this species may have robbed it of its natural protection to this coronavirous.

The immune system of animals may also be compromised by prolonged and unavoidable stressors present in the captive or wild environment (Munck et al. 1984). Disease outbreaks in free-ranging and captive animals have been associated with environmental stressors, presumed immunosuppression, and illness due to organisms of low pathogenicity (Fowler et al. 1980, Spraker et al. 1984, Bailey 1986). Prolonged environmental stress increases serum cortisol in captive bighorn sheep (Ovis canadensis) (Harlow et al. 1987), which could induce immunosuppression. Physiologic alterations owing to stress may cause fatal hemorrhagic gastroenteritis in some mustelid species (Wallach & Boever 1983; Williams et al., in press).

Iatrogenic diseases may also be important in endangered species; the best examples are induction of disease during immunization against infectious disease using modified live virus vaccines. The canine distemper vaccines are most often incriminated, and vaccine-induced canine distemper has been documented in black-footed ferrets (Carpenter et al. 1976), lesser pandas (Ailurus fulgens) (Bush et al. 1976, Itakura et al. 1979), and possibly in African cape hunting dogs (McCormick 1983) and maned wolves (Thomas-Baker 1985).

Humans may also be responsible for transmitting diseases to rare or endangered species. Tuberculosis caused by Mycobacterium tuberculosis has created many problems for managers of zoological parks (Montali 1978) and has prevented re-introduction of orangutans (Pongo pygmaeus) to the wild in Indonesia (Jones 1982). Measles may cause disease in primates and has caused nearly 100 percent mortality in several groups of colobus monkeys (Colobus guereza) (Hime et al. 1975, Mehren et al. 1979). Even species as taxonomically far
removed as ferrets may become ill when infected with human influenza virus (Bernard et al. 1984), and this virus could cause disease in black-footed ferrets (Thorne et al. 1985).

Summary

The saga of the black-footed ferret, along with other documented cases of disease in endangered species, provides an example from which lessons may be derived that should be important in the management of other endangered species. Foremost of these is that disease may have catastrophic effects upon a species when it exists in a single population. Timely consideration should be given to dividing a single population, either by translocation or by placing a few animals in captivity.

Precautions were taken to prevent introduction of disease into the free-ranging black-footed ferret population by researchers and managers. We believe these worthwhile and effective and that similar precautions could be tailored to other endangered species. These precautions, however, should not result in complacency, because other animals with which the endangered species shares its habitat are more likely sources of disease.

One precaution taken to protect the black-footed ferret that apparently failed was vaccination against canine distemper. However, this was due to difficulties associated with successfully vaccinating black-footed ferrets against canine distemper (Carpenter et al. 1976; Williams et al., in press). There were no illusions that vaccinated free-ranging black-footed ferrets would be completely protected from canine distemper; rather, it was hoped that exposure to canine distemper virus vaccine would result in some degree of primary immune response and possibly would modify the course of the disease in an individual exposed to the virus. Some endangered species likely can be effectively vaccinated against important diseases, and they should be vaccinated when handled for research or other purposes. Whether or not endangered species should be captured solely for the purpose of vaccination is more contentious, and such a decision should depend upon inherent dangers associated with capture, demonstrated safety and efficacy of the vaccine in question, predicted consequences of the disease, and potential for exposure of members of the endangered species to the disease.

Although isolation and quarantine of individual animals brought into captivity from the same population is generally not considered, had this been done when the first six black-footed ferrets were captured in 1985, there likely would be four additional animals in the captive population. Certainly, isolation and quarantine should be used before recently captured endangered species are formed into a single captive population. During this isolation period all individuals should be screened for as many pathogens of that species as possible.

Addendum

During the 1987 breeding season, nine of 11 black-footed ferret females were bred. The black-footed ferret reproductive cycle was partially characterized; most animals, including juveniles, demonstrated a willingness to participate in breeding activities, and successful pairing techniques were developed. One litter of six kits and one litter of two kits were born; one kit from the litter of two died, and the remaining seven captive-born black-footed ferrets were weaned. Although most breedings were not productive, there is reason for optimism about the future prospects for captive propagation of black-footed ferrets.

Literature Cited

Anderson, E., S. C. Forrest, T. W. Clark, and L. Richardson. 1986. Paleobiology, biogeography, and systematics of the black-footed ferret. Mustela nigripes (Audubon and Bachman), 1851. Great Basin Naturalist Memoirs 8:11–62.

Anderson, S. H., and D. B. Inkley. Black-footed ferret workshop proceedings. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.

Anonymous. 1984. Black-footed ferret captive breeding facility committee report. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.

Appel, M. J. G., E. P. J. Gibbs, S. J. Martin, V. ter Meulen, B. K. Rima, J. R. Stephenson, and W. P. Taylor. 1981. Morbillivirus diseases of animals and man. Pages 259–273 in E. Kurstak and C. Kurstak, editors. Comparative diagnosis of viral diseases. Vol. 4, Vertebrate animal and related viruses, part B—RNA viruses. Academic Press, New York, New York, USA.

Ashton, D. G. 1982. Malignant catarrhal fever. Veterinary clinical report. Scientific Report, the Zoological Society of London. Journal of Zoology (London) 197:82–83.

Bailey, J. A. 1986. The increase and die-off of Waterton Canyon bighorn sheep: biology, management and dismanagement. Proceedings of the Northern Wild Sheep and Goat Council 5:325–340.

Barnes, A. M. 1982. Surveillance and control of bubonic plague in the United States. Symposium of the Zoological Society of London. Journal of Zoology 202:237–270.

Bernard, S. L., J. R. Gorham, and I. M. Ryland. 1984. Biology and diseases of ferrets. Pages 385–397 in J. G. Fox, B. J. Cohen, and F. M. Loew, editors. Laboratory animal medicine. Academic Press, New York, New York, USA.

Budd, J. 1981. Distemper. Pages 31–44 in J. W. Davis, L. H. Karstad, and D. O. Trainer, editors. Infectious diseases of wild mammals, 2nd ed. Iowa State University Press, Ames, Iowa, USA.
Bush, M., R.J. Montali, D. Brownstein, A.E. James, and M.J.G. Appel. 1976. Vaccine-induced canine distemper in a lesser panda. Journal of the American Veterinary Medical Association 169:959–960.

Carpenter J.W. 1985. Captive breeding and management of black-footed ferrets. Pages 12.1–12.13 in S.A. Anderson and D.B. Inkley, editors. Black-footed ferret workshop proceedings. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.

Carpenter, J. W., W. M. G. Appel, R. C. Erickson, and M. N. Novilla. 1976. Fatal vaccine-induced canine distemper virus infection in black-footed ferrets. Journal of the American Veterinary Medical Association 169:961–964.

Carpenter, J. W., and C. N. Hillman. 1979. Husbandry, reproduction, and veterinary care of captive ferrets. Proceedings American Association Zoo Veterinarians (Washington D.C.) 36–47.

Carr, A. III. Introduction. 1986. Great Basin Naturalist Memoirs 8:1–7.

Castro, A. E., G. G. Daley, M.A. Zimmer, D. L. Whitenack, and J. Jensen. 1982. Malignant catarrhal fever in an Indian gaur and greater kudu: experimental transmission, isolation, and identification of a herpesvirus. American Journal of Veterinary Research 34:5–11.

Davidson, M. 1986. Canine distemper virus infection in the domestic ferret. Compendium on Continuing Education for the Practicing Veterinarian 8:448–453.

Dein, F. J., J. W. Carpenter, G. G. Clark, R. J. Montali, C. L. Crabb, T. F. Tsai, and D. E. Docherty. 1986. Mortality of captive whooping cranes caused by eastern equine encephalitis virus. Journal of the American Veterinary Medical Association 189:1006–1010.

Docherty, D. E., and D. J. Henning. 1980. The isolation of a herpesvirus from captive cranes with an inclusion body disease. Avian Diseases 24:278–283.

Docherty, D. E., and R. I. Romaine. 1983. Inclusion body disease of cranes: A serological follow-up to the 1978 die-off. Avian Diseases 27:830–835.

DonCarlos, M. W., B. Miller, and E. T. Thorne. Observations of the 1986 black-footed ferret (Mustela nigriceps) captive breeding program. In U. S. Seal, S. A. Anderson, M. Bogan, E. T. Thorne, editors. Reproductive biology of Black-footed Ferrets and small population biology as they relate to conservation. Yale University Press, New Haven, Connecticut, USA, in press.

Evermann, J. F., G. Burro, M. E. Roelke, A. J. McKeirnan, A. Greenlee, A. C. Ward, and M. L. Pfeifer. 1983. Diagnostic features of an epizootic of feline infectious peritonitis in captive cheetahs. Proceedings American Association of Veterinary Laboratory Diagnosticians 26:365–382.

Flath, D. L., and T. W. Clark. 1986. Historic status of black-footed ferret habitat in Montana. Great Basin Naturalist Memoirs 8:63–71.

Fletcher, K. C., A. K. Eugster, R. E. Schmidt, and G. B. Hubbard. 1979. Parvovirus infection in maned wolves. Journal of the American Veterinary Medical Association 175:897–900.

Forrest, S. C., D. E. Biggins, L. Richardson, T. W. Clark, T. M. Campbell, III, K. A. Fagerstone, and E. T. Thorne. Population attributes for the black-footed ferret (Mustela nigriceps) at Meeteetse, Wyoming, 1981–1985. Journal of Mammalogy, in press.

Fowler, M. E., N. Jacobsen, L. Erb, and S. McDonald. 1980. Stress-induced mixed infections in Columbian black-tailed deer. Pages 415–420 in R. J. Montali, G. Migaki, editors. The comparative pathology of zoo animals. Washington D.C.: Smithsonian Institution Press.

Gillespie, J. H., and J. F. Timoney. 1981. Hagan and Bruner's infectious diseases of domestic animals 7th Ed. Ithaca, NY: Cornell University Press.

Hall, E. R. 1981. The Mammals of North America. New York: John Wiley and Sons.

Hamdy, F. M., A. H. Dardiri, C. Mebus, R. E. Pierson, and D. Johnson. 1978. Etiology of a malignant catarrhal fever outbreak in Minnesota. Proceedings United States Animal Health Association 82:248–267.

Harlow, H. J., E. T. Thorne, E. S. Williams, E. L. Belden, and W. A. Gern. 1987. Cardiac frequency: a potential predictor of blood cortisol levels during acute and chronic stress exposure in Rocky Mountain bighorn sheep (Ovis canadensis canadensis). Canadian Journal of Zoology 65:2028–2035.

Hatkin, J. 1980. Endemic malignant catarrhal fever at the San Diego Wild Animal Park. Journal of Wildlife Diseases 16:439–445.

Henderson, R. R., P. F. Springer, and R. Adrian. 1974. The black-footed ferret in South Dakota. South Dakota Department of Game, Fish, and Parks Technical Bulletin no. 4.

Heuschele, W. P. 1982. Malignant catarrhal fever in wild ruminants—a review and current status report. Proceedings of the United States Animal Health Association 86:552–570.

Hillman, C. N., and J. W. Carpenter. 1983. Breeding biology and behavior of captive black-footed ferrets. International Zoo Yearbook 23:186–191.

Hime, J. M., I. F. Keymer, and C. J. Baxter. 1975. Measles in recently imported colobus monkeys (Colobus guereza). Veterinary Record 97:392.

Huck, R. A., A. Shand, P. J. Allsop, and A. B. Patterson. 1961. Malignant catarrh of deer. Veterinary Record 73:457–465.

Itakura, C., K. Nakamura, J. Nakatsuka, and M. Goto. 1979. Distemper in lesser pandas due to administration of a canine distemper live vaccine. Japanese Journal of Veterinary Science 41:561–566.

Jones, D. M. 1982. Conservation in relation to disease in Africa and Asia. Symposium of the Zoological Society of London 50:271–285.

Kurstak, E., and P. Tijssen. 1981. Animal parvoviruses: comparative aspects and diagnosis. In E. Kurstak and C. Kurstak, editors. Comparative diagnosis of viral diseases. Vol. 3. Vertebrate animal and related viruses, part A—DNA viruses. Academic Press, New York, New York, USA.
Mann, P. C., M. Bush, M. J. G. Appel, B. A. Beehler, and R. J. Montali. 1980. Canine parvovirus infection in South American canids. Journal of the American Veterinary Medical Association 177:779–783.

May, R. M. 1986. The cautionary tale of the black-footed ferret. Nature 320:13–14.

McCormick, A. E. 1983. Canine distemper in African cape hunting dogs (Lycaon pictus)—possibly vaccine induced. Journal of Zoo Animal Medicine 14:66–71.

Mehren, K. G., W. A. Rapke, I. K. Barker, S. S. Kalter, and D. Onderka. 1979. Outbreak of a viral disease in newly imported colobus monkeys. Titles and Abstracts, Wildlife Disease Association Conference, 32.

Montali, R. J., editor. 1978. Mycobacterial infections of zoo animals. Smithsonian Institution Press, Washington D.C., USA.

Munck, A., P. M. Guyer, and N. J. Holbrook. 1984. Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. Endocrine Reviews 5:25–44.

Nowak, R. M., and J. I. Paradiso. 1983. Pages 993–994 in Walker's mammals of the world, 4th ed. Vol. 2. Johns Hopkins University Press, Baltimore, Maryland, USA.

O'Brien, S. J., M. E. Roelke, L. Marker, A. Newman, C. A. Winkler, D. Meltzner, L. Colly, J. F. Evermann, M. Bush, and D. E. Wildt. 1985. Genetic basis for species vulnerability in the cheetah. Science 227:1428–1434.

O'Brien, S. J., D. E. Wildt, D. Goldman, C. R. Merril, and M. Bush. 1983. The cheetah is depauperate in genetic variation. Science 221:459–462.

Pfeifer, M. L., J. F. Evermann, M. E. Roelke, A. M. Gallina, R. L. Ott, and A. J. Keirnan. 1983. Feline infectious peritonitis in a captive cheetah. Journal of the American Veterinary Medical Association 183:1317–1319.

Plowright, W., R. D. Ferris, and G. R. Scott. 1960. Blue wildebeest and the aetiological agent of bovine malignant catarrhal fever. Nature 188:1167–1169.

Reese, A. 1985. An analysis of potential sites for black-footed ferret captive propagation facility. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.

Rossiter, P. B. 1981. Antibodies to malignant catarrhal fever virus in sheep sera. Journal of Comparative Pathology 91:303–311.

Schuh J. C. L., L. Sileo, L. M. Siegfried, and T. M. Yuill. 1986. Inclusion body disease of cranes: comparison of the pathologic findings in cranes with acquired versus experimentally induced disease. Journal of the American Veterinary Medical Association 189:993–996.

Senior, M., C. R. E. Halman, and E. H. Tong. 1962. An outbreak of malignant catarrh among the Père David deer. Veterinary Record 74:932–936.

Snyder, B., J. Thilsted, B. Burgess, and M. Richard. 1985. Pigeon herpesvirus mortalities in foster reared Mauritius pink pigeons. Pages 69–70 in Proceedings of the American Association of Zoo Veterinarians. Scottsdale, Arizona, USA.

Spraker, T. R., C. P. Hibler, G. G. Schoonveld, and W. S. Adney. 1984. Pathologic changes and microorganisms found in big-horn sheep during a stress-related die-off. Journal of Wildlife Diseases 20:319–327.

Thomas-Baker, B. 1985. Vaccination-induced distemper in maned wolves, vaccination-induced corneal opacity in a maned wolf. Page 53 in Proceedings of the American Association of Zoo Veterinarians. Scottsdale, Arizona, USA.

Thornc, E. T. 1987. Captive propagation of the black-footed ferret in Wyoming. Pages 419–425 in Regional conference proceedings of the American Association of Zoological Parks and Aquariums. Colorado Springs, Colorado, USA.

Thornc, E. T., and D. W. Belitsky. The black-footed ferret captive propagation effort in Wyoming. In U. S. Seal, S. H. Anderson, M. Bogan, and E. T. Thorne, editors. Reproductive biology of black-footed ferrets and small population biology as they relate to conservation. Yale University Press, New Haven, Connecticut, USA; in press.

Recommenda-