Chapter 10

Disease and Translocation Issues of Gray Wolves

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INTRODUCTION

This overview of pathogens of North American wolves includes health management considerations for translocations. These health issues encompass potential direct effects of a specific pathogen on wolves. It is also a discussion of pathogens present at potential relocation sites (referred to as endemic or enzootic pathogens or diseases), and the potential to move new diseases via translocation to a naive (i.e., unexposed) population, causing a new disease in existing populations. These diseases are referred to as epidemic (in people) or epizootic (in other animal species) pathogens or diseases. Once a new or novel pathogen is introduced into a wildlife population, control of the pathogen is problematic. We never just translocate a species; we translocate biological packages containing all the external and internal parasites, bacteria, fungi, and viruses associated with that animal. There is concern that an introduced disease will move beyond a local pack to the rest of the new environment and then spread to other packs and species. Disease movement by natural dispersal, dispersal via human intervention, and movement corridors into historic habitat is well documented.

Diseases and health anomalies in carnivores have occurred throughout recorded history and generally have a minimal impact on humans. Rabies is an exception. Severe and widespread rabies epizoodemics (i.e., rapid and severe disease outbreaks that occur in both people and other animals simultaneously) were reported in the late eighteenth century (1796) in the Americas in foxes and dogs, and distemper outbreaks occurred in dogs and cats as early as the late nineteenth century (Fleming 1975).

Diseases that influence species at the top of the food chain not only affect predator numbers directly but also indirectly, by spreading diseases that cause changes in prey populations. Describing each disease entity will provide insight into the transmission pathways that pathogens might travel between or within populations. The movement of disease via animal translocation can play a major role in the persistence of that disease in a population and can be a major concern when making decisions that affect rare endemic (i.e., restricted to one location) or founder animals or those with naive immune systems.

DISEASE OVERVIEW

Wolves (*Canis lupus*) have been exposed to a variety of viral, bacterial, fungal, and parasitic diseases throughout all areas of their range. Several publications have provided extensive overviews of the known diseases that affect free-ranging wolves (Mech 1970; Brand et al. 1995; Kreeger 2003). These authors identified important diseases carried by wolves, including those that cause high numbers of animals to be infected (known as morbidity) and high numbers of animals to die of the disease (in other words, mortality).

Wolves potentially carry diseases that are important in other carnivores but do not

appear pathologic in wolves. Multiple publications have documented diseases affecting gray wolves in North America using necropsy (i.e., autopsy of an animal), surveys for blood antibodies (or serological surveys), parasitic surveys, investigations into the factors affecting wolf health and illness (epidemiological investigations), or incidental observations from case reports (Chapman 1978; Todd et al. 1981; Carbyn 1982). Due to the naturally low densities of this top carnivore species, the large home range and distribution among packs within populations, and relatively secretive nature of wolves, large die-offs from disease might go undetected unless specific populations are being intensively monitored (Brand et al. 1995). Diseases and parasites have been shown to have significant impacts on wolf population recovery in Michigan, Minnesota, and Wisconsin (US Fish and Wildlife Service 2000). The risk of disease should be an important consideration in reintroduction decisions regarding founder animals and release sites for North American wolves.

VIRUSES

Viral diseases are the most important to carnivore populations from an epizootic perspective. Viral diseases affecting wolves in North America include rabies, canine parvovirus, canine distemper, infectious canine hepatitis, and oral papillomatosis. Epidemic and endemic rabies is predicted to be capable of causing population declines in wild carnivores. Canine parvovirus may affect wolf pup recruitment, based on circumstantial evidence in a captive wolf colony in Minnesota in which eleven of twelve pups succumbed to the disease (Mech and Fritts 1987). The potential exists for canine parvovirus to affect wolf pup survival and recruitment in wild populations.

Recent published reviews of rabies in North American wolves have been described by Brand et al. (1995) and Johnson (1995). Murie (1944), Cowan (1949), and Mech (1970) identified this important viral disease as one that could potentially limit population numbers. Rabies is a rhabdovirus (a type of bullet-shaped, enveloped virus with a single strand of RNA) that is generally confined to one species in a geographic area, although extension to other species is not uncommon. New diagnostic tests have enabled researchers to understand endemic and epidemic rabies. However, the role that rabies may play in regulating wolf populations is unknown (Brand et al. 1995). Historic and recent accounts of rabies in wolves (Weiler et al. 1995; Ballard and Krausman 1997) indicate that this disease will likely remain in wolf range for extended periods. Additionally, several authors have shown wolf packs being reduced due to the incidence of rabies (Chapman 1978; Davis et al. 1980; Theberge et al. 1994).

North American wolves are not considered reservoirs of rabies virus. In published cases, wolves were suspected of contracting the disease from other canid species including red foxes (*Vulpes vulpes*) and arctic foxes (*Alopex lagopus*) (Mech 1970; Rausch 1973; Ritter 1981; Theberge et al. 1994). The spread of rabies by wolves, though generally contained within individual packs (Chapman 1978), can occur when infected animals contact members of adjacent packs at their territory boundaries or via dispersing individuals.

Canine parvovirus was first detected in domestic dogs in 1978 and had spread worldwide by 1980 (Pollock 1984). Parvovirus is very stable in the environment and spread by direct contact and fecal contamination of the habitat. Once infected, canids are capable of periodically shedding the virus for many years. Based on retrospective studies of serological (blood serum) data, canine parvovirus likely entered wild coyote (*Canis latrans*) and wolf populations in North America sometime during 1978 or 1979 (Thomas et al. 1982; Barker et al. 1983) and possibly as early 1975 (Goyal et al. 1986).

Testing blood for diseases yields serological titers that indicate whether or not an animal has been exposed to a disease. Though serological titers in wild wolves have been reported as high as 65 percent in Minnesota (Mech 1986), Montana, and southeastern British Columbia (Johnson et al. 1994), no published reports of mortalities or clinical signs have been reported in wild populations (Brand et al. 1995).

Captive studies conducted by J. Zuba and reported by Brand et al. (1995) challenged a cohort of study animals with live canine parvovirus. Only 30 percent developed clinical signs and about 10 percent would have died without supportive care. In captive populations, canine parvovirus has been shown to carry high mortality rates, especially among young animals (Mech and Fritts 1987; Mech and Goyal 1993; Mech et al. 2008). Circumstantial evidence from population crashes in Isle Royale National Park during the 1980s indicates that the disease is capable of limiting wolf populations. This episode was coincidental with a canine parvovirus outbreak among neighboring domestic dogs (R. O. Peterson, unpubl., as reported in Brand et al. 1995). In a Minnesota study of wolves from 1973 to 2004, Mech et al. (2008) found that pup survival was reduced by 40 percent to 60 percent, limiting the population rate of increase to 4 percent compared with increases in other populations of 16 percent to 58 percent. Canine parvovirus was suspected to reduce gray wolf pup survival from 80 percent to 60 percent in populations residing in central Idaho and Yellowstone National Park (Gillin et al. 2000), with similar circumstantial evidence occurring in Glacier National Park, Montana (Johnson et al. 1994).

Canine distemper is another important viral disease of canids and other carnivores (Johnson et al. 1994). The disease is caused by a paramyxovirus (a type of single-strand RNA virus) closely related to measles and rinderpest. This disease affects domestic dogs at three to nine weeks of age (Gillespie and Carmichael 1968), and morbidity and mortality can be high in exposed unvaccinated animals. Despite the ubiquitous distribution of canine distemper, there are only two reports in the literature of mortality occurring in wild populations (Carbyn 1982; Peterson et al. 1984). Because recruitment (i.e., young animals surviving to adulthood) in North American wolf populations is generally good, canine distemper cannot be considered a significant mortality factor (Brand et al. 1995).

Infectious canine hepatitis (ICH) is an important disease in domestic dogs and has been reported via seropositive titers in Alaskan (Stephenson et al. 1982; Zarnke and Ballard 1987; Zarnke et al. 2004) and Canadian wolves (Choquette and Kuyt 1974). A DNA virus, canine adenovirus 1, causes ICH. In Alaskan populations, annual prevalence has reached 100 percent with up to 42 percent of the exposed animals being pups, suggesting exposure at an early age. Although infectious canine hepatitis appears to be enzootic in wolf populations, the percentage of wild wolves that test positive for exposure to this disease, called the seroprevalence, is uncorrelated with its occurrence in domestic dogs. No mortality from this disease has been reported in wolves.

Oral papillomatosis is a disease caused by a small, double-stranded DNA virus of the Papovaviridae family. It has been reported in wild populations of wolves and coyotes (Samuel et al. 1978). Although this disease causes severe oral tumors in coyotes (Trainer et al. 1968), it has not been documented to cause mortality in wild wolves or other canids and is not considered a threat to those populations.

BACTERIAL AND FUNGAL DISEASES

The most noted bacterial disease threats in North American populations of wolves are brucellosis (*Brucella* spp.), Lyme disease

(*Borrelia burgdorferi*), leptospirosis (*Leptospira* spp.), tularemia (*Francisella tularensis*), plague (*Yersinia pestis*), and bovine tuberculosis (*Mycobacterium bovis*). Of these, Lyme disease and plague are spread through the bite of infected fleas and ticks, whereas the other diseases are passed primarily through the exposure to or consumption of mammalian prey.

Lyme disease has the potential to infect wolves, but clinical disease has never been demonstrated (Kazmierczak et al. 1988). The bacterium is spread through the bite of infected ticks, principally of the genus *Ixodes damnii*. It is passed to other species through transmission via a life cycle involving small mammals such as the white-footed deer mouse (*Peromyscus leucopus*) that host immature ticks, and then to white-tailed deer (*Odocoileus virginianus*), the host of the adult ticks. In one study, two of seventy-eight wild wolves sampled in Wisconsin and Minnesota tested positive to exposure (i.e., were seropositive) with *B. burgdorferi* (Kazmierczak et al. 1988). When inoculating captive wolves, Kazmierczak (1988) saw only swollen lymph nodes (or lymphadenopathy) and no evidence of chronic lymph node inflammation (known as lymphadenitis), fever, and arthritis, as seen in domestic dogs (Lissman et al. 1984; Kornblatt et al. 1985). Although the effects on wolf reproduction are not known, abortion and fetal mortality have been reported in humans and horses (Schlesinger et al. 1985; Burgess et al. 1989).

Fungal diseases do not appear to play an important morbidity or mortality role in wild wolf populations. The only reported fatal case occurred in a wolf in Minnesota from the fungal disease blastomycosis (*Blastomyces dermatitidis*) (Thiel et al. 1987). This disease is enzootic and limited to the region encompassing Minnesota and Wisconsin and is most commonly diagnosed in domestic dogs in those states (Archer 1985).

HELMINTHS

Holmes and Podesta (1968), Mech (1970), and Archer et al. (1986) describe an array of parasites for which wolves serve as an important host species. These parasites include three species of spiny-headed worms (acanthocephala), nine species of flukes (trematodes), twenty-one species of tapeworms (cestodes), and twenty-four species of roundworms (nematodes). Craig and Craig (2005) conducted a definitive literature survey of helminth parasites of wolves and identified a total of seventy-two species from forty genera, 93 percent collected from the gastrointestinal tract. They found twenty-eight species of nematode, twenty-seven species of cestode, sixteen species of trematode, and one acanthocephalan. As a general observation, the majority of helminth infections cause little pathology among wolves, and apparently they are not a factor in regulating populations (Brand et al. 1995). Several species of note are described below.

Dog heartworm infection (*Dirofilaria immitis*) is caused by a nematode that inhabits the heart and pulmonary arteries of canid and several felid species, but is most prominent in domestic dogs. Several case history accounts of dog heartworm infection and fatalities from pathologic changes have occurred in wolves held in zoo collections where the parasite occurs enzootically (Hartley 1938; Coffin 1944; Pratt et al. 1981). This disease may have been partially responsible for the decline of red wolves (*Canis rufus*) in the southeastern United States (McCarley and Carley 1979). Mech and Fritts (1987) have expressed concern over the potential effects of *D. immitis* infection in free-ranging wolves in heartworm enzootic areas.

Dog hookworm (*Ancylostoma caninum*) is another internal parasite of canids. It causes ulcerative lesions on intestinal mucosa through its blood-feeding activities. In domestic dogs, emaciation accompanied by anemia (or a deficiency of red blood cells), diarrhea, and occasionally death can occur. Although this parasite has not been reported in gray wolves, it has been suspected of causing infection and deaths in red wolves (McCarley and Carley 1979; Custer and Pence 1981) and coyotes (Mitchell and Beasom 1974). Similar morbidity and mortality may occur in areas inhabited by gray wolves where the parasite is enzootic (Brand et al. 1995).

A liver fluke (*Metorchis conjunctus*) was found in wolves from Alberta (Holmes and Podesta 1968) and Saskatchewan (Wobeser et al. 1983). Spending a portion of its life cycle in fish, this trematode caused pathologic changes to the bile ducts and pancreas in several of the infected wolves. Health effects to infected wolves were not known from these observations (Wobeser et al. 1983), and population effects have not been documented.

Wild canids including wolves harbor a wide variety of cestode (tapeworm) populations, particularly from the genera *Taenia* and *Echinococcus*. *Echinococcus grunulosus* can cause hydatid disease in humans as an intermediate host. After inadvertently ingesting eggs, the eggs hatch and invade the circulatory system and lodge in various organs (liver and lungs). Human infection from *Tinea* spp. and *Echinococcus multiloculares* has also been documented.

Cestode populations within wolves segregate regionally by differences in the prey species they consume. From an individual animal and population perspective, tapeworms do not cause known negative pathologic changes because they do not feed on the host, but rather use nutrients of passing ingested food in the intestinal tract of the host.

Wolves should be monitored for other internal parasites common in wild canid species. *Capillaria* spp. and *Crenosoma* spp. are lungworms that could hinder wolf populations by causing chronic bronchitis or pneumonia.

ECTOPARASITES

Lack of published reports indicates infestations of external parasites (or ectoparasites) are rare in gray wolves. As might be expected in wild canid species, ticks (*Amblyomma americanum, Amblyomma maculatum, Dermacentor albipictus, D. variabilis, Ixodes* spp.) (Pence and Custer 1981; Archer et al. 1986), fleas (*Pulex simulans, Ctenocephalides canis*) (Skuratowicz 1981; Hristovski and Beliceska 1982), and occasional deerflies (*Lipoptena cervi*) (Itamies 1979) have been reported as pests on wolves. However the most notable ectoparasites occurring in wolf populations are from infestations of lice and mange mites.

Domestic dogs were likely the source of infection of the dog louse (*Trichodectes canis*) on gray wolves (Brand et al. 1995). The louse is transmitted by direct contact between infected and uninfected animals. Infected animals show varying degrees of hair loss (i.e., alopecia). Although dog lice occur throughout most of the wild gray wolf range in North America, there is scant evidence that the parasite causes negative effects on populations (Schwartz et al. 1983; Mech et al. 1985).

Sarcoptes scabei, known commonly as sarcoptic mange (or scabies), is found worldwide and transfers easily among a variety of host species (Sweatman 1971), including the gray wolf. The mite causes skin pathology by burrowing into the epidermis of infected animals. Mites are transferred to new hosts by direct contact between infected and noninfected individuals or contaminated objects such as scratching and rubbing posts. Classic clinical presentation of a severely infected individual usually includes extensive hair loss, lesions with crusting and exudate, and thickened, gray discolored skin. Todd et al. (1981) reported that wolves in advanced levels of infestation might show lower body weight and overall condition. Although the evidence is not substantial, there is reason to believe

that sarcoptic mange may be regulating some wild canid populations (Murie 1944; Cowan 1951; Green 1951; Todd et al. 1981).

DISEASES OF PREY SPECIES

Brucellosis, a highly contagious disease in many species, is caused by the bacterium from the genus *Brucella*. Of the five known *Brucella* species, a canine species, *Brucella canis*, is the only species endemic in domestic canids. It has not been documented in wild wolf populations.

The other species of *Brucella* affect domestic and wild hoofed animals (known also as ungulates). However, wolves in Alaska have been tested seropositive to *Brucella suis biovar* 4, which is present in infected caribou (*Rangifer tarandus*) herds (Pinigan and Zabrodin 1970; Neiland 1975; Zarnke and Ballard 1987). The effects of *Brucella* infection in wild wolf populations have not been documented; however, one might expect antibody titers where the disease occurs in their prey, such as the Greater Yellowstone Ecosystem or Wood Buffalo National Park, Alberta and Northwest Territories. Wolves are considered dead-end hosts for *Brucella abortus* because they are unable to transmit the disease.

Under experimental captive conditions, Neiland and Miller (1981) infected two pregnant female wolves with *Brucella suis biovar* 4. Although these animals showed no apparent clinical signs of disease, four of six offspring were born dead. *Brucella suis biovar* 4 was subsequently isolated from various organs and lymph nodes of the pups and bitches. Although *Brucella* was not determined to be the cause of death in the pups, the authors concluded that reproductive failure could possibly occur if infection were present during pregnancy.

Leptospirosis infection, of the bacterium *Leptospira* spp., is endemic in domestic hogs, cattle, and horses in parts of Minnesota and in moose (*Alces alces*) populations (Khan et al. 1991). Signs of disease in domestic animal populations range from undetectable to mortalities depending on the species, type of microorganism (i.e., serotype), and host (Brand et al. 1995). Wolves in Alaska (Zarnke and Ballard 1987) and in northern Minnesota (Khan et al. 1991) have tested serologically positive to the disease. However, clinical disease has not been documented in wild canids. The disease is spread among carnivores primarily through infected urine or via consumption of infected food (Reilly et al. 1970). Due to the potential of severity of leptospirosis in domestic dogs (Alston et al. 1958) and other species, Brand et al. (1995) felt concern was warranted for wolf reintroduction where leptospirosis was endemic in other carnivore species or in prey.

Tularemia is present in many lagomorph and rodent populations. The disease has caused clinical signs in coyotes and foxes (*Vulpes* spp.) including diarrhea, loss of appetite (or anorexia), and difficulty in breathing (Bell and Reilly 1981). However, clinical disease has not been documented in wolves, although some Alaskan populations have shown seroprevalance. Zarnke and Ballard (1987) felt that healthy adults recover from the disease.

In many areas where tularemia is found, the plague bacterium is also present. Plague is maintained in wild rodent populations and has not been reported in wolves. Antibody titers exist in regions of wolf range where plague is found in their prey. The plague organism is spread by fleas and can be devastating for prairie dog (*Cynomys ludovicianus* and *C. gunnisoni*) populations in the western United States. North America's most endangered mammal, the black-footed ferret (*Mustela nigripes*), is also at risk from a plague epizootic. Humans are also at risk from handling flea-infested animals. In the Greater Yellowstone Ecosystem, coyotes showed positive antibody titers to *Yersinia pestis* without associated disease (Gese et al. 1993).

Tuberculosis is a bacterial disease with three species of potential concern to wolves (*Mycobacterium tuberculosis*, *M. bovis*, and *M. avium*). The bovine bacilli found in domestic cattle and wild ungulates would be the most likely to produce infection in wolves. Any mammal can contract clinical disease through direct exposure (Thoen and Hines 1981; Tessaro 1986) to the bovine, human, or avian types. The only reported occurrence of bovine tuberculosis in wolves was from Riding Mountain National Park, Manitoba (Carbyn 1982). This case history account was limited but fatal to two wolf pups. The source of the infection was not determined. Tuberculosis, among other diseases, was believed to have been partially responsible for a decline in the wolf population from 1975 to 1978 in Riding Mountain National Park (Carbyn 1982).

Neospora caninum is a protozoan parasite identified in dogs and cattle (Bjerkas et al. 1984; Dubey et al. 1988) causing bovine abortions worldwide (Dubey 1999). The disease has more recently been described in other mammalian species including sheep, goats, horses, and deer (Dubey and Lindsay 1996), with more definitive studies identifying transmission of the parasite between wild canids and domestic species (Gondim et al. 2004). Dogs and coyotes were considered the definitive host (McAllister et al. 1998; Gondim 2004, 2006) in the sylvatic transmission cycle between canids and cervids. However, wolves may play a role as a host in areas where the parasite exists in prey species. In an Illinois study, Gondim et al. (2004) found a seroprevalence of 39 percent in free-ranging gray wolves, with lesser prevalences in coyote (11 percent), white-tailed deer (26 percent), and moose (13 percent). The risk of transmitting the parasite to domestic livestock may increase with the infection of multiple wild canid and ungulate species occupying shared ranges with domestic species.

Chronic wasting disease (CWD) is an emerging disease of elk (*Cervus elaphus*), moose, and deer (*Odocoileus* spp.) (Williams and Young 1980, 1982). First identified in northeastern Colorado and southern Wyoming, it is now recognized in wild cervid populations in eleven states and two providences. CWD is a fatal, transmissible spongiform encephalopathy (type of degenerative brain disease) considered unique to native North American cervids. The disease is caused by a prion that alters proteins, which ultimately degenerate specific neural regions of the brain leaving nonfunctional spaces, or spongiform areas.

CWD was first documented in 1968, but to date it has not controlled ungulate populations. Should that change, reduced prey numbers could impact carnivores dependent upon them. Wolves, however, are able to select alternative species from a diverse range of prey. Further, wolves are highly mobile, have large home ranges, and can move to areas of higher prey density. On the other hand, this might increase intraspecific strife. CWD has not been reported in scavengers of diseased ungulate prey species, although translocation of the prion through feces of the scavenger could spread the pathogen via environmental contamination transmission.

Wolves could prey on CWD-affected ungulate species early in the course of the disease. They would differentiate affected prey by their abnormal behaviors and remove them, perhaps before they contaminate the environment and spread the disease to additional animals. If canids are not susceptible to CWD, a hypothetical consequence of wolves in a CWD-infected habitat would be an increase in the health and vitality of the prey species. Model simulations based on conditions at Rocky Mountain National Park (i.e., high elk density) suggest wolves could have "potent effects" on the prevalence of CWD (Wild and Miller 2005).

A similar prion disease, bovine spongiform encephalopathy (BSE), jumped from infected cattle to humans, causing a fatal

brain disease (or encephalopathy) known as new variant Creutzfeldt-Jakob disease (known as mad cow disease in cattle). In experiments using BSE-infected food, domestic canids did not appear susceptible to contracting the disease (Southwood n.d.); however, domestic cats did contract the disease. Other carnivores (domestic ferret, *Mustela putorius furo*; raccoon, *Procyon lotor*; and skunks) have been shown to be susceptible to a similar prion disease known as transmissible mink encephalopathy (TME) (Eckroade et al. 1973).

CONCLUSIONS

Wolves or other wildlife species run the risk of becoming infected by diseases from domestic species when their home ranges take them close to human habitation. In Denali National Park and Preserve, Alaska, biologists have witnessed increased stress on wildlife populations due to domestic animal diseases and lice infestations (P. Owens, Denali National Park Research and Resources Division, pers. comm.). Canine parvovirus, canine distemper, and infectious canine hepatitis may have been transmitted to wild carnivores from the large domestic dog population (including sled dogs) outside the park (Elton 1931; Stevenson et al. 1982). This could be a concern for wolves in the Southern Rockies.

Although viral agents (rabies, canine distemper, parvovirus), bacterial agents (tuberculosis), and parasites (sarcoptic mange) have caused mortality with possible declines in local populations (Davis et al. 1980; Carbyn 1982), there appears to be little evidence that any one disease has historically controlled wolf populations. The territorial nature of wolves with minimal mixing of individuals may limit losses to a pack or two and spare most of the population. Many canine diseases originate from domestic sources, and these pathogens may be less well-adapted to wild hosts.

The role of disease and parasites in controlling wolf populations remains relatively unknown. Several authors have suggested a relationship between disease and population density in other canid populations (Todd et al. 1981; Debbie 1991; Fekadu 1991). This has yet to be shown in wolf populations; however, it could become an important factor, particularly in fragmented or island populations.

When animals are moved from one location to another, the bacteria, viruses, and parasites residing with them are also relocated. Disease consequences can be significant on wildlife populations and devastating to relocation or reintroduction programs. The questions then are how to limit disease outbreaks, particularly involving domestic species and wolves, and how to limit transporting diseases with relocated animals. Other important considerations in translocation issues involve wolves as potential carriers of pathogens. Wolves may harbor pathogens without signs of disease but cause morbidity and mortality to endemic animals with naive immune systems. Relocated animals may also be infected by disease pathogens from endemic species or may be affected indirectly either by new or enzootic diseases that cause morbidity and mortality in their prey.

The risks and costs associated with diseases and translocation programs have been documented by several authors: Karesh (1993), Lyles and Dobson (1993), Woodford (1993), Olney et al. (1994), and Woodford and Rossiter (1994). The greatest disease risks occur when species are exposed to new pathogens, are chronically stressed, or when herd immunity is not present (Lyles and Dobson 1993). To understand the basis of risk, fundamental issues to address include: (1) identifying known diseases that could be relocated or acquired at release sites by relocated animals; and (2) the consequences of inadvertent infection.

The ramifications of disease on legal or illegal animal reintroduction can be measured from case histories throughout the literature. These include examples of brucellosis in elk and bison (*Bison bison*) (Mohler 1917; Rush 1932; Tessaro et al. 1990), bovine tuberculosis in elk (Merritt 1991) and bison (Joly et al. 1998), respiratory disease in desert tortoises (*Gopherus agassizii*) (Jacobson et al. 1991), and whirling disease in trout (Hoffman 1970), to name a few. Managers should evaluate the risks involved from translocated disease by familiarizing themselves with occurrences cited in the literature and taking precautions to prevent human-assisted intrusion of a wildlife disease. These important issues of identifying the pathogens that may be released with relocated animals and the variety of related risks can be reduced with good planning.

The most effective preventive measure is to follow strict quarantine protocols (Woodford 2001). This should include identifying the entire range of health hazards associated with the translocation. A complete translocation assessment includes identifying and prioritizing all hazards, then, for each health hazard selected, determining the probability that the event will occur and the magnitude of negative consequences should it arise (Leighton 2002). These risk assessments should include identifying diseases carried within translocated animals and enzootic diseases residing at the release site. Then, following a systematic procedure, quantitative and qualitative health risk assessments can be determined.

Disease issues are also critical in decisions related to planning and designing habitat corridors through which species might travel and migrate. Corridors can function similarly to the physical transportation of an animal from one location to another with the same potential deleterious health effects on populations (Simberloff et al. 1992; Hess 1994).

Introducing diseases into uninfected areas and connecting several populations via habitat corridors can create herd immunity. This immunity is maintained in populations that experience repeated outbreaks of a pathogen (Dobson et al. 1999). Immunity by some or all individuals decreases the potential for a large-scale epidemic that could significantly reduce population numbers (Anderson and May 1991). Outbreaks of a pathogen are less frequent in isolated populations located in habitat islands, but those animals would be more susceptible, causing a high morbidity and mortality. Dobson et al. (1999) surmise that habitat corridors between habitat islands can thus permit the natural flow of pathogens, thereby maintaining a level of herd immunity and reducing the risk of epidemics.