

# Chapter 11

## Disease Management in Endangered Mammals

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### 11.1 Introduction

One quarter of all mammal species are considered threatened with extinction (IUCN 2007). The rate of loss of biodiversity is accelerating because increasing pressure from an expanding human population is shrinking natural habitat and over-exploiting wild animal populations. Although processes such as habitat loss and over-harvesting are usually identified as the major drivers of extinction, recent evidence suggests that disease can also be a significant threat to endangered species (Lyles and Dobson 1993; Daszak and Cunningham 1999; Daszak et al. 2000b; de Castro and Bolker

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2004; Choisy and Rohani 2006; Lips et al. 2006; Smith et al. 2006). Disease has already been documented as a cause of extinction of a land snail (*Partula turgida*) (Cunningham and Dazsak 1998), and several amphibian species (Schloegel et al. 2006; Skerrat et al. 2007). Diseases are also known to cause significant population declines, as illustrated by the impact of canine distemper virus in black-footed ferrets (*Mustela nigripes*) (Williams et al. 1988) and lions (*Panthera leo*) (Roelke-Parker et al. 1996), rabies virus in African wild dogs (*Lycaon pictus*) (Woodroffe and Ginsberg 1999), Ebola virus in apes (Leroy et al. 2004), squirrelpox virus in red squirrels (*Sciurus vulgaris*) (Rushton et al. 2006) and transmissible facial tumour disease in Tasmanian devils (*Sarcophilus harrisii*) (Pearse and Swift 2006).

A recent study identified 54 species of mammal for which disease was considered a threatening process (Table 11.1). The majority of such species (88%) were from the orders Artiodactyla or Carnivora, with families containing the most familiar and widespread livestock and companion animals (i.e. cattle, sheep, goats, pigs, dogs and cats) most represented. This is probably because of the close taxonomic relationship of these wild animals with domestic species, increasing the likelihood of pathogen transfer, and due to the widespread distribution of large populations of domestic species, allowing exposure of wildlife to domestic animal pathogens. Viruses and bacteria with broad host ranges that include domestic animals have been identified as most likely to threaten wild mammal populations (Table 11.2). Close contact was the predominant mode of transmission (75%) amongst the listed

**Table 11.1** Mammals for which parasites were identified as a major threat at either the species or subspecies level based on the IUCN Red List summary documentation (Pedersen et al. 2007)

Order	Family	Species	Common name
<b>Artiodactyla</b>	Bovidae	<i>Alcelaphus buselaphus</i>	Coke's Hartebeest
		<i>Antidorcas marsupialis</i>	Springbok
		<i>Beatragus hunteri</i>	Hirola
		<i>Bos frontalis</i>	Gaur
		<i>Bos grunniens</i>	Wild Yak
		<i>Bos javanicus</i>	Banteng
		<i>Bos sauveli</i>	Kouprey
		<i>Bubalus bubalis</i>	Asian Buffalo
		<i>Bubalus depressicornis</i>	Anoa
		<i>Bubalus mindorensis</i>	Tamaraw
		<i>Bubalus quarlesi</i>	Mountain Anoa
		<i>Budorcas taxicolor</i>	Takin
		<i>Connochaetes taurinus</i>	Blue Wildebeest
		<i>Damaliscus lunatus</i>	Tsessebe
		<i>Hemitragus jayakari</i>	Arabian Tahr
		<i>Ovis canadensis</i>	Bighorn Sheep
		<i>Ovis orientalis</i>	Punjab Urial
		<i>Procapra gutturosa</i>	Mongolian Gazelle
		<i>Syncerus caffer</i>	African Buffalo
		<i>Tragelaphus imberbis</i>	Lesser Kudu

(continued)

**Table 11.1** (continued)

Order	Family	Species	Common name		
<b>Artiodactyla</b>	Cervidae	<i>Dama dama</i>	Mesopotamian Fallow Deer		
		<i>Hippocamelus antisensis</i>	North Andean Deer		
		<i>Hippocamelus bisulcus</i>	Chilean Guemal		
		<i>Ozotoceros bezoarticus</i>	Pampas Deer		
<b>Artiodactyla</b>	Suidae	<i>Babyrousa babyrussa</i>	Babiroussa		
		<i>Phacochoerus aethiopicus</i>	Cape Warthog		
		<i>Sus cebifrons</i>	Visayan Warty Pig		
		<i>Sus philippensis</i>	Philippine Warty Pig		
<b>Artiodactyla</b>	Tayassuidae	<i>Catagonus wagneri</i>	Chacoan Peccary		
<b>Carnivora</b>	Canidae	<i>Alopex lagopus</i>	Arctic Fox		
		<i>Atelocynus microtis</i>	Short-eared Dog		
		<i>Canis lupus</i>	Gray Wolf		
		<i>Canis simensis</i>	Ethiopian Wolf		
		<i>Chrysocyon brachyurus</i>	Maned Wolf		
		<i>Cuon alpinus</i>	Dhole		
		<i>Lycaon pictus</i>	African Wild Dog		
		<i>Nyctereutes procyonoides</i>	Raccoon Dog		
		<i>Otocyon megalotis</i>	Bat-eared Fox		
		<i>Pseudalopex fulvipes</i>	Darwin's Fox		
		<i>Urocyon littoralis</i>	Channel Islands Fox		
		<i>Vulpes bengalensis</i>	Bengal Fox		
		<b>Carnivora</b>	Felidae	<i>Felis silvestris</i>	Wild Cat
				<i>Prionailurus bengalensis</i>	Iriomote Cat
<i>Puma concolor</i>	Florida Panther				
<b>Carnivora</b>	Mustelidae	<i>Lontra felina</i>	Marine Otter		
		<i>Lontra provocax</i>	Southern River Otter		
<b>Carnivora</b>	Otariidae	<i>Eumetopias jubatus</i>	Steller Sea Lion		
<b>Carnivora</b>	Phocidae	<i>Monachus monachus</i>	Mediterranean Monk Seal		
<b>Cetacea</b>	Delphinidae	<i>Cephalorhynchus hectori</i>	Maui's Dolphin		
<b>Dasyuromorphia</b>	Dasyuridae	<i>Dasyurus hallucatus</i>	Northern Quoll		
		<i>Parantechinus apicalis</i>	Southern Dibbler		
<b>Peramelemorphia</b>	Peramelidae	<i>Perameles gunnii</i>	Eastern Barred Bandicoot		
<b>Rodentia</b>	Sciuridae	<i>Cynomys parvidens</i>	Utah Prairie Dog		
<b>Sirenia</b>	Trichechidae	<i>Trichechus manatus</i>	West Indian Manatee		

pathogens, with other routes such as indirect or arthropod-vectorled transmission being considered less likely to cause extinction.

Disease may threaten an endangered mammal population by suppressing population growth rates, making them more vulnerable to extinction through stochastic factors. For example otodectic mange in the Mednyi arctic fox (*Alopex lagopus*) (Goltsman et al. 1996) and canine parvovirus in the gray wolf (*Canis lupus*) (Mech and Goyal 1995) reduced population growth by limiting recruitment. Synergistic interaction with other threatening processes, such as hunting, could increase the probability of population extinction (Choisy and Rohani 2006). Alternatively, disease may kill individuals more rapidly than they can reproduce, leading to deterministic extinction. This is most likely to occur in populations that are already small or fragmented.

**Table 11.2** Parasites identified as causing population declines or reduced host fitness in mammals listed on the IUCN Red List as threatened by pathogens. The numbers in each column reflect the number of mammal species threatened by each pathogen. Names of diseases are in parentheses. Table from Pedersen et al. (2007)

Parasite name	Carnivores	Artiodactyls	Other
<b>Viruses</b>			
Morbillivirus, canine distemper virus	10	0	0
Parvovirus, canine parvovirus	4	0	0
Vesivirus, feline calicivirus	1	0	0
Coronavirus, feline infectious peritonitis virus	1	0	0
Parvovirus, feline panleukopenia virus	1	0	0
Gammaretrovirus, feline leukemia virus	0	0	0
Aphovirus, foot-and-mouth disease virus	0	7	0
Lentivirus, jembrana disease virus	0	1	0
Morbillivirus, monk seal morbillivirus	1	0	0
Rhadinovirus, ovine herpesvirus 2	0	1	0
Varicellovirus, pseudorabies virus	1	0	0
Lyssavirus, rabies virus	9	2	0
Morbillivirus, rinderpest virus	0	7	0
<b>Bacteria</b>			
Bacillus anthracis (anthrax)	0	5	0
Chlamydia sp. (infectious keratoconjunctivitis)	0	1	0
Fusobacterium necrophorum (hoof rot)	0	2	0
Mannheimia haemolytica (pasteurellosis)	0	1	0
Mycoplasma conjunctivae (infectious keratoconjunctivitis)	0	1	0
Mycobacterium bovis (bovine tuberculosis)	0	2	0
Pasteurella spp. (pasteurellosis)	0	2	0
Yersinia pestis (plague)	0	0	1
<b>Helminths</b>			
Angiocaulus gubernaculatus (nematode)	1	0	0
Diocotophyma renale, giant kidney worm	1	0	0
Dirofilaria immitis, heartworm	1	0	0
Protostrongylus spp., lungworm	0	1	0
Taenia hydatigena, thin-necked bladderworm	0	1	0
<b>Arthropods</b>			
Otodectes cynotis, ear canker mite	1	0	0
Psoroptes sp. (psoroptic mange)	0	1	0
Sarcoptes scabiei (sarcoptic mange)	3	1	0
<b>Protozoa</b>			
Toxoplasma gondii (toxoplasmosis)	2	0	2
<b>Fungi</b>			
Encephalitozoon cuniculi (encephalitozoonosis)	1	0	0

Small and fragmented populations may themselves be more vulnerable to infection. Small populations might be below the critical threshold for pathogen maintenance, causing previously-endemic diseases to become locally extinct. When the population comes into contact with that pathogen again, the loss of herd immunity could result in

heightened morbidity and mortality (Cunningham 1996). Also, small fragmented populations are likely to have reduced genetic variability, even if the population size subsequently increases. Susceptibility to infectious disease and neoplasia (tumours) in Californian sea lions (*Zalophus californianus*), for example, was positively correlated with inbreeding (Acevedo-Whitehouse et al. 2003). The mechanism responsible for this enhanced susceptibility is unknown, but heterozygosity of the major histocompatibility complex (MHC) has been linked to effective immune response in other species (Penn 2002). For example, a reduction in the MHC region of the cheetah's (*Acinonyx jubatus*) genome after an historic population bottleneck may have contributed to the severity of an epidemic of feline infectious peritonitis in captive animals (Evermann et al. 1988). Tasmanian devils are another species in which low genetic diversity (Jones et al. 2004) has increased susceptibility to disease (Pycroft et al. 2007; Woods et al. 2007). An invariably lethal transmissible tumour, not recognised as 'non-self' by the host, is spreading through Tasmanian devil populations, with current trends suggesting extinction could occur within 20 years (McCallum et al. 2007).

Dealing with disease in endangered mammals can be considered a special case within wildlife disease management for several reasons. First, the goal of management is principally the conservation of biodiversity (i.e. prevention of the extinction of populations and maintenance of genetic diversity) rather than disease control or eradication. Indeed, interaction between hosts and parasites is crucial for the healthy functioning of ecosystems and parasites are important components of biodiversity *per se*. Many parasites are host specific and, when treating endangered species in small populations, the inadvertent extinction of parasites is possible. Disease management actions, therefore, must be compatible with the over-arching aim of biodiversity conservation in its broadest sense; this may influence the choice of approach when working with endangered species. Second, in the case of endangered species, sufficient knowledge of the ecology and epidemiology of the host-disease system may be particularly difficult to acquire. Such information can be critical to the effective control of disease in any wildlife population, and consequently ill-informed *ad hoc* interventions to manage disease in endangered species have often done more harm than good. Therefore, the management of disease threats to endangered species needs to be considered as an integral component of the overall conservation plan, subjected to careful scrutiny and provided with adequate financial and logistical support.

Identifying when disease poses a real threat to endangered wildlife populations, and when management or intervention is appropriate, can be challenging for many reasons. The epidemiology of disease in species of conservation concern is often poorly understood because the basic ecology, behaviour and population dynamics of the hosts are usually not well described (Plowright et al. 2008); diseased or dead animals are frequently difficult to detect; and consequently substantial effort and expense is required to estimate disease impact and prevalence. Indeed the true impact of a disease on a population can only be determined through manipulation of the host-parasite relationship, for example by treating or vaccinating a portion of the population. Furthermore, diagnosis of disease is often limited by an absence of diagnostic tests or, where these are available, tests which have not been validated for the species concerned: most diagnostic tests used for wild mammals have been

developed for use with domestic animals and may give poor or inaccurate results when used for wildlife

Despite the difficulties and expense, a thorough understanding of disease epidemiology, and the likely responses of host populations to management intervention, should ideally be gained prior to management intervention, to avoid wasted effort or even damaging interventions. Mathematical models can provide valuable insights into disease epidemiology and the potential impact of interventions, and as such is an important tool for those attempting to manage infectious disease threats in endangered mammal populations (see Chapter 4). In the context of endangered populations these outcomes are typically some measure of the likelihood of persistence of the population, in the face of varying levels of disease risk and over different time periods, or the quantitative demographic impact of disease on population abundance. Modelling infectious disease processes in these populations will be more uncertain. Traditional approaches using deterministic models predict the *average* progress of a disease through a population and often fail to capture key elements that influence the spread of infection in small populations. These elements depend on chance events in transmission. In models of small populations their inclusion will help to inform decision-makers of the range of possible outcomes associated with a disease outbreak. Such (stochastic) models have been successfully applied to the management of infectious disease risks to the world's most endangered canid species, the Ethiopian wolf (*Canis simensis*) (see Box 11.1).

**Box 11.1** Modelling and the management of disease threats in endangered populations: the case of the Ethiopian wolf

Fewer than 600 Ethiopian wolves (*Canis simensis*) persist in seven populations confined to remnant fragments of Afroalpine habitat above 3,000m, in the Ethiopian highlands. Within these fragments, wolves live in discrete packs of 3–13 adults that communally share and defend an exclusive territory. The largest population of wolves (around 300 adults) is found in the Bale Mountains National Park in southeast Ethiopia. In the park, Ethiopian wolves occur in several subpopulations connected by narrow corridors of suitable habitat. The park and surrounding area are also occupied by pastoralists, their livestock and domestic dogs (*Canis lupus familiaris*). These dogs act as reservoirs for a number of infectious diseases, since their high numbers allow several generalist canid pathogens, including rabies and canine distemper, to persist within their populations. Sporadic spillover of these pathogens into the sympatric wolf population has been responsible for a number of large outbreaks – indeed, rabies is recognized as the most immediate threat to the short-term persistence of the Bale wolf population. Management decisions to mitigate this threat have recently taken into account results from mathematical models, which predict the consequences of rabies introduction into the population, and the effect of various intervention strategies on the outcome of such an introduction. This potentially powerful approach successfully combines elements of demographic monitoring, disease surveillance, contingency planning and reactive vaccination.

Models of disease dynamics in Ethiopian wolf populations have progressed from simple population viability analysis (Mace and Sillero-Zubiri 1997) to a

sophisticated spatially-explicit demographically stochastic individual-based model (Haydon et al. 2006). The latter model incorporates the pack-based social structure of the wolves, an important advance as the composition and configuration of packs have been shown to play a critical role in the outcome of rabies introduction into the system. This model was able to quantify the threat posed by rabies to the persistence of wolf populations, an outcome that in itself was useful for galvanising support for a domestic dog rabies vaccination campaign in and around wolf habitats. The model has been used to make specific, practical recommendations to managers on the prevention of, and response to, future rabies outbreaks in the Bale Mountains wolf population. Traditional epidemiological theory is often used to predict the proportion of individuals that must be vaccinated in order to reduce the effective reproduction number ( $R$ ; see Chapter 3) of the agent to less than one and eliminate infectious disease from a population – an approach which generally requires the vaccination of the majority of individuals (in domestic dogs, the coverage required for the elimination of rabies is estimated at 70%). However, the first priority of conservation biologists may be to ensure the long-term persistence of an endangered population. This objective may not require total elimination of all outbreaks, but perhaps only the largest that might compromise long-term population viability. Stochastic epidemiological and demographic models of the Bale wolf metapopulation, suggested that vaccination of as few as 20–40% of wolves against rabies might be sufficient to eliminate the largest outbreaks, and thus prevent populations from reaching low densities from which they would be unlikely to recover (Haydon et al. 2002a). These findings suggested that prophylactic vaccination of the wolf population against rabies could be a feasible and worthwhile undertaking.

The model has also informed contingency plans to deal with potential future outbreaks by showing that the impact of epidemics could be reduced by low-coverage reactive vaccination campaigns even after discovery of the outbreak. Model results have shown that vaccination within the infected zone could be effective and reduce mortality. Long-term persistence of wolf populations could be further improved by focusing reactive vaccination in the habitat corridors between sub-populations.

The Ethiopian wolf rabies model predicts that around 40% of spillover events will ‘fade out’, requiring no management action. If however more than four individuals become infected, the probability that an epidemic will occur increases. Hence, the model provides managers with a trigger threshold, above which action should be taken. Following the diagnosis of rabies in several wolves in the Bale Mountains in 2003, a vaccination programme was implemented which entailed the physical capture and injectable vaccination of wolves (Knobel et al. 2008). The virus did not progress beyond the initially infected subpopulation, and results of simulations based on the developed model demonstrated that the probability of the disease spreading into unaffected areas would have been much greater in the absence of intervention. Given the controversy surrounding the handling of endangered African canids (Woodroffe 2001), such evidence added valuable support to the benefits of this intervention.

(continued)

**Box 11.1** (continued)

This case study clearly illustrates the potential utility of individual-based stochastic models in assisting managers of populations of endangered species in decision-making. The value of such models is dependent on the accuracy of the underlying data. A major strength of the model described here was the volume of detailed demographic and spatial data collected over a number of years by field staff of the Ethiopian Wolf Conservation Programme. This was enhanced by a major surveillance effort during the outbreak, which produced data on mortality patterns and the spatial distribution of cases. A detailed pre-existing database of genetic profiles of the animals within the outbreak area even allowed the pack membership of dead wolves to be ascertained. The ability of mathematical models to successfully inform management decisions for endangered populations thus depends on the synergistic interactions of field biologists and epidemiologists with modellers who have an understanding of the importance of underlying natural ecological processes to the outcome of pathogen introductions in small populations.

As described earlier in this book, approaches to disease management in wildlife can be categorised according to the proposed target of action. For those situations where the disease is better understood, interventions can be directed at the infectious agent through vaccination or medication (see Chapter 6 and Section 11.2); at the host population (see Chapter 7 and Section 11.3), or at the environment (see Chapter 8 and Section 11.4). Special cases arise when species are so valuable or endangered that animals are managed on an individual basis when they may require a combination of techniques (see Box 11.2), or when they are translocated as part of an integrated conservation plan (Section 11.5).

## 11.2 Targeting the Infectious Agent

Management actions targeting the infectious agent can involve (i) administration of anti-parasitic or antibiotic drugs or (ii) vaccination against the infectious agent. The use of anti-parasitic and antibiotic drugs in free ranging endangered mammals has been attempted on a few occasions, but with limited success. Treatment of ectoparasitic mites causing mange has been undertaken in cheetahs (Mwanzia et al. 1995), Mednyi arctic foxes (Goltsman et al. 1996), mountain gorillas (*Gorilla gorilla beringei*) (Graczyk et al. 2001) and southern hairy-nosed wombats (*Lasiorhinus latifrons*) (Ruykys et al. 2006). Individual cases showed a positive response to treatment but the long-term effects on populations are unknown. Intestinal and vascular nematodes have been treated with anthelmintics in red wolves (*Canis rufus*) (Phillips and Scheck 1991) and Florida panthers (*Puma concolor coryi*) (Roelke and Glass 1992).

**Box 11.2** The Mountain Gorilla Veterinary project

Some endangered mammals are considered so valuable that individuals are monitored and treated if they become ill. The Mountain Gorilla Veterinary Project (MGVP), a non-profit group that provides veterinary care to mountain gorillas (*Gorilla gorilla beringei*) (Cranfield et al. 2001; Cranfield et al. 2005), is a prime example of this disease management strategy. MGVP considers the health of the gorillas not in isolation, but as part of an ecosystem that includes sympatric species such as local domestic livestock, wildlife and human populations (Nizeyi et al. 1999, 2002; Graczyk et al. 2002a; Graczyk et al. 2002b; The Mountain Gorilla Veterinary Project 2002 Employee Health Group 2004).

The mountain gorilla exists in two, geographically distinct, island populations: the Virunga Massif, a small body of forest at the intersection of the borders of the Democratic Republic of Congo (DRC), Rwanda and Uganda, and the Bwindi Impenetrable Forest in Uganda. The total estimated population of 700–750 individuals is divided equally between these two sites.

In Rwanda and Uganda, the development of protected areas, in the form of patrolled national parks, and a robust tourist industry has helped to reduce the threat from habitat degradation and poaching, leaving zoonotic disease as the major threat to the health of the gorilla population (Homsey 1999). However, in the DRC, political instability, militia forces and groups of internally displaced persons are currently more immediate threats to both gorilla health and habitat.

The majority of MGVP's routine work consists of health monitoring, preventative health procedures, education, research and the dissemination of information. To do this, MGVP works in partnership with the regional Protected Area Authorities and non-governmental organizations, to provide ongoing monitoring of the gorilla groups, disease monitoring and vaccination of domestic livestock and companion animals adjacent to the national parks, and health monitoring in the form of an annual Employee Health Programme (EHP) (Ali et al. 2004) for those people who work with the gorillas.

Emergency care in the field is provided to gorillas in the event of human induced conditions that are considered to be life-threatening. A 'decision tree' was developed to assist field vets in their choice of action in each case. Cases are usually identified during routine health monitoring visits or from feedback from partner organisations. Subsequent intensive, focal animal monitoring, to establish the nature, degree and progression of disease, is then undertaken. Data such as morbidity, current social status, demographic information (e.g. age, sex, relative 'genetic value' of an individual), geographic and meteorological information (e.g. proximity and interaction with other groups, altitude, season), any relevant history (recent, or likely, transfer to or from the group), and the perceived risks and benefits of intervention to the individual and to the group, all contribute to the decision on whether to monitor, or immobilize and treat. The final decision to immobilise an animal for treatment rests with the local

(continued)

**Box 11.2** (continued)

protected area authority, and is based on factors such as the terrain, weather, time of day, and the availability of appropriate expertise.

The most significant causes of death in the mountain gorilla population are trauma and respiratory disease (Nutter et al. 2005). For example, the MGVP frequently treats injuries resulting from interactions with humans, such as snare removal and bacterial respiratory infections probably transmitted from humans. Intervention is more likely in 'high-risk' and 'high-value' cases such as infants.

Routine collection of blood, urine, faeces and tissue samples occurs during emergency interventions and at *post mortem* examination. All samples are stored in an in-country bio-bank of samples, with duplicates transported to the Biological Resource Center at the Maryland Zoo in Baltimore, USA. The international scientific community is encouraged to apply for access to samples for independent research projects. MGVP collaborates with various laboratories in Europe and the United States for rapid and accurate interpretation of pathological samples.

MGVP is also developing a contingency plan, to be enacted in the event of a catastrophe that threatens the survival of the gorillas. In collaboration with Mississippi State University, MGVP has developed the Internet-supported Management Program to Assist Conservation Technologies (IMPACT) database system, which integrates data collected on the gorillas (e.g. demographic, routine health monitoring, pathology: Cranfield and Minnis 2007). This database may be updated at any time via the Internet, to provide immediate, real-time information on health trends at the level of the individual, the group and the population. IMPACT has the potential to provide an early warning of disease trends associated with an outbreak.

Overall, the MGVP approach has proven to be effective, local political instability notwithstanding. During the period in which MGVP has been in operation there has been an overall increase in gorilla numbers (of 17% in the last 10 years), a significant milestone for the development of a sustainable population.

Both vaccination of the endangered host, and of domestic animal reservoir species, have been proposed as control strategies for minimising the transmission of pathogens to wildlife hosts. During the translocation of critically endangered hirola antelope (*Beatragus hunteri*) in Kenya in the mid 1990s there was an ongoing epizootic of rinderpest with virus circulating in the source and release areas (Kock, 2008). The translocated animals were vaccinated with the standard cattle vaccine without ill-effect and were monitored with no reports of dead animals with signs of the disease despite probable exposure (Butynski 2000). A second example was the vaccination of 65 mountain gorillas in the Virunga volcanoes region of Central Africa, during a measles-like respiratory disease outbreak in 1988 (Hastings et al. 1991). Signs of respiratory disease ceased after the vaccination program was initiated but because all non-pregnant animals had been treated, there was no control group so the role of measles vaccination in preventing the spread of this disease could not be rigorously evaluated. In this case it was considered impractical and unethical to withhold treatment from a control group

of gorillas and other interventions in highly endangered populations are likely to be faced with similar dilemmas. Nevertheless, it is important that wherever possible attempts are made to assess the efficacy of disease management actions, as such information is crucial to the development of future management plans.

Vaccination of the host is also not without its risks. For example, vaccinating African wild dogs before conducting adequate vaccine trials may have led to the failure to control a rabies epidemic in the Serengeti. Subsequent trials demonstrated that multiple doses of rabies vaccine might be required for protection from disease (Woodroffe 1997; Hofmeyr et al. 2004). The use of live canine distemper vaccines in black-footed ferrets has induced fatal canine distemper in the past (Carpenter et al. 1976). In contrast, the use of a killed canine distemper vaccine in the same species failed to protect against fatal distemper infection (Williams et al. 1988).

The control program enacted against rinderpest in Africa highlights the enormous impact that vaccination of domestic animals *can* have on the prevalence of disease in wild mammal populations. Rinderpest caused catastrophic losses of wildlife and livestock after introduction of the virus into Africa in the late 1800s, however widespread vaccination of domestic cattle led to a rapid decline in the incidence of the disease in wild bovids and a marked increase in their abundance (Plowright 1982). By decreasing the number of susceptible domestic animals below the threshold required to sustain rinderpest virus, the cattle vaccination campaigns effectively reduced the distribution of the virus affecting both cattle and wildlife (Rossiter 2001). A similar approach was initiated in the Serengeti ecosystem, in Tanzania, where domestic dogs were vaccinated against pathogens that threaten endangered African canids (see Box 11.3). Domestic dogs were

**Box 11.3** Managing disease threats from a domestic reservoir: rabies outbreaks in endangered African canids

Rabies has been responsible for a number of well-documented outbreaks in endangered African canids, including Ethiopian wolves (*Canis simensis*) and African wild dogs (*Lycaon pictus*). However, the virus appears incapable of persisting indefinitely within these populations, independent of other hosts. The high pathogenicity of the virus, coupled with small host population size, low connectivity between populations, and rapid transmission of the virus through packs facilitated by their social behaviour, ensure the rapid depletion of susceptible hosts and disappearance of the virus. Rabies epidemics in wild dogs and Ethiopian wolves are thus dependent on reintroduction of the virus from a population of one or more reservoir species. Prediction and prevention of these epidemics requires an understanding of the ecology of local reservoir hosts and the transmission dynamics of the virus within and between the reservoir and populations of endangered canids. Although rabies has a broad mammalian host range, within any given geographical area a single species is often principally responsible for its maintenance. Domestic dogs are the principal rabies hosts throughout most of the current distributions of African wild dogs and Ethiopian wolves.

(continued)

**Box 11.3** (continued)**1 No intervention**

In the face of a disease threat to an endangered population, a decision not to intervene may be valid. But this must be a proactive choice, based upon as full an understanding as possible of the consequences of inaction, rather than a decision made by default, through poor preparedness. In view of the shortage of detailed data on the epidemiology and dynamics of infectious disease outbreaks in wild populations, even if no direct intervention is contemplated managers should be prepared, in the event of pathogen spillover or an encroaching epidemic in the reservoir population, to collect as much information as possible on the spatial and temporal spread of disease, recent incidence in the reservoir population, clinical and pathological signs, morbidity and mortality rates, and molecular characteristics of the pathogen. Such data collection should be seen as the minimum adequate management response, and can be used to guide future disease management decisions. Utilising such information to develop mathematical models of possible outcomes of disease introduction into the target population (see Box 11.1) can be useful for future decision-making.

**2 Reducing incidence in the reservoir population**

Reducing the incidence of disease in the reservoir host population will decrease the force of infection acting on the population of interest. Three strategies can achieve this:

*Reducing the density of susceptible animals*

This can be achieved by reducing the survival rate of the population (e.g. culling of stray animals; limiting resource availability by for example burning, burying or otherwise reducing access to refuse), decreasing the fecundity of the population (through the sterilisation of females), or immunising susceptible animals through vaccination. Domestic dog populations can also be limited by reducing the need for people to keep dogs. The relative merit of each of these strategies will depend on local demographic and cultural circumstances, which will in turn affect their cost-effectiveness. In practice, lethal methods of controlling stray dog populations have met with limited success, and the resulting instability of dog populations and antagonism towards rabies control personnel within local communities may result in a net detrimental effect. The World Health Organization Expert Committee on Rabies (WHO 1992) concluded that removal of dogs should not be used in large-scale rabies control programmes unless ecological and socio-cultural studies show it to be feasible within the particular local context. Large-scale mass vaccination of dogs is now accepted as the control method of choice for rabies in most circumstances.

### *Eliminating infected animals from the population*

Because of the danger they pose to human health, local authorities and the public attempt to kill clinically rabid dogs in rabies endemic areas. This probably results in some reduction in transmission to susceptible animals, but in isolation is unlikely to alter the course of an outbreak, since rabid dogs only exhibit clinical signs for a few days. Such actions may also compromise human safety and animal welfare standards, and will potentially miss infected animals that do not exhibit classical clinical signs.

### *Reducing contact between susceptible and infected animals*

This can occur by encouraging owners to restrict the movement of their dogs by for example tying them up, confining them to a kennel or compound, and walking them on a leash. The adoption of these behaviours will however depend on the specific cultural reasons for dog ownership, implementation of education programmes, and possible enforcement by local legislation.

## **3 Reducing contact between reservoir and target populations**

This may be achieved through the confinement methods described above or, in more extreme cases, by fencing off populations of endangered canids. Construction and maintenance of fences is costly and is usually implemented for management purposes other than disease control, for example to reduce human-carnivore conflicts, to prevent human encroachment, or to reduce disease transmission from wildlife to livestock. Reduced disease transmission to wild canids has seldom, if ever, been a primary reason for fencing (although in some small reserves in southern Africa income generated from eco-tourism centred around African wild dogs contributes to the upkeep cost of fences). In addition to the economic cost, the ecological implications of fence construction must obviously also be considered.

## **4 Vaccination of target populations**

For both African wild dogs and Ethiopian wolves, effective vaccination regimens have been developed using commercially available inactivated domestic dog injectable vaccines. Hence, direct vaccination of endangered hosts against rabies is an option. Vaccination strategies may either be prophylactic (to prevent spillover) or reactive. As in all cases where an intervention is contemplated, the benefits of vaccination must be weighed up against the costs, both financial and in terms of risks to target and non-target species. Detailed contingency planning, ideally incorporating mathematical modelling of various outbreak and intervention scenarios, should be conducted in advance of spillover events. Improving delivery strategies for vaccines, particularly through the development of effective oral bait formulations, must be a priority for future research.

In all likelihood a combination of the above management options, dependent on the local context, will be necessary to ensure the persistence of all but the largest populations of Africa's endangered canids.

immunized against rabies, canine distemper, and parvovirus, with the goal of reducing disease outbreaks in lions, African wild dogs and bat-eared foxes (*Otocyon megalotis*) (Cleaveland et al. 2000; Cleaveland et al. 2003). Detailed plans have also been drawn up for the vaccination of Ethiopian wolves (*Canis simensis*) against rabies infection (see Box 11.1).

### 11.3 Targeting Hosts

Disease management action directed towards wild and domestic mammals has often included culling, under the assumption that a reduction in population density will reduce transmission rates. This is however almost always inappropriate when dealing with species of high conservation value. An exception is when infected individuals are culled to reduce the force of infection to susceptible individuals; as is the case during the management of facial tumour disease in Tasmanian devils (see Box 11.4).

#### **Box 11.4** Facial tumour disease in Tasmanian devils

On the Australian island state of Tasmania, devil facial tumour disease (DFTD) threatens the survival of the Tasmanian devil (*Sarcophilus harrisi*), an endemic and endangered marsupial carnivore. Predictions for its future are gloomy, with all populations likely to be affected within five years, followed by extinction of the species in 20–30 years if no action is taken to mitigate the spread of disease (Jones et al. 2007).

DFTD is an emerging infectious disease found exclusively in wild devil populations that appears to be invariably fatal to affected individuals. It was first observed in the mid 1990s and its increasing prevalence and geographic distribution became rapidly apparent (McCallum and Jones 2006). It is a transmissible neoplasm (tumour) that appears to be an infectious allograft (the tumour cells are the infective agent), and is most likely spread by biting (Pearse and Swift 2006).

The disease management strategy currently in place is a multi-faceted approach based on information gathering and risk minimisation. All components address the three possible management options: maintaining insurance populations isolated from the disease for reintroduction in the event of extinction in the wild; in situ management (disease suppression; development of vaccines); and detecting and spreading devils that are resistant to the disease (Jones et al. 2007).

A disease suppression trial is currently underway, whereby any animals captured in the target area showing signs of the invariably fatal tumour are removed and euthanased (Jones et al. 2007). In the first trial, an intensive trapping programme is being implemented on the large isolated Tasman and

Forestier peninsulas (a combined area of 360 km<sup>2</sup>), that are connected to mainland Tasmania by a single bridge. Site isolation, including the possibility of constructing a barrier to devil movement on the bridge that connects this peninsula to the mainland, reduces edge effects and will likely enhance the chance of disease eradication. It is too early to indicate whether disease suppression will be successful in either eradicating or controlling the disease, but initial reports indicate a reduction in the size of the tumours being detected (Jones et al. 2007).

Planning for the establishment of insurance populations of devils incorporates current knowledge of the epidemiology of this unique disease with the population biology of Tasmanian devils, in order to assess the risks and benefits of various translocation options. The genetic diversity of devils has already been reduced by about 50% (Jones et al. 2004), hence it is important to minimise any further reduction. There are currently separately managed captive populations of devils on the Australian mainland and Tasmania, and plans to translocate animals from disease free areas in western Tasmania to offshore islands. Close demographic and genetic monitoring of captive populations has the advantage of requiring a smaller effective and hence actual population size than translocated insurance populations as mating can be controlled to maximise genetic diversity. Captive management is more costly and labour intensive than management on offshore islands. Insurance populations on offshore islands also have the advantage of allowing a larger founder population size and the animals will retain their natural parasites and pathogens as well as behaviours that may be lost in captivity. The overall plan is for an insurance metapopulation comprised of multiple populations of captive and wild-living devils, with managed dispersal between populations with appropriate quarantine steps, to maintain a high level of genetic diversity for 50 years.

Where a disease that threatens an endangered mammal resides in a wild animal reservoir, it may be legitimate to cull the reservoir host in an effort to reduce the likelihood of transmission. For example, it has been suggested that culling introduced grey squirrels (*Sciurus carolinensis*) in the UK, could reduce transmission of squirrelpox virus (SQPV) (see Section 11.4) to the rare native red squirrel (Gurnell et al. 2006). However, culling can have complex effects on host behaviour that may influence transmission rates (see Chapter 7) so the wider ecological consequences of interventions should always be assessed before management action is implemented.

When the threat from disease is particularly severe, the establishment of 'insurance' populations, either in captivity, or free-living in isolation from the disease, may be necessary to prevent extinction (e.g. Williams et al. 1988). Caution must be exercised if disease vectors are involved, or if there is a long asymptomatic stage of infection, in which case thorough quarantine and testing is required before transferring individuals to the 'insurance' population.

## 11.4 Targeting the Environment

Close contact with domestic animals can risk disease transmission to endangered mammal hosts. Minimising such cross-species contact can be accomplished through the use of physical barriers, such as the buffer zones between agricultural areas and ranges of bighorn sheep (*Ovis canadensis*), which have been effective in reducing disease epidemics in susceptible wildlife (Jessup et al. 1991; Jessup et al. 1995). Conservation programmes may have policies that specifically seek to ensure that endangered species are not exposed to domestic animals. For example, domestic dogs are prevented from entering the remaining refuge areas of black-footed ferrets in Wyoming, to avoid transmission of canine distemper virus (CDV) (Williams et al. 1988); and the removal and subsequent banning of domestic dogs from Antarctica has been used to avoid possible transmission of CDV to pinnipeds (Anon 1994). Exposure to humans may pose particular disease threats to primates, and so tourists visiting habituated Mountain gorilla populations in the Virungas and Bwindi conservation areas in Central Africa are limited in group size (eight people), viewing time (one hour) and minimum distance (seven metres) to reduce direct and indirect contact (Ferris et al. 2005). Other measures to prevent transfer of disease from humans to gorillas include burying human faeces deeper than 30 cm and deterring gorillas from private land surrounding their habitat.

Another approach to reducing opportunities for inter-specific disease transmission is to limit temporal overlap in the use of shared water resources or grazing habitats. For example, separation of domestic cattle and bison (*Bison bison*) during the bison birthing season prevents cross-species transmission of brucellosis in the Greater Yellowstone ecosystem (Bienen and Tabor 2006). In Kruger National Park, South Africa, water holes provide focal points for the dissemination of anthrax (*Bacillus anthracis*) throughout ungulate populations. Control of this problem has been tackled by treating waterholes with antibiotics, which has successfully halted the spread of bacteria (Prins and Weyerhaeuser 1987).

The manipulation of habitat and landscape features has been used as an effective tool to make environments more attractive to species of conservation concern. Similarly, there may be opportunities to manage habitats to reduce disease transmission to endangered species, although such actions need to be consistent with the broader aims of natural habitat preservation. The presence of a squirrelpox virus (SQPV) reservoir in the grey squirrel population in England and Wales has been shown to accelerate the rate at which the rare native red squirrel has declined by 20-fold (Rushton et al. 2006). Minimising inter-specific contact is a crucial component of red squirrel conservation in Britain (Gurnell et al. 2006). Red and grey squirrels utilise forest habitats with differing efficiency. In particular the red squirrel, which is best adapted to mature boreal coniferous forests of Scots pine (*Pinus sylvestris*) and Norway spruce (*Picea abies*), is able to thrive in certain coniferous tree plantations, such as Sitka spruce (*Picea sitchensis*), which appear to be avoided by grey squirrels. The Kielder Forest is dominated by Sitka spruce and holds the largest remaining red squirrel population in England. This forest has been managed

to maximise its suitability for red squirrels through tree species selection whilst minimising the likelihood of incursions by grey squirrels by trapping them around forest edges and habitat corridors in particular. Plantation management specifically includes minimising pine and large seeded broadleaves around Sitka plantations (Lurz et al. 2003).

Managing the movement of endangered species between fragmented subpopulations to limit disease transmission has recently been debated in the disease ecology literature. The development and use of 'corridors' of suitable habitat to facilitate movement between small and fragmented populations is increasingly advocated as a means of reducing the deleterious effects of isolation. However, while connectivity diminishes the loss of genetic diversity and allows recolonisation of local populations, it can also increase the risks of disease transmission (Hess 1996a). Nevertheless, recent modelling studies suggest that when a reservoir host (domestic or wild) occupies the matrix between patches, corridors may have relatively little effect on transmission of pathogens between populations of the endangered host, and that corridors should therefore provide a net conservation benefit (Woodroffe 1999; Gog et al. 2002). These investigations were extended to examine the situation where the endangered host and reservoir species occupied the same patches (McCallum and Dobson 2002). All studies concluded that too little connectivity always leads to extinction of the endangered host and the benefit of increased landscape connection far outweighs the risk of increased disease transmission.

## 11.5 Translocation and Reintroduction

Conservation programmes for endangered species usually aim to increase the genetic diversity of small populations, by enhancing the gene flow between fragmented populations and restoring a species historical range after local extinction. Translocation of individuals between different populations, reintroductions and restocking are important tools in many such programmes. However, these activities may themselves present high-risk opportunities for disease transmission, with potentially devastating implications for endangered populations. Consequently, it is essential that the disease risks of all translocations are effectively managed.

### *11.5.1 Why Do Translocations Represent a Disease Risk?*

Animal translocations are thought to play a major role in the emergence of infectious diseases in wildlife (Daszak et al. 2000b; Williams et al. 2002a). Alien pathogens can be introduced with animals translocated into indigenous populations of the same or differing species where they may have a particularly severe impact if the recipients are naïve to infection (Cunningham 1996; De Leo and Dobson 2005). In the absence of effective immunity, the pathogen may cause disease and readily

spread with potentially disastrous consequences. Both domestic and wild animal translocations present a disease risk to endangered species. The rinderpest pandemic in African ungulates described above represents a severe example of the consequences of alien parasite introduction with a translocated domestic mammal (cattle transported from Europe to Africa). There are several well-described examples where the translocation of wild mammals has resulted in anthropogenic spread of infectious diseases such as: the transmission of bovine tuberculosis to a local naïve population of wood bison (*Bison bison anthabascae*) after the introduction of plains bison (*Bison bison bison*) into a National Park in Canada (Carbyn and Watson 2001); the spread of the giant liver fluke (*Fascioloides magna*) to European ungulates when infected elk (*Cervus elaphus*) were introduced into Italy from the USA (Haigh 1988); the introduction of rabies into the raccoon (*Procyon lotor*) population in parts of the eastern United States following the translocation of raccoons to supplement hunted populations (Anthony et al. 1990). Programmes in which captive-bred animals, or animals held away from their geographic region of origin, are translocated are thought to represent a greater risk of alien parasite introduction, particularly where they have been in contact with exotic species, for example in zoological collections (Kirkwood and Sainsbury 1997).

The potential exposure of translocated animals to endemic pathogens in recipient populations, to which they have inadequate immunity, represents another disease hazard of translocations. Animals that have had no exposure to one or more parasites present in the destination environment are likely to be naïve and more susceptible to parasites they encounter after translocation. A classic example is mortality in reintroduced captive-reared black-footed ferrets caused by canine distemper, which was endemic in the wild (Williams et al. 1988). Other examples include: the development of neurological disease in eastern woodrats (*Neotoma floridana*) due to infection with *Baylisascaris procyonis* (a neurotropic roundworm of raccoons) following their reintroduction to New York (Davidson and Nettles 1992); an outbreak of babesiosis in translocated sable antelope (*Hippotragus niger*) (McInnes et al. 1991); and mortality or disease due to cowdriosis (heartwater), trypanosomosis, babesiosis or theileriosis in African antelope, big horn sheep, mule deer (*Odocoileus hemionus*), white rhinoceros (*Ceratotherium simum*) and black rhinoceros (*Diceros bicornis*) (Kock et al. 2007; Kock et al. 1999b; Nijhof et al. 2003).

A further potential consequence of translocations is that pre-existing disease dynamics in the recipient ecosystem can be affected by the introduced species. By acting as new hosts, changing host-parasite dynamics through altering host density, or potentially forming new reservoirs of infection, translocated individuals could exacerbate disease caused by endemic pathogens. This scenario is most likely to occur among closely related wild and domestic species, since parasites are more likely to move between species of higher relatedness. For example, bacterial pneumonia caused by *Pasteurella* sp. resulted in high mortality rates in translocated bighorn sheep (Foreyt 1989), which were spatially correlated with high domestic sheep densities (Singer et al. 2001).

Translocation usually involves capture, transport, quarantine, introduction to a new environment, and subsequent competition for food, territory and mating oppor-

tunities. The associated stress experienced by individual animals can be considerable and may result in immunosuppression and greater susceptibility to infectious disease (Viggers et al. 1993; Kock et al. 2007).

### ***11.5.2 Captivity and Exposure to Pathogens***

The ex situ management of an endangered species may take place for the purpose of acquiring knowledge about the taxon, increasing public awareness of its plight, as a source for breeding and reintroduction, or any combination of these objectives. In critically endangered species, individuals from the few remaining populations are sometimes taken into captivity for captive breeding and reintroduction. Examples include the black-footed ferret in the USA (Thorne and Williams 1988; Williams et al. 1988; Dobson and Lyles 2000), Arabian oryx (*Oryx leucoryx*) in Oman (Spalding et al. 1999) and the golden lion tamarin (*Leontopithecus rosalia*) in Brazil (Gippoliti and Carpaneto 1997). Potential disease outbreaks in such small numbers of highly valuable individuals can have disastrous consequences for the success of conservation projects.

Unfortunately, disease-screening protocols are not always an inherent part of projects involving the captive management of endangered species. However, the time spent in captivity creates a situation of enhanced risk regarding the acquisition of novel diseases. The animals may be exposed to an array of pathogens that they would not normally encounter, such as those transmitted by commensal rodents that inhabit facilities and enclosures, or those carried by related host species in the direct vicinity, be they exotic animals in a zoo, domestic animals on neighbouring land or human caretakers. Pathogens with wide host ranges (which often include domestic animals) are the most likely to infect endangered species (Pedersen et al. 2007). For example, the grazing of domestic sheep along the perimeter fence of an endangered-species breeding centre resulted in transmission of capripox virus (pathogens of sheep, goats and cattle) to Arabian oryx reared for reintroduction purposes. Although only one case developed clinical signs, subsequent screening of the entire herd revealed a seroprevalence of 2% (Greth et al. 1992). However, contact with infected rodents in a captive breeding facility was identified as the source of outbreaks of toxoplasmosis and callitrichid hepatitis (caused by infection with lymphocytic choriomeningitis virus) in golden lion tamarins (Montali and Bush 1992). Captive animals also can be exposed to novel diseases via their food (e.g. an epidemic of toxoplasmosis decimated a captive colony of squirrel monkeys (*Saimiri sciureus*): Cunningham et al. 1992). A wide range of antelope and wild felid species died with neurodegenerative disease following exposure to the bovine spongiform encephalopathy (BSE) agent via commercially-available feed concentrate (bovids) or meat (felids) (Kirkwood & Cunningham 2006). As neither the degree of exposure of other zoo animals nor the biology of the disease (e.g. incubation period, transmissibility) in wild mammals is known, recommendations were made to minimise the risk of infected animals being translocated to disease-free regions or

released to the wild (Kirkwood & Cunningham 1994, Kirkwood & Cunningham 2006). An example where infection acquired in captivity did reach the wild occurred when a pet orang-utan (*Pongo pygmaeus*) was released despite having previously tested positive for tuberculosis, which it was suspected to have acquired from its captors (Bonner 1995). These examples illustrate that where adequate information on risks and appropriate screening are absent, there may be significant opportunities for the transfer of pathogens into areas where they may pose a threat to both indigenous and translocated species.

### 11.5.3 Disease Risk Analysis for Translocations

Knowledge regarding the prevalence of pathogens in wild populations and susceptibility to clinical disease is often lacking for endangered species. Also, for most pathogens of wild mammals, reliable *ante mortem* diagnostic tests are unavailable (Kirkwood and Sainsbury 1997). Often infections are subclinical (the hosts may not necessarily develop clinical disease), which makes detection of the pathogen even more difficult. Consequently, for both translocated animals and recipient populations, enhanced exposure to novel pathogens is a realistic possibility in any translocation project. Although precautions should be taken when undertaking translocations, a 'zero risk' approach is simply not possible.

Although the IUCN provide guidelines that advocate disease monitoring during translocations, if there is no legal obligation to carry out a disease risk analysis, this requirement will often be ignored. However, governments may not be aware of the potentially serious risks of wildlife translocations and therefore often have no statutory regulations on such movements.

Standard disease control methods for any translocation project should include strict quarantine procedures, comprehensive health examinations (including *post mortem* examinations) with appropriate laboratory screening tests to detect a wide range of possible pathogens, vaccination protocols where appropriate, and clinical examinations, including haematological and plasma biochemical analyses where possible, prior to release to assess body condition and anticipate survival in the wild (Montali et al. 1995). To aid the identification of those pathogens that could be important during translocation projects, a risk assessment can be performed. This identifies the diseases that are prevalent in donor and recipient populations. After the major disease risks have been identified, screening for selected pathogens can be incorporated into the translocation project and suitable measures can be identified in the event of an outbreak (e.g. treatment, vaccination, euthanasia). This protocol does, however, rely on previous health studies on the donor and recipient ecosystems, which are often absent or incomplete. The incorporation of such studies should be considered as part of a translocation programme.

A disease risk analysis can be broken down as follows: (Macdairmid and Pharo 2003; Murray 2004).

(i) *Hazard identification*

All known pathogens that could potentially be imported with the species concerned are listed.

(ii) *Risk assessment*

An assessment of risk is carried out on each pathogen identified as a hazard. This evaluates the likelihood and possible consequences, both biological and economic, of entry, establishment or spread of the pathogen to the area of reintroduction.

(iii) *Risk management*

Based on the results of the risk assessments, decisions are made with regards to disease management protocols for the translocation procedure. Screening for the diseases of greatest risk can be planned for both the animals to be translocated and the recipient population. *Post mortem* examinations should be performed on animals found dead in captivity or post-release. Following disease screening, appropriate measures, such as (prophylactic) treatment for certain pathogens and vaccination protocols, should be implemented. In some circumstances it may be considered appropriate to expose the translocated animals to low levels of diseases they might encounter in their new habitat to build up herd immunity. This approach was employed for the reintroduction of black rhinoceros in southern Africa, where the animals were temporarily held in low-density tsetse fly (*Glossina* spp.) areas to permit low exposure rates to *Trypanosoma* spp. prior to release (Kock et al. 1999b). Treatment for certain diseases may also be considered before release. Post-release health surveillance is an important component of risk management because the results of surveillance can be used to refine risk management protocols. Close monitoring of animals' health and behaviour can be achieved through differing methods depending on the species, for example by radio-tracking or trapping.

(iv) *Risk communication*

At all stages during the risk analysis process all stakeholders should be involved in discussions on the potential disease risks and their consequences for the translocation project.

In some cases, the risk analysis may identify a risk that is of such significance that the intended translocation project should be abandoned. For instance, bovine tuberculosis has been identified in black rhinoceroses held in captivity in Western zoos, but as yet not in those in the wild. As there are currently no sufficiently reliable *ante-mortem* screening methods to detect infection, the risk of introducing this pathogen to the free-ranging population outweighs the potential conservation benefits of translocation (Osofsky et al. 2001).

Other approaches have been advocated to minimise or avoid the disease risks associated with animal translocations and ex situ breeding. For instance, translocation of germplasm rather than entire animals can be undertaken. Although disease transfer is still possible (Philpot 1993), it is considerably less likely. Also, where animals are captive-bred for local release within their natural range, they experience continuous exposure to the climate and endemic pathogens of the area.

Programmes for the reintroduction and translocation of endangered species are expensive and time consuming, and may require specialist facilities. The potential

for negative outcomes in terms of transmission of novel pathogens to either the recipient population or translocated individuals is significant, and can have devastating consequences for the conservation project. Consequently, the extra effort and resources required to conduct sufficient research into the potential disease risks, to carry out the appropriate screening and to ensure adequate veterinary involvement throughout, constitute an essential investment in any translocation project.

## 11.6 Future Perspectives

In all likelihood more mammal species will become endangered throughout the world in the near future. At the same time the occurrence of new and emerging diseases are likely to increase. In fact, many of the same processes are likely to be driving both trends. Over-exploitation of natural resources, the disruption of ecosystems and continuing urban expansion bring humans and our livestock into increasingly closer contact with potential sources of disease from wildlife (see Chapter 1). Hence, we can expect to be more frequently challenged with the management of diseases in the small and fragmented populations of endangered mammals.

The imperative to act to safeguard the survival of many species, the heightened opportunities for disease transmission that these interventions incur and the impossibility of screening for all pathogens, mean that a 'zero-tolerance' approach to disease risk is unattainable. However, the thorough and systematic assessment of risk, based on current knowledge and the integration of disease management at all levels of conservation programmes provide the best available framework for continued action.

Most disease threats to endangered mammals are from well-known pathogens that also infect domestic mammals (Pedersen et al. 2007). However, recent experiences such as those in Tasmania (with devil facial tumour disease (DFTD)) and Central Africa (with Ebola and Marburg viruses: see Box 11.5) indicate that pathogens can arise from unexpected or unknown sources. This raises several points worthy of broader consideration in conservation biology and disease management. Firstly, early recognition of an infectious disease as the cause of population decline is crucial to development of a management plan, while identification of the causative agent is of lesser importance and hence syndromic surveillance can be effectively applied to detect emerging disease threats. Secondly, loss of genetic diversity can expose populations to unforeseen disease threats. With habitat loss and fragmentation increasingly leading to a reduction in genetic diversity of wild animal populations, more species may become susceptible to disease. Thirdly, both host-specific pathogens (e.g. DFTD), as well as the more familiar generalist pathogens (e.g. rabies and CDV) that reside in abundant reservoir species, are able to pose a significant extinction risk particularly when their transmission is frequency dependent (e.g. McCallum 2008).

**Box 11.5** Emerging disease, human health and endangered species: Ebola in Central Africa

Marburg and Ebola virus are members of the filoviridae that cause acute viral haemorrhagic disease (Pourrut et al. 2005) and are a source of current concern for the health of humans and endangered primates. In Central Africa, Ebola Zaire virus (EBOV) has killed over 1,300 people, and populations of great apes (gorillas and chimpanzees) have declined by 80% in some of their last strongholds in Central Africa (Walsh et al. 2003; Leroy et al. 2004). Following a human EBOV outbreak along the Gabon-Congo border in late 2001-early 2002, the first gorilla (*Gorilla gorilla gorilla*) carcass was found in June 2002, and by October 130 animals out of 143 had disappeared. Of the 32 carcasses found, 10/12 gorilla and 3/3 common chimpanzee (*Pan troglodytes troglodytes*) carcasses tested were positive to EBOV by PCR, antigen capture or immunohistochemical staining (Bermejo et al. 2006). This has led to the presumption that the dramatic decline of gorilla and chimpanzee populations in the region was due to EBOV. It is estimated that these populations will take at least 75 years to recover to pre-EBOV outbreak densities (Walsh et al. 2005).

Identification of the reservoir hosts of filoviruses has proved challenging. Despite infection being identified in both primates and duikers (*Cephalophus* sp.), neither is thought to be the reservoir host due to their high disease-related mortality rates. Serological and antigen assays have provided further evidence of this. In an outbreak of EBOV-Reston subtype in a captive primate facility in the Philippines in 1996, 12.5% (131/1051) of the animals were antigen positive, but only 0.2% (3/1732) were seropositive (Miranda et al. 1999; Miranda et al. 2002). The index human filovirus cases had previously been linked to caves or buildings with resident bats, one such case reported a sting or bite from an arthropod, and there was some indication that the virus resembled certain plant viruses. Experimental studies with EBOV were therefore undertaken using 24 species of plants and 19 species of vertebrates and invertebrates (Swanepoel et al. 1996). Infection of Angola free-tailed bats (*Mops condylurus*), little free-tailed bats (*Chaerephon pumilus*), and Wahlberg's epauletted fruit bats (*Epomophorus wahlbergi*) resulted in virus replication without death (Swanepoel et al. 1996). Subsequently EBOV RNA was recovered from the liver and spleen tissues of wild forest dwelling fruit bats, following an outbreak of infection in humans (Leroy et al. 2005). This was followed by the discovery of specific immunoglobulin IgM antibodies in the same bat species (the hammer-headed fruit bat (*Hypsignathus monstrosus*), Franquet's epauletted bat (*Epomops franqueti*) and the little collared fruit bat (*Myonycteris torquata*)). The PCR and serological findings suggested acute infection followed by seroconversion, and together the evidence strongly implicates fruit bats as reservoir hosts for these viruses.

(continued)

**Box 11.5** (continued)

It is unclear whether the fruit bat populations in Central Africa have a long standing association with EBOV and are endemically infected, or whether the virus may exhibit wave-like spread through the region (Walsh et al. 2005). Although the subtype EBOV-Zaire, probably diverged from EBOV-Ivory Coast subtype 700–1,300 years ago (Suzuki and Gojobori 1997), all Central African isolates identified subsequent to the Yambuku outbreak in 1976 are closely related descendents of a Yambuku-like virus suggesting a recent expansion in viral diversity (Walsh et al. 2005; Biek et al. 2006b). EBOV isolates from fruit bats show genetic variation, which suggests all strains in bats have descended from a common ancestor within the last 30 years (Biek et al. 2006b). Whether this is due to a genetic bottleneck, or there is another, as yet unidentified reservoir, is not known. There is, however, no evidence to date of an epidemic wave with associated spillovers occurring in other regions from where EBOV has been isolated. The death of chimpanzees (*Pan troglodytes verus*) in Tai National Park in Ivory Coast, suggest a single event (Le Guenno et al. 1999), with no local or regional epidemic causing further deaths in susceptible primate or duiker species.

Investigating EBOV in wild mammals has presented particular challenges in terms of diagnosing the cause of the decline in populations of endangered great apes, identifying the principal reservoir host and describing the pattern of infection. Another, perhaps even more challenging task, is to consider how to control or manage this virulent pathogen in an extensive and complex ecosystem such as the Central African forest. If the acute epidemic in Central Africa has been facilitated by the role of primates and duikers (Walsh et al. 2007), then it may quickly run out of susceptible hosts, particularly in those parks where over 80% of resident apes have already been lost (Walsh et al. 2003). This may also be true if the density of the putative fruit bat reservoir is low, the infectious period short and transmission is density dependent.

EBOV infection in African apes is an emerging zoonotic disease with potentially catastrophic consequences for endangered primates. There is compelling circumstantial evidence that EBOV has caused the decline of apes in the Congo-Gabon region, although the actual number of clinical cases diagnosed is small compared to the suggested level of mortality. There is little serological evidence of the disease in ape populations (Bermejo et al. 2006), however, this is unsurprising if apes are 'new' or spillover hosts with high mortality. When outbreaks occur in humans, survivors with detectable immunity are scarce (Busico et al. 1999; Jezek et al. 1999). It is therefore difficult to consider the potential merits of vaccination without a better understanding of the epidemiology and ecology of this disease. Even then, the practicality of vaccination, and impacts of intervention need to be carefully assessed. When the target population is small (e.g. a few tens of animals) vaccination may be a reasonable approach to consider, but where there are tens of thousands of animals in an extensive area, it may be impractical regardless of the conservation status of the species.

Wherever possible, efforts should be made to monitor and evaluate the efficacy of interventions to manage disease, to provide an evidence base for future work. This is true for the management of disease in any wildlife population, but for endangered species it takes on particular significance because of the potentially catastrophic effects of ineffective or counter-productive interventions. This may be particularly challenging for endangered species, as it is often not possible to collect scientifically robust data on the efficacy of interventions because there is no 'untreated' control group for comparison. The benefits of mathematical modelling are increasingly evident in these situations, particularly when epidemiological and host demographic data are available (see Box 11.1).

As recognition has increased for the role that parasites play in wildlife ecology and ecosystem health, including the value associated with their potential regulation of host numbers and contribution to biodiversity (Daszak and Kilpatrick 2008), so has the realisation that the health of humans, wild and domestic animals and ecosystems are inextricably connected (see Boxes 11.2 and 11.5). Hence, it is multidisciplinary teams that can best provide the necessarily broad range of knowledge and skills posed by the problems of the management of disease in wild mammals in the 21st century.