Good Medicine for Conservation Biology: the Intersection of Epidemiology and Conservation Theory

KEVIN D. LAFFERTY* AND LEAH R. GERBER†‡

*U.S. Geological Survey, University of California, Marine Science Institute, Santa Barbara, CA 93106-6150, U.S.A.
†National Center for Ecological Analysis and Synthesis, University of California, Santa Barbara, CA 93101-3351, U.S.A.

Abstract: *Infectious disease can be a concern for several aspects of conservation biology, such as determining threats to species, estimating population viability, and designing reserves, captive breeding, and recovery programs. Several measures are useful for describing infectious diseases in host populations, but it is not straightforward to determine the degree to which a particular disease may affect a host population. The most basic epidemiological theory suggests that populations should be least subject to host-specific infectious disease when they are at low abundance (paradoxically, the state at which they are in most need of conservation action). There are important exceptions, however, such as when a reservoir host exists or when Allee or stochastic effects occur. Several of the key threats to biodiversity—habitat alteration, introduced species, pollution, resource exploitation, and climate change—can facilitate and/or impair transmission of infectious disease. Common management tools such as population viability analysis rarely address infectious disease explicitly. We suggest that such an inclusion is both possible and warranted. Considerations of infectious disease may influence the way we determine whether a species is in need of protection and how we might design reserves and captive breeding programs. Examples from the literature suggest that (1) introduced pathogens can make abundant species rare and (2) diseases of domestic animals can dramatically affect rare species. For both scenarios, conditions that cause stress or reduce genetic variation may increase susceptibility to disease, whereas crowding and cross-species contact can increase transmission. Southern sea otters (*Enhydra lutris nereis*) make an interesting case study for consideration of the intersection of epidemiology and conservation because disease may be an important factor limiting the growth of otter populations. We conclude that pathogens are of increasing concern for conservation. Because many newly emerging pathogen dynamics often do not conform to the simplifying assumptions used in classic epidemiology, a detailed understanding of pathogen life history will illuminate the intersection of epidemiology and conservation theory.*

Buena Medicina para la Biología de la Conservación: la Intersección de la Epidemiología y la Teoría de Conservación

Resumen: *Las enfermedades infecciosas pueden ser una preocupación para varios aspectos de la biología de la conservación, tales como determinar amenazas para las especies, estimar la viabilidad poblacional, el diseño de reservas, la reproducción en cautiverio y los programas de recuperación. Varias medidas son útiles para describir enfermedades infecciosas en poblaciones huésped; sin embargo, no es sencillo determinar el grado en el que una enfermedad particular puede afectar a una población huésped. La teoría epidemiológica más elemental sugiere que las poblaciones debieran estar menos sujetas a enfermedades infecciosas específicas cuando su abundancia es baja (paradójicamente, el estado en el que requieren de mayor acción de conservación). Sin embargo, hay excepciones importantes como cuando existe un huésped reservorio o cuando ocurren efectos Allee o estocásticos. Varias de las principales amenazas a la biodiversidad (alteración de hábitat, especies introducidas, contaminación, explotación de recursos, cambio climático) pueden facilitar y/o impedir la transmisión de enfermedades infecciosas. Las herramientas comunes de manejo, tales como el análisis de viabilidad poblacional, raramente consideran enfermedades infecciosas explícitamente. Sugerimos que tal inclusión es tanto posible como justificada. Considerar enfermedades infecciosas puede influir en la forma en que determinamos si una especie requiere de protección y en como podemos diseñar reservas y programas de reproducción en cautiverio. Ejemplos de la literatura sugieren que (1) patógenos introducidos pueden hacer*

‡*Current address: Department of Biology, Arizona State University, Box 8715, Tempe, AZ 85287, U.S.A., email leah.gerber@asu.edu*
Paper submitted October 19, 2000; revised manuscript accepted June 13, 2001.

Conservation Biology, Pages 593-604
Volume 16, No. 3, June 2002

*que una especie abundante sea rara y (2) enfermedades de animales domésticos pueden afectar a especies raras. En ambos casos, las condiciones que producen estrés o que reducen la variación genética pueden incrementar la susceptibilidad, mientras que aglomeraciones y contacto trans-específico puede incrementar la transmisión. Las nutrias marinas (*Enhydra lutris nereis*) son un estudio de caso interesante para considerar la intersección de la epidemiología y la conservación porque la enfermedad puede ser un importante factor limitante del crecimiento de la población de nutrias. Concluimos que los patógenos son una preocupación para la conservación. Debido a que su dinámica a menudo no se ajusta a las suposiciones simplistas utilizadas en la epidemiología clásica, el entendimiento detallado de la historia de vida de los patógenos ayudará a explicar la intersección de la epidemiología y la teoría de la conservación.*

Introduction

The need to integrate theories from the fields of ecology, demography, taxonomy, and genetics to evaluate human effects on biological diversity and to develop practical approaches to prevent the extinction of species is central to conservation biology (Wilson 1992; Soulé 1996). Specific methods have been developed for determining the best strategies for protecting threatened species, designing nature reserves, and initiating breeding programs to maintain genetic variability in small populations (Primrack 1993). In addition, conservation biologists are gaining an increasing awareness of the role of disease in the management of species and ecosystems, captive breeding and reintroduction, and habitat restoration (Scott 1988; Cooper 1989; Lyles & Dobson 1993; Hess 1994; Real 1996; Dobson & McCallum 1997; Woodroffe 1999). Nonetheless, the effects of most infectious disease probably go unnoticed (Scott 1988), and we need more research on the population-level effects of diseases of wild organisms (Woodroffe 1999).

We summarize aspects of epidemiology that are relevant to conservation biology, note where epidemiology could explicitly interact with conservation theory, and provide examples of the degree to which infectious diseases (i.e., the consequences of parasitic organisms that harm their hosts) may interact with the main threats to biodiversity and conservation practices. Our population approach contrasts with the traditional veterinary focus on diagnosis and treatment (as noted by Lyles & Dobson 1993). We conclude with a case study of southern sea otters (*Enhydra lutris nereis*) that exemplifies some of the real-world complexities that emerge in efforts to infuse principles of epidemiology into conservation practice.

Influence of Key Principles of Epidemiology on Conservation Questions

How does one determine whether a disease will affect a population? Because most organisms are host to parasites and pathogens, it is important to accurately ascertain whether severe pathology is attributable to infectious disease before determining that the disease is of conservation concern. For example, of six viruses that infect lions (*Panthera leo*) in the Serengeti, only one, canine distemper virus, clearly decreases lion abundance (Packer et al. 1999). Pathology has historically been the domain of veterinary science in which veterinarians play the key role in addressing effects of disease.

The effect of an infectious disease on a host population does not increase linearly with its pathology in individual hosts. This is because infectious diseases that rapidly kill their hosts, as severe as they may seem, have a tendency to become extinct locally. For this reason, the effect of an infectious disease on host population density is expected to be highest at intermediate pathogenicity (Anderson 1979). One useful indirect indicator that an infectious disease is affecting a host population is the absolute difference between the prevalence (percentage of hosts infected) of diseased hosts in the dying (or morbid) subset of the population versus that in the entire population (McCallum & Dobson 1995). If the difference is large, then the infectious disease is likely affecting the host population dramatically.

Perhaps the most fundamental principal of epidemiology is that the spread of a directly transmitted infectious disease agent through a population increases with the density of susceptible and infectious hosts. Empirical comparative studies support the prediction that intensity and prevalence tend to increase with host population density (Anderson 1982; Anderson & May 1986; Arneberg et al. 1998). Most simple epidemiological models indicate a host-threshold density below which a parasite cannot invade a host population, suggesting that rare or depleted populations should be less subject to invasion by a host-specific infectious disease agent. Observations of the epidemiology of morbillivirus (e.g., measles) of humans (Black 1966) and cattle (Aune & Schladweiler 1992) support this prediction. In addition, the density-dependent nature of transmission makes infectious diseases unlikely to be agents of extinction (Dobson & May 1986). With these predictions in mind, it appears that

the main message from epidemiology to conservation biology is that, with the exception of factors that impair a host's ability to battle a normally benign infection, infectious diseases are irrelevant. Important exceptions to this general theory include disease agents with dynamics controlled by large populations of reservoir hosts.

So what are the conditions that make infectious diseases a concern for conservation? Changes in host susceptibility, such as increased susceptibility due to inbreeding, nutritional status, stress or immunosuppression, may increase the efficiency of transmission to new hosts (Scott 1988). Immune systems are costly to maintain, and animals that are stressed may need to divert energy from defense (Rigby & Moret 2000). Malnutrition (Beck & Levander 2000), toxins from pollution (Kahn 1990), and thermal stress from climate change (Harvell et al. 1999) are hypothesized to increase susceptibility to infectious diseases and may also affect host populations directly (the concept of a multiple stressor; Lafferty & Kuris 1999). Infectious diseases, even if they do not normally drive hosts extinct, might reduce abundant hosts to low densities where other agents of extinction, such as reproductive failure due to Allee effects or random stochastic events, could cause extinction. In addition, host behavior, through aggregation, sociality, or crowding, increases opportunity for contact even if absolute host numbers are low. Finally, the density of alternate hosts (sometimes called reservoir hosts) can greatly relax the extent to which transmission rate depends on the density of a single host species.

This raises the possibility of extinction due to infectious disease (Woodroffe 1999), particularly if the alternative host suffers little pathology. For example, because Mallard Ducks (*Anas Platyrhynchus*) are particularly resistant to duck plague, they can be important carriers of the disease and can initiate mass mortalities among other waterfowl (Friend 1987). Such an interaction among host species in a community is known as apparent competition (Greenman & Hudson 2000). Analogous to this are facultative parasites, such as fungi that can live entirely in the soil. Such fungi may be one cause of worldwide declines in amphibians, partially because they are able to persist even when amphibians are absent (Daszak et al. 1999).

Human Activities and the Effects of Disease on Natural Populations

Habitat alteration is perhaps the greatest threat to biodiversity. When the size of available habitat shrinks, remaining individuals may initially be crowded into abnormally high densities, placing populations at risk for epidemics. This risk is further magnified if crowding degrades the environment and reduces the resources that might improve disease resistance. The risk of infectious disease may be offset, however, by an eventual reduction in host population size or a loss in vectors or intermediate hosts associated with habitat degradation. For example, parasites with complex life cycles are usually less common in degraded habitats, presumably because they require a diversity of hosts (Lafferty 1997). Other forms of habitat degradation—particularly damming, irrigation, logging, and road construction—result in favorable conditions for some vectors and intermediate hosts, increasing the prevalence of infectious disease agents (Lafferty & Kuris 1999).

Isolation due to habitat fragmentation may reduce the flow of infectious disease agents among populations in the short term (Hess 1994), but it can promote a long-term loss in genetic diversity (Frankel & Soulé 1981) and a potential loss of corresponding evolutionary adaptations to infectious disease (Lyles & Dobson 1993; Coltman et al. 1999). Reduced exposure to infectious disease agents may also lower the immunocompetency of individuals, reducing herd immunity such that infectious disease outbreaks have devastating consequences (Lyles & Dobson 1993). Perhaps most important, habitat fragmentation leads to greater edge effects that may increase the potential for infectious disease agents to spread from agriculture and pets (e.g., canine distemper virus; Cleaveland et al. 2000).

Domestic plants and animals are often managed in dense monocultures conducive to transmission of infection (Knops et al. 1999). In some cases, these disease agents cross over to closely related wild species. Infectious diseases from domestic sheep have extirpated populations of bighorn sheep (*Ovis Canadensis*) (Goodson 1982), rinderpest (brought to East Africa with cattle) has devastated native ungulates (Dobson 1995), several infectious diseases from domestic poultry have affected wildfowl (Friend 1987), black-head disease from domestic turkeys contributed to the extinction of the Heath Hen (*Tympanuchus cupido cupido*) (Simberloff 1986), and fisheries have introduced whirling disease into native salmonid populations (Hedrick et al. 1998) and crayfish plague into Europe (Alderman 1996). Brucellosis, probably brought to America from domesticated cattle, also infects native bison (*Bison bison bison*) and elk (*Cervus elaphus*) which, ironically, may be culled to protect cattle (Dobson & Meagher 1996). Tropical birds from the pet trade have introduced infectious diseases to wild birds (Cooper 1993). Domestic dogs have brought canine parvovirus, sarcoptic mange, rabies, and distemper to wildlife (Daszak et al. 2000). For instance, canine distemper virus led to the death of 35% of the lions in the Serengeti (Roelke-Parker et al. 1996). Recently, there have been efforts to vaccinate domestic dogs to protect wildlife from canine diseases (Woodroffe 1999). Humans, the most widespread of all species, can transmit measles to mountain gorillas and polio to

chimpanzees (Daszak et al. 2000). However, while pathogens such as fungi and viruses are routinely introduced to control insect pests, there have been no documented cases of non-target effects of conservation concern from these agents (Goettel & Hajek 2001).

Subsidized native species may increase in abundance, altering patterns of disease concomitant with habitat loss associated with human development of the landscape. For example, raccoons (*Procyon lotor*) adapt well to human settlements and can become dense in urbanized areas. Florida panthers (*Puma concolor coryi*) are at a greater risk of contracting rabies when they feed on raccoons than if they feed on deer. Efforts to restore deer populations have successfully reduced panther exposure to rabies (Roelke et al. 1991). Raccoons are also the final host for a nematode (*Baylisascaris procyonis*) that is pathogenic for other mammals that serve as intermediate hosts. The regionally endangered Allegheny woodrat (*Neotoma magister*) collects raccoon feces for nest-building material and, as a result, suffers such high mortality that woodrats apparently cannot persist where raccoons are common (LoGiudice 2000).

Exotic species invasions occur as a consequence of global trade and travel. When these species become successfully established in new habitats, they may affect native species through the processes of predation and competition, which may have indirect effects on native infectious diseases. One of the very reasons for the success of introduced species may be their release from natural enemies such as parasites (Lafferty & Kuris 1996). This is particularly likely if (1) an introduced species arrives in small numbers so that infectious disease agents quickly go extinct, (2) required intermediate hosts or vectors are absent in the invaded region or, (3) a species arrives uninfected, as might seeds or larvae in ballast-water transport (Lafferty & Kuris 1996). In addition, local infectious-disease agents may not be able to infect an introduced species with which they have not coevolved (Cornell & Hawkins 1994; contrary to Sakanari & Moser 1990). Despite this general pattern, when infected species invade, their disease agents may spread through closely related hosts that have no evolved defenses (Culver 1999).

Pollution is a growing contributor to habitat degradation and can interact with disease, sometimes in unpredictable ways. Acid precipitation negatively affects trematodes because of the effect of low pH on snails that act as intermediate hosts (Marcogliese & Cone 1996). Heavy-metal pollution reduces the abundance of parasites with complex life cycles, perhaps because parasites are more sensitive than their hosts or because infected hosts die more easily (Lafferty 1997). In contrast, all studied forms of pollution increase ciliate parasites in fish (Lafferty 1997) because these parasites are particularly successful in stressed hosts (Kahn 1990). For substances that bioaccumulate, the effects are likely to be greater for top predators (which often suffer from several other threats). Eutrophication and thermal effluent also increase parasitism, but this may be due to the epidemiological consequences of increased host density, not host susceptibility (Lafferty 1997). For example, eutrophication increases the abundance of freshwater snails (*Helisoma* spp.), which increases the abundance of the trematode *Ribeiroia ondatrae*, which increases parasitism of amphibians, which leads to limb deformation and high rates of mortality (Johnson & Lunde 2002).

Resource exploitation, such as hunting or fishing, may reduce the persistence of an infectious disease if it drives target hosts below a threshold density (Dobson & May 1987). This is particularly true if human actions selectively remove infected individuals, as is seen in the high proportion of infected moose (*Alces alces*) killed by hunters (Rau & Caron 1979). This effect also occurs in a variety of fisheries (reviewed by Lafferty & Kuris 1999). The reverse may also occur if a fishery selectively avoids infected individuals, as in the fishery for Tanner crab (*Chionoecetes bairdi*) affected by parasitic dinoflagellates (Kuris & Lafferty 1992), and as when humans move infected individuals around in the process of exploiting them (Culver & Kuris 2001). There are several possibilities for indirect effects related to exploitation. The removal of a top predator enables prey densities to increase such that epizootics may occur (Lafferty & Kushner 2000), and the removal of a prey base may force predators to move into areas where they may have increased contact with infectious disease (e.g., rinderpest; Dobson 1995).

Epidemiological Principles and Conservation Practice

Captive Breeding

Captive breeding and reintroduction as a mechanism to conserve imperiled species is complicated by infectious disease. An unfortunate example is the extinction of the remnant population of a Polynesian tree snail by a microsporidian (Daszak & Cunningham 1999). Similarly, one of the last remaining breeding colonies of black-footed ferrets (*Mustela nigrepes*) was decimated after individuals infected with canine distemper virus were unknowingly added (Williams 1986). There are several factors associated with captive breeding that increase a species' contact with infectious disease agents. For example, high population density may have helped a herpes virus spread rapidly through a captive breeding colony of cranes (Docherty & Romaine 1983), significantly reducing their survival (Carpenter et al. 1980). Contaminated food sources can expose captive animals to protozoans, prions, and viruses (Daszak et al. 2000). Cross-species contact, particularly in zoos, also increases the

chance of infectious-disease outbreaks. The virus that causes catarrhal fever can spread in zoos from virus-tolerant wildebeest to endangered ungulates such as Pere David's deer and Indian gaur (Thorne & Williams 1988). Foster mothers of a different species may pass disease agents to foster offspring (Carpenter et al. 1980; Cooper 1989). If animals are stressed in captivity, their susceptibility may be higher as well (Real 1996).

In light of these problems, Derrickson and Snyder (1992) recommend that captive-breeding programs occur in replicated localities within the species' native range and away from sites with arthropod vectors, so as to avoid exposure to exotic and native infectious-disease agents and to provide insurance against disaster. In addition, staff should avoid caring for other species that might transmit disease. Based on these principles, the captive-breeding program for the black-footed ferret was eventually successful, and breeding centers were replicated in different areas to prevent the possibility of infectious disease decimating a large portion of the remaining individuals.

Reintroduction

The effects of infectious disease may also influence the success of reintroduction programs (Viggers et al. 1993; Cunningham 1996). Once animals are infected in captivity, it may be impossible to release them, as with Arabian oryx (*Oryx leucoryx*) infected with bluetongue virus and orangutans (*Pongo pymaeus*) infected with human tuberculosis. Dobson and McCallum (1997) describe a case study of the Bali Mynah (*Leucopsar rothschildi*), one of the most threatened birds in the world with only about 30 individuals in the wild. Around 750 Bali Mynahs live in zoos and will form the basis for a reintroduction program. The majority of these captive birds test positive for shedding *oocysts* of the coccidian *Isospora rothschildi*, the cause of atoxoplasmosis. In this case, the question of whether wild birds are infected with atoxoplasmosis and the extent to which release of infected birds poses a risk to the wild population has yet to be resolved. At present, reared birds face a 1-year quarantine prior to release, and it has been proposed that they be released only to areas lacking wild Bali Mynah birds. Viggers et al. (1993) provide a series of measures to reduce the risk of disease in reintroduction programs, including quarantine, diagnosis, and cure. Ironically, lack of exposure to wild infectious diseases may reduce immunocompetency; similarly, inbreeding and a lack of natural selection may reduce genetic resistance and variation (Cunningham 1996). In cases where there are only a few infectious diseases of concern, vaccination may be useful. For example, captive-bred White-tailed Sea Eagles can be vaccinated against *Clostidium botulinum*, an important cause of mortality in the wild (Dobson & McCallum 1997). In light of the cost of vaccination, however, it is worth considering that there is, as yet, no evidence that it has been effective in protecting any threatened mammals. This is partly due to the fact that effectiveness is often difficult to monitor, because of an understandable unwillingness to leave some animals unvaccinated to serve as controls (Woodroffe 1999).

Reserves

One approach to conserving biodiversity is to establish reserves so that populations of rare species (particularly exploited species) might recover or so that some portion of the habitat can persist in a relatively pristine state (Myers & Mertz 1997; Lauck et al. 1998). In theory, corridors can influence the spread of infectious disease among reserves (Hess 1994). Hess (1994) used models to show that, for intermediate rates of disease-induced mortality, infectious diseases could move along corridors and produce severe negative consequences; under most conditions, however, corridors provide a buffer to extinction. If reserves are successful in achieving high densities of a threatened species, they may make the population more vulnerable to epidemics. Similarly, parasites associated with high host diversity are likely to increase in reserves. For example, trematode parasites of snails attain higher densities in salt-marsh reserves than in degraded areas, presumably because the shorebirds and other hosts they require to complete their life cycles are more abundant there (Lafferty 1997). An indirect example of the potential effect of reserves is the possibility that dense, multispecies aggregations of wild birds created by feeding stations increase disease transmission (Daszak et al. 2000). Reserves do not increase the prevalence of infectious disease in all cases. In California, for example, spiny lobsters (*Panulirus interruptus*) are heavily fished, which leads to increased densities of their prey, the sea urchin (*Strongylocentrotus purpuratus*) (Lafferty & Kushner 2000). In turn, these urchins overgraze the kelp forest, leading to starvation and frequent bacterial epidemics of urchins. In a marine reserve that protects spiny lobsters from harvest, the lobsters prey on sea urchins, and the urchin population apparently becomes too small for infectious-disease outbreaks. Reserves may buffer wild animals from domestic animals that are the source of infectious diseases; for example, isolating bighorn sheep from domestic sheep is the most effective way of protecting bighorn sheep from disease (Woodroffe 1999).

Population Viability Analysis and Risk Analysis

Population viability analysis (PVA) is sometimes useful for risk-classification of species under the U.S. Endangered Species Act (ESA) and the World Conservation Union (IUCN) Red List of Threatened and Endangered

Wildlife. In the few cases in which PVAs include infectious disease, disease occurs as a stochastic, density-independent reduction in the population growth rate in a manner similar to how one might incorporate bad weather. Very different results are likely if infectious disease is explicitly considered. Disease can influence the risk of population decline to low densities in a few distinct ways. As mentioned previously, while a host-specific agent of infectious disease is likely to go locally extinct before causing a local extinction of its host, it may be capable of driving the host density to low levels before it does, making the population susceptible to stochastic events. In this sense, disease, like any catastrophe, should always decrease the expected time to extinction. Furthermore, because the epidemic spread of an infectious disease in a population is increasingly unlikely at low densities, infectious diseases might have less of an effect in small populations than would a density-independent mortality event. In addition, stochastic events that co-occur with an epidemic may eliminate the pathogen from the local host population. The level of sophistication of PVA models has increased such that compensatory density-dependent survival can be incorporated, a phenomenon that what one would expect in an epidemic.

Synthesis and Patterns from the Literature

Our review suggests some general patterns regarding the intersection of epidemiology and conservation theory. We briefly synthesize infectious-disease considerations—density, susceptibility, exposure—as they pertain to conservation theory and practice. Because infectious-disease transmission generally increases with the density of the target species, species in decline should suffer less from host-specific infectious diseases. Exceptions include cases where habitat fragmentation or captivity result in increased contact and disease spread among individuals of a declining species, suggesting that disease transmission should be of concern in conservation strategies that increase density, such as captive breeding, zoos, restoration, and reserves. A number of factors, including poor nutrition, pollution, and climate change, may increase susceptibility or the chance that an individual host will become infected once exposed. If stressors also decrease population density, it will be difficult to predict their net influence on disease dynamics. Most important, an increase in, or introduction of, species that can act as reservoir hosts, intermediate hosts, or vectors is likely to increase the importance of disease as a cause of species declines and a barrier to recovery.

We reviewed several studies of infectious diseases of hosts that were, according to the authors, of conservation concern. We tabulated information from a subset of cases that we believed provided sufficient evidence that infectious disease reduced population density or inhibited recovery. For each of the resultant 29 examples, we recorded the host, infectious-disease agent, source of the agent, consequences of the disease at the population level, and actions taken to control the disease and results of these actions (Table 1). We divided the examples into two general categories: (1) species initially at natural densities that were decimated by an epidemic and (2) species that were rare and put into increased jeopardy by an infectious disease. The majority of examples were of viruses (12), the most prevalent of which was canine distemper virus, followed by fungi (6), bacteria (4), helminths (3), protozoans, (3) and an arthropod (1).

For common species decimated by an epidemic, the source of the disease was usually novel or unknown previous to the epidemic. Sources for these epidemics were most often intentionally introduced species. For rare species with inhibited recovery, the source of the epidemic was usually a domestic animal, most commonly dogs. Actions to reduce the effects of disease were attempted in half of the examples. Such actions were taken on the part of the infected individual (chemotherapy, vaccination), reservoir population (vaccination, culling), or target population (vaccination, culling). The outcomes of such actions were not well understood. Treating sick individuals usually was not intended to control disease at the population level. Vaccinations, in particular, had unclear effects at the population level, perhaps because of the difficulty of establishing controls. Culling was the action most commonly associated with reducing disease, a testament to the importance of host or reservoir density in driving disease dynamics.

Role of Disease in Recovery of the Southern Sea Otter

Overexploitation by the fur trade nearly led to the extinction of southern sea otters. Such a bottleneck probably had as great an effect on otter-specific diseases as it did on otters themselves. After hunting ceased at the turn of the last century, otters increased in numbers and expanded their range (Estes 1990). For northern sea otters, (*Enhydra lutris*) annual rates of growth have been 17–20%, whereas the growth of southern sea otters has been much slower (approximately 5%). Since 1995, southern sea otter numbers have declined abruptly at a rate of 2–3% per year, primarily because of an increase in mortality rates among young adults, calling into question the proposal that the population be considered for delisting (U.S. Fish and Wildlife Service 2000).

We discuss the role of disease in the conservation of southern sea otters, because this species' situation illustrates how particular case studies are dominated as much

Table 1. Infectious diseases that have influenced host species of conservation concern.*

Type	*Transmission*	*Disease agent*	*Host (scientific name)*	*Source*	*Consequences*	*Actions; results*	Reference
Cause decline of naturally dense populations							
bacteria	STD	chlamydia	Koala (*Phascolarctos cinereus*)	native	decreased birth rate	chemotherapy; individual treatment	Cork et al. 2000
bacteria	unknown	withering syndrome	black abalone (*Haliotis cracherodii*)	unknown	>90% population reduction	fishery closure; none	Lafferty & Kuris 1993
fungus	opportunistic	aspergillosis	sea fans (several species)	soil	80% population reduction	none	Nagelkerken 1996, cited by Harvell et al. 1999
fungus	direct	bark fungus	American beech (*Fagus grandifolia*)	introduced beech	30–50% population reduction	chemotherapy; individual treatment	Real 1996
fungus	direct	blight	American chestnut (*Castanea dentata*)	introduced chestnut	near extinction	none	Real 1996
fungus	opportunistic	chytrid fungus	frogs (several taxa)	soil	increased mortality	none	Laurance et al. 1996, cited by Woodroffe 1999; Woodroffe 1999
fungus	direct	crayfish plague	European crayfish (several taxa)	introduced crayfish	extirpations	none	Alderman 1996, cited by Daszak et al. 2000
fungus	vector	Dutch elm disease	American elm (*Ulmus americana*)	introduced elm wood	>50% population reduction	culling; control	Woodroffe 1999
protozoan	vector	avian malaria	Hawaiian land birds (several taxa)	introduced mosquitoes	29 species extinct, 17 endangered	none	Real 1996; Van Riper et al. 1986, cited by Real 1996
virus	vector/direct	avian pox	Hawaiian land birds (several taxa)	introduced birds	30 species extinct, 17 endangered	none	Warner 1969
virus	vector	louping ill	Red Grouse (*Lagopus lagopus*)	domestic sheep	>50% population reduction	cull hares; effective	Hudson et al. 1995
virus	direct	parapoxvirus	red squirrel (*Sciurus vulgaris*)	introduced grey squirrel	>90% population reduction	none	Rushton et al. 2000
virus	direct	rabies	wolf (*Canis lupus*)	Arctic fox	>60% population reduction	none	Chapman 1978
virus	direct	rinderpest	African ungulates (several taxa)	domestic cattle	>80% population reduction	control rinderpest in cattle; effective	Woodroffe 1999; Plowright 1982, cited by Woodroffe 1999
Inhibit recovery of small populations							
arthropod	direct	scabies	bighorn sheep (*Ovis canadensis*)	domestic sheep	80% population reduction	chemotherapy, buffer from sheep; unclear	Woodroffe 1999; Jessup et al. 1991, cited by Woodroffe 1999
bacteria	direct	mycoplasmosis	desert tortoise (*Gopherus agassizii*)	captive tortoises	90% population reduction	none	Woodroffe 1999; Jacobson 1994, cited by Woodroffe 1999
bacteria	direct	cholera	bighorn sheep (*Ovis canadensis*)	domestic sheep	local extinction	none	Woodroffe 1999; Plowright 1982, cited by Woodroffe 1999
helminth	trophic	acanthocephalan peritonitis	southern sea otter (*Enhydra lutris nereis*)	native birds	increased mortality	none	this paper
helminth	direct	heterakiasis	Grey Partridge (*Perdix perdix*)	domestic pheasant	>90% reduction	none	Tompkins et al. 2000
helminth	trophic	larval migrans	Allegheny wood rat (*Neotoma magister*)	subsidized raccoons	local extinction	none	LoGiudice 2000
protozoan	direct	black-head	Heath Hen (*Tympanychus cupido*)	domestic turkeys	extinction	none	Simberloff 1986
protozoan	direct	steinhausiosis	partula snails (*Partula turgida*)	unknown	extinction	none	Daszak & Cunningham 1999, in Daszak et al. 2000
virus	direct	canine distemper	African lion (*Panthera leo*)	domestic dogs	33% reduction	vaccinate domestic dogs; unclear	Woodroffe 1999; Roelke-Parker et al. 1996
virus	direct	canine distemper	African wild dog (*Lycaon pictus*)	domestic dogs/jackal	local extinction	vaccine, vaccinate domestic dogs; unclear	Woodroffe 1999; LoGiudice 2000
virus	direct	canine distemper	black-footed ferret (*Mustela nigripes*)	live vaccine	>90% reduction	switch vaccine, successful	Woodroffe 1999; LoGiudice 2000
virus	direct	canine distemper	black-footed ferret (*Mustela nigripes*)	domestic dogs?	>90% reduction	vaccine, ban domestic dogs; unclear	Woodroffe 1999; Williams et al. 1998, cited by Woodroffe 1999
virus	direct	parvovirus	wolf (*Canis lupus*)	domestic dogs	reduced recovery	none	Woodroffe 1999; Mech & Goyal 1995, cited by Woodroffe 1999
virus	direct	rabies	Ethiopian wolf (*Canis simensis*)	domestic dogs	>50% density	vaccinate domestic dogs; unclear	Woodroffe 1999; Laurenson et al. 1997, cited by Woodroffe 1999
virus	direct	rabies	African wild dog (*Lycaon pictus*)	domestic dogs/jackal	local extinction	vaccine, vaccinate dogs; unclear	Woodroffe 1999; Kat et al. 1995, cited by Woodroffe 1999

**Examples classified either by causing a decline of a naturally dense population or by inhibiting the recovery of small populations. Available information, including type of disease, mode of transmission, disease agent, host species, source of disease, known consequence for the host populations, actions taken to rectify the disease effect, and known results of these actions, is recorded for each disease.*

by peculiarities as by general theory. Many decades of necropsies have provided insight into otter diseases and other sources of mortality. Although there is some question about the extent to which information collected from stranded otters provides a measure of causes of mortality, infectious disease is associated with lethal pathology in nearly half of stranded animals (Thomas & Cole 1996), indicating that infectious disease may be responsible for the slow annual growth rates of the California population. Otters are accidental hosts for several unusual diseases for which they serve as a dead-end for transmission. Many otter deaths (12%) result from bacterial infections (e.g., from an infected wound), typically *Streptococcus* (Thomas & Cole 1996). Because these are secondary infections, we do not discuss them further except to note that susceptibility to bacterial infections might be higher in immunologically compromised individuals.

As top predators, sea otters, like other marine mammals, bioaccumulate lipophillic toxins. Such contaminants can affect the mammalian immune system; for example, harbor seals (*Phoca vitulina*) fed fish from polluted areas had lower killer-cell activity, decreased responses to T- and B-cell mitogens, and depressed antibody responses (DeStewart et al. 1996). It has been speculated that such immunosuppression can be a cofactor in mass mortalities associated with morbillivirus (Van Loveren et al. 2000). Correlative evidence supports the hypothesis that marine contaminants may increase an otter's susceptibility to infectious diseases. Otters with high levels of DDT (Nakata et al. 1998) and Butyltin (Kannan et al. 1998) may be more likely to maintain infectious disease, perhaps because these pollutants impair immune function in marine mammals (Swartz et al. 1994). With the exception of PCBs, many contaminant levels are higher off the coast of California than off Alaska (Bacon et al. 1999), where disease is thought to be a less important source of mortality (Margolis et al. 1997; but see Rausch 1953). Controlled experiments with mink as a model may allow a better determination of the role of contaminants in affecting otter immune systems and the corresponding susceptibility to infectious diseases.

Valley Fever (Coccidioidomycosis)

Valley fever, an unusual disease found in humans (and other mammals, including, occasionally, sea otters), is caused by the inhalation of spores of the soil fungus *Coccidioides immitis*. Thomas and Cole (1996) report valley fever as the cause of 4% of otter deaths sampled. The emergence of valley fever as an otter disease corresponds with an increase in human cases in California. Fungal spores become airborne with dust from construction and agriculture, activities that are common along much of the otter's range. Inhalation of spores can lead to fatal pneumonia. Immunosuppression may also play a role in this disease, at least for humans. It is a frequent opportunistic infection among HIV patients, whereas most healthy people exhibit no symptoms. Efforts to reduce the exposure of humans to valley fever (e.g., soil treatments) may indirectly protect sea otters from dust containing fungal spores.

Protozoal Encephalitis

Sea otters serve as intermediate hosts for two potentially pathogenic protozoans, the coccidian *Sarcosystis neurona*, which has introduced opossums (*Didelphis virginiana*) as definitive hosts (Lindsay et al. 2000), and *Toxoplasma gondii*, which has cats as definitive hosts. *Toxoplasma gondii* infects 22% of otters, and toxoplasmosis is the cause of death in 3% (Cole et al. 2000) to 8.5% (Thomas & Cole 1996). Most mammals (including humans) and birds can serve as intermediate hosts if they consume oocysts from cat feces or tissue cysts contained in the flesh of infected intermediate hosts. The effect of toxoplasmosis varies widely among hosts. Although we do not know how sea otters consume cysts, cat feces can enter coastal waters from storm drains or sewage. Otters might ingest oocysts directly or, as suggested by Cole et al. (2000), filter-feeding invertebrates may act to concentrate viable oocysts transmittable to otters during a predation event. In addition, some otters may become infected by tissue cysts if they consume birds, such as gulls, that have become infected by foraging on human refuse. Although otters only infrequently prey on birds (Riedman & Estes 1988), the potential for exposure could easily accumulate over an otter's life span. The dependency between immunocompetency and toxoplasmosis is not known for otters but would be worth investigating, given the frequency at which toxoplasmosis is an opportunistic disease among immunosuppressed humans. Better waste-disposal practices and a reduction in the contaminants responsible for immunosuppression might reduce the effects of toxoplasmosis in the otter population.

Acanthocephalan Peritonitis

The most frequent cause of death from disease among otters (14% of all mortalities) occurs when otters become infected with acanthocephalan worms (*Polymorphus kenti*, a shorebird parasite) by consuming the intermediate host, the sand crab (*Emerita analoga*; Hennessy & Moorejohn 1977; Thomas & Cole 1996). Ingestion of sand crabs also exposes otters to trematodes that may cause pathology. Most sand crabs are infected with a number of larval worms (Lafferty & Torchin 1997), and otters can eat several of these small crabs in a foraging bout. When a predator consumes infected crabs, the worms excyst in the stomach and attempt to lodge themselves in the intestinal wall of their host. Although

the worms never mature in an otter, they sometimes penetrate the intestine, leading to contamination of the body cavity with bacteria (peritonitis), a condition that is often fatal (Thomas & Cole 1996).

Historical records indicate that acanthocephalan peritonitis was rare in southern sea otters, probably because the animals did not usually feed in the shallow, sandy habitat where sand crabs occur, preferring, instead, large invertebrates in rocky reefs and kelp forests. But as otter densities increase toward their carrying capacity in a region, their foraging can shift to less favored prey species and habitat, such as sand crabs in sandy areas (Stephenson 1977). In addition, as otters have expanded their range to the south, they have encountered long stretches of sandy habitat where sand crabs are often the most abundant macroinvertebrate (Dugan et al. 2000). The proportion of dead otters harboring acanthocephalans appears to have increased steadily over the last three decades (Hochberg 1998). To determine the extent to which exposure to acanthocephalans might affect population trends, we compared existing data on otter mortality (U.S. Geological Survey stranding database, courtesy of J. Estes) with the (incomplete) 1968–1989 acanthocephalan records from carcasses (Hochberg 1998). The proportion of the population found dead in a particular year increased with the proportion of animals with acanthocephalans ($r = 0.508$, $n = 18$, $p < 0.05$). Because the percentage of otters found dead in a particular year is negatively associated with population growth rates in the following year ($r = -0.440$, $n = 31$, $p < 0.05$), our analysis suggests that acanthocephalans may contribute to variation in the population trends of sea otters. More detailed analyses that partition otter mortality among its various sources are needed to better account for this pattern and are underway.

The risk of exposure to acanthocephalans may vary between age classes and locations. Pups and juveniles are most likely to die from peritonitis (Thomas & Cole 1996), suggesting that inexperience or foraging inefficiency might lead young otters to feed on sand crabs. Otter populations might feed more on secondarily preferred items, such as sand crabs, in places where they have exhausted their preferred foods of mollusks and sea urchins (Estes et al. 1981; Ostfeld 1982). Humans may have increased the prevalence of this disease by competing with otters for food, causing them to seek alternative prey. Such an increase in parasitism by nonotter parasites occurred in Alaska following a shift in the diet of otters to fishes and crabs—which serve as intermediate hosts for seal nematodes and shorebird trematodes, respectively—after otters overexploited sea urchins and mollusks (Rausch 1953). The prevalence of acanthocephalans in a sand crab population varies greatly at a local scale (Lafferty & Torchin 1997) and with the density of the shorebird population (N. Smith, unpublished data). Limiting acanthocephalan transmission by controlling final host birds on sandy beaches may not be desirable given concern that these birds are currently declining due to human disturbance (Howe et al. 1989).

Diseases in Sea Otters and Human Alteration of the Environment

Our sea otter case study demonstrates that the effects of disease can be idiosyncratic and highlights some of the unexpected consequences that can occur when a species that was reduced in range and abundance begins reoccupying its historical range, coming into contact with novel diseases. Although disease contributes to the unusually high mortality experienced by southern sea otters and perhaps limits the growth rate of the population, few of the disease risks to otters follow simple predictions of epidemiology. The diseases that cause the most mortality in otters are accidental and seem to be newly emerged. In some cases, humans may have influenced the spread and emergence of these new diseases.

Perhaps the biggest benefit to be gained by understanding the role of disease in southern otters is that it will help focus attention on the sources of mortality that will influence recovery efforts. Although little action can be taken to specifically buffer the otter population from disease, other independent events may alter the disease landscape. Improved control of sewage disposal into coastal areas may decrease the prevalence of bacterial infections and perhaps reduce bioaccumulation of contaminants, which could improve otter immune defenses. Airborne fungal spores that cause valley fever may be reduced by soil treatment and restrictions on agricultural and construction activities intended to protect human health. Increased disturbance of birds by human recreation on beaches (Lafferty 2001) might result in fewer shorebirds and their parasites, which would reduce the prevalence of acanthocephalan peritonitis in sea otters. Although this paints a seemingly rosy future, as otters continue to expand their range south, their preferred prey—abalone and sea urchins—are host to a larval gnathostome nematode (*Echinocephalus pseudouncinatus*) that uses elasmobranchs as definitive hosts (Millemann 1963). In mammals, gnathostomes are known to migrate through the tissues and can cause brain damage (Miyazaki 1960). Thus, diseases may continue to emerge along with further expansion of the sea otter's range.

Conclusions

Many examples indicate that infectious disease is a concern for conservation efforts. This has been most evident and, consequently, most confronted in efforts to develop captive-breeding programs for endangered species. Most lacking (and perhaps easiest to rectify) are

theoretical approaches for considering infectious disease in analyses of extinction risk. We emphasize that this is an area for a fruitful intersection between conservation biology and epidemiology. The likely infectious diseases of concern for rare species are those with broad host ranges or for which the species of concern is an accidental host for emerging diseases fostered by introduced species, habitat degradation, climate change, and pollution. Monitoring for disease and reducing crowding, inbreeding, and selection for susceptibility will help conservation biologists begin to understand and minimize disease risks.

Acknowledgments

This work was supported by the National Center for Ecological Analysis and Synthesis, funded by the National Science Foundation (grant DEB-94-21535), the University of California-Santa Barbara, the California Resources Agency, and the California Environmental Protection Agency. A. Dobson, J. Estes, P. Hudson, P. Kareiva, A. Kuris, C. Mitchell, M. McCarthy, R. Ostfeld, J. Sabo, M. Samuel, M. Torchin, G. VanBlaricom, and two referees provided insightful comments on earlier drafts of this paper.

Literature Cited

Alderman, D. L. 1996. Geographical spread of bacterial and fungal diseases of crustaceans. Revue Scientifique et Technique de l Office International des Epizooties **15:**602-632.

Anderson, R. M. 1979. Parasite pathogenicity and the depression of host population equilibria. Nature **279:**150-152.

Anderson, R. M. 1982. Epidemiology. Pages 204-251 in F. E. G. Cox, editors. Modern parasitology. Blackwell Scientific Publications, Oxford, United Kingdom.

Anderson, R. M., and R. M. May. 1986. The invasion, persistence and spread of infectious diseases within animal and plant communities. Philosophical Transactions of the Royal Society of London, Series B **314:**533-570.

Arneberg, P., A. Skorping, B. Grenfell, and A. F. Read. 1998. Host densities as determinants of abundance in parasite communities. Proceedings of the Royal Society of London, Series B **265:**1283-1289.

Aune, K., and P. Schladweiler. 1992. Wildlife laboratory annual report. Montana Department of Fisheries Wildlife and Parks, Helena.

Bacon, C. E., W. M. Jarman, J. E. Estes, M. Simon, and R. J. Norstrom. 1999. Comparison of organochlorine contaminants among sea otter (*Enhydra lutris*) populations in California and Alaska. Environmental Toxicology and Chemistry **18:**452-458.

Beck, M. A., and O. A. Levander. 2000. Host nutritional status and its effect on a viral pathogen. Journal of Infectious Diseases **182:**S93-S96.

Black, F. L. 1966. Measles endemicity in insular populations: critical community size and its evolutionary implication. Journal of Theoretical Biology **11:**207-211.

Carpenter, J. W., T. R. Spraker, and M. N. Novilla. 1980. Disseminated visceral coccidiosis in Whooping Cranes. Journal of the American Veterinary Medicine Association **177:**845-848.

Chapman, R. C. 1978. Rabies: decimation of a wolf pack in Arctic Alaska. Science **201:**365-367.

Cleaveland, S., M. G. J. Appel, W. S. K. Chalmers, C. Chillingworth, M. Kaare, and C. Dye. 2000. Serological and demographic evidence for domestic dogs as a source of canine distemper virus infection for Serengeti wildlife. Veterinary Microbiology **72:**217-227.

Cole, R. A., D. S. Lindsay, D. K. Howe, C. L. Roderick, J. P. Dubey, N. J. Thomas, and L. A. Baeten. 2000. Biological and molecular characterizations of *Toxoplasma gondii* strains obtained from southern sea otters (*Enhydra lutris nereis*). Journal of Parasitology **86:**526-530.

Coltman, D. W., J. G. Pilkington, J. A. Smith, and J. M. Pemberton. 1999. Parasite-mediated selection against inbred Soay sheep in a free-living, island population. Evolution **53:**1259-1267.

Cooper, J. E. 1989. The role of pathogens in threatened populations. International Comittee for Bird Conservation Technical Bulletin **10:**51-59.

Cooper, J. E. 1993. Historical survey of disease in birds. Journal of Zoo and Wildlife Medicine **24:**256-264.

Cork, S. J., T. W. Clark, and N. Mazur. 2000. Conclusions and recommendations for koala conservation. Conservation Biology **14:** 702-704.

Cornell, H. V., and B. A. Hawkins. 1994. Patterns of parasitoid accumulation on introduced herbivores. Pages 77-89 in B. A. Hawkins and W. Sheehan, editors. Parasitoid community ecology. Oxford University Press, New York.

Culver, C. C. 1999. The aperture of marine gastropods: factors precluding settlement of fouling organisms. Ph.D. dissertation. University of California, Santa Barbara.

Culver, C. S., and A. M. Kuris. 2001. The sabellid pest of abalone: the first eradication of an established introduced marine bioinvader. Pages 100-101 in J. Pederson editor. Marine bioinvasions. Massachusetts Institute of Technology Sea Grant, Cambridge, Massachusetts.

Cunningham, A. A. 1996. Disease risks of wildlife translocations. Conservation Biology **10:**349-353.

Daszak, P., and A. A. Cunningham. 1999. Extinction by infection. Trends in Ecology and Evolution **14:**279.

Daszak, P., L. Berger, A. A. Cunningham, A. D. Hyatt, D. E. Green, and R. Speare. 1999. Emerging infectious diseases and amphibian population declines. Emerging Infectious Diseases **5:**735-748.

Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2000. Emerging infectious diseases of wildlife: threats to biodiversity and human health. Science **287:**443-449.

Derrickson, S. R., and N. F. R. Snyder. 1992. Potentials and limits of captive breeding in parrot conservation. Pages 133-163 in S. R. Beissinger and N. F. R. Snyder, editors. New world parrots in crisis. Smithsonian Institution Press, Washington, D.C.

DeStewart, R. L., P. S. Ross, J. G. Voss, and A. D. M. E. Osterhaus. 1996. Impaired immunity in harbour seals (*Phoca vitulina*) fed environmentally contaminated herring. Veterinary Quarterly **18:**S127-S128.

Dobson, A. P. 1995. Rinderpest in the Serengeti ecosystem: the ecology and control of keystone virus. Pages 518-519 in R. E. Junge, editor. Proceedings of a joint conference. American Association of Zoo Veterinarians (AAZY), Wildlife Disease Association, and American Association of Wildlife Veterinarians. AAZV, East Lansing, Michigan.

Dobson, A. P., and R. M. May. 1987. Patterns of invasions by pathogens and parasites. Pages 58-76 in H. A. Mooney, and J. A. Drake, editors. Ecology of biological invasions of North America and Hawaii. Ecological studies 58. Springer, New York.

Dobson, A. P., and H. McCallum. 1997. The role of parasites in bird conservation. Pages 155-173 in D. H. Clayton and J. Moore, editors. Host-parasite evolution: general principles and avian models. Oxford University Press, Oxford, United Kingdom.

Dobson, A., and M. Meagher. 1996. The population dynamics of brucellosis in the Yellowstone National Park. Ecology **77:**1026-1036.

Docherty, D. E., and R. I. Romaine. 1983. Inclusion body disease of cranes: a serological follow-up to the 1978 die-off. Biological Conservation **54:**33-45.

Dugan, J. E., D. M. Hubbard, D. L. Martin, J. M. Engle, D. M. Richards, G. E. Davis, K. D. Lafferty, and R. F. Ambrose 2000. Macrofauna communities of exposed sandy beaches on the Southern California

mainland and Channel Islands. Pages 339–346 in D. R. Brown, K. L. Mitchell, and H. W. Chang editors, Proceedings of the fifth California islands symposium. Publication 99-0038. Minerals Management Service, Santa Barbara, CA.

Estes, J. A. 1990. Growth and equilibrium in sea otter populations. Journal of Animal Ecology **59:**385–401.

Estes, J. A., R. J. Jameson, and A. M. Johnson. 1981. Food selection and some foraging tactics of sea otters. Pages 606–641 in J. A. Chapman and D. Pursley, editors. Worldwide Furbearer Conference proceedings, 1980. Worldwide Furbearer Conference, Frostburg, Maryland.

Frankel, O. H., and M. E. Soulé. 1981. Conservation and evolution. Cambridge University Press, Cambridge, United Kingdom.

Friend, M. 1987. Field guide to wildlife diseases. 1. General field procedures and diseases of migratory birds. U.S. Fish and Wildlife Service, Washington, D.C.

Goettel, M.S., and A.E. Hajek. 2001. Evaluation of non-target effects of pathogens used for management of arthropods. Pages 81–98 in E. Wajnberg, J.K. Scott and P.C. Quimby, editors. Evaluating indirect ecological effects of biological control. CABI, Oxford, United Kingdom.

Goodson, N. J. 1982. Effects of domestic sheep grazing on bighorn sheep populations: a review. Proceedings of the Biennial Symposium of Northern Wild Sheep and Goat Council **3:**287–313.

Greenman J. V., and P. J. Hudson. 2000. Parasite-mediated and direct competition in a two-host shared macroparasite system. Theoretical Population Biology **57:**13–34.

Harvell, C.D., K. Kim, J. M. Burkholder, R. R. Colwell, P. R. Epstein, D. J. Grimes, E. E. Hofmann, E. K. Lipp, a. D. M. E. Osterhaus, R. M. Overstreet, J. W. Porter, G. W. Smith, and G. R. Vasta. 1999. Emerging marine diseases: climate links and anthropogenic factors. Science **285:**1505–1510.

Hedrick, R. P., M. El-Matbouli, M. A. Adkison, and E. MacConnell. 1998. Whirling disease: re-emergence among wild trout. Immunological Reviews **166:**365–376.

Hennessy, S. L., and G. V. Moorejohn. 1977. Acanthocephalan parasites of the sea otter, *Enhydra lutris*, off coastal California. California Fish and Game Bulletin **63:**268–272.

Hess, G. R. 1994. Conservation corridors and contagious disease: a cautionary note. Conservation Biology **8:**256–262.

Hochberg, F. G. 1998. Acanthocephalan parasites of the southern sea otter (*Enhydra lutris nereis*) and the sand crabs (*Emerita analoga* and *Blepharipoda occidentalis*) off California. Report. U.S. Geological Survey, National Wildlife Health Center, Madison, Wisconsin.

Howe, M. A., P. H. Geissler, and B. A. Harrington, 1989. Population trends of North American shorebirds based on the International Shorebird Survey. Biological Conservation **49:**185–200.

Hudson, P. J., R. Norman, M. K. Laurenson, D. Newborn, M. Gaunt, L. Jones, H. Reid, E. Gould, R. Bowers, and A. Dobson. 1995. Persistence and transmission of tick-borne viruses: *Ixodes ricinus* and louping-ill virus in Red Grouse populations. Parasitology **111:**S49–S58.

Johnson, P. T. J., and K. B. Lunde. 2002. Trematode parasites and amphibian limb malformations in the western United States: are they a concern? In M. J. Lannoo, editor. Status and conservation of US amphibians. In press. University of California Press, Berkeley.

Kahn, R. A. 1990. Parasitism in marine fish after chronic exposure to petroleum hydrocarbons in the laboratory and to the Exxon Valdez oil spill. Bulletin of Environmental Contamination and Toxicology **44:**759–763.

Kannan, K. K., S. Guruge, N. J. Thomas, S. Tanabe, and J. P. Giesy. 1998. Butyltin residues in southern sea otters (*Enhydra lutris nereis*) found dead along California coastal waters. Environmental Science and Technology **32:**1169–1175.

Knops, J. M. H., D. Tilman, N. M. Haddad, S. Naeem, C. E. Mitchell, J. Haarstad, M. E. Ritchie, K. M. Howe, P. B. Reich, E. Siemann, and J. Groth. 1999. Effects of plant species richness on invasion dynamics, disease outbreaks, insect abundances and diversity. Ecology Letters **2:**286–299.

Kuris, A. M., and K. D. Lafferty. 1992. Modelling crustacean fisheries: effects of parasites on management strategies. Canadian Journal of Fisheries and Aquatic Sciences **49:**327–336.

Lafferty, K. D. 1997. Environmental parasitology: what can parasites tell us about human impacts on the environment? Parasitology Today **13:**251–255.

Lafferty, K. D. 2001. Birds at a southern California beach: seasonality, habitat use and disturbance by human activity. Biodiversity and Conservation **10:**1–14.

Lafferty, K. D., and A. M. Kuris. 1993. Mass mortality of abalone (*Haliotis cracherodit*) on the California Channel Islands: tests of epidemiological hypotheses. Marine Ecology Progress Series **96:**239–248.

Lafferty, K. D., and A. M. Kuris. 1996. Biological control of marine pests. Ecology **77:**1989–2000.

Lafferty, K. D., and A. M. Kuris. 1999. How environmental stress affects the impacts of parasites. Limnology and Oceanography **44:**564–590.

Lafferty, K. D., and D. Kushner. 2000. Population regulation of the purple sea urchin (*Strongylocentrotus purpuratus*) at the California Channel Islands. Pages 379–381 in D. R. Brown, K. L. Mitchell, and H. W. Chang, editors. Proceedings of the fifth California Islands symposium. Publication 99-0038. Minerals Management Service, Carmarillo, California.

Lafferty, K. D., and M. E. Torchin. 1997. Parasites of the sand crabs, *Emerita analoga* and *Blepharipoda occidentalis*, and the risk of infection for foraging sea otters, *Enhydra lutris*. Report. U.S. Geological Survey, National Wildlife Health Center, Madison, Wisconsin.

Lauck, T., C. W. Clarke, M. Mangel, and G. R. Munro. 1998. Implementing the precautionary principles in fisheries management through marine reserves. Ecological Applications **8:**S72–S78.

Lindsay, D. S., N. J. Thomas, and J. P. Dubey. 2000. Biological characterisation of *Sarcocystis neurona* isolated from a southern sea otter (*Enhydra lutris nereis*). International Journal for Parasitology **30:**617–624.

LoGiudice, K. 2000. *Baylisascaris procyonis* and the decline of the Allegheny woodrat (*Neotoma magister*). Ph.D. dissertation. Rutgers University, New Brunswick, New Jersey.

Lyles, A. M., and A. P. Dobson. 1993. Infectious disease and intensive management: population dynamics, threatened hosts and their parasites. Journal of Zoo and Wildlife Medicine **24:** 315–326.

Marcogliese, D. J., and D. K. Cone. 1996. On the distribution and abundance of eel parasites in Nova Scotia: influence of pH. Journal of Parasitology **82:**389–399.

Margolis, L., J. M. Groff, S. C. Johnson, T. E. McDonald, M. L. Kent, and R. B. Blaylock. 1997. Helminth parasites of sea otters (*Enhydra lutris*) from Prince William Sound, Alaska: comparisons with other populations of sea otters and comments on the origin of their parasites. Journal of the Helminthological Society of Washington **64:**161–168.

McCallum, H., and A. P. Dobson. 1995. Detecting disease and parasite threats to endangered species. Trends in Ecology and Evolution **10:** 190–194.

Millemann, R. E. 1963. Studies on the taxonomy and life history of Echinocephalid worms (Nematoda : Spiruroidea) with a complete description of *Echinocephalus pseudouncinatus* Millimann, 1951. Journal of Parasitology **49:**754–764.

Miyazaki, I. 1960. On the genus *Gnathostoma* and human gnathostomiasis, with special reference to Japan. Experimental Parasitology **9:**338–370.

Myers, R. A., and G. Mertz. 1997. The limits of exploitation: a precautionary approach. Ecological Applications **8:**S165–S169.

Nakata, H., K. Kannan, L. Jing, N. Thomas, S. Tanabe, and J. P. Giesy. 1998. Accumulation pattern of organochlorine pesticides and polychlorinated biphenyls in southern sea otters (*Enhydra lutris nereis*) found stranded along coastal California, USA. Environmental Pollution **103:**45–53.

Ostfeld, R. S. 1982. Foraging strategies and prey switching in the California sea otter. Oecologia **53:**170–178.

Packer, C., S. Altizer, M. Appel, E. Brown, J. Martenson, S. J. O'Brien, M. Roelke-Parker, R. Hoffman-Lehmann, and H. Lutz. 1999. Viruses

of the Serengeti: patterns of infection and mortality in African lions. Journal of Animal Ecology **68:**1161-1178.

Primrack, R. B. 1993. Essentials of conservation biology. Sinaur Associates, Sunderland, Massachusetts.

Rau, M. E., and F. R. Caron. 1979. Parasite induced susceptibility of moose to hunting. Canadian Journal of Zoology **57:**2466-2468.

Rausch, R. 1953. Studies on the helminth fauna of Alaska. XIII. Disease in the sea otter, with special referene to helminth parasites. Ecology **34:**584-604.

Real, L. A. 1996. Sustainability and the ecology of infectious disease. Bioscience **46:**88-97.

Riedman, M. L., and J. A. Estes. 1988. Predation on seabirds by sea otters. Canadian Journal of Zoology **66:**1396-1402.

Rigby, M. C., and Y. Moret. 2000. Life-history trade-offs with immune defenses. Pages 129-142 in R. Poulin, S. Morand, and A. Skorping, editors. Evolutionary biology of host-parasite relationships: theory meets reality. Elsevier Science, Amsterdam.

Roelke, M. E., D. J. Forrester, E. R. Jacobson, and G. V. Kollias. 1991. Rationale for surveillance and prevention of infectious and parasitic disease transmission among free-ranging and captive Florida panthers (*Felis concolor coryi*). Pages 185-190 in R. E. Junge, editor. Proceedings of the annual meeting of the American Association of Zoo Veterinarians [AAZV]. AAZV, East Lansing, Michigan.

Roelke-Parker, M. E., L. Munson, C. Packer, R. Kock, S. Cleaveland, M. Carpenter, S. J. O'Brien, A. Pospischil, R.Hofmann-Lehmann, H. Lutz, G. L. M. Mwamengele, M. N. Magasa, G. A. Machange, B. A. Summers, and M. J. G. Appel. 1996. A canine distemper virus epidemic in Serengeti lions (*Panthera leo*). Nature **379:**441-445.

Rushton, S. P., P. W. W. W. Lurz, J. Gurnell, and R. Fuller. 2000. Modelling the spatial dynamics of parapoxvirus disease in red and grey squirrels: a possible cause of the decline in the red squirrel in the UK? Journal of Applied Ecology **37:**997-1012.

Sakanari, J. A., and M. Moser. 1990. Adaptation of an introduced host to an indigenous parasite. Journal of Parasitology **76:**420-423.

Scott, M. E. 1988. The impact of infection and disease on animal populations: implications for conservation biology. Conservation Biology **2:**40-56.

Simberloff, D. A. 1986. The proximate causes of extinction. Pages 259-276 in D. M. Raup and D. Jablonski, editors. Patterns and processes in the history of life. Springer-Verlag, Berlin.

Soulé, M. E., editor. 1996. Conservation biology: the science of scarcity and diversity. Sinauer Associates, Sunderland, Massachusetts.

Stephenson, M. D. 1977. Sea otter predation on Pismo clams in Monterey Bay. California Fish and Game Bulletin **63:**117-120.

Swartz, R. L, P. S. Ross, L. J. Vedder, H. H. Timmerman, S. Heisterkamp, H. V. Loveren, J. G. Vos, P. J. H. Reijnders, and A. D. M. E. Osterhaus. 1994. Impairment of immune function in harbor seals (*Phoca vitulina*) feeding on fish from polluted waters. Ambio **23:**155-159.

Thomas, N. J., and R. A. Cole. 1996. The risk of disease and threats to the wild population. Endangered Species Update **13:**24-28.

Thorne, E. T., and E. S. Williams. 1988. Disease and endangered species: the black-footed ferret as a recent example. Conservation Biology **2:**66-74.

Tompkins, D. M., J. V. Greenman, P. A. Robertson, and P. J. Hudson. 2000. The role of shared parasites in the exclusion of wildlife hosts: *Heterakis gallinarum* in the Ring-necked Pheasant and the Grey Partridge. Journal of Animal Ecology **69:**829-840.

U. S. Fish and Wildlife Service (USFWS). 2000. Draft revised recovery plan for the southern sea otter (*Enhydra lutris nereis*). USFWS, Portland, Oregon.

Van Loveren, H., P. S. Ross, A. D. M. E. Osterhaus, and J. G. Vos. 2000. Contaminant-induced immunosuppression and mass mortalities among harbor seals. Toxicology Letters **112:**319-324.

Van Riper, C., III, S. G. van Riper, M. L. Goff, and M. Laird. 1986. The epitzootiology and ecological significance of malaria in Hawaiian land birds. Ecological Monographs **56:**327-344.

Viggers, K. L., D. B. Lindenmayer, and D. M. Spratt. 1993. The importance of disease in reintroduction programmes. Wildlife Research **20:**687-698.

Warner, R. E. 1969. The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. Condor **70:**101-120.

Williams, T. 1986. The final ferret fiasco. Audubon **88:**110-119.

Wilson, E. O. 1992. The diversity of life. The Belknap Press of Harvard University Press, Cambridge, Massachusetts.

Woodroffe, R. 1999. Managing disease threats to wild mammals. Animal Conservation **2:**185-193.