IS DISEASE INCREASING OR DECREASING, AND DOES IT IMPACT OR MAINTAIN BIODIVERSITY?

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ABSTRACT: Disease is a growing concern in conservation biology. Alteration to habitats can favor intermediate hosts such as mosquitoes. Stress may increase susceptibility to infectious disease. Alterations to trophic cascades can indirectly favor the emergence of diseases on those species that increase in abundance. Most notably, pathogen pollution, by introduced species, can expose hosts to diseases for which they have no coevolved resistance. These effects can impair conservation by making formerly common species rare or inhibiting rare species from recovering. In addition, managing wildlife diseases can have undesirable environmental impacts. Because conservation biology focuses on threats, less attention has been paid to the concept that environmental change will lead to declines in disease. Because the spread, persistence and impact of infectious diseases tend to increase with host density, rare species of concern should suffer less from host-specific infectious disease than common species. Parasites may themselves be sensitive to environmental change and suffer direct mortality or interruption of life cycles. This loss of disease may be of concern in instances where diseases are important for biodiversity. A lack of disease may allow common species (and particularly introduced species) to exclude rare species. A loss of trophically transmitted parasites may impair predators. In conclusion, environmental change will hamper some diseases and favor others in ways that may be of substantial concern for conservation biology.

CONSERVATION AND DISEASE

Diseases can impact hosts and, because epidemics will generally increase the likelihood that a species will go extinct (Lafferty and Gerber, 2002), it is logical for conservation biologists to be concerned with diseases. There are several alarming examples of how infectious diseases have drastically reduced the abundance of once-common species and impaired the recovery of rare species (Daszak et al., 2000). These have occurred despite the general expectation that rare species should be at less risk to disease than common species (because the spread, persistence, and impact of infectious diseases is inefficient at low host density [Lyles and Dobson, 1993]). In this article, I argue that the intersection between conservation and disease is complex. In some cases, impacts to ecosystems aid infectious disease, and these diseases may impair the conservation of species of concern. In other cases, diseases may be decreasingly prevalent; we may even see that impacts to ecosystems impair diseases or that diseases aid the conservation of species of concern. Instead of a balanced, integrated treatment of the available evidence, I have organized this article like an English essay assignment designed to illustrate that each topic has 2 sides. Part 1 argues that disease is increasing and impacts biodiversity. Part 2 advocates that diseases are declining and foster biodiversity.

PART 1. DISEASE IS INCREASING AND IMPACTS BIODIVERSITY

Impacts to ecosystems aid disease

Some environmental impacts favor transmission. Alterations to habitats can increase infectious diseases of humans (and probably diseases of wildlife as well). Most examples of this relate to generating habitats for vectors of disease or intermediate hosts. Damming for irrigation and electricity creates habitats for snails and black flies, thereby increasing schistosomiasis and filariasis, respectively (Heyneman, 1979; Gryseels et al., 1994). Deforestation and road construction create puddles and ditches, making good habitat for some Anopheles spp. mosquitoes, the vectors for malaria (Smith, 1981; Desowitz, 1991). Habitat fragmentation can increase edge effects, increasing contacts between wildlife and disease (Holmes, 1996). Oddly, loss of biodiversity can lead some pathogens to concentrate in remaining host populations (Schmid and Ostfeld, 2001). Eutrophication and thermal effluent can raise rates of parasitism when the associated increase in productivity increases the abundance of intermediate hosts. For instance, eutrophication of an urban lake in Moscow improved conditions for snails that host avian blood flukes, causing outbreaks of swimmer’s itch (Beer and German, 1993). Similarly, fish parasite richness increased with eutrophication in Finnish lakes (Valtonen et al., 1997). In a temperate pond, thermal effluent acts similar to eutrophication by extending the breeding season for snails, increasing food supply, and attracting birds, which all lead to an increase in trematodes (Sankurathri and Holmes, 1976). As such, environmental changes increase, so should disease.

Stress may increase host susceptibility to disease. Pollution, malnutrition, and rising temperatures are examples of stressors hypothesized to increase disease (Khan, 1990; Harvell et al., 2002). Stressed animals may lack sufficient energy to maintain adequate immune defenses (Rigby and Moret, 2000), increasing susceptibility to infection (Scott, 1988; Holmes, 1996). For example, several stressors (oil pollution, and pulp mill, thermal, and industrial effluents) increase the prevalence and intensity of parasitic fish ciliates (Lafferty, 1997) because toxic conditions compromise a fish’s ability to impair the production of mucus that protects against ciliates (Khan, 1987). Stress may also impose differential mortality on infected hosts (Guth et al., 1977; Stadnichenko et al., 1995). For instance, amphipods infected with larval anacanthocephalans are much more susceptible to the toxic effects of cadmium (Brown and Pascoe, 1989). Mathematical models in preparation suggest that stress will increase noninfectious, allogenic, or generalist diseases. As stressors increase in the environment, individual hosts will become more susceptible to disease.

Environmental change can indirectly affect species through trophic cascades. A common impact to ecosystems is the removal of top predators; this can have subsequent indirect effects on lower trophic levels (Pace et al., 1999). A 20-yr, 16-site study in southern California reveals how reducing predators that drive trophic cascades can indirectly increase disease in prey.
Historically, predators (spiny lobsters, *Panulirus interruptus*) kept herbivorous sea urchins (*Strongylocentrotus purpuratus*) at low densities, allowing kelp forests to thrive (Tegner and Levin, 1983; Harrell and Reed, 1985; Harrell and Pearse, 1987; Estes and Duggins, 1995; Dayton et al., 1998; Pinnegar et al., 2000; Tegner and Dayton, 2000). Where fishers removed the main predators, urchin populations increased and overgrazed algae. In 1992, an urchin-specific bacterial disease (Gilles and Pearse, 1986) entered some of the study sites. Epidemics were more probable and led to higher mortality in dense urchin populations. In comparison, urchins in a marine reserve rarely suffered from disease. Here, predators limited urchin densities to levels where disease transmission was inefficient. These results indicate how global reduction in top predators may favor the emergence of new diseases in prey species.

Introduced species can bring new diseases. Exotic species invade unintentionally as a consequence of global trade and travel and some become successfully established. Disease agents of these introduced species may spread through closely related hosts that have no evolved defenses. To address the importance of this pathway, a recent review tabulated information on 29 examples where infectious disease reduced host population density or inhibited recovery: where common species were decimated by an epidemic, the source of the disease was usually an intentionally introduced species (Lafferty and Gerber, 2002). We should continue to see new diseases emerge in association with the rapid pace of new introductions.

**Diseases impair conservation**

Some common species have become rare as a consequence of disease. Common species are sensitive to epidemics of host-specific diseases. An epidemic, even if it does not cause the extinction of a species, may reduce the formerly common species to densities where other factors, such as demographic stochasticity, can cause extinction. As mentioned previously, such cases occur when introduced species bring infectious diseases to native host populations with no evolved resistance. Introduced diseases will add new hosts to endangered species lists.

Some rare species fail to recover because of disease. There are 2 main circumstances where rare species may be impacted by infectious disease. The first is where an increase in susceptibility makes it easier for diseases to invade a small population. In particular, small populations are likely to be inbred. Infectious diseases should be better able to spread through genetically homogeneous populations, and inbreeding depression may impair natural defenses to infection (Cunningham, 1996). The other circumstance that renders rare species susceptible to infectious disease is when the operable host density is inflated by the presence of a reservoir species. Domestic animals are a main source of diseases that inhibit the recovery of rare species (Lafferty and Gerber, 2002). This is particularly problematic when the reservoir host population does not suffer appreciable mortality from infection in comparison with the target species of concern. In some cases, the target species may not be an appropriate host for the infectious disease, yet still suffers pathology (in this case a reservoir is absolutely required for the disease to persist). The case of the southern sea otter illustrates how non-otter parasitic diseases supported by reservoirs lead to increased mortality and could inhibit recovery of this threatened species because the diseases that cause the most mortality in otters are accidental and, seemingly, newly emerged (Lafferty and Gerber, 2002). In some cases, humans may have influenced the spread and emergence of these new diseases. The abundance of raccoons is believed to have exploded due to their adaptability to the human landscape. Raccoons are the final host for a nematode (*Baylisascaris procyonis*) that is pathogenic for the many species of mammals and birds that commonly serve as intermediate hosts (Evans, 2002). One of these, the regionally endangered Allegheny woodrat (*Neotoma magister*), collects raccoon feces for nest-building material and, as a result, experiences such high mortality that reintroduced woodrats apparently cannot persist where raccoons are common (L Giudice, 2000).

Some diseases are intensively managed. There are several examples where disease management has had environmental impacts. Filling of wetlands was 1 of the most effective means of malaria control in countries such as the United States, Italy, Israel, and the Panama Canal Zone (Kitron and Spielman, 1989: Desowitz, 1991) and has had widespread ecological impacts that are now of conservation concern. In many countries, the loss of such habitat for this and other reasons has been sufficiently severe to foster conservation measures to protect wetlands (Tiner, 1984; Field et al., 1991). In a similar vein, pesticides against disease vectors often have nontarget effects; although the ecological impacts of pesticides used for agriculture far outweighed the effects of dithiothreitol used for mosquito control (Desowitz, 1991). Another potential conservation impact of disease is the culling of wildlife to control zoonotic disease. Brucellosis, probably introduced to North America with domesticated cattle, infects native bison and elk, which may be culled to protect cattle (Dobson and Meagher, 1996). Because society values human and veterinary health over conservation, impacts to the environment associated with disease management are likely to continue.

**PART 2. DISEASE IS DECLINING AND IMPORTANT FOR BIODIVERSITY**

**Impacts to ecosystems impair disease**

Environmental change has caused some species to become rare. Below a certain host–threshold density, epidemics of host-specific infectious diseases are unlikely because hosts usually die or recover before they contact a susceptible host and transmit the disease (Anderson and May, 1986). The impact of a disease on host population growth rate is also believed to increase with host density (Swinton, 1998). Mathematical models in preparation indicate that, for infectious diseases with classic dynamics, we should generally expect a negative association between stress and disease in a host population if the stressor has a negative effect on host population density and disease transmission requires contact among hosts. For these reasons, host-specific diseases are less likely to be a source of extinction for rare species.

Environmental impacts can impair transmission. Parasites with complex life cycles require a sufficient abundance of a diverse set of hosts. Impacts to ecosystems that reduce biodiversity, therefore, should generally reduce parasitism by breaking transmission (Robson and Williams, 1970; Pohley, 1976;
Hughes and Answer, 1982). Larval digene species richness has declined over several decades in some Michigan lakes in association with an increase in human disturbance and a reduction in shorebird populations (Keas and Blankespoor, 1997). Similarly, in disturbed areas, populations of the horn snail (Cerithidea californica) have a lower prevalence and diversity of larval digenes than do snail populations in adjacent undisturbed sites (Huspeni and Lafferty, 2003). This is probably due to a decreased abundance of birds in degraded areas, a hypothesis supported by experiments that find that improving and creating habitat can increase the prevalence of trematodes over time (Huspeni and Lafferty, 2003). Loss of parasites is an unseen component of the global biodiversity crisis.

Stress can eliminate disease. Poor environmental quality can hurt parasites. In particular, pollution negatively affects most intestinal helminths (Lafferty, 1997). Fish parasites are particularly good indicators of pollution (MacKenzie et al., 1995). As an example, fish tapeworms are more sensitive to selenium than their fish hosts (Riggs et al., 1987). In this case, the toxin is analogous to a drug that, at appropriate doses, will kill the parasite before the patient. Free-living parasite stages may be sensitive to pollution (Evans, 1982). For instance, trace metals reduce the survival of digenean cercariae and miracidia, leading to a lower prevalence of digenese in snails (Siddall and de Cler, 1994). Increased pollution should increase parasite mortality rates, leading to fewer disease.

**Diseases aid conservation**

Some common species become sick and rare. Parasites may mediate interactions between competitors (Hudson and Greenman, 1998). If numerically dominant hosts are also competitively dominant, parasitism could facilitate diversity because simple theory indicates that host-specific diseases should exploit abundant host species. For example, laboratory experiments and field observations indicate that the presence of a parasitoid wasp allows the coexistence of 2 fruit fly species (Bouletreau et al., 1991). This density-dependent effect of parasitism is a possible mechanism for maintaining high-diversity communities (Janzen, 1970; Combes, 1996). Humans are an increasingly common species and a dominant competitor for space. When humans or their livestock or both are limited by infectious diseases, native habitat is spared from human exploitation. Tick- and fly-borne diseases have prevented cattle grazing in large regions of Africa, thereby conserving native game animals that might otherwise be displaced (Ford, 1971). The ability of parasites to regulate common species should favor rare species of conservation concern.

As mentioned previously, environmental change can indirectly affect species through trophic cascades. If the ease of preying on parasitized prey is high and the cost of being parasitized is low, predators could benefit from trophically transmitted parasites that modify the behavior of intermediate hosts (Holmes and Bethel, 1972; Moore, 1984; Lafferty, 1992; Lafferty and Morris, 1996; Lafferty, 1999). There may even be theoretical conditions where the parasite is necessary for the predator population to establish (Freedman, 1990). Therefore, if the extirpation of a predator leads to the extirpation of trophically transmitted parasites, predator reintroductions may fail without the parasite simply because food may be too hard to catch. The presence of parasites may aid the persistence of increasingly rare top predators in ecosystems.

Introduced species do not necessarily bring all their natural enemies with them. After habitat loss, the introduction of alien species is the greatest threat to conservation (Vitousek, 1990; Wilcove et al., 1998). A common hypothesis posited for the success of introduced species is a release from natural enemies (Elton, 1958; Dobson, 1988; Schoener and Spiller, 1995). Introduced species might invade without their native parasites if they arrive as uninfected stages or because a low initial number of invaders is statistically unlikely to contain all the parasites from the native range. Parasites that do arrive with introduced species may soon go extinct if required intermediate hosts are absent or if initial small population sizes of the invading host limit the efficiency of transmission (Torchin et al., 2002). Furthermore, native parasites may not colonize introduced species due to a lack of coevolutionary history. Introduced animals leave an average of 84% of their parasite species behind; in addition, native parasites do not sufficiently colonize introduced species to make up for this release from natural enemies so that introduced populations have fewer than half the parasites species compared with native populations of the same species (Torchin et al., 2003). Comparisons for plant pathogens reveal the same pattern (Mitchell and Power, 2003). A release from natural enemies could greatly facilitate the establishment and subsequent impacts of an introduced species. In the European green crab, release from parasites (parasitic castrators in particular) is likely responsible for the large body size and high density that introduced populations of green crabs attain (Torchin et al., 2001). Modern biological control, applied effectively and relatively safely in agriculture, is essentially a means to redress the release of introduced species from natural enemies by artificially introducing host-specific natural enemies that were left behind by invaders (Debach, 1974).

**CONCLUSION**

Diseases are of conservation concern because they can drive common species to low levels, impair the recovery of rare species, and they necessitate management actions that impact the environment. Global change leading to habitat alteration, stress, loss of top predators, and introduced species will cause new diseases to emerge. Diseases are also sensitive to global change because rarer hosts impair transmission of host-specific parasites and stress increases parasite mortality. Losses of parasites may reduce biodiversity and top predators and facilitate invasions.

Although I have presented 2 opposing opinions about the topic of conservation and disease, these opinions are compatible in that neither contradicts the other. The existence of these opinions in our field is exemplified by presentations at the Tenth International Congress of Parasitology (ICOPA X) at which a version of this article was presented in a plenary session. In the ICOPA X program, there were 64 presentations related to ecology of parasites. Of these, roughly half were concerned with the question of how changes in the environment affect parasites, an indication that this question is now of major concern to ecological parasitologists. Two thirds of these environmental studies focused on emerging diseases or increases in infectious disease associated with environmental change. One
third found that changes in the environment reduced infectious disease. This distribution implies that parasitologists are studying the full range of issues related to conservation and disease and, for this, our discipline should be commended. However, the vast majority of the presentations focused on a single side of the issue: few of the presentations acknowledged that environmental change could increase or decrease infectious disease, depending on the disease and the type of change. Embracing the complex interactions between environmental change, disease, and conservation will aid in parasitology’s contribution to much needed conservation efforts.

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LITERATURE CITED


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