Diseases, Conservation and

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Glossary

Definitive host For parasites with complex lifecycles, this is the host in which the parasite reproduces sexually (also called the *primary host*).

Epidemic Characterizes a sudden increase in parasite prevalence or intensity beyond that which is normally present.

Helminth One of several classes of parasitic worms: nematodes, cestodes, trematodes (monogeneans and digeneans), and acanthocephalans.

Intensity The number of parasites per infected host; a related measure, abundance, refers to the average parasite load of the entire population (including uninfected individuals).

Intermediate host For parasites with complex lifecycles, earlier parts of their life cycle are completed within intermediate hosts (also called the *secondary host*).

Prevalence The proportion of hosts in a population that are infected or diseased.

Reservoir host A host organism that can carry and transmit a parasite or pathogen to other host species while suffering little to no negative effects of disease.

R_0 The basic reproductive ratio of a parasite or pathogen; for a microparasite, this describes the number of new infections generated by a single infected host entering an entirely susceptible population; for a macroparasite, R_0 is the number of adult offspring produced by a single adult parasite over its lifespan.

Vector An animal that transmits parasites among definitive hosts; for example, mosquitoes are vectors of malaria.

Virulence Disease-induced mortality rate, or the harm caused by parasites to individual hosts.

Types of Parasites and Causes of Disease

Parasites are organisms that live in or on and obtain resources from a host, usually to the host's detriment. The terms parasite, pathogen and disease are often used interchangeably; however, disease refers to the pathogenic condition of a host, including the signs and symptoms of infection, whereas parasites and pathogens are the disease-causing agents. Infectious diseases can be caused either by microparasites (such as viruses, bacteria, protozoa, and fungi) or by larger macroparasites, including worms and arthropods. The distinction between macro- and microparasites is particularly useful to ecologists and epidemiologists because these groups differ in the degree of within-host replication, their ability to generate a lasting adaptive immune response, and how they are quantified. They also differ in their impacts on host populations (**Table 1**).

Disease outbreaks require the presence of a susceptible host population, an infectious pathogen, and favorable environmental conditions; these three factors are often referred to as the disease triangle (Wobeser, 2007). It follows that disease prevalence or infection intensity can increase when changing environmental conditions favor pathogen transmission or host susceptibility. In many cases, these environmental modifications are caused by human activities that lead to host crowding, habitat degradation, and host stress. Other environmental changes can cause novel pathogen introductions or enhance opportunities for cross-species transmission through changes in the composition of ecological communities or shifts in the geographic distribution of a host, pathogen, or vector species.

Basic Epidemiological Principles

Since the pioneering work of Anderson and May in the late 1970s, ecologists have shown increasing interest in the spread and impacts of parasites at the population and community levels. A general understanding of host–pathogen population ecology can illuminate problems in conservation biology ranging from detecting disease threats for endangered species to predicting how human impacts on landscapes will affect pathogen invasion and persistence. Epidemiologists quantify and model the spread of infectious diseases over time and space to identify parameters that influence their prevalence and population-level effects. Models are used in epidemiology to detail how processes operating at the level of individuals (such as infection, recovery, and death) translate into population-level patterns such as changes in the numbers of susceptible and infected hosts. Epidemiological models differ from models of other antagonistic interactions (such as between predators and prey) because pathogens and parasites do not necessarily kill their hosts. Also, a single host can be infected by many parasites at once, and recovered hosts can develop a long-term immunity to reinfection by some pathogens. It is useful to address models designed for micro- versus macroparasites separately to identify basic principles that govern parasite spread and quantify their effects on host populations.

Microparasites

Mathematical models for microparasites divide the host population into categories that reflect their stage of exposure,

Table 1 General characteristics, examples, and ecological properties of micro- and macroparasites

	Microparasites	*Macroparasites*
Representative taxa	Viruses, bacteria, protozoa, fungi, microsporidians	Helminths (e.g., nematodes, cestodes, and acanthocephalans), arthropods (e.g., mites, ticks, and lice)
Size and reproduction	Small, unicellular, short generation times and rapid replication within individual hosts	Large, multicellular, longer generation times, usually no direct replication within individual hosts
Transmission of infectious stages	Transmission *via* direct contact (e.g., venereal and vertical), vectors, or contaminated air/soil/water	Complex life cycles and intermediate hosts are common; can also be transmitted by vectors, close contact, or ingestion of material contaminated by feces
Effects on adaptive host immunity	In many cases, temporary or lasting host immunity develops quickly and protects against reinfection	Some acquired immunity, but antigenic diversity of parasites usually too high for host to mount lasting adaptive immune response
Effects on host fitness	Disease can be acute or chronic, may have strong effects on host survival or fecundity	Depends on the number of parasites within the host (can affect mortality or fecundity, usually chronic infection with cumulative, sublethal effects)
Quantification in host populations	Prevalence, seroprevalence, incidence	Prevalence, intensity, degree of aggregation in individual hosts
Frequency of epidemics	Common	Rare

including susceptible (S), infected (I), and recovered/immune (R) classes (**Figure 1**). These compartment models track changes in the number of hosts within each category, but ignore the number of parasites within each host. SIR models are commonly used in modeling directly transmitted microparasites of vertebrates, and have been developed and analyzed extensively (e.g., Anderson and May, 1991). The model shown in **Figure 1** makes many assumptions that can be relaxed. For example, hosts are uninfected at birth, infection increases host mortality but does not affect host fecundity, and host populations are large enough that stochastic processes (random events that affect populations in unforeseeable ways) can be ignored. For pathogens for which hosts do not acquire immunity to reinfection (e.g., many plant and insect pathogens and vertebrate diseases such as tuberculosis and brucellosis), the recovered/immune class (R) is eliminated, and the equations simplify to a SI or SIS model.

The basic SIR model gives rise to several key principles that have consequences for the spread of infectious diseases in wild populations. Of fundamental importance is a value called R_0, the basic reproductive ratio of the pathogen. R_0 describes the initial rate of pathogen increase in a previously unexposed host population. This parameter is estimated by multiplying the expected number of new infections from a single infected host by the average duration of infectiousness. For the SIR model in **Figure 1**,

$$R_0 = \frac{\beta S}{\alpha + b + \nu} \qquad (1)$$

R_0 must exceed 1.0 for a pathogen to invade and spread. The form of eqn (1) suggests that pathogens with relatively high transmission (β), low virulence (α), and low host recovery (ν) are most likely to establish in host populations. For pathogens that can successfully invade, a common dynamical outcome is an ''epidemic curve'' (**Figure 1**, orange line), whereby the number of infected hosts firsts increases, then decreases as the pathogen spreads through the population. At the same time, the numbers of susceptible hosts decline as they transition to the infected class, and the number of recovered hosts gradually increases. The general shape of the epidemic curve produced by simple compartment models is mirrored by many real-world epidemics including influenza in humans and phocine distemper in harbor seals (reviewed in Anderson and May, 1991; Hudson *et al.*, 2002).

The SIR model shown in **Figure 1** is useful for pathogens with density-dependent transmission, a process in which transmission (measured by the rate new susceptibles are infected per unit time) increases directly with host population density. Specifically, for density-dependent transmission there exists a threshold density, N_T, of hosts below which the pathogen cannot persist ($R_0<1$) in a host population. Assuming that the population is large and homogeneously mixed, this value is

$$N_T = \frac{\alpha + b + \nu}{\beta} \qquad (2)$$

Pathogens that are highly virulent (high α) or have lower transmission (low β) are likely to require much higher host densities for establishment than those that are highly transmissible and relatively benign.

For some pathogens, transmission can remain relatively constant over a range of host densities, a process termed frequency-dependent transmission. A key result of frequency-dependent transmission is that there is no threshold density for pathogen invasion (so in theory, such pathogens can persist at arbitrarily low host densities). Pathogens spread by direct contact (such as touching), aerosol droplets (coughing or sneezing) or indirect contact (ingesting fecally contaminated food or water) are expected to show density-dependent transmission. By comparison, transmission of sexually transmitted diseases and some vector-borne diseases is thought to be frequency dependent. Importantly, field and experimental studies of pathogens affecting mice, voles, frogs, ladybird beetles, and wildflowers have shown that the transmission of most pathogens probably falls between these two extremes.

Analysis of simple microparasite models can generate important insights for considering pathogen risks to wild or captive populations. For example, models suggest that the population-level impacts of an infectious disease will depend

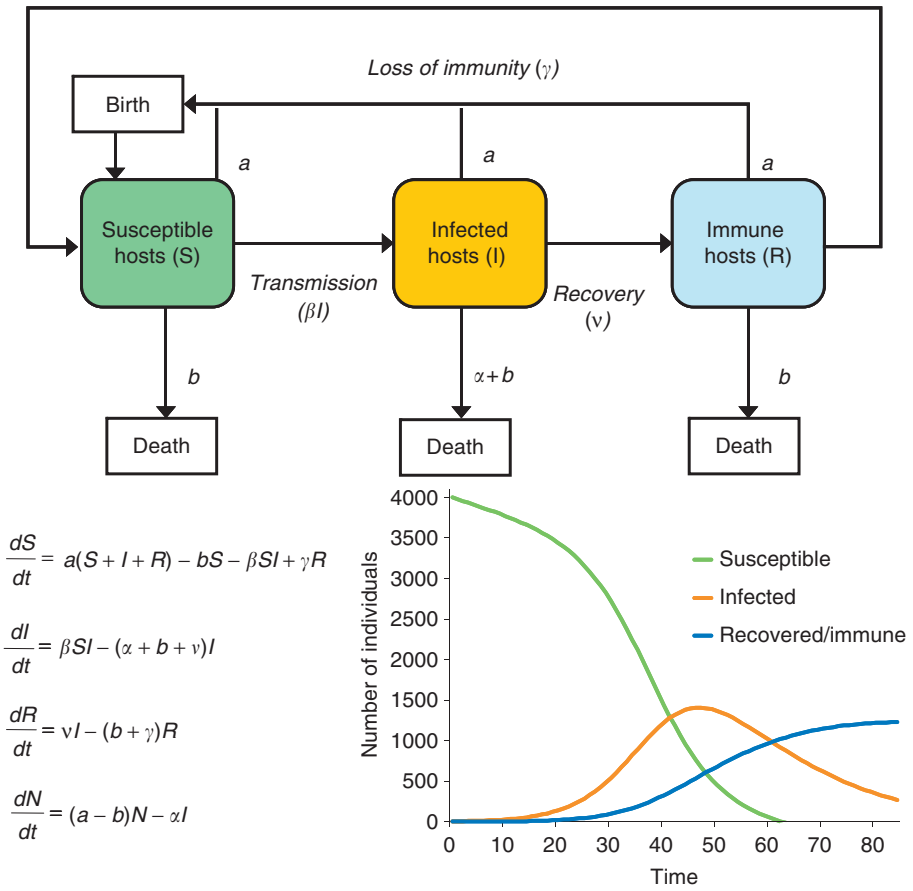

Figure 1 SIR compartment model for directly transmitted microparasitic disease. Diagram shows a population containing susceptible hosts (S), infected hosts that can transmit the parasite to others (I), and recovered or immune hosts that are no longer infected (R). Total host population size ($N = S + I + R$. Susceptible hosts arise from birth or immunity loss at per capita rates a and γ, respectively. Individuals leave the susceptible class through natural mortality (rate b) or by acquiring the parasite at rate β after encountering an infected host. Hosts leave the infected category through natural death or disease-induced mortality (rates b and α, respectively) or through recovery (rate ν) to an immune state. Differential equations that track changes in the numbers of susceptible, infected and recovered hosts are shown in the bottom left panel. Representative dynamics (changes in numbers over time) are graphed in the bottom right panel for the following parameter values: $a = 0.1$, $b = 0.05$, $\beta = 0.0006$, $\alpha = 0.3$, $\nu = 0.3$, $\gamma = 0.005$.

on several factors, including pathogen effects on individual host fitness. In general, for pathogens that lower host survival, those with intermediate virulence should have the largest negative impacts on host populations. If pathogens are too virulent, infected hosts will die before they cause many new infections, whereas nonvirulent pathogens can become highly prevalent but have minimal population-level impacts. For similar reasons, models also predict that sterilizing pathogens (those that reduce fecundity) can cause greater reductions in host population size than those that reduce survival (Anderson and May, 1991). Models can generate important predictions for pathogen control, including effects of vaccination or culling on the probability of pathogen eradication (*see* Intervention Methods).

Finally, the spread and impacts of pathogens will depend on a number of ecological and genetic heterogeneities. Unlike the simple homogeneous populations described in **Figure 1**, natural host populations tend to be stratified by age, sex, social rank, or clumped spatially due to fluctuating resources or habitat fragmentation. This heterogeneity can have large impacts on pathogen transmission and, as a result, on disease control efforts. Heterogeneity also arises when pathogens can be transmitted between several different host species. For these multihost pathogens, some host species are reservoirs or amplifiers (their presence increases transmission of the pathogen), whereas others may lower transmission or be unusually susceptible to pathogen-induced mortality. The identity and abundance of different host species has been shown to be important in the transmission of plant diseases such as sudden oak death and animal diseases such as brucellosis, Lyme disease, and chytridiomycosis (*see* Habitat Management).

Macroparasites

In contrast to microparasites, macroparasites typically cause persistent infections (**Table 1**), in large part because the host's immune response is often incomplete or short-lived.

Ecologists keep track of the number of adult macroparasites per host because the outcomes of macroparasite infections (e.g., the survival and fecundity of both hosts and macroparasites) depend strongly on infection intensity. The distribution of parasites among hosts almost always shows evidence for aggregation or clumping which means that most hosts have few or no parasites, and a few hosts have many.

Fundamental macroparasite models developed by Anderson and May (1991) have been modified by Dobson and Hudson (1992) and others to consider the presence of free-living infective stages, arrested parasite development, and parasites with complex life cycles (which can include multiple intermediate hosts and a definitive host). These models typically track the density of the entire host population (H), the abundance of adult parasites within hosts (P), and the number of free-living parasite stages (W) in the external environment (**Figure 2**). The model further assumes that parasites are aggregated within hosts according to the negative binomial distribution, where the degree of aggregation varies inversely with k. As indicated by the equations in **Figure 2**, the mortality of adult parasites is affected by within-host clustering, with parasite mortality increasing when k is small (and parasites are highly aggregated).

The basic reproductive ratio of macroparasites is the product of the mean number of new infections produced by a single adult parasite and the average life expectancy of adult and larval stages:

$$R_0 = \frac{\beta\lambda H}{(\mu + b + \alpha)(\gamma + \beta H)} \qquad (3)$$

As with microparasites, eqn (3) must exceed 1.0 for the parasite to establish when rare. Therefore, parasite invasion

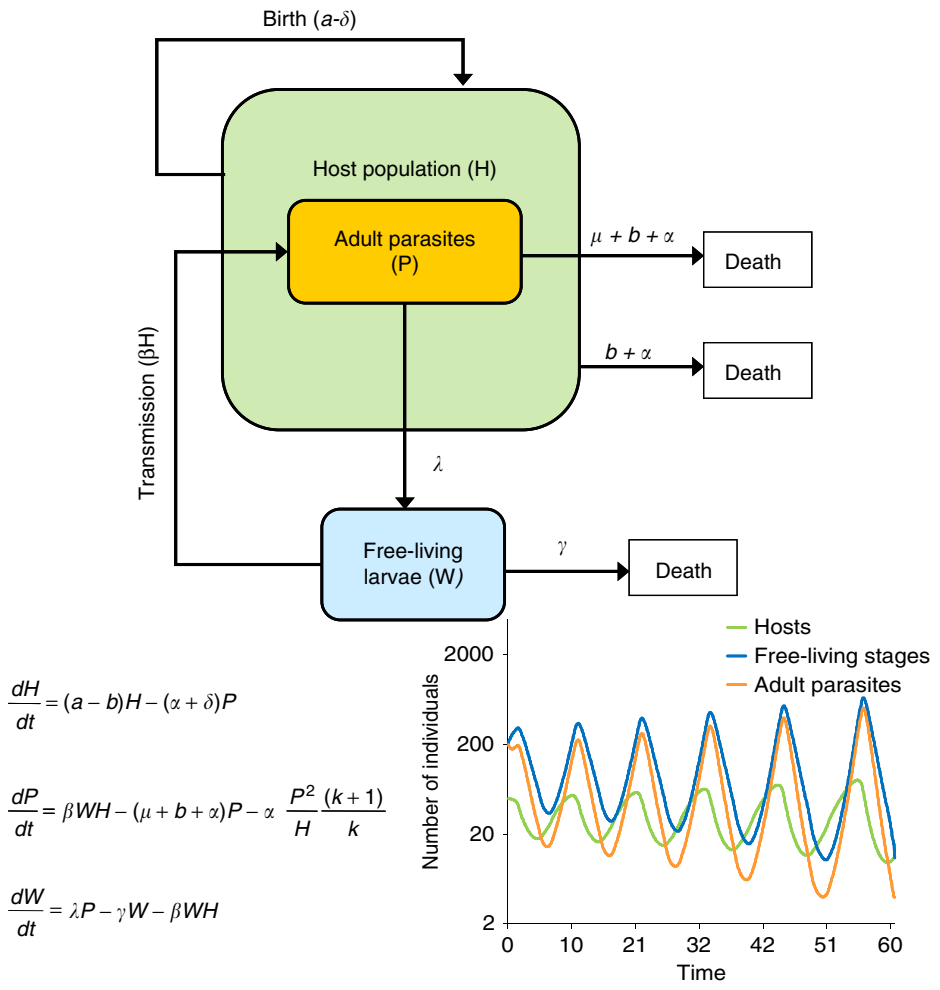

Figure 2 Box diagram of host and parasite life cycle for macroparasitic infections, showing a host population of size H, collectively harboring an adult parasite population of size P. As in the microparasite model (**Figure 1**), per capita host birth and death rates are denoted by a and b, respectively. δ and α are the per capita host sterility and mortality rates induced by each individual parasite and assume that overall host death rate increases linearly with parasite burden. Adult parasites give birth to free-living infective stages at rate λ and die at rates μ (background mortality), b (host mortality), and α (disease-induced mortality). Thus, the model assumes that when hosts die, so do their parasites. Free-living egg and larval stages die at rate γ, and transmission occurs when hosts eat these stages at rate β, thus giving rise to new adult infections. The aggregation parameter k corresponds to the negative binomial distribution, such that low k values indicate highly aggregated parasite distributions (in which most parasites are harbored by a few hosts). Representative dynamics (changes in numbers over time) are graphed in the bottom right panel for the following parameter values: $a = 0.9$, $b = 0.55$, $\beta = 0.1$, $\alpha = 0.03$, $\delta = 0.06$, $\lambda = 11$, $\mu = 0.4$, $\gamma = 1$, $k = 0.315$.

and persistence depend strongly on the rate of production of eggs or larval stages (λ), the rate at which parasites are consumed by hosts (β), and the survival of free-living infective stages ($1-\mu$). The macroparasite model shown in **Figure 2** is also associated with a threshold host population necessary to sustain infection. Because larval macroparasites often have long-lived resistant stages and adult worms can live for years within their hosts, many macroparasites can persist at lower host population densities than directly transmitted microparasites.

The effects of macroparasite virulence on host population thresholds and their ability to regulate host populations depend on the degree of parasite aggregation. Parasites of intermediate virulence (α) will depress host density more than those of low or high virulence, and parasite impacts will be maximized when aggregation is low (so that parasites are distributed across a greater number of host individuals). Importantly, highly aggregated parasite distributions tend to stabilize host–macroparasite interactions, whereas random or regular parasite distributions tend to destabilize them, leading to population cycles in host and parasite abundance (**Figure 2**). When parasites reduce host fecundity (i.e., $\delta>0$) this can further destabilize the host–parasite interaction and increase the probability of parasite-induced host population cycles.

Field studies also support a role for macroparasites in wildlife population dynamics, although their effects are often more subtle than the dramatic population declines seen in response to some microparasitic diseases. Perhaps the best evidence comes from a handful of field experiments where researchers treated a fraction of animals or a subset of populations with antiparasitic drugs. This approach has been useful in demonstrating impacts of nematode parasites on host survival and population sizes of feral Soay sheep, white-footed mice, and red grouse (with several examples reviewed in Hudson *et al.*, 2002). In the case of red grouse, for example, cecal nematodes are only weakly aggregated among hosts and high parasite loads cause reduced fecundity in grouse. In addition, treating 20% or more of a local population to remove parasites was sufficient to halt periodic population crashes that occurred every 4–8 years (and *see* Alternative Interventions). This work suggests that macroparasites should not be overlooked as important causes of wildlife declines, either alone or together with other factors such as food limitation or harsh environmental conditions.

Case studies of macroparasite infections further emphasize the importance of sublethal effects of parasites for infection outcomes and host–parasite population dynamics. In the case of red grouse, for example, negative effects of cecal nematodes on host breeding success (and not survival) drive population cycles in abundance over time. Moreover, grouse that carry heavy infestations of cecal nematodes are more vulnerable to predation by red foxes and raptors. These effects of parasites on host fitness would be easy to miss, but their implications for host and parasite population dynamics are extremely important. In other examples, parasites have been observed to manipulate key behaviors of hosts as diverse as ants, amphipods, and fishes, making them behave in ways that increase their risk of being consumed by a predator and thereby improving the chances that the parasite will be transmitted to its definitive host (Moore, 2002). Another subtle effect of parasitism occurs when infection by one parasite species affects host susceptibility to other pathogens. In the case of African buffalo, Jolles *et al.* (2005) showed that infections by parasitic worms were negatively associated with the probability of bovine tuberculosis infection, as might be expected if coinfected animals experience sharp declines in body condition and greater mortality. The authors used a population dynamic model to show that high mortality of coinfected hosts (as might be caused by failure of the immune system to adequately control both parasite types) qualitatively captured the observed patterns of disease in free-ranging buffalo populations. Thus, the sublethal and cumulative effects of parasites can impact host population dynamics and community-level interactions in substantial and unexpected ways.

Infectious Diseases as Threats to Biological Diversity

Introduced Parasites and Species Declines

Exotic diseases and parasites are increasingly recognized as important factors driving population declines and geographic range contractions in many organisms (**Table 2**). Owing to their high rates of spread and potentially devastating effects on host populations, a handful of pathogens are now considered the greatest threats to the survival of some endangered species. Parasitic organisms have been shown to impact host populations in a variety of ways. Most directly, disease-induced mortality can reduce host population sizes below a threshold necessary for maintenance and growth. In very small populations, differential mortality between male and female hosts can sufficiently distort sex ratios or shift host life history to affect future reproduction. This is illustrated by a facial tumor disease infecting Tasmanian devils that spreads when infectious cancer cells are transmitted through aggressive encounters, especially among older males. Mortality caused by this disease has resulted in declines of up to 50% in Tasmanian devil populations. Moreover, as older, more dominant males succumb to this disease, researchers have observed a shift in breeding phenology such that devils are reaching sexual maturity sooner and are breeding at a younger age than before the epidemic. Tasmanian devils may eventually come to rely on single, early reproductive bouts as opposed to engaging in multiple reproductive efforts over a longer time period.

A recently discovered terrestrial fungus, *Geomyces destructans*, causes White Nose Syndrome (WNS) in multiple bat species of the eastern United States (**Figure 3(a)**). Following initial reports in New York state in the winter of 2006–2007, WNS spread rapidly, devastating populations of the little brown bat (*Myotis lucifugus*) in eastern North America. Once among the most abundant bat species in North America, local population declines of *M. lucifugus* have exceeded 75%, with bat mortality reaching 100% in some hibernation caves. Perhaps one of the best-studied examples of parasite-induced population declines involves a chytrid fungus, *Batrachochytrium dendrobatidis*, which causes amphibian chytridiomycosis in hundreds of frogs, toads, and salamander species worldwide. Multiple amphibian populations from North America, Central America, South America, Australia, and several island

Table 2 Selected disease outbreaks associated with declines in natural populations

Host species	*Disease (parasitic agent)*	*Location*	*Comments*
Plants			
American chestnut (*Castanea dentata*)	Chestnut blight (*Cryphonectria parasitica*)	Eastern North America	Caused massive extinction of dominant hardwood species
Flowering dogwood (*Cornus florida*)	Anthracnose bight (*Discula destructiva*)	North America	Fungus decimated dogwood populations throughout native range
Several native plant species	Fungal dieback (*Phytophthora cinnamoni*)	Western Australia	Responsible for large-scale diebacks and permanent plant community shifts
American elm (*Ulmus americana*)	Dutch elm disease (*Ceratocystis ulmi*)	North America	Pathogen introduced from Asia, spread by bark beetles
Multiple woody species including live oaks and tanoaks	Sudden oak death (*Phytophthora ramorum*)	Western North America	Introduced from Asia; wide host range
Invertebrates			
Elkhorn coral (*Acropora palmata*)	White pox disease (*Serratia marcescens*)	Caribbean Sea	Bacterial pathogen traced to human wastewater
Oysters (*Crassostrea virginica*)	Protozoan parasite (*Perkinsus marinus*)	Atlantic coast of North America	Warming ocean temperatures facilitated parasite spread along the coast
Fish			
Rainbow trout, salmon (*Salmo spp.*)	Whirling disease (*Myxobolus cerebralis*)	Montana	Introduced with stocked fish
Aral Sea sturgeon (*Acipenser nudiventis*)	Monogenean trematode (*Nitzschia sturionis*)	Aral Sea, former USSR	Introduced with stocked Caspian sturgeon
Amphibians			
Multiple species of frogs, toads, salamanders	Digenean trematode (*Riberoia ondatrae*)	North America	Causes limb malformations during metamorphosis; linked with eutrophication
Multiple species of frogs, toads, salamanders	Saprolegniasis *Saprolegnia* spp (oomycete)	North America	Lethal to amphibian embryos; linked with increasing UV-B radiation, decreased rainfall and stocking nonnative fishes
Reptiles			
Desert tortoise (*Gopherus agassizii*)	Upper respiratory tract syndrome (*Mycoplasma agassizii*)	Mojave Desert	Introduction through released pets
Birds			
Hawaiian honeycreepers	Avian malaria (*Plasmodium relictum*)	Hawaii	Implicated in the extinction of several Hawaiian forest birds
House finches (*Carpodacus mexicanus*)	Bacterial conjunctivitis (*Mycoplasma gallisepticum*)	North America	Likely transfer from domesticated poultry
Mammals			
Harbor seals (*Phoca vitulina*)	Phocine distemper virus (*Morbillivirus*)	North Sea	Outbreaks likely initiated by contact with Harp seals
Sea otter, some wild felids, red fox	Toxoplasmosis (*Toxoplasma gondii*)	Pacific Ocean and the Pacific coast of North America	Associated with ground surface run-off contaminated by fecal matter from infected felines
Gorillas, Chimpanzees	Ebola virus (*Filoviridae*)	Multiple countries in Central and sub-Saharan Africa	Associated with severe population declines of wild primates

countries have experienced severe declines, with up to 50% of species vanishing over a matter of months in some stream communities of Central America (**Figure 4**).

In addition to causing precipitous declines of initially abundant host populations, parasites can also threaten already-endangered species with extinction. For example, critically endangered black-footed ferrets in western N. America succumbed to an outbreak of canine distemper in the late 1980s, with only 18 individuals remaining for population recovery through vaccination and captive breeding efforts. It is important to keep in mind that such heavily endangered populations are not likely to sustain most parasites in the long run simply because remaining host populations are too small. This is particularly true for parasites that specialize on a single host species and for highly virulent parasites. However, parasites that infect multiple host species (i.e., generalist parasites) often pose the greatest conservation concern because they can persist in reservoir hosts and their transmission is not limited

Figure 3 Examples of infectious diseases that have caused population declines. (a) White nose syndrome caused by the fungus *Geomyces destructans* is illustrated by white spots on the noses and ear membranes of infected little brown bats (*Myotis lucifugus*); (b) Barley yellow dwarf virus causes yellowing of infected grasses compared to healthy grass; (c) Eurasian red squirrel (*Sciurus vulgaris*) severely infected by parapox virus introduced by gray squirrels into the UK; (d) Elkhorn coral (*Acropora palmata*) infected with white pox disease (white spots) caused by the human enteric bacterium *Serratia marcescens*.

by the small population sizes of many endangered species. As a case in point, rabies is a generalist pathogen that can be maintained in domesticated dog populations; in recent decades, rabies outbreaks have caused the near-extinction of local populations of endangered carnivores in parts of Africa, including African wild dogs and Ethiopian wolves. Additionally, theoretical studies suggest that parasites with frequency dependent transmission will cause severe problems for endangered species or species with small local population sizes (de Castro and Bolker, 2005) because they can continue to drive down host populations even after they have been drastically reduced in number.

Invasive Species and Pathogen Introductions

A number of introduced or exotic pathogens have caused catastrophic declines of plant and animal species. In epidemics where new pathogens are introduced into previously unexposed host populations, the disease often progresses rapidly through immunologically naive hosts and can cause mass mortalities. One of the best known examples is the introduction of the exotic malaria parasite *Plasmodium relictum* and its mosquito vector into the Hawaiian Islands. Although most nonnative birds on Hawaii showed a combination of resistance and tolerance to this infection, the parasite was

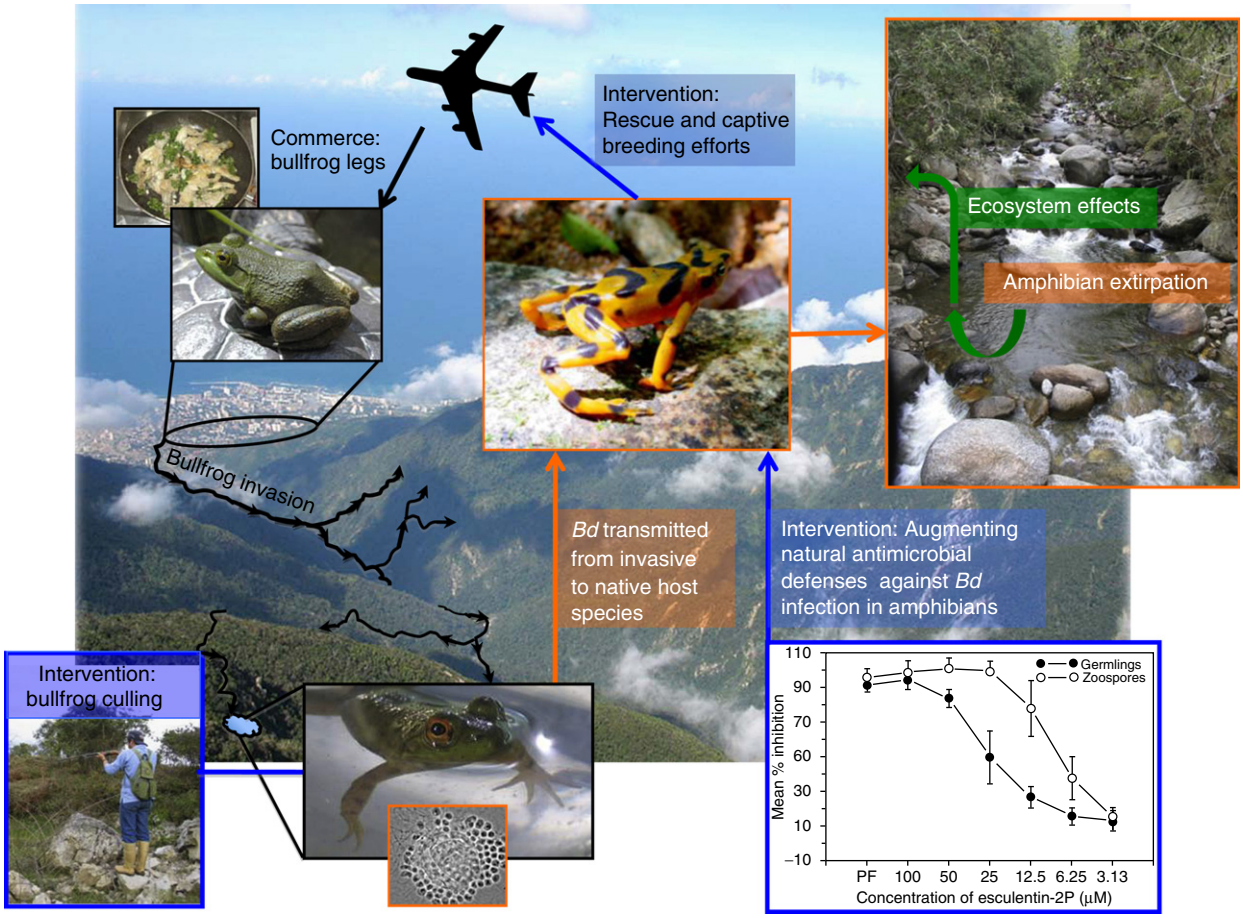

Figure 4 Amphibian chytridiomycosis can spread to native frog populations through contact with reservoir species such as the American bullfrog (*Lithobates catesbeianus*). Bullfrogs are disseminated globally to support the culinary industry for frog legs, and can be heavily infected with *Batrachochytrium dendrobatidis* (*Bd*). Bullfrogs that escape captivity can invade and contaminate new habitats with *Bd*. Chytridiomycosis epidemics can extirpate multiple amphibian species with dramatic effects on community structure and ecosystem function. Intervention efforts include continuous monitoring of amphibian populations, focused culling of established bullfrog populations, and alternative tactics such as augmenting antimicrobial peptides found on amphibian skin that can confer protection from *Bd*. The blue box (lower right) shows the mean percent of *Bd* growth inhibited by increasing concentrations of one antimicrobial peptide, esculentin. Amphibian species that are highly susceptible to lethal *Bd* infection have also been rescued to other locations for captive breeding and postepidemic reintroduction. Photo credits: bullfrog and cooked frog legs, Wikimedia Commons; infected *Atelopus zeteki* (center), Brenes R; inset *Bd* zoospores, Longcore JE; all other photos, Han BA).

extremely pathogenic to native bird species. Avian malaria epidemics throughout the Hawaiian Islands caused high mortality in the native birds, essentially clearing the lower island elevations of the native avifauna and contributing to the extinctions of several endemic bird species. In addition to species extirpations, introduced pathogens have facilitated drastic changes to community composition through invasion and displacement of native species. For example, Barley and Cereal Yellow Dwarf Viruses (**Figure 3(b)**) have enabled invasive annual grasses to outcompete and replace native perennial bunchgrasses in California. A parapox virus that is highly lethal to native red squirrels (**Figure 3(c)**) was introduced by invasive gray squirrels, and has accelerated the replacement of red squirrels by gray squirrels throughout much of the UK. Currently there is intensive research to determine whether invasive American bullfrogs are transmitting amphibian chytridiomycosis (**Figure 4**) to native amphibian populations in western North America, Central, and South America. Dissemination of the amphibian chytrid fungus *via* invasive reservoir species could be particularly devastating in areas such as the Venezuelan Andes, one of the world's biodiversity hotspots for endemic amphibians.

Negative Community and Ecosystem Consequences

The negative impacts of parasites and pathogens extend beyond their direct effects on hosts. Host population declines can generate secondary effects that ripple through natural communities, in severe cases triggering secondary extinctions termed "extinction cascades." Examples of such community-wide effects have been reported from both marine and terrestrial ecosystems. For example, the disappearance of long-spined sea urchins (*Diadema antillarum*) throughout the

Caribbean Sea by an unidentified pathogen caused a shift from coral- to algae-dominated reef communities (Harvell *et al.*, 1999). Similarly, the virtual disappearance of the American chestnut caused by the introduced fungal pathogen *Endothia parasitica* led to the extinction of eight Lepidopteran species that fed predominantly on this tree species.

Direct and indirect effects of infectious diseases can also damage the way an ecosystem functions. For example, the extirpation of adult amphibians by chytridiomycosis in Panama was linked to dramatic declines in amphibian larvae in stream communities. In the absence of tadpoles as filter feeders, grazers, and detritivores, studies report dramatic decreases in the quality and quantity of fine organic particulate matter suspended in the water column (i.e., seston, made up of periphyton, tadpole feces and bacteria). These changes are likely to affect communities downstream, which receive an influx of energy through the particulate matter generated by headwater communities. Ecosystem impacts can occur even when host mortality or species declines are not apparent. For example, experimental manipulations of intact grassland communities demonstrate that infection by foliar fungal pathogens affects net primary productivity and soil respiration by reducing root biomass, photosynthetic capacity, and leaf longevity in multiple grasses (*Andropogon*, *Poa*, and *Schizachytrium* spp.) (Mitchell, 2003).

Anthropogenic Drivers of Disease Emergence in Nature

In recent decades, a growing number of pathogen outbreaks in natural systems have been attributed to human activities. In particular, economic development influences disease emergence through changes in land use and human population growth. These activities can increase the risk of pathogen spread in natural populations through habitat destruction or fragmentation, pollution or other forms of habitat degradation, climate change, and global commerce.

Loss and Fragmentation of Natural Habitats

Humans destroy natural habitats in a plethora of ways, many of which have been summarized under the term habitat fragmentation – the process of reduction and isolation of a continuous natural habitat into smaller patches. Habitat fragmentation is of great conservation importance because it affects native communities directly by removing individuals and their resources, and indirectly through changes in community composition and species interactions. In terms of host–pathogen interactions, the reduction or subdivision of a host population can allow hosts in some patches to escape infection, especially if patches are isolated. Furthermore, if remaining host subpopulations are relatively small they may fall below the critical host threshold required for disease persistence (N_T). Consequently, one possible result of habitat fragmentation may be the decline or extinction of a parasite. Conversely, it is also possible that hosts moving between habitat fragments will continue to spread infection. Between these two contrasting scenarios, the benefits of increasing connectivity – namely the increased likelihood of species persistence and the potential for recolonization of locally extinct patches – appear to outweigh the potential costs of pathogen transmission between fragmented habitats on a landscape (McCallum and Dobson, 2002).

From a different perspective, habitat fragmentation can increase disease risks to native biota by providing opportunities for contact with domesticated crops, livestock, humans, and other native species crowded into adjacent habitats. For example, endangered red colobus monkeys living on the edge of forest fragments in Kibale National Park were exposed more frequently to multiple infections and had a higher prevalence and intensity of certain parasites, including harmful parasitic worms and protozoa, than monkeys living in the forest interior (Chapman *et al.*, 2006). The authors speculated that interactions with humans and domesticated species on the forest edge could cause greater exposure to some generalist parasites. Other work in this same region showed that forest fragmentation caused greater transmission of enteric bacteria among humans, livestock, and three wild primate species; this was partly associated with crop raiding behaviors of wild primates living in small fragments or on the edge of reserves.

Habitat Degradation and Pollution

Degradation of natural habitats takes many forms and has frequently been associated with pathogen outbreaks in wildlife and humans. Human activities such as logging, overexploitation of game and fisheries, erosion, and pollution can trigger pathogen epidemics. For example, direct injury to trees as a result of logging can increase disease in native plant communities. Many scientists suggest that the phocine distemper epidemic that spread through the seal populations in the North Sea can be attributed to the depletion of the fish stocks (by commercial fishing) in the North Atlantic ocean, leading to aberrant migrations of harp seals seeking food. In addition, wastewater run-off (which includes human sewage and agricultural byproducts like pesticides and fertilizers) into the ocean has been linked with toxoplasmosis in southern sea otters along the California coast, aspergillosis in Gorgonian sea fans in the Gulf of Mexico, and white pox in Elkhorn coral in the Caribbean sea (**Figure 3(d)**) among other diseases (reviewed in Harvell *et al.*, 1999). Across the eastern seaboard of the United States and in the Gulf of Mexico, eutrophication due to excess run-off from agricultural fertilizers has led to outbreaks of *Pfiesteria piscicida*, an aggressive protozoan that kills large numbers of fish. In freshwater ecosystems, the spread and duration of avian cholera outbreaks in waterfowl populations is greatly facilitated by pollution from nutrient-rich run-off into wetlands. This disease, caused by the introduced bacterium *Pasteurella multocida*, has become the second most important cause of waterfowl die-offs in North America.

Global Climate Change and Disease

Because the development and transmission of many parasites depend on environmental conditions, it is not surprising that

anthropogenic climate change is expected to influence the distribution, prevalence, and severity of pathogens in natural ecosystems. A number of recent examples illustrate that even small changes in climate can speed up parasite generation times, shift the geographic ranges of both hosts and vectors, and propagate complex trophic cascades leading indirectly to disease emergence. In the Arctic, warming temperatures appear to enable lungworm parasites to mature twice as quickly within their slug intermediate host. Infection severity in muskoxen (the definitive host) is postulated to increase as climate continues to warm. Coral reef ecosystems are among the most severely impacted by small increases in temperature, which can trigger coral bleaching (expulsion of symbiotic algae from the coral) and enhance the growth rates of a suite of opportunistic infections that have led to massive coral die-offs. Although such ocean warming events can occur naturally in association with El Niño-Southern Oscillation events, their frequency and severity have increased during the past 20 years. As a result of warming oceans, Caribbean yellow band disease has increased in prevalence and virulence to endanger all four coral species in the genus *Montastraea*, the most important reef building corals of this region (Harvell *et al.*, 2009). Because so many marine organisms depend on habitat provided by coral reefs, and because reefs can take many years to recover, the consequences of such massive die-offs are profound and long-lasting.

As climate warms, species range shifts will also bring new species in contact with each other and with new parasites. For example, the distribution of some animal diseases (such as the previously discussed *P. relictum*, and *B. dendrobatidis*) could lead to declines of many endangered bird and amphibian species surviving in higher elevations. Since global climate impacts all biological interactions, some ecologists have noted that the net effects of changing climate can increase disease in some regions (as exemplified above) and decrease disease in others. Predicting changing biological interactions as a result of changing climatic conditions is therefore an increasingly important goal for scientists and wildlife managers.

Worldwide Commerce and Travel

Worldwide commerce and animal trafficking are probably the most important causes of new disease outbreaks in wildlife populations. This traffic, which has dramatically increased in the past few decades, includes international trade of live animal and plant stock for commercial breeding purposes, for zoos and animal parks, for the pet trade and hunting, and for laboratory research. The situation is exacerbated by the staggering amounts of international transport of fruit, vegetables, and various animal parts which can facilitate the spread of pathogens. For example, most of the major pathogens that cause disease epidemics in wild birds in North America were probably introduced by humans. Both avian cholera (a highly virulent disease caused by the bacterium *P. multocida*) and duck plague (caused by a herpesvirus) appeared first in domesticated bird flocks and spread to wild waterfowl. Today these diseases are responsible for the deaths of tens of thousands of wild waterfowl each year in the United States alone. Similarly, avian malaria was introduced to Hawaii with imported birds, and brucellosis (caused by the bacterium *Brucella abortus*) was introduced to North America by domesticated livestock (and has since spread to wild deer, elk, and buffalo populations).

Disease spread into wildlife populations is further facilitated by the continual breakdown of barriers between farmed animals and wild animals. For example, aquaculture practices create crowded conditions where farmed Pacific salmon become heavily parasitized by sea lice (*Lepeophtheirus salmonis*). Wild juvenile salmon returning to open waters from spawning grounds travel migratory routes in close proximity to salmon farms, and become infected by sea lice on their way back out to the open sea. Similarly, there is concern that international demand for farmed bullfrogs is contributing to the spread of amphibian chytridiomycosis (**Figure 4**). As bullfrogs can carry very high pathogen loads with no clinical signs of infection, escaped bullfrogs that invade new habitats may transmit the chytrid fungus to native species that are less resistant to disease.

Sparse regulation of the growing exotic pet trade has exacerbated risks of novel pathogen introductions. Many exotic pets comprise a mix of captive reared and wild caught animals, which can facilitate the transmission of infectious diseases acquired from different environments. Currently there are no screening procedures enforced for exotic animals, especially for animals ordered over the internet. In 2007, the number of exotic animals imported into the US numbered over 37 million, reflecting a staggering diversity of reptiles, amphibians, rodents, primates, and various wild cat species.

Transport and commerce also pose a risk for the spread of invertebrate diseases due to the growing popularity of raising and releasing insects such as ants, ladybird beetles, bees, and butterflies for the purposes of biological control, pollination, education activities or for release at special events. Not surprisingly, the parasite communities of most insect species and disease risks for wild insect populations remain largely unknown. Honey bees are probably the best-studied insect species in terms of their infectious diseases, but disease threats have also caused concern for wild bumble bee species (*Bombus* spp.) in recent years. Since the 1990s, colonies of several native bumble bee species have been mass-produced and distributed for the pollination of greenhouse crops in North America. The movement of bumble bees from Europe to the US is thought to have introduced an exotic strain of a microsporidian parasite that is highly virulent to some North American bumble bees. This parasite appears to have caused the near-extinction of at least one bumble bee species (*Bombus affinis*). Furthermore, the prevalence of this and other pathogens was shown to be higher in commercially reared bumble bees than in nearby wild populations. Furthermore, infections in wild bees declined with increasing distance from industrial greenhouses, strongly implicating human rearing operations in the disease-induced declines of these pollinator species.

Positive Role for Pathogens in Biodiversity and Conservation

Although the risks they pose to threatened species are apparent, parasites and pathogens can also maintain biodiversity, in

part through their effects on the outcome of other species interactions. Indeed, recent advances in the field of disease ecology show that parasites can maintain diverse communities through their effects on competition, food web structure, and trophic interactions. Other work suggests that over longer timescales, parasites can promote evolutionary diversification among hosts. These interactions support the idea that parasites are integral components of ecological communities and can even mediate ecosystem processes. Although the conservation of parasites is almost never considered to be a priority, a key point highlighted by this section is that not all aspects of parasitism are necessarily negative; a strong argument can be made for conserving endemic parasites and pathogens.

Parasites, Species Interactions, and Biodiversity

One way that parasites can enhance biodiversity is by altering the outcome of competition between host species. In this case, generalist pathogens that are relatively benign to one species can lower the density of other hosts for which they are more pathogenic, potentially reversing the outcome of resource competition. Apparent competition is a related phenomenon whereby two or more hosts not directly competing for resources are affected by the same parasite to different degrees. In some cases, parasites shared between host species can threaten highly susceptible native species with extinction, as discussed *in* Infectious Diseases as Threats to Biological Diversity (e.g., red squirrel–gray squirrel parapoxvirus). In other cases, parasites can promote biodiversity by having disproportionate negative effects on otherwise dominant species, thus allowing many species to coexist at relatively low densities. Several case studies support the role of pathogens in determining plant and animal community structure and modifying the outcome of competition. For example, malaria parasites have been shown to facilitate coexistence between two *Anolis* lizard species on the Caribbean island of St Maarten; in parts of the island where lizard malaria is absent, only the dominant competitor was found, whereas the competitively inferior species could coexist at sites where malaria was present. Other studies found that pathogenic soil fungi can be more harmful to dominant tallgrass prairie species, facilitating the coexistence of less competitive species. Soil-borne diseases have also been implicated in the rate and direction of succession in plant communities and in determining patterns of seedling recruitment in tropical rain forests.

When parasites attack dominant herbivore species in a community, epidemics can cause major shifts in community composition through secondary effects on plant recruitment and abundance or on the density of predators and other natural enemies. One example involves the introduction of rinderpest in the Africa; this viral disease of cattle was introduced in the late 1800s and spread to native ungulate species including wildebeest and African buffalo, causing mortality rates of up to 80%. Starting in the 1950s, a vaccination campaign in cattle eventually eliminated the virus from wildlife, leading to a gradual rise in the numbers of wildebeest and other ungulate species. The growing numbers of herbivores affected species at other trophic levels: with the recovery of wildebeest populations, predator populations increased (including lions and hyenas) and the biomass of vegetation (especially grasses) declined. This example illustrates how the effects of pathogens on dominant or keystone host species can propagate through food webs and alter community structure.

From a different perspective, evolutionary adaptations of hosts and pathogens over long timescales could increase biodiversity through cospeciation (the joint divergence of two or more interacting lineages) and phylogenetic diversification. For example, one comparative study of wild primates and their pathogens showed that primate host species from more diverse lineages harbored a greater number of parasite species (including viruses, protozoa, and helminths) (Nunn *et al.*, 2004). This pattern could be caused by an evolutionary arms race between hosts and pathogens, whereby hosts mount greater resistance against infection, and parasites evolve higher virulence and transmissibility. Other explanations are possible, however, including the idea that parasites infecting primates from more diverse lineages experience greater opportunities for diversification themselves through cross-species transmission and host shifting. As a final possibility, coextinctions of hosts and parasites might drive these associations. Parasite lineages could lose transmission opportunities and go extinct themselves as their hosts decline in population size, or as their geographic ranges shrink.

Pathogens and Host Genetic Diversity

In many ways, the maintenance of genetic diversity within species parallels the coexistence of multiple species in ecological communities and should enable hosts to evolve in response to changing environments and ecological perturbations. In terms of host–pathogen interactions, parasites are likely to be powerful selective agents because they can spread rapidly and cause significant negative effects on host fitness. Host species that are continually exposed to a diverse array of parasites should therefore harbor a variety of resistance alleles or a repertoire of inducible defenses. A growing number of examples from wild populations, including parasitic worms infecting Soay sheep and snails, and fungal pathogens affecting plants show how parasites can favor allelic diversity or sexual recombination in their hosts (Altizer and Pedersen, 2008).

Given the strong selective pressures imposed by parasites and the benefits of host resistance traits, an important question is why aren't all individuals resistant to infectious diseases? Simple host–parasite models show that genetic variation underlying host resistance traits can be maintained by balancing selection or by costs of resistance traits for host survival or reproduction. Balancing selection simply refers to processes that can maintain genetic variation over time, including heterozygote advantage, frequency-dependent selection, and selection pressures that fluctuate over time or space. For example, under frequency-dependent selection, rare host genotypes are resistant to pathogen genotypes that attack more common host types; this advantage held by rare alleles can lead to cycles in both host and parasite allelic frequencies over time, resulting in the maintenance of host genetic variation in the long-term. The phenomenon of parasites tracking

common host genotypes has been demonstrated by studies of parasitic trematodes infecting freshwater snails in New Zealand and bacterial pathogens of *Daphnia* in European ponds, and is critical for arguments concerning the role of parasites in favoring sexual reproduction in their hosts.

The major histocompatibility locus (MHC) in vertebrates provides a notable example of extreme polymorphism and diversity of rare alleles maintained by balancing selection. Immune proteins coded by MHC genes can recognize and bind to pathogen proteins (antigens) inside infected host cells, and transport these antigens to cell outer membranes. Here, they are presented to other immune cells that destroy infected host cells and initiate antibody production. Specific MHC molecules preferentially bind to specific pathogen peptides, and hence different MHC alleles confer resistance to different pathogens. In natural populations, MHC class I and II genes show enormous variation, and even species with low overall genetic diversity can show high genetic variation at MHC genes. In one extreme example, Aguilar *et al*. (2004) demonstrated high levels of variation across five MHC loci in a population of the San Nicolas Island fox with essentially no genetic diversity across selectively neutral loci. These findings led authors to conclude that intense balancing selection had maintained MHC variation in the face of past bottlenecks.

In plants, gene-for-gene coevolution has also been demonstrated to promote a high diversity of resistance and virulence alleles. For example, long-term field studies of the interaction between wild flax and flax rust in natural populations in Australia indicate that many alleles can persist among metapopulations, and the distribution of genotypes can change rapidly during individual epidemics. Collectively, these examples emphasize that pathogens commonly exert selection pressures on their hosts, in many cases leading to genetic heterogeneity over space and time.

Parasites and Ecosystem Function

Parasites and pathogens have long been excluded from studies of food webs (maps of feeding interactions in an ecological community) and ecosystem function (flows of energy and nutrients), in large part due to their small size and assumptions that they contribute little biomass to ecosystems. However, several recent studies have challenged this view by showing that parasites can dramatically alter the connectivity and stability of ecological food webs, and can represent a surprising fraction of biomass in real-world ecosystems. For example, parasites are involved in more food web links than predators in some estuarine and salt marsh ecosystems. Moreover, adding parasites to food webs increased measures of food web connectance (the number of actual links between species relative to the total links possible) by up to 93%. Indeed, parasites can account for 3–13% of the biomass of their free-living plant and animal counterparts in some estuarine ecosystems. Some parasitic worms like trematodes had biomass levels comparable to those of fishes and small arthropods, and their collective biomass was greater than that of top predators (namely shorebirds) (Kuris *et al*., 2008). A similar study of plant fungal pathogens in a tallgrass prairie ecosystem showed that the estimated pathogen biomass was comparable to that of herbivores, and that pathogen effects on grass biomass appeared to be stronger than the effects of herbivory (Mitchell, 2003). Other researchers have pointed out that host death caused by pathogens can liberate carbon and nutrients from host cells and tissues, increasing their turnover in natural ecosystems. Not surprisingly, however, the effects of pathogens on biogeochemical cycling remains relatively unexplored. More generally, a growing number of studies point to parasites as key players controlling properties of food webs and biomass production, and suggest that parasites should be viewed as essential components of ecosystem function rather than as nuisances that interfere with management objectives.

Parasites Can Inform Conservation of Threatened Species

Pathogens can reveal valuable information about host population size and movement in ways that enhance conservation efforts. A handful of case studies show that the evolution of some pathogens can be rapid enough to reveal cryptic host geographic isolation and historical contact patterns. For example, an analysis of sequence variation in the feline immunodeficiency viruses of cougars in western N. America (Biek *et al*., 2006) showed that different pathogen lineages dominated in different parts of the cougars' range, indicating population structure in the host that could not be uncovered by analysis of molecular markers in the cougars themselves. Genetic analysis further revealed that the spatial occurrence of viral lineages is expanding, most likely due to increases in cougar population size. In another study, three species of whale lice revealed historical separation of populations of endangered right whales (Kaliszewska *et al*., 2005). Genetic analyses of parasite mitochondrial DNA showed that whale populations in the North Atlantic, North Pacific, and southern oceans diverged several million years ago, following the formation of the Isthmus of Panama. High genetic diversity among lice in the currently small populations of North Atlantic right whales also indicated that their host population sizes probably numbered in the tens of thousands before the modern era of commercial whaling. This work illustrates the usefulness of pathogen molecular markers for understanding historical population size, contemporary population movements and geographic structuring of their hosts.

Managing Diseases and Biodiversity in the Future

Parasites and pathogens that pose significant threats to conservation programs can be divided roughly into two categories. The overwhelming majority of epidemics begin with the introduction of a disease into a naïve population. A second category of concern are cases where environmental changes (such as habitat loss and pollution) shift host relationships with endemic parasites. Effective disease prevention must therefore address both of these areas by limiting the introduction of novel pathogens and mitigating large-scale environmental changes that facilitate the emergence of endemic disease or cross-species transmission with domesticated species and humans. Monitoring and disease screening programs, quarantine and vaccination regimes, and attention to

captive breeding programs comprise common approaches to preventing or managing disease outbreaks in wild populations.

Monitoring Populations for Infection

Most pathogens are discovered after epidemics have already spread through wild populations. A powerful tool in the management of diseases is thus to monitor threatened populations for signs of infection before disease-induced mortality occurs. Several tools exist to monitor host populations and track the spread of an epidemic, and the efficacy of a monitoring program will increase with the number of host animals included in the screening (Wobeser, 2007).

Parasite prevalence and intensity are often monitored by examining blood, tissue, and fecal samples in animals. The presence of blood parasites (e.g., trypanosomes, malaria, and filarial nematodes), anemia, elevated leukocyte levels, and pathogen-specific antibodies can all be detected from relatively small volumes of blood. Antibody assays (e.g., enzyme-linked immunosorbent assay) can show evidence of current and past infection. Molecular techniques based on polymerase chain reaction tests can reveal the presence and intensity of infection in host blood or tissue by identifying and quantifying pathogen genetic material. In addition, many microbial pathogens can be cultured directly from tissue showing signs of infection or from swabs taken from the mouth, ears, eyes, nose, genitals, anus, or skin. Scans of feces will also provide information on the diversity of intestinal parasites in an individual as well as fecal egg counts per host as a measure of infection intensity.

Hosts that are hunted or culled for other purposes are frequently examined for internal and external parasites. Parasitic arthropods (e.g., ticks, mites, fleas, and biting dipterans) are also monitored because they represent key vectors for many infectious diseases and can decrease the condition of their hosts by drawing on host resources. Combining infection-related data from animals that are opportunistically sampled over a long time period can be useful for examining temporal trends. Long-term monitoring over large spatial scales is usually time consuming and expensive, but such studies often provide important information on the conditions leading to elevated disease prevalence, and are invaluable to developing effective disease management and prevention strategies for wild populations.

Assessing Disease Threats

Screening programs will verify the presence of a particular pathogen in a wild population, but do not provide information about population-level consequences resulting from infection. Epidemiological models outlined in the section on Basic Epidemiological Principles indicate that this determination requires information on both the prevalence of a pathogen (γ) and its effects on host fitness (α and δ). It is important to note that *post mortem* examinations are often performed to determine the effects of disease on individual hosts. However, the presence of a pathogen in dead animals does not necessarily indicate the cause of death, nor does it reveal subtle disease effects on host behavior or fecundity. Ideally, captive or wild hosts should be monitored throughout the course of infection to compare survival and fecundity between infected and uninfected hosts. Experimental manipulation of parasite loads in natural populations remains the most direct way of assessing the effects of pathogens on host populations.

Intervention Methods

Historically, diseases in wild populations have drawn the attention of wildlife managers only after an epidemic severely threatened the host population or when a pathogen threatens agricultural crops or livestock. In general, the types of management regimes used to limit disease spread vary depending on the type of pathogen, the threat it poses to host populations, and the availability of financial resources. Management regimes also depend on the existence of agricultural or veterinary information and tools, which often do not exist for free-living plant and animal populations. That being said, pathogen outbreaks in recent years have allowed researchers to employ some of the more common intervention methods available and test their efficacy in reducing pathogen spread and impacts.

Vaccination, Culling, and Quarantine

Several intervention measures center on decreasing the number of contacts between susceptible and infectious host individuals to prevent new infections. Viral infections and, less frequently, bacterial infections in vertebrate animals can be effectively controlled by vaccinations, which confer a period of immunity and essentially transfer susceptible hosts directly to the resistant/immune class (*see* Microparasites and **Figure 1**). However, the cost of vaccinations and lack of testing (for safety and efficacy) in most wildlife species may severely limit the success of population-wide disease control plans. Epidemiological models can be helpful in determining where to concentrate finite vaccination resources, and what proportion of the population to target to limit or eradicate disease. A frequent goal of vaccination is to treat enough hosts to prevent pathogen invasion, or to achieve local eradication. This critical vaccination threshold is $1-1/R_0$, which reduces R_0 below 1. Vaccinations have been successfully administered to slow the spread of rabies in European foxes and North American raccoons, and vaccination of domesticated reservoir hosts (cattle and domestic dogs) has reduced the threats of rinderpest and canine distemper outbreaks in wild ungulates and carnivores in east Africa. A key benefit from vaccination is that even when susceptible hosts are not vaccinated they are less likely to be infected because they are surrounded by immune individuals. It is also important to note that even if too few hosts are vaccinated to eradicate a pathogen, low-coverage vaccination (of a small fraction of hosts) can still prevent local host extinction, as recently illustrated by vaccination of Ethiopian wolf populations against a potentially devastating rabies virus outbreak.

Culling is the removal of host individuals by lethal means. Culling can be indiscriminate or targeted solely toward infected individuals and is most often used when transmission is

believed to be density-dependent. The goal is to reduce host densities below the threshold needed for parasite persistence, N_T. Selective culling targeted toward infected individuals is analogous to intensifying parasite-induced mortality (α; **Figure 1**). This strategy can effectively decrease disease prevalence and lower R_0. Selective culling has been implemented to counter the spread of certain tree diseases (e.g., Dutch elm disease). Although less frequently applied to vertebrate populations, routine culling has been applied to some populations of Cape buffalo in South African national parks when individuals are found with tuberculosis. In Venezuela, invasive American bullfrogs are routinely culled to slow the spread of amphibian chytridiomycosis to native species (**Figure 4**).

Quarantine involves the isolation and care of infected individuals from a population currently experiencing an outbreak, with the goal of decreasing contact rates between infectious and susceptible individuals. Although commonly employed to slow or stop human disease outbreaks, this approach is less often applied to wild populations. An exception occurs when host populations are dangerously low in numbers such that every member may be of great value and worth rehabilitating. This was the case during a canine distemper epidemic in Wyoming black-footed ferrets (*noted in* Introduced Parasites and Species Declines), and a similar situation may arise if future epidemics affect endangered African apes, Ethiopian wolves, or African wild dog populations.

Alternative Interventions

Administering focused treatment regimes of antiparasitic drugs could be the best option for infectious diseases in some populations, although little quantitative data exist on the success of these approaches. Drugs and other forms of chemotherapy are most frequently administered for bacterial, fungal, helminth, and ectoparasitic infections. This method of disease control least effectively addresses the ultimate cause of a disease and may be extremely costly for population-wide control measures (e.g., systemic fungicides to counter tree blights). Other examples of targeted intervention include the treatment of mites causing mange in cheetahs, Arctic foxes, mountain gorillas, and wombats, but the long-term efficacy of these treatments is still unknown. Recently, researchers discovered that the microbial community and a wide array of antimicrobial peptides covering the surface of amphibian skin can confer protection against chytridiomycosis in some frog species (**Figure 4**). The possibility of augmenting this unique form of innate immunity to help conserve amphibian populations is an active area of research.

Habitat Management

Management regimes that address the ultimate causes of disease outbreaks have the greatest potential for removing disease threats but are also the most difficult to implement. Indeed, the ultimate causes of marine invertebrate diseases, namely, pollution and ocean temperature changes, are so global and diffuse in origin as to be impossible to confront in any single species management plan. Managing terrestrial habitats may alleviate some disease-related conservation problems. Two such examples are the proposed creation of a bovid-free land zone around Yellowstone National Park to prevent contact between bison and cattle, and the removal of feral pigs from the Hawaiian Islands (because their activities increase mosquito-breeding areas and elevate the transmission of avian malaria).

An understanding of disease ecology is also pertinent to the design of habitat reserves. For example, how large should reserves be to prevent host crowding and limit disease transmission? Do corridors between reserves increase the threat of pathogen transfer among locations, or does host dispersal among habitats facilitate the spread of resistance genes or aid in parasite avoidance? Maintaining species richness and genetic diversity within reserves is also critical to limiting threats from disease. In particular, high species diversity may buffer natural communities from devastating epidemics, and habitats that are restored with genetically diverse stock may be less susceptible to pathogen invasion. In terms of host species diversity, this can be particularly important in slowing the spread of multihost pathogens. One hypothesis that has gained support from studies of both plant and animal communities is the dilution effect, which predicts a pattern whereby disease risk decreases with increasing host species diversity through one of several underlying mechanisms (Keesing *et al.*, 2006). This buffering effect of host diversity can be especially important for pathogens spread by vectors that feed on several host species, but for which only a few host species effectively amplify the pathogen. The best-studied examples are the Lyme disease bacterium and West Nile virus, two pathogens that are transmitted between dozens of animal species by ticks and mosquitoes, respectively. In both cases, the presence of a diverse mammal and bird community, respectively, reduces the fraction of vectors that feed on highly competent hosts, and lowers infection prevalence in vectors. Thus, habitat management practices that maintain diverse vertebrate communities (such as larger reserve sizes and corridors that connect existing reserves) could lower the risk of pathogen transmission to wildlife and humans.

Concerns for Captive Breeding Programs

Conservation efforts rely increasingly on captive breeding programs to augment and restore free-living populations. Because captive animals are particularly susceptible to infections, pathogens represent a significant concern of captive breeding managers. Captive animals may acquire novel infections from unrelated species kept in the same pen, foster parents, or from individuals of the same species or closely related species. For example, captive African elephants kept in mixed collections have been infected with a lethal herpesvirus that occurs without disease symptoms in their Asian elephant pen mates. Captive-bred hatchlings of the endangered Mauritius pink pigeon contracted and succumbed to a herpesvirus infection that their foster parents (domestic rock doves) were carrying without ill effects. Several human diseases, such as measles, tuberculosis, herpesvirus, and influenza are highly virulent for nonhuman primates, especially gorillas, chimpanzees and other apes, which poses a concern for zoo and sanctuary animals exposed to potentially infected humans (and for wild animals exposed to ecotourism groups).

Many animals in captive breeding programs are often held close together, a practice that poses two disease-related risks.

First, animals are likely to become stressed and hence more susceptible to infection (particularly those that are territorial or normally persist at low densities in the wild). Second, crowding in pens or cages can elevate host densities above the threshold necessary for virulent pathogens to invade, and will also increase transmission rates (e.g., hosts may reinfect themselves by ingesting the eggs of their own parasitic nematodes released into their pen). Vector-borne diseases or parasites with complex life cycles may be less of a concern to captive breeding programs because of the likely absence of intermediate hosts (or vectors) that are necessary for transmission.

Finally, additional complications exacerbating disease problems in zoos and captive breeding programs stem from inbreeding depression, or the genetic impoverishment of a captive colony due to loss of diversity and the expression of deleterious recessive alleles. This loss of genetic variability leads to homogeneous captive populations that can be very susceptible to a variety of pathogens, as implicated in the high mortality that captive cheetah populations experienced due to a feline infectious peritonitis virus. Hence, genetic and ecological problems can operate in synergy to reduce population size and diminish heterozygosity, leading populations toward increased disease susceptibility, and possible extinction. In conclusion, as captive breeding programs expand, disease-related problems are likely to become even more prevalent and will require serious precautions including screening and treatment, suitable housing and animal care, and separating potentially infected individuals (including some humans) from healthy captive populations.

Equal Rights for Parasites?

Parasites are an integral part of life on earth, with their biodiversity projected to be significantly greater than the species richness of free-living hosts, but biologists have uncovered only a tiny percentage of this diversity. Many micro- and macroparasites that live uniquely on threatened host species could go extinct long before their hosts, and this poses a distinct threat for parasite extinction. As with most taxa, we do not have accurate numbers of how many species of parasitic organisms might be affected by future extinctions. But in contrast to many other threatened species, the conservation of parasites is virtually never considered a primary goal of conservation strategies. This is most likely due to their small body size and cryptic nature, and because parasites tend to draw the most public attention during epidemics where large numbers of hosts die from infection.

Many parasites have fascinating life cycles and have evolved incredible strategies for dispersing their progeny to new hosts. Some parasites have become sources of pharmaceutically important products; for example, a tick salivary gland component (calreticulin) has been used in dogs to treat thrombosis and heart disease. Perhaps more importantly, as humans disturb natural balances and break transmission barriers among species, pathogen outbreaks among rare or threatened host species will continue to occur. Because pathogens are a major factor promoting both genetic and species diversity in natural communities, conservation strategies that result in the loss of native parasites might ultimately rob host populations of genetic diversity needed to respond to future epidemics. An important question that has yet to be answered is "do healthy populations have a diverse community of parasites?" If the answer is "yes," then maintaining endemic parasite populations, and hence the ability of wild populations to respond evolutionarily to parasite-mediated selection, could be one of the best long-term strategies for mitigating the risks of infectious diseases.

See also: Biodiversity and Human Health. Biodiversity, Evolution and. Captive Breeding and Reintroduction. Climate Change and Ecology, Synergism of. Climate Change: Anticipating and Adapting to the Impacts on Terrestrial Species. El Niño and Biodiversity. Endangered Amphibians. Estuarine Ecosystems. Hemiparasitism. Implications of Urbanization for Conservation and Biodiversity Protection. *In Situ*, *Ex Situ* Conservation. Loss of Biodiversity, Overview. Microbial Biodiversity. Microorganisms (Microbes), Role of. Parasitism. Population Diversity, Overview. Species Interactions. Traditional Conservation Practices. Wildlife Management. Worms, Nematoda. Worms, Platyhelminthes

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