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The Role of Infectious Diseases in Natural Communities

WHAT INTRODUCED SPECIES TELL US

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Mathematical models provide many predictions about the effects of parasites on host populations, but these predictions have been challenging to test. Controlled parasite addition and removal experiments have provided some of the most valuable insights into theoretical predictions. Like these experiments, species invasions may involve the addition and removal of infectious disease agents and may therefore add to our understanding of the effects of infectious disease. For example, species that invade without their parasites give us an idea of how populations perform in the absence of parasites, whereas biological control programs and introduced diseases can provide before-and-after comparisons. Studies of introduced species indicate that parasites can reduce host performance and may dramatically reduce host abundance. These effects on host species can have indirect effects on ecosystems. Species introductions can result in novel host-parasite contacts, which provide insight into how host-parasite evolution drives host specificity and indicate that host-parasite evolution can occur in only a few generations.

Introduction

Following the introduction of livestock with rinderpest into colonial Africa, diseased carcasses, bloated vultures, and starving predators populated the plains of the Serengeti. For the ranchers and local officials, the effect of infectious disease on the savanna ecosystem was impossible to overlook. Among modern ecologists, the role of infectious disease is gaining increasing appreciation, largely through the lens of species introductions. This chapter reviews how introduced species, such as the morbillivirus that causes rinderpest, can lend considerable insight into the role of infectious diseases in host populations.

We use the term "infectious disease" to describe the pathological consequences of infection by parasites and pathogens. Disease is a pervasive element of all natural communities (Kennedy et al. 1986; Dobson et al. 1992), and the parasitic mode of life is probably the most popular of all consumer strategies (Price 1980; Toft 1986). Nearly all animal taxa have parasitic representatives, and most free-living species (and many parasite species) are hosts for infectious disease agents. At the most basic level, parasites (and pathogens) are consumers that live in physical association with their hosts. Although all parasites negatively affect the vital rates (e.g., birth, growth, death) of their hosts by consuming host energy, there is a vast diversity of parasitic strategies, including those of typical parasites (e.g., intestinal tapeworms), pathogens (e.g., many viruses, fungi, and protozoa), parasitoids (e.g., some wasps and nematodes), parasitic castrators (e.g., rhizocephalan barnacles), trophically transmitted parasites (e.g., larval acanthocephalans), and, by some considerations, micropredators (e.g., herbivorous insects) (Lafferty and Kuris 2002). Depending on the type of infectious agent, hosts can suffer a variety of consequences, including higher mortality, slower growth, lower fecundity, altered behavior, and lower social status.

Before discussing the utility of introduced species as a tool to understand host-parasite dynamics, we provide background information on existing paradigms and traditional tools for testing them. Changes in the vital rates of infected hosts can have a diverse array of effects at the population level as well as on natural selection. Here, we are specifically interested in the effect of infectious diseases on host population dynamics. An obvious prediction is that infectious disease agents with strong effects on host vital rates could noticeably reduce the mean equilibrium density of their hosts. Infectious diseases that reduce host density may have indirect effects by facilitating trophic cascades or mediating competition. Finally, due to the likelihood of density-dependent feedback between host and parasite populations, there is potential for infectious diseases to alter the stability of host populations.

In addition to valuable studies of the pathological effects of infectious diseases and the natural history of infectious disease agents, research into the population-level effects of parasites and pathogens comes from theoretical mathematical models, correlations in nature, and experiments in the labora-

tory and field. For example, simple mass action equations (Bernoulli 1760) used in "microparasite" disease models track *s*usceptible, *e*xposed, *i*nfectious, and *r*ecovered hosts (SEIR models), but not parasite intensities (the number of parasite individuals within an infected host) (Ross 1916; Kermack and Mc-Kendrick 1927; May 2000). Similarly, models of parasitoids used for assessing the effects of biological control agents are based on predator-prey models. Finally, intensity-dependent "macroparasite" models have proved most effective in understanding the relationship between parasite intensity and host pathology.

Perhaps the most fundamental principle of epidemiology revealed by models is that the spread of a directly transmitted infectious disease agent through a population increases with the density of susceptible and infectious hosts. Most simple epidemiological models indicate that there is a host threshold density below which a parasite cannot sustain itself within a host population (Anderson and May 1979). This feature is key to understanding the parasite release hypothesis for species invasions because introduced species usually experience a population bottleneck in the early stages of an invasion. In addition, the density-dependent nature of transmission makes infectious diseases unlikely to be agents of extinction except where alternative hosts are present (Dobson and May 1986).

The best evidence for the hypothesis that parasites can affect host populations comes from experiments in which parasites are added to an uninfected host population or removed from an infected host population (Scott and Dobson 1989). This has been done repeatedly in the laboratory, often leading to the conclusion that parasites can limit host abundance (Greenwood et al. 1936; Park 1948; Stiven 1964; Keymer 1981; Lanciani 1982; Anderson and Crombie 1984; Scott and Anderson 1984; Scott 1987). This approach is much more difficult to apply to natural populations due to logistic and ethical constraints.

One parasite species for which field experiments have been applied successfully is the parasitic nematode *Trichostrongylus tenuis* in one of its host species, the red grouse (*Lagopus lagopus scoticus*) (Hudson and Dobson 1989; Dobson and Hudson 1992; Hudson et al. 1992). The host species is a game bird restricted to the upland areas of northern Britain. The population dynamics of red grouse exhibit sustained cycles of abundance with a period of about 5 to 6 years, and variation in grouse fecundity is associated with these cycles (Jenkins et al. 1967). Empirical studies (Potts et al. 1984; Hudson et al. 1985) and mathematical models (Dobson and Hudson 1992) suggest that decreased fecundity associated with nematode infection can cause grouse populations to cycle. Hudson et al. (1998) tested this hypothesis using a parasite removal experiment. By treating a significant proportion of a grouse population with drugs (reducing the number of nematodes in infected hosts), they increased grouse fecundity and thus changed the long-term population dynamics such that grouse populations no longer suffered periodic crashes (Hudson et al. 1998).

Although such experiments are very handy for testing predictions, it is difficult to manipulate and monitor disease agents in a safe and controlled manner. Introduced species can help us to circumvent the difficulty of designing and implementing field experiments, as they present a rich set of inadvertent "natural" field experiments that we can use to expand our understanding of the role of parasites in natural communities. In this chapter, we examine species introductions with respect to parasites and disease agents in several ways. We begin by exploring what happens when introduced species leave their parasites behind. We next consider the effects of biological control. Finally, we consider, through a detailed examination of four case studies, the effects of accidentally introduced disease agents.

Unintentional Parasite Removals: Introduced Species and Escape from Natural Enemies

Species introductions can provide an opportunity to look at how host populations perform without parasites, albeit with substantially less control than field experiments. In this section, we address the evidence that introduced species can serve as parasite removal experiments and then summarize the inferences made possible by comparative approaches.

Growing evidence indicates that introduced plants and animals escape most of their native parasites and pathogens. Parasites may be absent from the host founder population, die out soon after the invasion (due to low host density), or fail to complete their life cycles in the new environment (Dobson and May 1986; Cornell and Hawkins 1993; Kennedy 1993; Torchin et al. 2001; Mitchell and Power 2003; Torchin et al. 2003; Ricklefs, this volume). Over time, and as they spread, introduced species accumulate new parasites, but these generally amount to only a fraction of those they escape, perhaps because native parasites lack a coevolutionary history with introduced species (Cornell and Hawkins 1993; Mitchell and Power 2003; Torchin et al. 2003). The resulting decrease in parasitism may explain why some introduced species proliferate in their new environment and become abundant pests (Torchin et al. 2001; Mitchell and Power 2003). Freed from the effects of old host-parasite associations and occasionally establishing new ones, introduced species provide "natural experiments" that can be used to reveal the extent to which parasites control host populations and structure ecological communities. Geographically widespread species and those that have invaded multiple regions allow particularly comprehensive analyses with replication.

Available evidence from plants and animals suggests that only a fraction of the parasite species that infect species in their native range will infect those same species' populations in their introduced range (Torchin and Mitchell 2004). Parasite species richness generally decreases by 63%–77% in introduced plant populations compared with populations in their native range, while introduced animal populations are infected with roughly half the number of para-

TABLE 5.1 *Parasite release experienced by introduced species in different taxonomic groups*

Taxonomic group	Sample size (no. of species)	Species released (%)	Mean release (SR)	S.E.	Mean release (P)	S.E.	Source
Plants	473	100	0.77	—	—	—	Mitchell and Power 2003
Insects[a]	87	67	0.63	0.04	0.6	0.1	Cornell and Hawkins 1994
Crustaceans	3	100	0.86	0.14	0.93	0.06	Torchin et al. 2003
Mollusks	7	100	0.56	0.09	0.44	0.25	Torchin et al. 2003
Fishes	6	100	0.76	0.11	0.89	0.03	Torchin et al. 2003
Amphibians and reptiles	3	100	0.57	0.14	0.38	0.07	Torchin et al. 2003
Birds	3	100	0.36	0.14	0.41	0.22	Torchin et al. 2003
Mammals	4	100	0.29	0.12	0.12	0.13	Torchin et al. 2003

Note: Parasite release is represented by the proportion $(N − I)/N$, where N is the value for the native range and I is the value for the introduced range. These values are calculated for standardized parasite species richness (SR), the proportion of parasite species found in the introduced range out of the total number found in the native range, and for average parasite prevalence (P), the proportion of the population infected.

[a]Data based on medians where the range of values was reported ($N = 22$); three of these had higher prevalence in the introduced range, and these data were included in the calculation of release.

site species found in native populations (Cornell and Hawkins 1993; Torchin et al. 2003). All things being equal, this lower diversity of parasites should add up to a reduced effect of parasitism (though this depends on which parasites are left behind and the abundance of natural enemies that remain with or colonize to the host species). An additional measure of parasite release is that parasite prevalence (percentage of individuals infected) in introduced animal populations is also typically less than half that in native populations (Cornell and Hawkins 1993; Torchin et al. 2003). Similarly, introduced plants are less frequently infected with pathogens compared with native populations (Torchin and Mitchell 2004). Introduced populations of all taxonomic groups, including plants, insects, crustaceans, mollusks, fishes, amphibians and reptiles, birds, and mammals exhibit 29%–86% parasite release compared with native populations (Table 5.1).

Species adapt to the abiotic environment in which they have evolved, and an inappropriate match of abiotic conditions probably explains why many invading species fail to establish successful populations. Those species that do become established sometimes become pests and exhibit greater densities and biomasses than in their native range (Table 5.2). Information on body size indicates that introduced populations may also exhibit a larger average body size than native populations (Torchin et al. 2001; Grosholz and Ruiz 2003). Grosholz and Ruiz (2003) examined 19 introduced invertebrate species and found that

TABLE 5.2 *Evidence for increases in demographic parameters (following parasite release) of introduced species in their naturalized range, as compared with their native range*

Taxonomic group	Species	Parameter compared	Mean % increase (response range)	Evidence for parasite release?	Source
Plants	*Prunus serotina*	Density (m^2)[a]	86 (80–92)	Yes	Reinhard et al. 2003
	Lythrum salicaria	Biomass (g)	157	—	Blossey and Notzhold 1995
	Lythrum salicaria	Height (cm)	41	—	Blossey and Notzhold 1995
Crustaceans	*Carcinus maenas*	Biomass (kg)	59	Yes	Torchin et al. 2001
	Carcinus maenas	Mean size (mm)	29	Yes	Torchin et al. 2001
Mammals	6 species	Density (km^2)	424 (61–735)	Yes	Freeland 1993
Amphibian	*Bufo marinus*	Density (100 m^2)	7400	Yes	Lampo and Bayliss 1996a
Marine invertebrates	19 species	Max. size (mm)	13 (8–45)	—	Grosholz and Ruiz 2003

[a]Density was calculated with nearest-neighbor techniques; values show a decrease in nearest-neighbor distance and hence an increase in density.

63% had a significantly larger body size in introduced populations than in native populations. In some cases, losses of natural enemies have been implicated as a potential cause of the increased demographic performance of introduced populations. Natural enemies are an aspect of the native environment that is inherently hostile. Losses of parasites, therefore, could explain the increased demographic performance of introduced species. Still, the increased performance of introduced species and its link to natural enemies is a topic in need of substantial research.

One study specifically addressed (1) how a parasite can affect the body size and abundance of its natural host and (2) how loss of this parasite (through invasion) can result in larger sizes and greater abundances. By comparing multiple native and introduced populations of the European green crab (*Carcinus maenas*), Torchin et al. (2001) demonstrated that a particular group of parasites, parasitic castrators (a rhizocephalan barnacle and an entoniscid isopod), explained 64% of the variation in mean crab size and 36% of the variation in crab biomass in native crab populations. Parasitic castrators do not infect any of the introduced populations, which exhibit significantly higher biomass and larger body sizes compared with native populations (Torchin et al. 2001). Similarly, introduced cane toads (*Bufo marinus*) in Australia reach densities two orders of magnitude higher (1000–2000/100 m^2 vs. 20/100 m^2) than native populations in South America (Lampo and Bayliss 1996a,b; Lampo and DeLeo

1998). Australian populations harbor fewer than 30% of the helminth parasites found in native populations (Barton 1997). In addition, they lack ectoparasites that may control toad density in South America (Lampo and Bayliss 1996a,b; Lampo and DeLeo 1998). Introduced plant populations may also experience demographic release compared with native populations. They are more likely to become noxious weeds if released from pathogens (Mitchell and Power 2003). This finding suggests that pathogens may limit native plant populations as well.

In addition to providing insight into the role of parasites in host demography, biological invasions can indicate the extent to which parasites mediate interactions among free-living species. Parasites can alter competitive dynamics among hosts (Hudson and Greenman 1998). This effect can be addressed by comparing competing native and introduced species. For example, in California's San Joaquin Valley, a suite of native parasitoids attacks the native leafhopper (*Erythroneura variabilis*). An invasive congener (*E. elegantula*) is attacked much less frequently, causing the invader to replace the native (Settle and Wilson 1990). A similar interaction has been demonstrated experimentally in microcosms (Aliabadi and Juliano 2002). Here, introduction of Asian tiger mosquitoes (*Aedes albopictus*) infected with gregarine parasites does not affect the survivorship of a native mosquito (*Ochlerotatus triseriatus*). However, addition of uninfected tiger mosquitoes causes the invader to outcompete the native by reducing its survivorship. Thus, escaping this parasite may give the invading tiger mosquito a competitive advantage and facilitate its spread (Aliabadi and Juliano 2002).

Finally, in addition to their direct effects on host survivorship, parasites may have indirect effects by facilitating changes in their host's behavior. For example, the presence of parasitoid flies alters the behavior of fire ants (*Solenopsis invicta*), reducing their competitive ability in their native Brazil (Orr et al. 1995). Release from the fly may explain the competitive dominance of the fire ant over native North American ant species. Evidence of reduced parasitism facilitating competitive interactions in invading plants is less clear. Blaney and Kotanen (2001) found no difference in the effect of fungal pathogens on seed recovery (survivorship) of native and in introduced plants. However, Klironomos (2002) demonstrated that soil pathogens significantly reduced growth of native plant species, but did not reduce growth of introduced plants (see also Callaway et al., this volume).

Escape from natural enemies could have many implications for evolution and speciation. One main benefit of colonizing a new location may be escape from coevolved natural enemies such as infectious disease agents. At geologic time scales, colonization of new locations by species is commonplace. Although the risks of failure are high, the payoff includes short-term access to abundant resources and a longer-term freedom from parasites and pathogens. These benefits have broad consequences for biodiversity. Natural invasions at remote locations establish reproductive isolation among populations, which in turn can lead to speciation. The extent to which release from parasites facilitates the

success of isolated populations could conceivably influence speciation rates. On a longer time scale, taxon cycle theory predicts that eventually natural enemies will catch up with released species and erode their advantage (see Ricklefs, this volume).

The association between release from parasites and pathogens and subsequent performance in terms of body size and density provides substantial support for the hypothesis that coevolved parasites strongly affect the demography of their hosts through various pathogenic effects. Because this pattern appears in all taxa considered so far, it speaks to the evident generality of the role of infectious disease in natural populations.

Intentional Parasite Additions: Biological Control Effects on Target Host Populations

Parasite addition experiments are powerful tools for determining the effects of parasites. In this section, we argue that many classic biological control programs are essentially parasite addition experiments. Here, the host is usually an unintentionally introduced pest and the natural enemy is intentionally introduced. These large-scale field manipulations, when successful, demonstrate that infectious agents can control host populations. Although this approach provides numerous examples and powerful insights, we also consider its limitations.

The release of introduced species from natural enemies, as described above, has led some species to become pests, affecting populations of native species, altering community structure, or exerting a negative economic impact on human activities (often on managed, agricultural species). Biological control seeks to reduce the abundance of exotic species that are pests (e.g., exotic insect pests, terrestrial and aquatic plants, a few vertebrates, and some medically important mollusks) to an economically or culturally acceptable level by reconstructing a few elements of the natural control of the pest where it was native. The infectious natural enemies employed in control campaigns include many parasitoids (against insects), some microbial pathogens (against vertebrates and insects), a few parasitic castrators (against snails that can serve as vectors for human disease), and herbivorous insects (against weeds). Such natural enemies usually reduce pest density by directly influencing pest mortality or reproductive rates. A few manipulations in ponds have demonstrated that biological control of freshwater snails can be achieved by parasitic castrators (trematodes) and that these parasites can competitively exclude other trematodes that cause human disease (Lie and Ow-Yang 1973; Nassi et al. 1979; Lafferty 2002).

Although biological control agents have frequently been shown to control target host populations, there are few examples of eradication or extinction of targeted pests. The most frequently cited example of extinction of a pest by its control agent is that of the coconut moth, *Levuana iridescens*. The control

agent, a tachinid, *Bessa remota*, rapidly made the coconut moth vanishingly rare (Tothill et al. 1930). However, a recent examination of this case history suggests that *L. iridescens* is probably not extinct (Kuris 2003).

Overall, only a minority of biological control efforts meet their control goals. This frequent lack of "success" is partly a result of the very high standard set for control. However, the variation in success across taxa, habitats, functional groups, and life histories underscores the concept that the effects of infectious diseases on host population dynamics, while occasionally dramatic, vary greatly, even for natural enemies that can kill or castrate a host individual. The sources of this variation are under active investigation and include the searching efficiency of the infective natural enemy, its host specificity, age structure, refugia, interactions with other mortality sources, competition among infectious agents, and the type of infectious agent (parasite, pathogen, parasitoid, parasitic castrator) (Hall et al. 1980; Murdoch et al. 1985, 1987, 2003; Kuris and Lafferty 1992; Ehler 1998; Begon et al. 1999; Bellows and Hassell 1999; Briggs et al. 1999; Shea et al. 2000).

The frequent success of biological control programs against Homoptera, such as aphids, scale insects, and whiteflies (Hall et al. 1980; Ehler 1998), emphasizes the importance of the relative scale of recruitment to host and parasite populations (Kuris and Lafferty 1992). As Murdoch et al. (1985, 2003) have shown for the red scale insect (*Aonidiella aurantii*) and the parasitoid *Aphytis melinus*, host populations are regulated because the parasitoid operates over a much larger spatial scale than does its host. Infecting many host populations sustains the parasitoid population at a sufficient density to significantly reduce most host populations at a particular moment (while preventing either the parasite or the host from going locally extinct). Consequently, this parasite does not closely track each host population and maintain stable local equilibria. We suggest that this may be a common feature of host-parasite population dynamics revealed through analysis of biological control. Infectious agents with relatively open recruitment will be most likely to control host populations (Kuris and Lafferty 1992).

Some biological control campaigns have produced unintended effects on nontarget species. This is generally undesirable because native species that are not pests may be affected. Among the best-documented studies are those of the tachinid parasitoid *Compsilura concinna*, introduced to North America to control the European gypsy moth (*Lymantria dispar*) (Boettner et al. 2000). Although the tachinid does frequently parasitize the gypsy moth to the extent that populations decline, gypsy moths remain abundant in New England forests. Thus, high gypsy moth populations sustain the abundance of the tachinid, which has broad host specificity across the lepidopterans. Consequently, this generalist biological control agent can drive other (native) hosts to very low densities. Even a host-specific biological control agent can affect native species through indirect pathways (Pearson and Callaway 2003) under a limited set of conditions (Thomas et al. 2004).

Biological control projects afford an excellent opportunity to examine host specificity. Using Combes's (2001) perspective on host specificity, a successful parasite must encounter a host and be compatible with it (Combes describes these processes as a series of filters). Encountering a host requires appropriate behavior, habitat, and temporal patterns. Compatibility requires completing development (by feeding on the host) and surmounting the immunological defenses of the host. While the question of compatibility is easily amenable to experimental investigation and is generally emphasized in analyses of host specificity, host encounter is generally only partially revealed by patterns of host use in nature. Intentional releases of infectious agents in biological control programs offer perhaps the most substantial body of evidence for the importance of host encounter because actual infection in the field can be compared with the results of compatibility studies in the laboratory. Particularly for biological control agents of weeds, but recently for other agents as well, laboratory analyses of compatibility are routinely conducted and included in risk assessment before deployment. When the encounter filter is experimentally removed in the laboratory, a specialist can often parasitize a wide range of compatible hosts. Studies following the release of biological control agents show, however, that many agents infect fewer species in nature than when placed in test arenas (Sands 1997), suggesting that limited host encounter could greatly determine host specificity.

The evolutionary interactions between vertebrate hosts and microbial pathogens have been most strikingly demonstrated through biological control campaigns. To mitigate the ecological and economic impacts of the European rabbits (*Oryctolagus cuniculus*) that became abundant exotic pests in Australia, a rabbit-specific myxoma virus was released. This virus was a relatively avirulent pathogen of South American rabbits (*Silvilagus brasiliensis*) (Hoddle 1999), but it swept through Australian rabbit populations, causing very high mortality rates. This resulted in strong selection for immune response to the virus in rabbits (Fenner and Ratcliffe 1965; Hoddle 1999). More importantly, as rabbits became rare (often decreasing below the transmission threshold), the virus experienced selection for reduced virulence, as measured by exposing laboratory rabbits to viruses taken from the wild in successive years (Fenner and Ratcliffe 1965). This case history revealed the close interplay between selective pressures and host and parasite population dynamics. The accidental release in Australia of a rabbit-specific calicivirus from Europe has begun to replay some of these dynamics as rabbit populations have experienced dramatic declines in some areas (Hoddle 1999). It will now be interesting to examine interactions in a two-pathogen one-host system.

Despite the oversimplification of agricultural systems (where many biological control experiments are performed), the overall conclusion from consideration of biological control studies is that infectious diseases can exert a significant and strong effect on host populations. However, as predicted by simple epidemiological models, biological control rarely leads to host extinc-

tion because pathogens decline in importance as hosts become rare. The ability of an infectious disease organism to persist in times or places of low host availability may determine the need for use of alternative hosts or broad dissemination. Where host specificity is low, there is the potential for broad impacts on the community of susceptible hosts (e.g., parasite-mediated changes in competitive ability). Finally, observations of novel contacts between pests and biological control agents show how virulence and host defenses can evolve.

Unintentional Parasite Additions: Effects of Introduced Pathogens on Host Populations

Perhaps the most notable insights into the role of infectious disease have come from introduced diseases. Increases in global trade have allowed diseases to cross geographic barriers and attack naïve hosts. In addition, human encroachment on wildlife habitat has led to more opportunities for transmission of disease to new hosts. Although the vast majority of disease introductions probably fail, those that succeed can have dramatic effects on new hosts, underscoring the potential for infectious diseases to influence not only host population dynamics but also entire communities. Below, we present four case studies that have shed considerable light on the effects of infectious disease on entire communities.

Introduction of avian pox and avian malaria to the endemic Hawaiian avifauna

The largest loss of the native Hawaiian avifauna occurred following the arrival and colonization of the Hawaiian Islands by Polynesians (James and Olson 1991; Olson and James 1991). Since Europeans arrived, however, the Hawaiian Islands have lost additional native species while gaining more than 125 exotic bird species, over 60 of which have become naturalized (van Riper and Scott 2001; Sax et al. 2002). This trend toward biotic homogenization of the Hawaiian avifauna can be explained in part by the post-European extinction event that peaked in the early twentieth century. At this time, the endemic Hawaiian avifauna undoubtedly experienced losses due to habitat destruction, introduced predators, and competitors. However, Warner (1969) cogently examined the evidence for the influence of habitat destruction, competition, and predation and rejected these as the primary cause of this extinction event. Warner observed that native birds of several species were common in disturbed areas in the nineteenth century, that they readily consumed a wide variety of introduced plants, and that large tracts of native vegetation remained in some lowland and most upland areas. When the die-off occurred, it was rapid and nearly total in lowland areas; by 1902, native lowland forests were largely silent. During this brief period, diseased native birds were unusually common. Their symp-

TABLE 5.3   *Prevalence and effects of avian pox and avian malaria on the native and exotic avifauna of the Hawaiian Islands*

Species	Altitude (m)	Disease[a]	Prevalence
NATIVE			
Chasiempis sandwichensis	1900	Avian pox[1]	1.0%
	1550	Avian pox[1]	40.0%
	300–2400	Avian pox[2]	19.5%
		Avian malaria[3]	6.0%
Hemignathus virens	>1600	Avian malaria[3]	Seropositive
	<1600	Avian malaria[3]	Seropositive
	300–2400	Avian malaria[3]	7.3%
		Avian pox[2]	17.6%
Vestiaria coccinea	300–2400	Avian malaria[3]	6.1%
		Avian pox[2]	16.8%
Myadestes obscurus	300–2400	Avian malaria[3]	2.1%
		Avian pox[2]	24.3%
Himatione sanguinea	300–2400	Avian malaria[3]	29.2%
		Avian pox[2]	34.9%
Telespiza cantans	Experimental	Avian malaria[3]	Seropositive
Paroreomyza montana	Experimental	Avian malaria[4]	Seropositive
EXOTIC			
Carpodacus mexicanus	300–2400	Avian malaria[3]	11.6%
		Avian pox[2]	21.5%
Passer domesticus	300–2400	Avian malaria[3]	11.4%
		Avian pox[2]	7.4%
Cardinalis cardinalis	300–2400	Avian malaria[3]	2.2%
		Avian pox[2]	2.0%
Zosterops japonicus	300–2400	Avian malaria[3]	0.9%
		Avian pox[2]	2.2%
Lonchura punctulata	300–2400	Avian malaria[3]	2.5%
		Avian pox[2]	0.0%

Note: All studies were conducted on Mauna Lao Volcano, except that of *Chasiempis sandwichensis*, which was conducted on Mauna Kea.

Sources: 1, VanderWerf 2001; 2, van Riper et al. 2002; 3, van Riper et al. 1986; 4, Jarvi et al. 2001

[a]Avian pox = poxvirus avium; avian malaria = *Plasmodium relictum capistranoae*.

[b]IUCN Red List category (www.redlist.org). NL = no listing.

toms were consistent with avian pox and possibly other diseases. These observations are all consistent with the hypothesis that a disease swept through these populations. The epidemic affected many endemic bird species, perhaps because all these species were naïve to diseases common on continents.

TABLE 5.3 *(continued)*

Species	Morbidity	Mortality	Status[b]
NATIVE			
Chasiempis sandwichensis			Vulnerable
	70% deformity		
	9.1% lesions		
Hemignathus virens		66.0%	NL
		20.0%	
	10.6% lesions		
Vestiaria coccinea			Lower risk
	10.3% lesions		
Myadestes obscurus			Vulnerable
	20.3% lesions		
Himatione sanguinea			NL
	14.1% lesions		
Telespiza cantans		100.0%	Vulnerable
Paroreomyza montana		75.0%	Vulnerable
EXOTIC			
Carpodacus mexicanus			NL
	6.3% lesions		
Passer domesticus			NL
	2.5% lesions		
Cardinalis cardinalis			NL
Zosterops japonicus			NL
	1.4% lesions		
Lonchura punctulata			NL

The avian diseases in question probably arrived with domesticated chickens imported in 1901, which probably carried avian poxvirus, and with caged passerines, brought in the early 1920s, which came with avian malaria (van Riper and Scott 2001; van Riper et al. 2002). These infectious diseases had "visited" Hawaii for millennia in migratory birds, but without a vector, were not able to infect native birds. The vector was supplied in 1826 when the ship *Wellington* drained its dregs, releasing mosquito larvae from the west coast of Mexico into a stream near Lahaina. The mosquito *Culex quinquefasciatus* was soon ubiquitous throughout the Hawaiian Islands at elevations below 1650 m.

A series of experiments with Laysan finches (*Telespiza cantans*) showed how devastating these vectored diseases were in naïve birds. Laysan (along with Nihoa) is a low, upwind island. It is still mosquito-free and retains large populations of its native birds. Warner (1969) brought Laysan finches to Honolulu and established an experiment in which some birds were exposed to mosquitoes in an unscreened cage and others protected from exposure in a screened cage. In 15 days, all the unscreened birds had died; all the screened birds were alive. Further experiments showed that an exposure as brief as 3 days was lethal. Blood analysis revealed unusually high parasitaemias for both *Plasmodium* (two species) and a species of *Haemoproteus*.

The loss of native birds was greatest in the lowlands, in the wettest areas, and during the rainy season. The effects of this extinction event are evident from IUCN's (2002) species status reports. Limitations in mosquito distributions (particularly along elevation gradients) appear to have spared the extant native avifauna from extinction due to avian pox and avian malaria (van Riper et al. 1986). It should be noted that mosquitoes are now less abundant than in the past due to partially effective mosquito abatement practices.

Pathogens infect exotic birds on Hawaii as well, but their effect on those birds is relatively minimal. This difference is a probably a result of differential exposure to vectors and resistance to disease. For example, Warner (1969) showed that the number of mosquitoes feeding on Laysan finches was 5–10 times greater than on introduced white-eyes (*Zosterops palpebrosa*). The prevalence of avian pox and avian malaria, and consequent levels of morbidity and mortality, are all significantly higher in natives than in exotics (Table 5.3). Whereas five of the seven native species listed in Table 5.3 are classified as vulnerable or at risk, none of the exotic species achieve this ranking at a global scale, and all are purportedly in stable condition on the Hawaiian Islands (IUCN 2002).

In short, the evidence strongly supports vectored blood pathogens as the primary cause of the extinctions of Hawaiian endemic avifauna, and the restriction of remnant populations to high altitudes and upwind islands, that occurred in the twentieth century. The roles of other exotic pests and habitat alterations appear to have been relatively unimportant during this time.

The effects of avian pox and avian malaria on the Hawaiian avifauna illustrate several attributes of vector-transmitted diseases that might otherwise have remained elusive had the pathogens not been introduced. The first is that host species distributions can be altered by the distributions of vectors (which, in this case, have environmental tolerances far narrower than the host species). Second, in a community in which multiple hosts share a common parasite, it is possible for the parasite to facilitate host-specific extinctions, so long as other host reservoirs persist. In the case of Hawaiian birds, it appears that the exotic hosts of avian pox and avian malaria, in part because they are relatively unaffected (and thereby remain common and infected), act as a substantial source of infection for native hosts. Finally, the differential susceptibility of exotic and naïve native hosts illustrates how hosts can evolve defenses that limit impacts at the individual, population, and species level.

Community response to the introduction and eradication of African rinderpest

In 1889, the Italian army imported cattle carrying rinderpest virus (a morbillivirus related to measles) from India to the Horn of Africa. Within a year, the pathogen infected a number of native species (Spinage 2003). Spreading at a rate of 500 km per year, rinderpest epidemics caused mass mortality in domestic and wild artiodactyls from Egypt to South Africa (Plowright 1982). By the turn of the twentieth century, the African rinderpest epidemic had claimed about 90% of the East African domestic cattle population and about 95% of the Serengeti buffalo (*Syncerus caffer*) and wildebeest (*Connochaetes taurinus*) populations (Plowright 1982; Spinage 2003). During a rinderpest outbreak between 1959 and 1961, the annual mortality of first-year wildebeest increased significantly to about 85%, with predation accounting for 45% and rinderpest accounting for 40% of all deaths. Numerous other species also succumbed to the disease (Plowright 1982). Rinderpest continued to suppress domestic and wild artiodactyls until a vaccine was introduced and the virus was largely eradicated (Spinage 2003). Although only domestic cattle were vaccinated, the disease disappeared from the wild species, implying that cattle were, in fact, the main reservoir. This outcome demonstrates the importance of reservoir hosts in maintaining and facilitating the spread of infectious diseases to other species, an issue central to how diseases can affect rare species (Lafferty and Gerber 2002). After 1961, when clinical rinderpest was absent from the Serengeti, wildlife populations experienced rapid recovery (Plowright 1982; Spinage 2003). Within a decade, the wildebeest population increased from 260,000 to 700,000 individuals, and buffalo increased from 30,000 to more than 60,000 individuals (Plowright 1982). These increases were largely attributed to the reduction in juvenile mortality following rinderpest control (Plowright 1982). This response implies that rinderpest suppressed artiodactyl populations to densities far lower than the Serengeti habitat could support (Sinclair 1979; Dobson 1995).

Given this strong suppression of artiodactyl populations, it is perhaps not surprising that rinderpest control had corresponding effects on the population dynamics and trophic structure of the Serengeti ecosystem as a whole (Sinclair 1979; Plowright 1982; Dobson 1995; Tompkins et al. 2001). Increases in the abundance of ungulate species (following rinderpest control) led to an increase in the density of carnivores, particularly lions (*Panthera leo*) and hyenas (*Crocuta crocuta*). These increases in carnivore abundance were matched by decreases in the abundance of gazelles, most likely due to increased predation pressure. The most dramatic demographic change following rinderpest control occurred in wild dogs (*Lycaon pictus*), whose numbers declined from about 500 to eventual local extinction, a likely consequence of increased competition with recovering lion and hyena populations. Furthermore, the changes in the numbers of grazing artiodactyls would certainly have had an effect on plant biomass and composition. There is even evidence to suggest that the decline in browsers, particularly impala (*Aepyceros melampus*), during the first pandemic may have

allowed a large recruitment pulse in many tree species (Prins and Weyer-haeuser 1987). For example, the acacia stands in many parts of the ecosystem are remarkably even in their area and size distribution and appear to result from a narrow window of recruitment during grazer population minima. Col-lectively, the trophic cascades and other regulatory effects initiated by rinder-pest virus on the East African ecosystem suggest that certain viruses can play keystone roles in ecosystem functioning and structure.

Evolution of host specificity and host switching in distemper viruses

The devastating epidemics of another morbillivirus, canine distemper virus (CDV), in Serengeti predators (Table 5.4) provide additional insight into the effects of disease on population dynamics. Plowright (1982) recorded that when he developed the rinderpest vaccine in Nairobi, dead cattle were disposed of by supplying them to the local dog owners. Distemper effectively disappeared from the domestic dog population at this time! This observation implies that exposure to rinderpest in infected carcasses may have caused cross-immunity to distemper in canids. Therefore, it may be that loss of rinderpest from wilde-beest subsequently increased the susceptibility of carnivores to distemper.

Between 1984 and 1988, CDV prevalence in the Serengeti lion population declined to zero from about 75%, a value believed to reflect a previously unde-

TABLE 5.4   *Emergent epidemics of distemper viruses in novel species*

Species	Location	Prevalence (%)	Morbidity	Mortality[a]	Source
Baikal seal	Lake Baikal	71.4	100% lesions	18,000 total	Grachev et al. 1989; Ohashi et al. 2001
Caspian Sea	Caspian Sea	75.0–100.0	100% lesions	>10,000 total	Kennedy et al. 2000
Harbor seal[b]	Europe	47.0–100.0	Acute CDV symptoms	60% regional, >17,000 total	Osterhaus and Vedder 1988; Heide-Jorgensen et al. 1992
African wild dog	Masai Mara, Kenya	4.0–75.0	Decreased appetite, diarrhea, listlessness	21%–50% regional	Alexander and Appel 1994
Serengeti lion	Serengeti, Tanzania	85.5–99.0	> 94% lesions	30% total	Roelke-Parker et al. 1996; Haas et al. 1996
Spotted hyena	Serengeti, Tanzania	99.0	100% lesions	—	Haas et al. 1996

[a]Percentage or absolute mortality resulting from epidemics.
[b]Values reported for phocine distemper virus (PDV).

tected epidemic (Packer et al. 1999). In 1994, another epidemic occurred, infecting the majority of the population in less than 3 months and reducing fecundity and survival in all age groups (Table 5.4; Cleaveland et al. 2000). Interestingly, evidence suggests that the 1980s epidemic corresponded with only 70 susceptible individuals, whereas the 1994 epidemic did not occur until 100% of the population (250 individuals) was susceptible (Packer et al. 1999). This discrepancy implies that CDV existed in other species in the early 1990s, so that the pool of susceptible hosts in the ecosystem was far larger than it had been in the 1980s (Packer et al. 1999). Indeed, other Serengeti species, including spotted hyenas and wild dogs, also experienced high morbidity and mortality from CDV in 1994 (Table 5.4; Cleaveland et al. 2000). This cross-species transmission may have been facilitated by the severe drought conditions that plagued the Serengeti throughout 1993 and increased the probability of contact between domestic dogs (the purported source of the disease), lions, and spotted hyenas at waterholes and carcasses (Cleaveland et al. 2000).

Since 1988, CDV and related morbilliviruses, such as phocine distemper virus (PDV), have also emerged in a number of marine hosts (see Table 5.4). The majority of recent CDV and PDV epidemics are attributed to interspecific virus transfer between species that do not typically experience close contact. As in the Serengeti epidemic, there is speculation that domestic dogs are the source of morbillivirus in marine mammals. For example, the introduction of sled dogs to Antarctica in 1955 is believed to be responsible for a mass mortality event that was induced by CDV and caused a 97% decline in the local crabeater seal (*Lobodon carcinophagus*) population (Bengston and Boveng 1991).

In 1987–1988, an epidemic of PDV occurred for the first time in European harbor seals (*Phoca vitulina vitulina*) (Osterhaus and Vedder 1988). Several hypotheses have been proposed to explain the origin of the virus, including incipient contact with harp seals (*Phoca groenlandica*) and terrestrial canids (Heide-Jorgensen and Harkonen 1992). The virus infected more than 90% of the total European harbor seal population, causing about 60% mortality in most regions (see Table 5.4; Osterhaus and Vedder 1988; Heide-Jorgensen and Harkonen 1992). Population recovery was rapid following 1988, with a 6%–12% increase in population size per year at the epicenter in Danish waters and more than a fourfold increase between 1989 and 2000 in the Wadden Sea (Jensen et al. 2002). In 2002, when the harbor seal population exceeded pre-epidemic levels and the majority of individuals were again susceptible to PDV, another outbreak occurred. The 2002 epidemic mirrored the 1988 epidemic in timing, geography, rate of spread, morbidity, and mortality (Jensen et al. 2002). Recent theory, although controversial, predicts a 1%–18% risk that recurrent outbreaks will reduce the European harbor seal population by 90% (Harding et al. 2002; Lonergan and Harwood 2003).

It is evident from the emergence of distemper virus in European harbor seals and Serengeti carnivores that unusual contact events between typically segregated species have the potential to spread disease to novel species groups,

even across the land-sea interface. Such host switching can occur rapidly and with devastating impacts on the novel host.

Indirect effects of disease on competitive interactions in red squirrels and gray partridges

At the turn of the twentieth century, the gray squirrel (*Sciurus carolinensis*), a native of North America, was introduced to the United Kingdom as a wildlife novelty and, like many exotics, experienced a rapid range expansion (Middleton 1930). Since its introduction, the gray squirrel has "replaced" the native red squirrel (*S. vulgaris*) throughout much of its range, primarily through competition for food resources (MacKinnon 1978; Bryce 1997; O'Teangane et al. 2000). Evidence supporting this replacement is apparent in Table 5.5, which shows that carrying capacity, growth rate, reproductive rate, and competitive effect are all greater for the gray squirrel. However, recent theory demonstrates that parapoxvirus, which was introduced by the gray squirrel, may also be contributing to the decline in red squirrel abundance (Tompkins et al. 2003). Whereas gray squirrels are resistant to the effects of parapoxvirus, red squirrels experience considerable virus-induced mortality. Tompkins et al. (2003) theorized that competition-mediated replacement of local red squirrel populations by gray squirrels could occur within 15 years, but when the effects of parapoxvirus are incorporated, replacement time drops to only 6 years. Furthermore, without the effects of parapoxvirus, natural rates of competition alone cannot explain the decline in red squirrel populations or gray squirrel range expansion (Tompkins et al. 2003).

A similar effect is apparent in the wild gray partridge (*Perdix perdix*) in the United Kingdom. Although declines in gray partridge populations over the last 40 years have been largely attributed to changes in agricultural regimes (Potts 1986), there is evidence that released pheasants (*Phasianus colchicus*) may

TABLE 5.5   *Variation in life history parameters for red and gray squirrels*

Parameter	Red squirrel	Gray squirrel
Carrying capacity	12/km^2	16/km^2
Net growth rate	0.61/year	0.82/year
Maximum reproductive rate	1.0/year	1.2/year
Competitive effect[a]	0.61	1.65
Natural mortality rate	0.40/year	0.40/year
Mortality rate due to virus	0.26/year	Resistant

Source: From Tompkins et al. 2003.

Note: Red squirrels are native to the UK. Gray squirrels were introduced together with parapoxvirus, which has a negative impact on naïve red squirrel populations.

[a] Competitive effect of one squirrel species on the other.

also be a contributing factor. Pheasants appear to be the driving force behind the spread of a cecal nematode (*Heterakis gallinarum*) that induces morbidity in gray partridge populations, but not in the pheasants themselves (Tompkins et al. 2000). Theoretical research by Tompkins et al. (2000) predicts parasite-mediated competition between the species, whereby partridges are excluded from regions where they overlap with pheasants due to the negative effects of *H. gallinarum*.

These examples illustrate how infectious disease can mediate competitive interactions, usually to the disadvantage of the host that is naïve to the pathogen. Although these two examples suggest an advantage for introduced species, it is also reasonable to expect that introduced species will face challenges from the new diseases they encounter in the areas they invade (though, except for agricultural introductions, it is difficult to observe cases in which diseases prevent establishment of an invader).

Conclusions

Species introductions can provide considerable insight into the role of infectious diseases in nature. We can use host species introductions to understand the dynamics of populations and communities in the absence of disease (disease removal experiments). In this case, the introduction may be replicated spatially or temporally, and the control can be the introduced species in its native range (with its native parasites). Similarly, we can use pathogen introductions to understand the dynamics of populations and communities in the presence of disease (disease addition experiments). Here, the control can be the status of the host population before the disease invades or after the disease is eradicated (as in the case of rinderpest) or other host populations that have not been exposed to the pathogen. Despite the large number of possible comparisons to be made, most of our knowledge is anecdotal and nonsystematic. An exception is the biological control literature, in which careful comparisons are often made and conclusions are easier to reach. Unfortunately, biological control is typically carried out in simplified agricultural settings, making it difficult to extrapolate what the effects of natural enemies would be in natural settings (Hawkins et al. 1999).

In spite of the limitations that exist, disease additions and removals lead to several ecological and evolutionary generalizations. The first ecological generalization is that not all infectious disease organisms have dramatic effects on host populations. This is most obvious from studies of biological control programs, in which even parasites that are chosen for their high potential to affect the host population often fail at regulating the host to the extent that economic damage becomes insignificant. Second, some infectious diseases do have appreciable effects on hosts. By affecting host vital rates, they can cause host performance (body size, density, and biomass) to decline. Third, the densities of populations subject to disease can fall, sometimes to low levels, but typically

not to extinction. This pattern may be a result of reduced parasite transmission at low host densities in the absence of alternative hosts or of restricted environmental tolerances of disease vectors. Fourth, effects on hosts can be broad (and potentially lead to extinction) if the disease is able to infect several different species, which may occur if the presence of a novel host creates opportunities for host switching. When more than one host species is affected, rare hosts can be differentially affected because disease transmission does not decline as the density of the rare species drops. Fifth, if the host plays a keystone role in the community, the disease may have considerable indirect effects. These effects may take the form of trophic cascades, in which hosts unaffected by the disease (competitors or prey) may gain a competitive advantage. Sixth, if a pathogenic disease has a different geographic distribution from the host (due to the distribution of a vector, for example), the host distribution may shift away from areas where the risk of infection is high. Finally, release from infectious diseases (via long-distance dispersal and species introduction) may have substantial benefits for the host species with respect to population abundance, growth rates, and so forth. Several evolutionary insights arise as well. For instance, novel encounters between hosts and infectious disease agents probably rarely result in disease. When they do, naïve hosts may suffer substantially, but natural selection can rapidly select for host resistance. In addition, release from parasites may have played a role in speciation events by aiding the performance of recently isolated host species.

Although many of these insights are speculative and based on only one or a few examples, more systematic study of introduced species and infectious disease may help us ascertain the generalities of these insights. In particular, it remains to be determined (1) how frequently disease effects are demographically or ecologically important, (2) how important the effects of disease are relative to those of other factors, and (3) what types of hosts and ecosystems are most affected. Researchers have been able to uncover valuable insights into infectious disease processes through their study of introduced species thus far. In our opinion, however, these investigators have just scratched the surface of what is possible.

Acknowledgments

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