Infectious Disease Ecology

Effects of Ecosystems on Disease and of Disease on Ecosystems

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CHAPTER EIGHT

Invasion Biology and Parasitic Infections

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Summary

Parasitic infections can strongly affect invasion success and the impact of invasive species on native biota. A key mechanism facilitating invasion is escape from regulation by natural enemies—the enemy release hypothesis. The level and duration of release depend on the types of parasites lost and gained, with highly regulating acute infections most likely to be lost and, over time, pathogenic RNA viruses likely to be gained. The rate at which hosts accumulate parasites depends on multiple factors, including the biotic resistance of the community and the ecosystem changes induced by the invasive species themselves. We discuss several examples of how invasive species may increase parasite susceptibility of the community by increasing parasite reservoir densities or by altering parasite flow via apparent competition. We then consider the evolutionary implications on a longer time scale if the susceptibility of invasive species is enhanced by loss of parasite resistance. Finally, we discuss whether parasites should be considered a special class of invader and conclude by identifying approaches, challenges, and priorities for future research in parasite dynamics of introduced species.

Introduction

With an estimated cost of U.S. $120 billion in environmental damage and economic loss in the United States alone (Pimentel et al. 2005), invasive species and their ecosystem effects are a major focus of research in ecology (Elton 1958; Kolar and Lodge 2001; Shea and Chesson 2002). Invasive species are those that have been moved beyond natural dispersal barriers and introduced into habitat outside their native range in which they become established and prolific, often with substantial consequences for native biota, human health, and ecosystem functioning (Kolar and

Lodge 2001; Vitousek et al. 1997). Not all introduced species become invasive, and as such, a focus in invasion ecology has been to determine the mechanisms facilitating species invasions. One of the major hypotheses is that of enemy release, which posits that in their native range, populations are regulated by enemies, but these enemies are reduced in number or absent from populations outside their natural dispersal range, thereby allowing introduced species to escape regulatory forces and become invasive (Darwin 1859; Elton 1958; Hierro et al. 2005). The enemy release hypothesis tends to focus on predators as enemies, but recent research recognizes the role of parasites as enemies and has documented high loss of parasites from host species in invaded compared with native ranges (Mitchell and Power 2003; Torchin et al. 2003, Torchin and Mitchell 2004).

We start this chapter by reviewing the empirical evidence for enemy release with respect to parasites and posit which parasites are likely to be lost and gained and the implications this has for host invasion success. We further explore the intersection between the ecology of species invasions and parasitic infections by examining what effect the parasites of introduced species may have on native biota and the knock-on ecosystem effects. In addition, we address evolutionary aspects of introduced species and their parasite fauna. Finally, we consider parasites as invaders themselves. Our aim is a review of invasive species and their interactions with parasites and the impact this has for native biota, ecosystem functioning, evolution, and human health. Throughout this chapter, we refer to parasites and pathogens as simply "parasites" (see Hall et al., chapter 10, this volume, for definitions) unless explicitly stated otherwise, and "enemies" in this context refers specifically to parasites, rather than also including predators.

The Enemy Release Hypothesis

To cite the enemy release hypothesis as a mechanism for facilitating successful invasion of introduced species, a combination of both escape and release from parasites must be demonstrated. *Enemy escape* quantifies the extent to which the parasite diversity and prevalence of introduced species are reduced relative to those in the native range. European plants that become established in the United States are infected by, on average, 77% fewer pathogen species than in their native range (Mitchell and Power 2003), whereas introduced animals are, on average, infected by 53% fewer helminth species than in their native range (Torchin et al. 2003). For successful invasion, we must determine whether this documented escape translates into *release*, which would require that the par-

asites have measurable negative impacts on species abundance or fitness so that escape from them results in increased success of the invaders. From disease ecology studies, we know that parasites cause harm to and regulate their host populations in terms of altering host density, fecundity, or growth (Hudson et al. 2002; Tompkins and Begon 1999; Torchin et al. 2001). Bringing together both invasion and disease ecology, we find evidence for an increase in demographic parameters in introduced species that have been released from parasites (Lafferty et al. 2005). A well-studied example is that of the European green crab, which performed better in an invaded range than the average parasitized European population but similar to an uninfected European population (Torchin et al. 2001). More empirical examples are required, but challenges exist in documenting enemy release empirically with respect to the role of parasites, as opposed to other enemies.

One challenge in documenting enemy release is that unsuccessful invasions often go unnoticed and assessment must rely on comparisons of established populations or species in their native versus introduced ranges (but see Jeschke and Strayer 2005; Suarez et al. 2005). This requires comprehensive and rigorous sampling of parasites in both the native and introduced range. Furthermore, given the complex number of ecological processes that influence the success of invaders, how do we measure the importance of enemy release by parasites relative to predators and other variables that influence invasion success? The approach thus far has been to use common garden or reciprocal transplant experiments and biogeographical studies, which have yielded insights into the success of introduced species with specific reference to parasites. However, both approaches suffer from a number of experimental difficulties.

Biogeographical studies (e.g., Mitchell and Power 2003; Torchin et al. 2001, 2003) suffer from confounding variables that are difficult to control for and usually involve a small number of randomly chosen populations inhabiting a restricted area in the native range of the species (e.g., Edwards et al. 2003; Grigulis et al. 2001; Reinhart et al. 2003; but see Buckley et al. 2003). As a result, it is difficult to discern to what extent the results are highly specific to the study sites. Often when several populations are studied in the same area in the native range, important differences in the role of enemies appear (Callaway et al. 2004). However, in contrast, Reinhart et al. (2005) sampled black cherry populations widely from throughout their range in eastern North America and found a consistent negative effect of soil-borne pathogens on seedling survival. Common garden experiments have been less frequently applied with respect to elucidating the effect of parasites as enemies and have the issue of being potentially confounded by variation among species in traits such as resistance and tolerance.

With these issues in mind, we suggest that conclusive evidence of enemy release would require replicated experimental manipulations and descriptive studies of two types. The first would be removal experiments, where one could show for a specific parasite that removal from the host in its native range resulted in demographic release, that is, greater host fitness, an increase in population size, or a range expansion. Some evidence for this exists with macroparasites (Tompkins and Begon 1999), and although these experiments have not focused specifically on hosts that are invasive species, they do provide evidence of enemy release with respect to parasites. The second type of conclusive evidence would be addition experiments in which parasites, when introduced into invasive populations, resulted in lower fitness, decreased population growth, or range contraction of the invader. Such "experiments" are commonly done during biological control (Lafferty et al. 2005). However, the parasites used in biocontrol often are not native to the invasive species in their home range. For example, myxomavirus, endemic to South America, is used to control European rabbits invasive in Australia.

In summary, the challenge is to disentangle the relative importance of an array of interactions with parasites, other enemies, and environmental factors to which an invasive species is exposed in its introduced range as compared with those in its native range. Given the broad range of species that occur in natural communities and the diverse ways these interact with other biotic and abiotic elements of the environment, we might not expect simple pairwise host-parasite interactions, and the effect of a single enemy, in our case a parasite, may be diluted (Keane and Crawley 2002). More demographic data demonstrating strong population-level suppression by parasites is necessary to evaluate the ecological significance of parasites in the enemy release hypothesis. Given the potential limitations of experimental tests, we advocate a pluralistic approach to the study of invaders using a combination of molecular techniques, field surveys, experiments, and historical records to understand the role of parasites in facilitating species invasions.

Parasites Lost and Gained

Invasion success can be a function of escaping parasitic regulation during the different transition phases, which include transport, establishment, and spread (Jeschke and Strayer 2005; Kolar and Lodge 2001). Considering the variation in host regulation associated with different parasites an important question to ask is, which types of parasites might be lost and gained during invasion? For example, if highly regulatory

parasites are lost during host transport, then we may expect immediate demographic release and increases in fecundity and survival, thus facilitating invasion. Conversely, if regulatory parasites are gained, then we may expect reduced invasion success.

Parasites Lost

Based on current knowledge of the ecology of parasites and invasive species, we can make predictions concerning the ecological characteristics of parasites that will be lost (table 8.1). The first phase of invasion, transport, typically occurs with a subset of the host population, and this founder population is expected to be devoid of parasites that are rare, or found at low prevalence, in the host population in its native range (Torchin et al. 2003). If these rare parasites do not have great regulatory control over the host population, then demographic release will be minimal (Colautti et al. 2004).

TABLE 8.1
Parasites predicted to be lost and gained during the invasion process for plants and animals

	Animals	*Plants*
Lost	Ectoparasites and the vector- borne diseases that they transmit	Aphids/nematode vectors and the pathogens they transmit
Lost	Specialists	Specialists
Lost	Rare parasites, low prevalence	Rare parasites, low prevalence
Lost	Acute immunizing pathogens (large threshold and short infectious period)	Pathogens that cause high mortality
Lost	Complex life cycles (e.g., trophically transmitted macroparasites)	Parasites with obligate alternative hosts
Retained	Vertically transmitted parasites (rare in animals)	Seed-borne viruses and some fungal pathogens
Retained	Retroviruses	Retroviruses
Retained	Generalists	Generalists
Gained	RNA viruses	RNA viruses
Gained	Generalists	Generalists
Gained	Parasites and pathogens of phylogenetically similar host species	Parasites and pathogens of phylogenetically similar host species

Indeed, it is the common or prevalent macroparasites that tend to be regulatory (Hudson et al. 2002), but these too may be lost or reduced in intensity, because subsampling hosts from aggregated host-macroparasite distributions tends to sample individuals with low parasite intensity (Shaw et al. 1998). We may expect parasite loss to be accentuated further if the process of selecting invaders favors healthy individuals that are free of parasitic infection, for example functional groups within the population that have low exposure or have acquired immunity to infection.

Immature stages have different (often fewer) parasites than do adults. For example, sexually mature male rodents have been identified as key hosts in the persistence of tick-borne disease (Perkins et al. 2003). Generally, for vertebrate hosts the intensity of macroparasites is male biased (Moore and Wilson 2002); thus if a deliberate introduction favors females, then parasite diversity will be further reduced within the founder population. For plants, a key issue is whether transport occurs as a photosynthetic plant or as a dormant seed or spore, as the latter generally have a smaller subset of parasites than do adults (e.g., Molloy et al. 1997). Adult plants are likely to be transported in soil with a much greater likelihood that associated parasites of leaves, flowers, or roots are simultaneously transported (Keane and Crawley 2002). Invasive plants are more likely to escape from fungal than from viral pathogens (Mitchell and Power 2003), in part because fungal parasites tend to be more host specific, but also because viruses can often persist in seeds and be vertically transmitted (Torchin and Mitchell 2004). Therefore, the mode of parasite transmission has an impact on the likelihood of loss, and retroviruses and other endogenous or vertically transmitted parasites will be extremely difficult to escape from. However, in plants, many vertically transmitted microbes tend to be mutualistic and not parasitic, and so movement of seeds compared with whole plants is much more likely to result in enemy escape.

Intuitively, we expect the high mortality associated with host transport, coupled with parasitic infection, could cause host mortality to be amplified, particularly if the infection is acute (Moller 2005). Therefore, acute infections of high virulence and short infectious periods are likely to be lost, along with their hosts, in the transport stage. Acute infections, which include many microparasitic diseases, are often highly regulating, and release from them may partly explain the increased demographic capability of invaders (figure 8.1).

After transport, some parasites could be lost during establishment, in part due to thresholds for invasion. Parasites require a threshold population size to establish and persist (Lloyd-Smith et al. 2005). However, founder populations of invading species typically fluctuate around

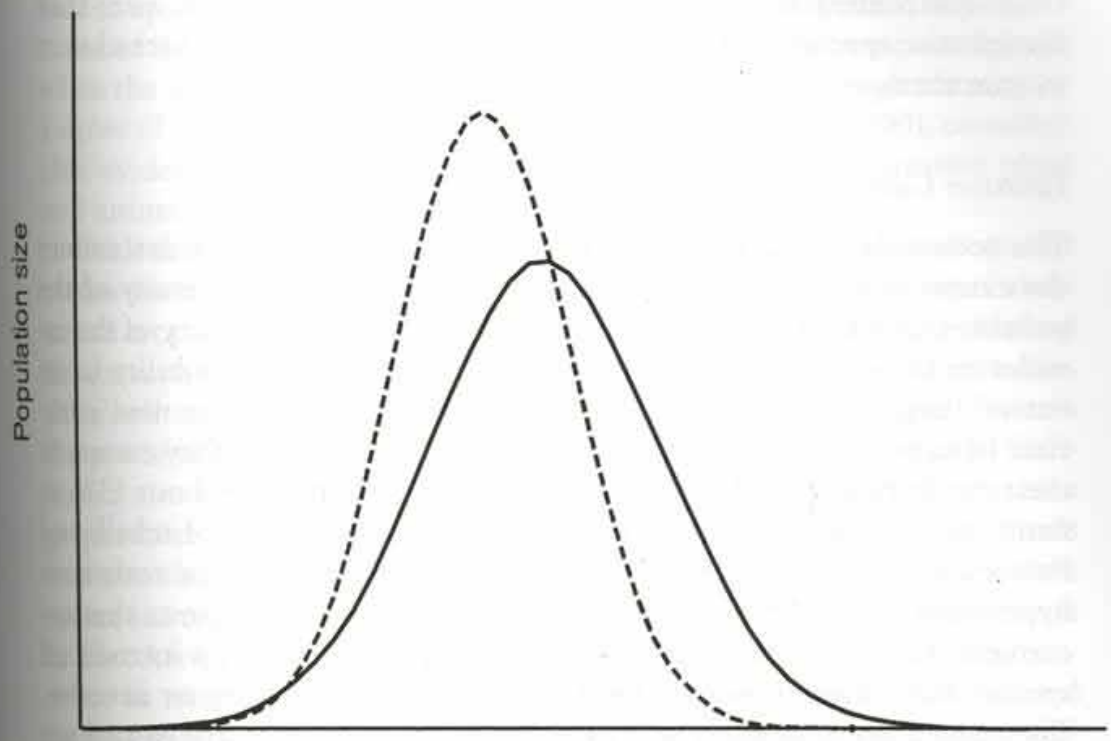

Figure 8.1. A schematic of the change in size over time of an introduced population that experiences parasite release and so increased growth during invasion, followed by parasite regulation coupled with a reduction in population size. Two comparable potential outcomes are shown. The solid line represents moderate parasite escape and release, followed by moderate biotic resistance of the invaded community; the dashed line represents high parasite escape and release, followed by high biotic resistance, or rapid accumulation of regulatory parasites.

relatively small numbers, and therefore parasites cannot establish and persist before the invader reaches population densities that would support endemicity (e.g., de Castro and Bolker 2005).

For hosts with complex life cycles, appropriate alternative hosts may not be present in the invaded range, precluding parasite establishment. Ectoparasites and other vectors also may be lost, since those with short feeding periods and seasonality in host biting behavior (e.g., ticks in temperate zones) are unlikely to be transported with the introduced hosts. Consequently, vector-borne pathogens will be especially vulnerable to loss. Additionally, if the abiotic conditions in the new habitat are not suitable for free-living parasitic stages, then they will be lost during establishment. Indeed, any form of complex life cycle parasite or specialist requirements suggests that specialist parasites will be lost over generalists (Cornell and Hawkins 1993; Kennedy and Bush 1994). The majority of parasites are lost during introduction and establishment.

Once established and spreading or increasing in population size, then the invasive species are no longer likely to lose parasites but instead start to gain them.

Parasites Gained

The accumulation of parasites during the invasion process determines the extent of net release, which in turn depends on the diversity of the parasite community in the invaded range and the susceptibility of the invader to these parasites, which may differ from its susceptibility in its native range. On average, animal invaders accumulate four new parasites from the invaded range, less than a third as many as they escape in their native range (Torchin et al. 2003). Plants accumulate about 13% as many new fungal and viral pathogens as they escape (Mitchell and Power 2003). This accumulation process is key to the biotic resistance hypothesis (Elton 1958; Maron and Vila 2001), which suggests that accumulation of enemies (both parasites and predators) by an introduced species may often prevent them from becoming damaging or invasive. The rate of accumulation and the type of parasites that are gained are likely to have profound effects on whether the species can become established or invasive and the time period over which an invasive species may remain so.

During the establishment phase, parasites from local communities can start to colonize the introduced hosts. Many introduced species might disappear before they are even noticed due to high parasite pressure, particularly if the local community exhibits high biotic resistance (Elton 1958). The phylogenetic similarity between introduced species and resident native species may determine the likelihood of colonization by preadapted parasites. An introduced species from an unrepresented genus or family should have a lower probability of parasite accumulation than an introduced species with many close relatives. However, close relatives might also indicate greater suitability of local environments for the introduced species (Mack 1996).

Encounter with potential natural enemies will increase as the invader expands its range. Parasites with broad host ranges and little host or vector specificity should be more likely to colonize than highly specific parasites (see table 8.1). Examples include RNA viruses, parasites with generalist vectors, or other generalist parasites. If there are no preadapted or generalist pathogens, the probability of de novo pathogen evolution by random mutation will increase as the local abundance of the introduced species increases (e.g., Antia and Koella 2004). Thus, the more successful an invasive species is, the greater the chance that a novel virulent pathogen will arise. Once a pathogen establishes, it may rapidly increase and

lead to epidemics and major die-offs in the introduced species, in part because of the high density of susceptible hosts. This may be especially true when the introduced species is genetically uniform. Although natural examples of this are few (but see Hochachka and Dhondt 2000), considerable evidence is available from the biological control of invasive plant and animal species (e.g., Fenner and Fantini 1999).

Evidence for the accumulation of parasites by introduced species comes from many sources: well-documented cases of infection of nonnative crop species and domesticated animals (Scheffer 2003), published databases and disease indices (Farr et al. 1989), and comparative studies (Mitchell and Power 2003; Torchin et al. 2003). For example, Pierce's disease of the grape, which caused several devastating epidemics in California vineyards, is caused by a bacterial pathogen common in native grapes from the southeastern United States. Fire blight of pome orchards was also thought to have arisen from native pathogens adapted to native rosaceous trees (Scheffer 2003). Similar examples exist in animals: cattle in East Africa succumb to sleeping sickness caused by trypanosome parasites in native ungulates, and introduced house finches in the United States are attacked by a native pathogen causing mycoplasma conjunctivitis (Dhondt et al. 2005).

Intuitively, release from natural enemies may be temporary, as introduced species gradually accumulate resident parasites (see table 8.1). Parasites from the invader's native range may eventually colonize introduced host populations through repeated introductions or long-distance or human-assisted dispersal. Colonization leads to accumulated gains over time of parasites and corresponding declines in invasive species abundance, and the extent and timing of the decline are a function of the likelihood of acquiring regulatory parasites (see figure 8.1). Therefore, a key question is, will parasite communities of introduced populations ever "catch up" to those of the native populations, and if so, how long does this take, and will the acquired community of parasites limit the fitness of the host species as effectively as in their native range?

For some species, accumulation of parasites can be a relatively rapid process leading to no differences in parasite diversity or infection levels between native and introduced hosts, and so host regulation is also expected to be rapid. For example, Clay (1995) found that introduced grasses in the United States actually supported more fungal pathogens than native grasses, although introduced species also had larger geographical ranges, which was the primary predictor of pathogen load. Strong and Levin (1975) examined species richness of parasitic fungi of British trees and found that there was no difference in fungal species richness between introduced and native trees when corrected for range.

They suggested that fungal species richness rapidly saturates over ecological time (i.e., over several hundred years). However, the agents with the greatest potential to have long-term control may not necessarily be the ones with the most dramatic immediate effects. Thus, for example, floral smuts that reduce fecundity are predicted to have an increasingly detrimental effect as host longevity increases, while for pathogens that affect mortality, causing intermediate levels of mortality will reduce population sizes significantly more in the long term, than pathogens that are more destructive (Thrall and Burdon 2004). Data from agricultural experiment stations or forestry plantations might prove useful, given the intensive records of plant introductions. Longer-term historical records may provide some solace in light of the disruption caused by recently introduced species, many of which have become problematic only in the past 20–30 years. Introduced species may cause much ecological damage in the meantime but nevertheless offer many opportunities for examining the longer-term dynamics of disease.

Effects of Introduced Species on Parasite Dynamics in Native Hosts

Introduced species, regardless of the pathogens they lose or gain, can have large indirect impacts on native hosts by changing the characteristics of the ecosystem, by amplifying disease or vector populations, or by changing the spatial distribution of hosts.

Ecosystem-Level Changes

Introduced species can affect ecosystem properties, such as productivity, nutrient status, water balance, physical structure, and disturbance regime (e.g., Cox 1999; Dukes and Mooney 2004; Mack et al. 2000; Eviner and Likens, chapter 12, this volume). Modification of ecosystem functions change the rules of the game for every other species in the community (National Research Council 2002), which ultimately may alter host-parasite dynamics. The consequences of ecosystem alterations by introduced species on the dynamics of native parasites seem little studied, but we can suggest some plausible possibilities based on well-studied introduced organisms. Introduced earthworms in eastern North America destroy the forest litter layer, changing the water and nutrient content of the upper soil layers (Hendrix and Bohlen 2002), thereby potentially altering conditions for transmission and survival of soil-borne plant pathogens. Another example is that of zebra mussels, widely introduced in Europe and North America, which produce nutrient-rich

(Roditi et al. 1997) and presumably microanaerobic conditions in their beds. This may favor anaerobic bacteria such as *Clostridium* and contribute to the recent rise in Type E botulism in Lake Erie (New York Sea Grant 2003). Finally, introduced grasses change the frequency of fires (D'Antonio and Vitousek 1992), which ought to affect the timing and severity of disease caused by plant pathogens. Similarly, parasite outbreaks in native coniferous forests make them more prone to devastating wildfires.

Amplification of Reservoir Host Populations

Invasive species may affect parasite dynamics by amplifying disease or vector populations. For example, in the highly invaded California grasslands, introduced annual grasses attract and amplify the fecundity of cereal aphids that vector barley and cereal yellow dwarf viruses (B/CYDVs). The presence of these introduced grasses has been found to more than double the incidence of barley and cereal yellow dwarf infection in nearby native bunchgrasses (Malmstrom, McCullough, et al. 2005). Because B/CYDV infection can stunt bunchgrasses and increase their mortality (Malmstrom, Hughes, et al. 2005), these findings indicate that virus-mediated apparent competition has the capacity to influence interactions between native and introduced species and contribute to the decline of the natives. In contrast, introduced tall fescue grass is widely distributed and commonly endophyte infected, and is more resistant to aphids as a result (Siegel 1990). Resistance to aphids has the knock-on effect of reduced aphid-borne virus pressure on neighboring native grasses. A good example of an introduced domestic species that has enlarged the pathogen reservoir population comes from the Serengeti, where domestic cattle increased rinderpest prevalence in wild ungulates, a result discovered when cattle vaccination indirectly lowered rinderpest incidence in wild hosts (Sinclair 1979).

Another mechanism by which introduced species can enlarge the reservoir population is by serving as alternative hosts for parasites with a multihost life history strategy. For example, the fungus *Cronartium ribicola*, which causes the devastating white pine blister rust, requires two hosts—one *Pinus*, one *Ribes*—to complete its life cycle. In the United States, the presence of cultivated and escaped *Ribes* (currants and gooseberries) has such a strong influence on white pine blister rust incidence in nearby pines that horticultural use of *Ribes* is severely restricted (Maloy 1997). Similarly, pheasants introduced into the United Kingdom have been shown to successfully feed ticks and support Lyme bacterium transmission, thus contributing to the reservoir host population for Lyme disease (Kurtenbach et al. 1998).

Changes to Host Spatial Distribution or Density

Native host spatial distribution, density, and host contact structure within and among populations and communities can be altered by species introductions. For example, impacts of invasive weeds on soil communities could alter ecosystem functions, with secondary impacts on native host abundances (Kourtev et al. 2002). Thus, changes in parasite dynamics are expected partly through indirect impacts on host population structure, including total host abundance or density, and the movements of hosts or vectors in the environment. One obvious way that introduced host species can have an impact on native community structure is through population explosions. Such invasions may lead to net increases in species diversity, although a decrease is usually more common. A decrease in species diversity can lead to an upsurge in parasite levels primarily by concentrating infection within the most competent reservoir hosts, an amplification effect (Mitchell et al. 2002, 2003; see also Clay et al., chapter 7, and Begon, chapter 7, this volume). Interestingly, as shown by Mitchell and colleagues, it is not just parasite abundance and disease prevalence that may change but also the relative prevalence of particular types of pathogens—that is, the parasite community. The converse of this observation is the basis for the use of varietal mixtures in cereal crops, where parasite levels may be significantly reduced by the construction of random three-component mixtures of susceptible and resistant varieties (Wolfe 1985). Other possible effects of changes in community composition following invasions include shifts in patterns of herbivory or predation, which if they increase or reduce a reservoir host population might further alter disease dynamics.

An intriguing but little considered impact of an invasive species is the potential collateral impact the invading species's parasites may have on the native species's own suite of host-specific parasites (e.g., Torchin et al. 2005). Although this may be minimal when the native species is still reasonably abundant, in the case of introduced pathogens like chestnut blight, *Cryphonectria parasitica*, or root rot, *Phytophthora cinnamomi*, that have devastated the entire population of their host species (chestnut, *Castanea dentata*, and Brown's banksia, *Banksia brownii*), any host-specific native parasites must have undergone a devastating (and undocumented!) decline if not extinction.

Evolutionary Considerations

A range of evolutionary processes should affect the vulnerability of introduced species to parasite accumulation. First, most invasive popula-

tions are likely to originate from a few founders, thus limiting their genetic diversity and increasing their susceptibility to parasites (Sakai et al. 2001). Second, introduced species that are released from parasites might reallocate resources away from parasite defense and into growth and reproduction (Colautti et al. 2004; Wolfe et al. 2004), thereby potentially allowing the species to become invasive (Siemann and Rogers 2001; Tilman 1999). Together, these ideas suggest that low overall genetic diversity combined with evolutionary reductions in parasite defense should make introduced species vulnerable targets for future epidemics (DeWalt et al. 2004, Knevel et al. 2004; Reinhart et al. 2003).

Population Bottlenecks and Parasite Susceptibility

Genetic variation tends to be reduced in introduced populations, particularly if colonists come from a single source population or undergo an establishment phase during which population sizes remain small (Sakai et al. 2001). Founder events resulting in extreme genetic drift and inbreeding could lower the fitness of introduced populations and limit their ability to adapt to future challenges (Lee 2002). Host populations characterized by loss of allelic diversity or reduced heterozygosity may be unable to respond evolutionarily to new threats imposed by parasites (Lande 1988; Lyles and Dobson 1993). This issue is more commonly raised in the context of agricultural systems (e.g., Elton 1958) or species of conservation concern (e.g., Acevedo-Whitehouse et al. 2003; Thorne and Williams 1988). However, similar problems could apply to populations of introduced species with low genetic diversity, as has been suggested for the unusually high susceptibility of introduced house finches in eastern North America to ongoing outbreaks of mycoplasmal conjunctivitis (Dhondt et al. 2005).

Yet, despite the fact that many introduced species probably show reduced genetic diversity in their invaded versus native ranges, this has yet to be widely established. A recent comparison of the loss of allelic diversity and heterozygosity in twenty-nine introduced animal species found that, on average, there is little reduction in genetic diversity (Wares et al. 2005). In plants, there may even be greater genetic variation in introduced populations than in populations where they are native (Novack and Mack 2005). In some cases, repeated introductions from multiple native sites could actually cause blending of alleles from different geographic locations in the new habitat, leading to greater genetic variation, rather than less, in the introduced range (as has been demonstrated with brown anole lizards; Kolbe et al. 2004). Hybridization in the new range could also lead to hosts with novel gene combinations that are highly resistant to parasite infections, and such genetically variable populations

could serve as problematic sources of introduction for other vulnerable locations (Sakai et al. 2001).

Evolution of Increased Competitive Ability

Invasive species often are larger, more abundant, and more vigorous in their introduced range relative to their native range (Crawley 1987; Grosholz and Ruiz 2003). One explanation for this observation is that following release from their natural enemies, introduced species experience increased growth and reproduction. A related idea, known as the EICA (evolution of increased competitive ability) hypothesis, states that because defenses are often costly and organisms have limited resources, introduced species should adapt to the loss of natural enemies by allocating more energy to growth and reproduction and investing less in pathogen resistance or immune defense (Blossey and Nötzold 1995). This hypothesis predicts that in the native range, growth and reproduction should be lower, natural enemies should be common, and investment in defenses high, whereas in the new range, natural enemies should be less common or absent, defenses should be low, and growth and reproduction should be greater (Wolfe et al. 2004). Furthermore, these phenotypic differences should be genetically based, and parasites and other natural enemies should preferentially attack the invasive phenotypes, two predictions that can be tested using comprehensive common garden and reciprocal transplant experiments. Some recent studies provide support for genetic divergence in enemy defense and reproductive strategies between native and introduced populations of weeds and trees (Siemann and Rogers 2001; Wolfe et al. 2004). Under this scenario, if pathogens are lost from introduced populations, the frequency of resistance should decline over evolutionary time scales, potentially setting the stage for future disease outbreaks.

The EICA hypothesis assumes that resistance is costly—and further depends on whether resistance traits are targeted against generalist or specialist enemies (Joshi and Vrieling 2005). Many studies have demonstrated that resistance-conferring host traits are in fact costly in terms of reductions in growth rates, fecundity, competitive ability, or body size (Simms and Rausher 1987). Invasive species might lose protection against specialist parasites, since these are most likely to be lost, and instead shift resources into defenses against generalist parasites (Joshi and Vrieling 2005), which are likely to be gained in the invaded range.

Importantly, species invasions offer new opportunities to understand the strength of parasites as agents of selection, particularly with respect to the evolution and maintenance of host defenses in the wild (Altizer et al. 2003). Field monitoring studies, reciprocal transplant experiments,

and common garden studies of introduced species from locations in the invaded and native range provide prime opportunities for researchers to measure host investment in parasite defenses relative to growth and reproduction, and to compare populations exposed to different levels of attack by a range of specialist and generalist parasites. At the present time, comprehensive studies of the biology of invasive species in both their native and introduced ranges are surprisingly rare (but see Reinhart et al. 2003, 2005 for a counterexample of detailed studies of parasite regulation of black cherry in its native and invasive ranges), despite the potential insights that can be gained from such comparisons. Furthermore, as has been demonstrated by a growing number of "virgin ground" epidemics, emerging pathogens often cause high case fatality rates and stunning reductions in host abundance (reviewed in Daszak et al. 2000). If these epidemics can be buffered by the genetic composition of host populations, then studies of disease outbreaks in populations of introduced species will provide new perspectives on the role of host genetic diversity and investment in immune defense in the outcomes of host-parasite interactions.

Parasite Evolution in Introduced Species

We have reviewed how invasion may be facilitated because parasites, particularly those causing acute infectious diseases, will be lost in transit. An important area for the future ought to focus on the evolutionary adaptive processes of parasites in both invading and native species in the community in the invaded range. Disease-causing pathogens generally have short generation times and high replication rates, and hence a great ability for fast evolution (Frank 2002). The literature on virulence-transmission trade-offs and the rapid evolution of the myxomatosis-causing DNA virus of rabbits provides an excellent testimony (Fenner 1983). Another interesting example is the extreme rate of evolution in the form of both gene loss and genomic organization of *Bordetella pertussis*—the whooping cough–causing bacterium—following its emergence in humans from its ancestral commensal of various mammals (Bjornstad and Harvill 2005; Parkhill et al. 2003). Over and above that, disease-causing RNA viruses may have even higher evolutionary rates because of the high mutation rates during RNA transcription (Grenfell et al. 2004).

There are at least four reasons why studies focused on the postinvasion evolution of parasites might be particularly interesting. The first is the great evolutionary potential of many parasites. The second reason is that the effects of a parasite on the host population depend critically on its virulence (Anderson and May 1978) and infectious period (Grenfell

2001), so that evolutionary changes in these two parameters can drive ecological change at the population level. The third and fourth reasons are that selective gradients for changes in virulence and infectious periods appear to be particularly steep when (3) genetic diversity within the host population is low and (4) contact networks are altered. In terms of contact networks, transmission rates will likely change whenever host densities are higher *or* lower in the invaded range than in the native habitats range, at least for directly transmitted pathogens. Changes in contact rates will generally alter the optimal pathogen strategy because of virulence-transmission trade-offs (Ebert 1998; Frank 2002). The introduction of a species may be enough to alter the contact network of a species. Indeed, several recent theoretical studies have shown that subtle changes in social networks can greatly alter evolution toward enhanced or diminished virulence (Boots et al. 2004; Read and Keeling 2003). The impact of any parasite that *does* survive the invasion process on the introduced host populations' growth and regulation may therefore rapidly diverge from the effects of the same pathogen in the host's native range.

Parasites as Introduced Species

Finally, it is important to keep in mind that parasites themselves can be a special class of introduced species (see also Hudson et al., chapter 16, this volume). Research questions in this area focus on the intersection of two important predictive frameworks: factors that affect the success of introduced species and factors that govern emerging diseases. Introduced parasites will represent a subset of both of these groups.

The introduction of parasites with invading hosts is the most important and widespread driver of disease emergence worldwide (Anderson et al. 2004; Bauer 1991; Daszak et al. 2000; Dobson and May 1986). Such introduced diseases can have a devastating impact on immunologically naïve host populations, often with enormous consequences for human health, the economy, and wildlife conservation. Diseases of humans and wildlife have been traded across the globe with increasing frequency for centuries; common examples of relevance to human health include smallpox, typhus, yellow fever, cholera, schistosomiasis, SARS, West Nile virus, HIV, and influenza. These same patterns are evident in animals and plants. Select examples for animals include salmonid whirling disease, chronic wasting disease, rinderpest, shrimp whitespot disease, crayfish plague, avian malaria, avian cholera, and duck plague. Common examples for plants include wooly hemlock adelgid, dogwood anthracnose, beech bark disease, white pine blister rust, oak wilt, and numerous others.

Unfortunately, even basic epidemiological information is lacking for most pathogens, native or introduced. For example, Taylor et al. (2001) reported that the basic transmission mode was unknown for more than 200 human pathogens, precluding any comparative analyses of the basic reproduction number, R_0. Information on host use, native geographic range, propagule pressure, and failed invasions is correspondingly lacking for many plant and animal pathogens. As such, attempts to analyze introduced pathogens through stages of the invasion process, while very promising, and to develop more quantitative predictions have lagged behind recent efforts with free-living groups, including fish, plants, and birds (see Kolar and Lodge 2001, 2002).

Recent reviews of disease emergence in humans (Taylor et al. 2001), vertebrate wildlife (Dobson and Foufopoulos 2001), and plants (Anderson et al. 2004) offer important first steps to understanding how disease emergence intersects with stages of the invasion process. Do these different stages represent different sorts of barriers for parasites than for other invasive species? Clearly, arrival doesn't lead to establishment for many introduced species (e.g., failed introductions for biocontrol). Is the probability of getting from one stage of the invasion process different for parasites than for plants and animals? What features promote success at each stage? Do plant parasites possess life-history features that make them more or less likely to invade than animal parasites? For example, wind-blown spores may be capable of dispersal over thousands of kilometers (Brown and Hovmoller 2002). Comparative studies could provide a useful approach to answering some of these questions, and are likely to provide further insights into factors influencing the invasion process.

The arrival of parasites into a novel environment is dependent on the invading host population, and we have posited which parasites are most likely to be present in that population. However, important questions remain, including the following: Are parasites a special class of invaders, or do they share features common to other invasive species, such as a generalist host range and simple life cycle? And are the routes by which parasites arrive different in some qualitative way from the ways in which other invaders arrive, so that the probability of successful invasion might also different?

Different types of investigative or applied approaches are more likely to be useful at different stages of an invasion process. For example, the earliest stages of invasion following arrival may be most amenable to eradication, especially if there are sufficiently effective quarantine and monitoring programs in place. Although it is difficult to envisage using experimental manipulations to study early processes associated with parasite arrival, the use of high-resolution molecular markers to study

within-host variation or to trace pathways of infection back to sources can be effective. As parasites become established and spread into multiple host populations, and eradication becomes less likely, the applied emphasis may shift from eradication to long-term control. Population and metapopulation modeling approaches become more valuable: one can begin to study general dynamical properties of the system and to test predictions empirically. Developing an understanding of the underlying patterns of host resistance and pathogen infectivity and aggressiveness as part of evolutionary studies (which may include modeling, population genetic, and phylogenetic approaches, as well as experimental studies) will be critical for explaining underlying patterns of disease incidence and prevalence. The lack of empirical evidence is particularly surprising in light of the potential for such variation to affect not only disease dynamics and prevalence, but also when or where new diseases emerge (e.g., canine parvovirus; Parrish 1999). Increasingly, this lack of knowledge has led to calls for an integrated approach to disease management that would incorporate both ecological and evolutionary processes. Addressing such questions will be essential if we are to develop a predictive understanding of diseases as invaders.

Conclusions

Questions concerning the role of parasites in the success of species invasions bring together two rapidly developing research fields, the ecology of species invasions and the population biology of infectious diseases. Recent years have seen an increase in the number of quantitative studies concerning the ecology of invasive species (reviewed in Colautti et al. 2004; Kolar and Lodge 2001), as well as factors determining the spread and impacts of parasites and the infectious diseases they cause in natural systems (reviewed in Hudson et al. 2002). A growing number of studies point to the role of parasites in regulating hosts and driving population dynamics, such that their removal could in part explain increased growth, abundance, and size of exotic organisms in novel habitats. As such, there exists an experimental niche in terms of bringing these disciplines together with regard to specifically assessing the role of parasites in the enemy release hypothesis. However, confounding variables create difficulties for examining empirical evidence of the role that parasites alone, compared with other enemies, play in facilitating or inhibiting the invasion process. Most studies of assessing enemy release thus far have taken comparative approaches, with release of biological control agents providing an alternative set of opportunities to assess the impacts of different parasites on host regulation.

Given the potential limitations of experimental tests of enemy release in introduced populations, we advocate a synergistic approach to study the role of parasites in invasion biology using a combination of molecular techniques, field surveys, experiments, historical records, and multivariate models. Molecular techniques can be used to test for evidence of multiple introductions in the introduced range as well as for genetic variation in both the native and introduced ranges (e.g., founder effects in the introduced range). Field surveys help quantify both enemy pressure and geographic variation in size and fitness of the invader across the native and introduced ranges. Historical records can provide further information on both the scope and timing of the introduction and the number of potential introduction events. Multivariate models that predict the success of invaders (at species or population level) based on overlap between native and introduced range, time since introduction, parasite burden, population density, climate, and existing genetic variation will provide a more comprehensive review of the importance of parasite release in determining invader success. Not only is there a need for further experimental approaches, such as manipulation of hosts and parasites in both natural and invaded range, but there is also a need to incorporate developing areas of disease ecology, such as interactions of parasite species within hosts.

Complementary to determining the role of parasites in enemy release is determining which types of parasites would be lost and gained during invasion. In this chapter we have speculated that highly regulatory parasites are most likely to be lost, but they could also be rapidly gained, especially RNA viruses. Therefore the rate at which parasite accumulations occur will determine the time period under which the invasive host population becomes regulated by parasites (see figure 8.1). We speculate that this time frame may be hundreds of years, but it may be accelerated by high propagule pressure, which in turn will depend on the level of biotic resistance of the invaded community. This could be high for invaded communities that contain phylogenetically close species, or low for species that invade phylogenetically distant communities, for example, deliberate introductions to distant and remote islands.

Owing to the potentially long time period over which accumulation of parasites and thus regulation of invasive species occur, it is essential to take into account evolutionary theory. This suggests that, at first, resource allocation away from costly parasite defense should enhance invasive species growth rates, but that founder effects and loss of resistance should increase host susceptibility to parasitic infection in the long term.

Careful consideration should be given to the changes in ecosystem functioning that invasive species may cause. Ultimately these indirect changes can affect host-parasite dynamics not just of the invading species but also of the native host community and its parasite fauna. For instance,

invasive species can increase the reservoir host density, facilitating the persistence of some parasites or increasing transmission through parasite-mediated competition. Indirect effects brought about by invasions could form the focus of future research, for example changes in native reservoir host density though trophic interactions with invasive species. An overall focus for future work is to move beyond case studies, speculation, and inference-based retrospective studies to develop a general understanding of key processes and patterns that reflect the interface between infectious diseases and biology of species invasions. As global trade and travel increase, the number of accidental and deliberate introductions is expected to rise, providing further impetus to elucidate the role of parasites and pathogens in host regulation and the importance this has for invasive species and ecosystem functioning.

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