CHAPTER NINE
Effects of Disease on Community Interactions and Food Web Structure

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SUMMARY

INFECTIOUS DISEASES CAN BE POWERFUL forces in natural populations. When diseases affect influential species, the consequences of disease can ramify through communities. For instance, parasites can reverse the outcome of competition and, therefore, alter biodiversity. They may aid or buffer against biological invasions. Parasites permeate food webs and may change communities by altering predator-prey interactions. In particular, they may alter trophic cascades. Infectious human diseases probably limited our influence on the environment in the past. Humans' continued escape from disease through history has contributed to our having the largest influence over natural communities of any species.

INTRODUCTION

This chapter considers how parasitism might affect interacting species. The possibilities are many and involve entire food webs. Before branching out into the complexities of communities, it is worth considering the wide range of effects that parasites have on individuals (In this chapter, I usually use the term parasite to refer to agents of infectious disease). Many parasites do so little that they rate only as an inconvenience. Others have subtle but noticeable effects on host growth and competitive ability, perhaps through host investment in immune function, reduced attractiveness, slowed growth, or increased susceptibility to predators or stress. Sometimes the effect on the host depends on the parasite's life history, of which there are several discrete types (Lafferty and Kuris 2002). Most parasites kill the host at their own peril. However, some, like parasitoids, can be deadly, and this helps them transfer to new hosts. Others, like trematodes in snails, may have no discernible effect on longevity but block reproduction. Because this chapter focuses on the effects of parasites on communities, I consider only those parasites that greatly affect host populations.

For host-specific parasites (and many parasites evolve host specificity), effects at the host population level are a function of the effects of parasitism on infected individuals and the pattern of spread between infected and uninfected individuals. Transmission requires that an infected individual or infective stage contact an uninfected host. The more uninfected hosts that are available, the more likely it is that a parasite will pass its offspring to a new host before the parasite dies, where death of a parasite is usually a result of host defense, host death, or, for free-living stages, too much time waiting in a hostile environment. If a parasite does invade a host population, it will spread until the contact rate between infected and susceptible hosts drops because the epidemic runs short of susceptible hosts and infected hosts die or become immune. This means abundant species are more susceptible to infectious disease. Density-dependent transmission is a recurring theme of this chapter. Parasites that affect host populations are the ones most likely to have impacts at the ecosystem level, so long as the hosts they affect play important roles in an ecosystem (see Collinge et al., chapter 6, this volume). For example, parasites can interact with food webs when they affect species involved in trophic cascades. The next several sections of this chapter take a food web approach to understanding how disease can indirectly affect communities by altering species interactions.

COMPETITION

There are many nonexclusive explanations for how some communities can support many species, including the tendency for overlapping species to diverge in their resource use. It is more difficult to explain the co-existence of similar species because most simple models predict that competitive dominants will always exclude subordinate species. Three factors can help prevent competitive exclusion: indiscriminant disturbance at intermediate levels (Connell 1978), rare species advantage (Roughgarden and Feldman 1975), and impacts to competitive dominants (Paine 1966). Parasites help promote diversity if they differentially affect dominant or common species. Parasites may also reduce biological diversity (particularly as measured by heterogeneity) if they differentially affect subordinate species or lead to apparent competition. Given the increased homogenization of biotic communities through species introductions, it is also worth considering how parasites might help or hinder the invasion of competitive dominants (or generalist predators) that could reduce biodiversity.

Density-dependent transmission allows parasites to disproportionately affect common species (figure 9.1a). This helps maintain rarer
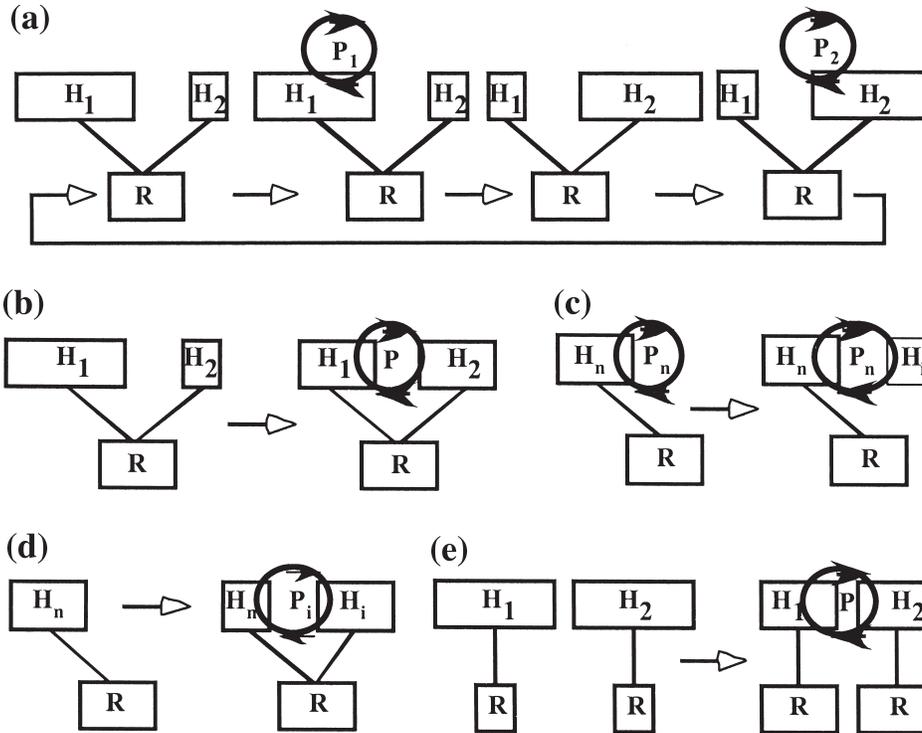
Figure 9.1. Interactions between infectious disease and competition. In this and subsequent figures, rectangles represent free-living species, with the volume of the rectangle proportional to the abundance of the species. Lines from one species to another represent a trophic link of a consumer (above) to a resource (below). Basal resources are indicated as R. Some free-living species are hosts (H) for parasites (P). Open horizontal arrows represent a comparison between two states (often uninfected vs. infected). (a) The parasite attacks an abundant species, releasing a rare species from competition, but another parasite attacks the released species when it becomes abundant. This prevents either species from becoming overwhelmingly common. (b) A shared parasite interferes with competitive dominance, allowing a subordinate species to succeed in areas where the parasite occurs. (c) A native species's (Hn) parasites (Pn) impede an invader (Hi). (d) A parasite (Pi) of an invader (Hi) disproportionately affects a native competitor (Hn), aiding the establishment of the invader. (e) Apparent competition through a shared parasite between two normally noncompeting species (H1 and H2).

competitors, thereby promoting coexistence and stability (Dobson et al. 2005). For example, natural enemies may maintain forest tree diversity (Wright 2002). This can occur if fungal pathogens disproportionately affect seedlings near conspecifics (Augspurger 1984; Packer and Clay 2000). (Clay et al., in chapter 7, this volume, discuss the idea that parasites beget host diversity in more detail.) The above scenario largely assumes parasites are host specific. When hosts share parasites, parasites can be competitive weapons. If subordinate species are tolerant or resistant to infection, parasites could help maintain them in a community (figure 9.1b). In a classic experiment, Park (1948) found that one flour beetle species (*Tribolium castaneum*) could competitively exclude another (*T. confusum*). A sporozoan parasite infects both species but has a bigger impact on the dominant species, reversing the outcome of competitive exclusion. In a more recent study, the fruit fly *Drosophila melanogaster* consistently outcompeted *D. simulans* in vials. Adding a parasitoid to the vial that slightly preferred the dominant *D. melanogaster* allowed the two species to coexist (Bouletreau et al. 1991). Similarly, in two separate studies of different species assemblages, competing amphipods may coexist in nature because a trematode reverses their relative population growth rates (Jensen et al. 1998; Thomas et al. 1995). A natural enemy might evolve to prefer competitive dominants if these dominants are more common and, therefore, the parasite encounters them more frequently. Still, parasite-mediated competition can differentially reduce subordinate species; a larval tapeworm shared by Park's flour beetles increased the rate at which the dominant beetle excluded the subordinate beetle (Yan et al. 1998).

Parasites could tip the balance in competitive interactions between native and introduced species (see also Perkins et al., chapter 8, this volume). On average, an invasive animal species has sixteen recorded parasite species in its home range but brings only three of these to invaded regions, where it picks up an additional four parasite species (Torchin et al. 2003). Leaving parasites behind could give invaders an advantage over natives saddled with a full parasite burden (Torchin and Mitchell 2004). Alternatively, if the invader has no coevolved history with the few new parasites it acquires, it might lack specific defenses, and infection could limit the invasion (figure 9.1c). For example, domestic cattle are very sensitive to the tsetse fly–transmitted trypanosome that causes sleeping sickness. This prevents their introduction to large parts of Africa, where cattle herding would likely exclude native grazers. Similarly, a meningeal nematode of white-tailed deer is highly pathogenic to other cervids and prevents their establishment in whitetail areas (Anderson 1972). We know that most invasions fail, and parasite defense could be one reason.

Of those three parasites that an average invader brings, some likely serve as handicaps, but others could serve as weapons (figure 9.1d). In the United Kingdom, the introduced grey squirrel competes with the native red squirrel. The grey squirrel is a good competitor for food on its own (Bryce 1997; MacKinnon 1978; O'Teangane et al. 2000), but it is aided by the parapoxvirus (Tompkins et al. 2003). This parapoxvirus is a relatively benign disease of grey squirrels. As grey squirrels expand into new habitat, they bring their pathogen along. Red squirrels are naïve hosts and suffer higher pathology. The parapoxvirus can persist even as red squirrels become rare because the more tolerant grey squirrels serve as a reservoir for the virus. A parallel situation occurs when caecal nematodes (*Heterakis gallinarum*) aid introduced pheasants (*Phasianus colchicus*) in their competition with grey partridges (*Perdix perdix*). Here, the effects of competitive exclusion center even more on the effect of the parasite on the native species (Tompkins et al. 2000).

Parasites can cause two species to interact indirectly even if these species do not compete for resources. This is known as apparent competition (figure 9.1e). Holt (1977) formalized this concept, and many derivations are possible. Apparent competition occurs because one host (the more tolerant or resistant) helps maintain the abundance of a natural enemy, which then differentially affects the second species.

Multiple hosts can also affect parasitism (see Power and Flecker, chapter 4, this volume). The predominance of host specificity suggests that host diversity will beget parasite diversity (Hechinger and Lafferty 2005). In other words, communities rich in hosts should also be rich in parasites. However, host diversity can also dilute transmission of a particular infectious agent if some hosts are infected but not suitable (see Begon, chapter 1, this volume).

Predation

Between 1846 and 1850, a pathogenic fungus transformed the human ecosystem of Ireland (Donnelly 2001). The resulting Irish potato famine serves as an allegory for how parasites can compete with predators for common food resources and alter entire communities. The adoption of agricultural monocultures increases plant density while reducing species and genetic diversity (Wolfe 2000). This provides a disease with a dense and uniform population through which to spread, conditions that are also conducive to the evolution of high virulence. In the 1800s, Irish peasant farmers produced cash crops (meat, dairy, grain) to pay rent to British landowners. They fed themselves on potatoes. This strategy allowed the population to nearly double in forty years, with two million acres planted

(a)

(b)

(c)

(d)

(e)
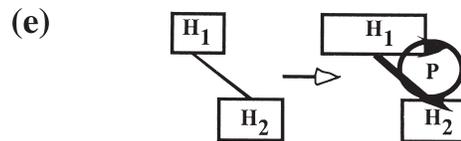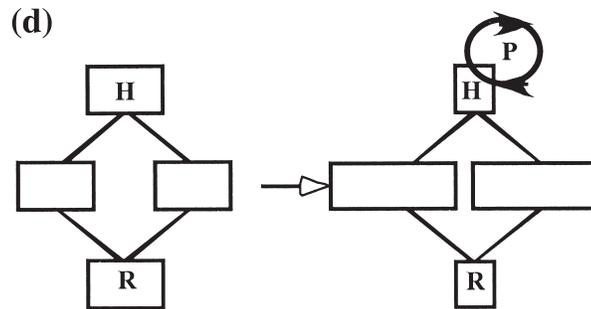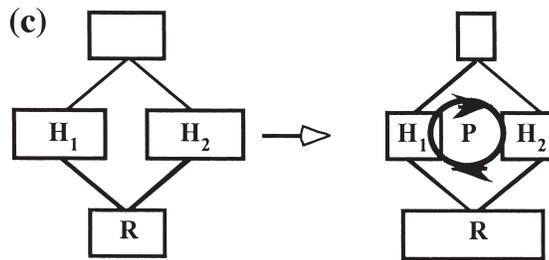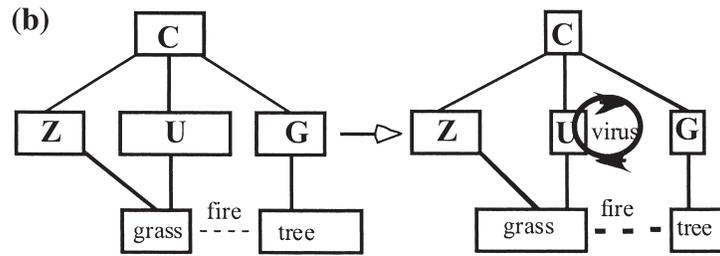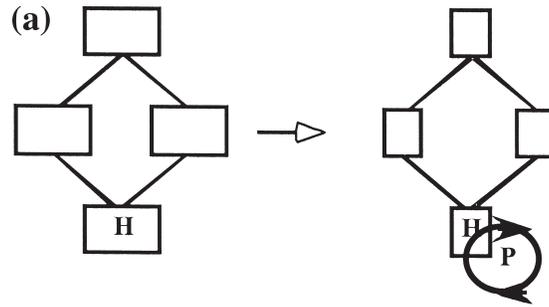
in potatoes. In 1845, a warm, wet winter favored fungal spread, and blight left potatoes rotting in the fields. Many peasants starved, succumbed to disease, or immigrated to North America. This illustrated how when disease affects lower trophic levels, bottom-up effects may cascade through a web (figure 9.2a).

A similar example is the loss of the American chestnut tree to blight. This tree was a dominant species, and chestnuts were an important source of food for wildlife in eastern deciduous forests. This was particularly true for the massive flocks of passenger pigeons (Schorger 1955). In the 1900s, an introduced fungus led to the gradual and near extirpation of chestnuts over 90 million acres of forest, eliminating an important food resource for animals. Although the impacts of chestnut blight on wildlife were not well quantified, seven species of moth that were specific to chestnut went extinct (Opler 1978).

If disease affects species in the midtrophic level, effects may propagate up, down, and sideways through the food web. For example, myxomavirus was introduced to England and Australia with the realized hope of releasing sheep from competition with rabbits (Fenner and Ratcliffe 1965; Minchella and Scott 1991). Rinderpest (a morbillivirus related to measles) in East African ungulates also illustrates how a parasite can alter food webs (Dobson 1995; Plowright 1982; Sinclair 1979; Tompkins et al. 2001). The rinderpest epidemics of the 1800s caused mass mortality in domestic and wild artiodactyls throughout Africa (figure 9.2b). This change indirectly reduced top predators such as lions and hyenas and altered vegetation structure via grazing and fire (Plowright 1982). A vaccine was introduced into cattle, and by 1961, the ecosystem had experienced rapid recovery (Plowright 1982; Spinage 2003), including an increase in predators such as lions and hyenas, and decreases in some prey, such as gazelles, and competitors, such as wild dogs (Dublin et al. 1990; McNaughton 1992).

Figure 9.2. Interactions between infectious disease and predation. (a) Comparison of the effect of a parasite (P) on a basal taxon. The disease reduces the plant population, depleting resources for species at higher trophic levels. (b) Serengeti food web before and after rinderpest (circle). Boxes with letters indicate carnivores (C), zebra (Z), ungulates (U), and giraffe (G). (c) Comparison of the effect of a parasite on a midlevel consumer. The disease reduces the grazer population, depleting resources for species at higher trophic levels and releasing basal taxa from grazing. (d) Effect of a parasite on a predator. Reduction in predator density releases prey and affects basal taxa through a trophic cascade. (e) Effect of a parasite that cycles between predator and prey and makes prey easier for the predator to catch. Predators increase in abundance and prey decrease (many other indirect effects are possible).

Sea urchins are herbivores that can exert an enormous effect on plant communities (figure 9.2c). On tropical reefs, sea urchins, along with other grazers, help minimize the standing stock of algae. This allows invertebrates, such as corals, to dominate and form reefs. In the 1980s, an apparently infectious disease swept through sea urchin populations in the Caribbean. The near extirpation of sea urchins, coupled with over-fishing of herbivorous fishes, allowed algae to grow up and choke coral reefs, adding to the worldwide decline in this ecosystem (Lessios 1988). At higher latitudes, where kelps create cathedral-like forests filled with fishes, people value algae more and urchins less. Overfishing of sea urchin predators (e.g., sea otters, spiny lobsters) increases the density of sea urchins, which then reduce kelp forests to "barrens" (Lafferty 2004). But high densities of urchins in these barrens promote epidemics of bacterial disease that can reduce urchin densities and push barren reefs back toward kelp forests (Behrens and Lafferty 2004).

Disease also affects top predators (figure 9.2d). In the temperate reefs of California, parasites of the southern sea otter are primarily from land (*Toxoplasma gondii* from cats, acanthocephalan worms from shorebirds, and terrestrial fungus) and do not have otter-to-otter transmission. Mortality rates from these parasites are high, and high mortality appears to be a main reason that otters have failed to expand their range to the south (Lafferty and Gerber 2002), releasing sea urchins from an important predator through much of the otter's former range. In a better studied example, a Scandinavian outbreak of sarcoptic mange (caused by mites) in the late 1970s through the 1980s reduced the density of red foxes. Prey (rodents, rabbits, ground birds, deer) increased as a result and then declined after the epidemic waned and fox populations recovered (Lindstrom et al. 1994).

The fate of most parasites is tied to that of their hosts. If their hosts die, this is usually a bad thing for host and parasite alike (see Holt et al., chapter 15, this volume). In nature, hosts get eaten. This puts tremendous evolutionary pressure on parasites to survive the ingestion process by establishing in the predator (Lafferty 1999). Perhaps as a result, many parasitic species have complex life cycles in which a final host must eat an intermediate host. In such life cycles, the parasite must wait for the ingestion of the intermediate host by final hosts. However, not all parasites are patient. Some parasites manipulate the behavior or appearance of the intermediate host to increase the rate at which a predator host will catch and eat it (Moore 2002). For instance, in southern California estuaries, the most common trematode, *Euhaplorchis californiensis*, encysts on the brain of killifish; the worms alter the fish's behavior, making it shimmy and swim to the surface. These fish are ten to thirty times more likely to be eaten by birds, the final host of the worm (Lafferty and Morris 1996).

In this system, the worm essentially dictates which fishes live and die. They also provide an easy snack for egrets and herons, which otherwise might have to work harder for a living. Some mathematical models indicate that such parasite-increased trophic transmission can reduce prey density; it can also increase predator density so long as the energetic costs of parasitism for the predator are not too severe (figure 9.2e) (Lafferty 1992). Other mathematical models suggest that predators may depend on parasites to supply them with easy prey (Freedman 1990).

PARASITISM

Parasites can interact with each other. Some parasites have parasites, and some compete for host resources with other parasite species. For larval trematodes, competition for resources within the snail is intense, and trematodes have special morphological and behavioral adaptations for interspecific interactions. For example, adding dominant trematode species to ponds can exclude subordinate trematode species (Lafferty 2002; Lie and Ow-Yang 1973; Nassi et al. 1979). In this example, the subordinate species are pathogenic to humans, and a consequence of this parasite-parasite interaction is improved human health (figure 9.3). Parasites can interact with the host, often via the immune system, to displace other parasites or alter their pathogenic effects on the host in various directions (Cox 2001). In shrimp, infection with one virus can reduce the effect of a second (Hedrick et al. 1994; Tang et al. 2003). In mosquitoes, filarial worms increase susceptibility to equine encephalitis virus (Vaughan et al. 1999) but decrease the development of malaria parasites (Albuquerque and Ham 1995). Despite all the potential for parasite-parasite interactions, few studies have considered what this means at the community level. Interactions between two morbilliviruses, rinderpest and canine distemper virus, are one possible example
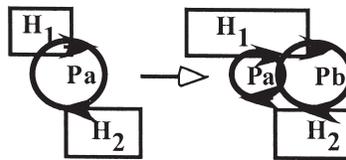

Figure 9.3 Interactions among infectious diseases. The effect of a nonvirulent parasite (Pb) on a virulent parasite (Pb) is modeled. Here, the nonvirulent parasite releases the final host from pathology through competition with the virulent parasite in the intermediate host.

of how two closely related pathogens can interact to alter communities. Anecdotal evidence suggests that carnivores develop some immunity to distemper when they feed on prey with rinderpest (Plowright 1968). This might explain the disappearance of canine distemper during the rinderpest epidemic and its resurgence in recent years (Dobson and Hudson 1986; Roelke-Parker et al. 1996). Outbreaks of distemper virus have led to periodic crashes in top predators in East Africa, with subsequent benefits for lower trophic levels.

Mutualism/Facilitation

Sometimes, by altering their host, parasites can alter communities dependent on these hosts or their actions. In one case, such manipulations can have dramatic and unexpected consequences for communities. The trematode *Curtuteria australis* reduces the ability of cockles to bury into New Zealand mudflats (perhaps this increases an infected clam's vulnerability to predation by final host birds) (Thomas et al. 1998). The shells of infected clams stick up out of the mud and provide a hard substrate for sessile invertebrates, such as limpets, that otherwise could not persist in the soft sediment (figure 9.4). Parasites can affect substrate-forming species as well, shifting communities in the opposite direction. For instance, trematodes reduce populations of a tube-building corophiid amphipod, thereby destabilizing the sediment and altering the faunal composition of a Danish mudflat (Mouritsen and Poulin 2002).

Food Web Topology

Food webs form a conceptual framework for the study of ecology, and substantial theory has developed on how the topological structure of food webs alters the flow of energy through an ecosystem and the resilience of
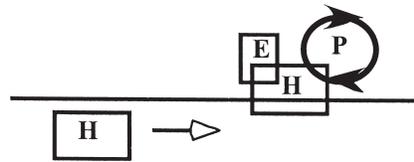

Figure 9.4 Effects of infectious disease on ecosystem engineers. The parasite makes the host sit above the surface of the sediment, where it is colonized by an epibiont (E) requiring hard substrate (which normally could not live in the habitat).

a community to change (or stability) that might occur following the addition of a new species or extirpation of an existing species. Few topological food webs (with the exception of parasitoid webs) have included parasites (Cohen et al. 1993; Polis 1991). This is because it is difficult to obtain good quantitative information on parasites in natural communities. Still, many ecologists acknowledge the potential importance of parasites in food webs and advocate their inclusion (Cohen et al. 1993; Marcogliese 2003; Marcogliese and Cone 1997; Polis 1991).

We can make some basic predictions about the effect of parasites on food webs. Parasites add links and species to food webs. This has the potential to change the linkage density or connectance of a food web. Connectance may alter the stability of a food web because it describes how strongly species are interconnected, helping to predict whether species additions or deletions will greatly alter other species. For example, Memmott et al. (2000) found that adding parasitoids to a food web decreased connectance, because most parasitoids interacted with one or a few hosts. Typical parasites are less restricted in their host ranges than are parasitoids, and we can expect that parasites that interact with many hosts will make a web more highly connected. For this reason, typical parasites are unlikely to act like parasitoids or predators in food webs. Parasites, as consumers of consumers, will tend to lengthen food-chain length (Huxham et al. 1995). In this way, they are very much like predators or parasitoids.

Existing insight into the role of typical parasites comes from estuarine food webs (see review in Sukhdeo and Hernandez 2004). In the Ythan Estuary food web, the addition of parasites slightly decreases connectance (Huxham et al. 1995). Adding parasites to a food web for a New Zealand mudflat yielded similar results (Thompson et al. 2005). By looking at the effect of each parasite species, this study found that most parasites only mildly decrease connectance. One generalist trematode, however, strongly increased connectance.

Most attempts to add parasites to food webs have considered that they operate similar to top predators. However, parasites differ from predators in several ways, the most notable being their intimate association with their prey and their relatively low biomass. Sukhdeo and Hernandez (2004) quantified the biomass of acanthocephalans in a food chain and compared this with predictions for predators. They found that the acanthocephalan population had the biomass expected from a top predator species, but that, because individual body size is very small, their abundance was much higher than expected for a top predator. Huxam et al. (1995) realized that parasites were not equivalent to top predators and predicted that including the full range of parasite links would add to connectance of food webs.

Lafferty et al. (2006) incorporated parasites into the Carpinteria salt marsh food web by using subwebs. This food web includes (1) a predator-prey subweb (this is what constitutes most published food webs) and (2) a host-parasite subweb (corresponding to previously published food webs with parasites included). In addition, a third subweb contains links where predators eat parasites. As mentioned previously, this happens in most predator-prey interactions because prey animals often have parasites in or on them. Sometimes the parasites are digested, but in a third of the links in the Carpinteria salt marsh food web, parasites can use the predator as a host. In addition, many parasites have free-living stages that may be fed on. The predator-parasite subweb contains the highest linkage density of all the subwebs in the Carpinteria Salt Marsh food web. A fourth subweb, parasite-parasite, completes the 2×2 matrix of subwebs. Including all four subwebs, connectance is three times higher with parasites than without parasites. Therefore, parasites have opposite effects on food web connectance than hitherto appreciated.

In the Carpinteria Salt Marsh food web, parasites have twice the number of hosts as predators have prey. Although top predators have few natural enemies in the predator-prey subweb, they are disproportionately attacked by parasites. For this reason, consumers at mid-trophic levels have the most natural enemies because they have a substantial number of predators and parasites compared with lower trophic levels, which have relatively few parasites, and upper trophic levels, which have few predators.

Although relatively little is known about parasites in food webs, the studies published to date indicate that parasites are likely to be worth including. They may make up most links in a food web and, at least for generalist species, may be more densely linked in webs than predators. In the Carpinteria Salt Marsh, two-thirds of the links occur in the parasite subwebs. It would seem that no food web is complete without parasites.

HUMANS

For parasites to affect communities, or even ecosystems, the parasite must alter the abundance of a host that plays an important role in the community. No host fits this better than humans. Parasites probably still affect human population densities, particularly in tropical regions. Historically, infectious diseases were a greater source of human mortality than combat. For instance, in the Spanish-American War, 10% of Spanish troop deaths occurred in battle, while 90% occurred as a result of malaria, dysentery, and other diseases (Cardona 1998). As humans

spread out of Africa, they escaped some parasites and inadvertently used others as weapons against human competitors. When Native Americans colonized the New World, they found a Shangri La of abundant wildlife and, presumably, few infectious diseases; their explosive spread and growth contributed to dramatic impacts on the faunal composition of the Americas (Flannery 2001). In turn, the number of Native Americans killed by the conquistadors' muskets and swords is miniscule compared with the number killed by infectious diseases of European origin (Diamond 1997). Epidemics in native populations presumably aided European colonization and deforestation of the New World. Modern pharmaceuticals and medical science have greatly decreased the impact of infectious disease, increasing the ability of humans to dominate the globe. We are now undisputedly Earth's dominant species in our consumption, distribution, and effects on biotic and abiotic conditions. Our escape from parasites has greatly fostered this outcome.

Conclusions

Because parasites are common, it is worth considering them alongside other, more obvious consumers. The consideration of infectious agents in food webs is increasingly under way. This will allow a more complete appreciation of food webs and better inform how consumer-resource interactions affect food web stability and the evolution of consumer strategies.

There are several approaches for considering these effects, and they mirror what has been used to decipher the effects of consumer interactions in food webs. The best place to start is to identify species that have disproportionate roles in food webs—foundation species, keystone predators, and the like—and then consider which parasites might alter their population dynamics.

Additionally, one can use the food web modules presented in the figures to develop mathematical models of the population dynamics of indirect effects. Many of these exist specifically for infectious diseases (see Holt, chapter 15, this volume). A unified modeling framework applied to all the modules in the figures would permit a comparative analysis. To date, most modeling efforts use microparasite models, because these are the most likely to yield analytical solutions. To broaden the value obtained from theory, it would be worth while attempting to model the entire range of consumer strategies and then compare their effects (Lafferty and Kuris 2002). This would help determine whether infectious disease is just another type of consumer-resource interaction (see chapter 10, this volume). Obvious points of entry into understanding the differences

between parasitism and other consumer strategies are the tendency toward host specialization by infectious agents and the coevolutionary responses (e.g., immune systems) specifically evolved to combat infectious pathogens.

Experimental manipulation of parasites to determine their effects on ecosystems is difficult, but the few attempts to do so have led to important insights on host population dynamics. Manipulations of parasites to investigate indirect effects have mostly been confined to laboratory investigations of apparent competition. However, because introduced species can bring parasites with them or leave them behind (see Perkins et al., chapter 8, this volume), species introductions serve as unintended experiments and have led to considerable insight into the role of infectious diseases in ecosystems (Lafferty et al. 2005). The example of the introduction of rinderpest to the Serengeti illustrates best how an infectious pathogen can dramatically change an ecosystem. We are just at the beginning of an exciting and challenging quest to uncover the role of parasites in community interactions and food webs.

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