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The Role of Infectious Diseases in Marine Communities

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Marine ecologists recognize that infectious diseases play an important role in ocean ecosystems. This role may have increased in some host taxa over time (Ward and Lafferty 2004). We begin this chapter by introducing infectious agents and their relationships with their hosts in marine systems. We then put infectious disease agents in the perspective of marine biodiversity and discuss the various factors that affect parasites. Specifically, we introduce some basic epidemiological concepts, including the effects of stress and free-living diversity on parasites. Following this, we give brief consideration to communities of parasites within their hosts, particularly as these can lead to general insights into community ecology. We also give examples of how infectious diseases affect host populations, scaling up to marine communities. Finally, we present examples of marine infectious diseases that impair conservation and fisheries.

An Introduction to Infectious Diseases

There are many types of infectious organisms in the ocean, and many are unfamiliar to most ecologists. We begin this section by describing their trophic strategies (i.e., what is a parasite?), discussing parasite life cycles, and giving an overview of the major taxonomic groups that include parasites. We consider the number of parasite species per free-living species, with examples of the diversity of bacteriophages and parasites of fishes, and what determines the abundance of parasites in ecosystems.

Trophic Strategies

Infectious diseases are caused by infectious agents. These infectious agents are called parasites, and they differ in trophic strategy from predators in that they attack just one resource during a particular parasitic life stage. They differ in trophic strategy from decomposers by attacking living resources. Parasites have a diversity of trophic strategies, and we use these strategies to help understand their contribution to community ecology. An ecological classification scheme for infectious agents considers aspects of the consumer–resource interaction (**Figure 5.1**; Lafferty and Kuris 2002). For instance, pathogens (microparasites) build up in numbers from a single infection event, whereas typical parasites (macroparasites) recruit to a host and grow, but generally do not multiply asexually. Then there are parasitoids (like most bacteriophages) that kill the host as part of their development, parasitic castrators that prevent their host from reproducing, and trophically transmitted parasites that can move to the next stage of the life cycle only when a predator host eats a prey host.

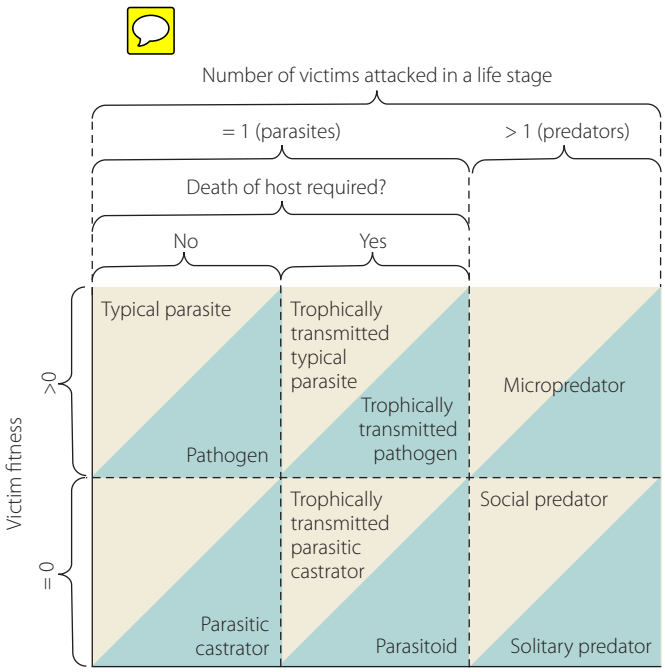


Figure 5.1 Seven types of parasitism and three types of predation separated by four life history dichotomies. Intensity-dependent relationships are above the diagonal line; intensity-independent ones are below. This results in ten trophic strategies of natural enemies. Note that the three predation strategies combine to four types of predators: micropredators, facultative micropredators, solitary predators, and facultative social predators. Victim fitness = 0 indicates that the interaction either kills the victim or blocks victim reproduction, whereas victim fitness > 0 indicates that the victim typically survives the interaction and can reproduce. (After Lafferty and Kuris 2002.)

Life cycles

To complete its life cycle, a parasite must recruit to a new host. After that host, the next life stage of the parasite might use a different host species. For such multi-host life cycles, the host in which reproduction occurs is called the definitive (or final) host, and the host in which larval forms occur is called the intermediate host. Transmission between hosts occurs via three main routes:

FREE-LIVING STAGE Many parasites have an infective stage that exits the host through a wound or orifice. Once outside the host, these infective stages are often nonfeeding and can be short-lived (like a virus) or persist in a dormant state (like a nematode egg). To get around, they can be passive (like an egg) or active (like a swimming larva). Such infective stages can be transmitted directly between hosts. However, direct contact is not as common a mode of infection in the ocean as it is on land, perhaps because the aquatic environment is more amenable to free-living stages (McCallum et al. 2004). Free-living stages can spread through the water, where they simply contact and penetrate host skin or are accidentally ingested. Alternatively, viral, bacterial, protozoan, and metazoan parasites (like copepods and monogeneans) can use a free-living stage to transmit between hosts.

VECTOR Some parasites require a vector to move the infective stage from host to host. Such infective stages usually circulate in the host's blood and are taken as a blood meal by a biting leech, fish louse, or isopod. In the vector, the parasite might develop further and often must move from the gut of the vector into the salivary glands or other organs near the mouth of the vector. When the vector bites a new host, the infective stages can be transferred during the blood meal. Examples of ~~marine vectors~~ include some viruses, filarial worms, and trypanosomes of fishes.

TROPHIC TRANSMISSION Some parasites have a larval stage in a prey host (often in the tissues) and become reproductive in a predator host (often in the gut). Predator–prey transmission is a common way for cestodes, nematodes, and trematodes to reach a final host. These parasites can sometimes use a series of intermediate hosts.

Taxonomic groups

Parasitism has evolved in many lineages, sometimes more than once. Here is a brief list of how parasitism is distributed from viruses to vertebrates. All viruses and phages are parasitic. Many bacteria are parasitic, including ~~many~~ that are opportunistic (normally free-living, but able to be parasitic). A few dinoflagellates are parasitic, and some can cause disease in fishes and invertebrates. The Apicomplexa are entirely parasitic and always have an intracellular stage. The ciliated Protozoa are mostly a free-living group with a few parasitic members. The amoebae are a diverse group of primarily free-living species. Some free-living amoebae can cause secondary infections in wounds, and a few are obligatory parasites. Like amoebae, slime molds can be opportunistic parasites, primarily infecting plant groups. Most fungal infections in marine organisms are also opportunistic. However, microsporidians are a parasitic group of fungi. None of the animals we call rotifers are parasitic, but an entirely parasitic clade of the rotifers is the Acanthocephala (until recently thought to be its own phylum). Some cnidarian species are parasites of other cnidarians and the eggs of fish, and there is a derived parasitic clade of the Cnidaria, called the Myxozoa, that parasitize fishes. Most of the flatworms are parasitic, with three fully parasitic groups: the monogeneans, trematodes, and cestodes. In addition, the Turbellaria are a large group of flatworms with a few parasitic members. Most nematode species are free-living, but parasitism evolved independently several times in this group. Few molluscs are parasitic. The most common parasitic molluscs are eulimid snails that parasitize echinoderms. Most marine annelids and arthropods are not parasitic, but parasitism occurs in some crustacean groups, such as the isopods, barnacles, and the copepod-like branchiurans. Almost no chordates are parasitic. However, pearlfish of the genus

Encheliophis live in the cloaca of sea cucumbers and eat the host's gonads (Parmentier and Vandewalle 2005). The echinoderms are the only major group of marine animals with no known parasitic members.

Parasites as a Part of Marine Biodiversity

Biodiversity has a positive connotation for most people. Coral reefs, with myriad colorful fishes and invertebrates, are a prime example. Although parasites are less visible and less appealing, they are a part of biodiversity. Overall, 40% of known metazoan species are parasitic (Rohde 2005). But a lack of sampling for parasites means that 40% is probably an underestimate. For instance, of the 110 intertidal decapod crustaceans on the U.S. west coast, only 15% have been investigated for more than one type of parasite species, and 68% have not had even minimal examination for parasites (Kuris 2007). Among well-studied land animals, there are, on average, eight helminth (wormlike) species per mammal species and nine helminth species per bird species (along with an untabulated number of described parasites from other taxonomic groups; Dobson et al. 2008). Humans, the best-known hosts, have more than a hundred host-specific (having only one host species per parasite stage) metazoan parasites that are common (Kuris 2012). The ocean is one big playground for parasites.

Marine bacteriophages are the most diverse life form in the ocean. These small viruses infect marine bacteria. Their diversity has become easier to quantify thanks to advances in metagenomics. Phages are specific to their bacterial hosts (Suttle 2007), and some estimates put bacteriophage diversity at hundreds of thousands of genotypes (Angly et al. 2006).

We know more about fish parasites than about the parasites of any other marine taxon because fisheries biologists and aquaculturists have long been interested in fish health. That said, only about 12% of fish species have been checked for one or more parasite species (Strona and Lafferty 2012). This is because most fish species are not caught for food and therefore remain unexamined for parasites.

How many parasites do fishes have? FishPEST (Strona and Lafferty 2012) tabulates more than 3000 known helminth species from fewer than 3000 examined marine fish species. On average, one helminth species is reported from 2.8 examined marine fishes, while 5.9 helminth species parasitize one marine fish species (a number slightly lower than reported for birds and mammals). There are more helminths per fish species for estuarine (8.8) and freshwater fishes (6.7) than for marine fishes. No matter the system, fishes are host to a rich parasite community, and many fish parasites remain to be discovered.

Even if they dominate species richness, parasites are always smaller than their hosts, so one could argue that they might be relatively insignificant at the ecosystem level. In one of the few ecosystem-level studies, parasites made up about 1% of the free-living biomass in estuaries (Kuris et al. 2008). That might not sound like much biomass, but one common group, the trematodes, exceeds the biomass of estuarine birds (**Figure 5.2**). The reason birds and trematodes are comparable is that they are on similar trophic levels. In general, after accounting for trophic level, parasites are as abundant as similar free-living species. In other words, parasites tend to have as much biomass as other consumers the top of the food chain. Parasites cannot be discounted from community ecology just because they are small in size (Hechinger et al. 2011a).

In conclusion, ~~just knowing taxonomy is not enough to understand a parasite's role in community ecology.~~ There are at least as many parasitic species as there are free-living species. There might be orders of magnitude more parasitic species than we think. This diversity of parasites represents a variety of trophic strategies and life cycles. Knowing these details is helpful for making predictions about the potential effects of parasites. And parasites are just as abundant as free-living species of the same body size and trophic level. In fact, the most numerous life forms on the planet are bacteriophages (10^{30}, or one nonillion), with a combined biomass equal to more than a million blue whales (Abedon 2001). Presently, when we look at a marine ecosystem, it is the top predators that get our notice: the sharks and marine mammals. If we could

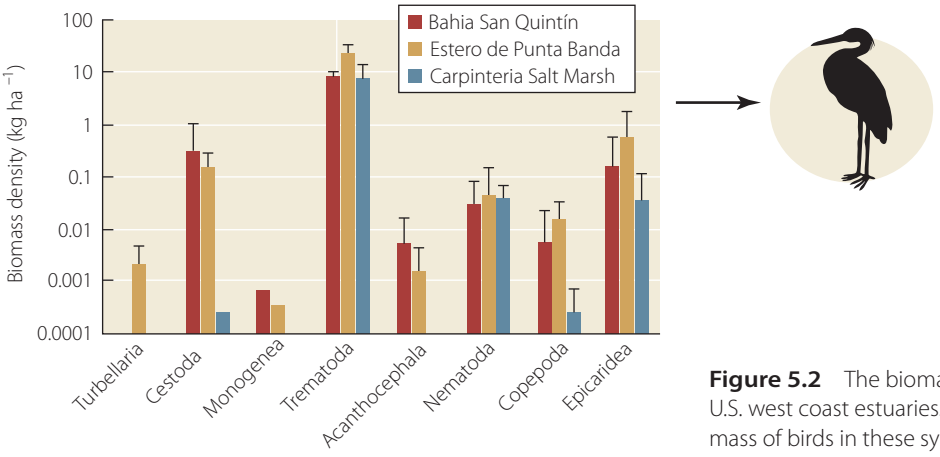


Figure 5.2 The biomass density of various parasitic taxa in three U.S. west coast estuaries. The icon on the right represents the biomass of birds in these systems. (After Kuris et al. 2008.)

see parasites as easily, we would notice that, in aggregate, they are just as abundant and as worthy of attention.

Basic Epidemiology

Most of the key points we make in this chapter are based on basic epidemiological principles. Epidemiology considers the population biology of infectious diseases and their hosts. In this section we explain how host population density affects parasite transmission. We describe intensity-dependent effects (where intensity refers to the number of parasite per host) and the implications of whether parasites affect mortality or reproduction.

A key epidemiological factor for community ecology is contact rate. Contact, in the simplest theoretical models, scales linearly with susceptible host density [this is akin to assuming a type 1 (non-saturating) functional response in a consumer–resource model]. As a result, a parasite can invade only a susceptible host population that is over a hypothetical minimum density. This host threshold density has several implications for community ecology, which we discuss on p. XX. As an example, studies in southern California kelp forests show that the probability of bacterial epizootics at a site increases with sea urchin density, and that below a minimum density, epizootics do not occur (**Figure 5.3**; Lafferty 2004). This finding is consistent with simple epidemiological models of density-dependent transmission (where the probability of an epidemic increases with host density).

There are many other possible relationships between transmission and host density. For instance, some types of contacts might saturate at high host density, or they might remain high at low density for social species. In addition, the presence of alternative host species decouples transmission of the parasite from a single host species. We return to this important point on p. XX.

Parasites take nutrition from their host, but often the impacts of a parasite on the host are disproportionate to the energy that the parasite takes. If consumption occurs in a key organ or tissue, it might not take much to impair the host seriously. An example is a copepod that infects the eyes of flatfishes (Kabata 1969). A little damage to this key organ affects the host far more than if the copepod's consumption were distributed generally throughout the fish's body. Perhaps the most sophisticated mode of energy drain is seen in parasitic castrators, such as larval trematodes in molluscs and rhizocephalan barnacles in crabs. These parasites maximize the amount of energy they take from their host while minimizing the extent to which they shorten the host's (and their own) life span (Lafferty and Kuris 2009b). One additional effect of parasites is that they force the host to invest in immune defenses. Immunity is not well understood in marine species, but its cost can dwarf the direct cost of parasitism (Careau et al. 2010).

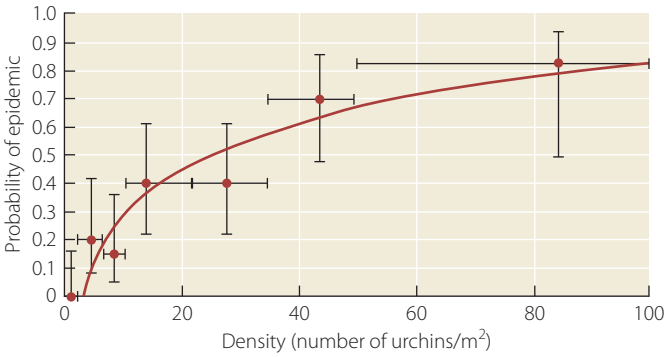


Figure 5.3 Plot of epizootic frequency versus sea urchin density. The estimated host threshold density is about 3 urchins per square meter. (After Lafferty 2004.)

The details of how a parasite affects its host are important when trying to estimate the effects of the parasite on the host population. A parasite that slows growth or eliminates reproduction can lead to a different dynamic than one that increases mortality. In particular, increased mortality can decrease the spread of parasites. Such virulent parasites have a shorter time to contact susceptible hosts than do avirulent parasites. This means that virulent parasites require higher host densities, or they must be more effective at producing infective stages.

An important characteristic of typical parasites is that most parasites in the population occur in just a few host individuals (Shaw et al. 1998). This aggregated parasite distribution increases the regulation of parasite populations if the most infected hosts are the most likely to die and, therefore, take a disproportionate number of the parasites with them (Anderson and May 1985). Another consequence of parasite intensity is that crowding can lead to intra- and interspecific competition for host resources.

In contrast to the classic parasite transmission described above, there are many opportunistic parasites that can cause infectious diseases. In humans, for instance, we are familiar with the potential for wounds to become infected by bacteria under unsanitary conditions. The bacteria that infect wounds have a very different dynamic than parasites that use direct, trophic, or vector transmission. Most importantly, their prevalence (proportion infected) in a host population is much more dependent on environmental factors, such as stress and injury, than on host density thresholds. Many coral diseases appear to fit this pattern, which, as we discuss on p. XX, may explain why they appear to be associated with environmental drivers (Ruiz-Moreno et al. 2012). Other coral diseases meet the assumptions of density-dependent transmission, such as prevalence being higher where coral cover or density is high (Bruno et al. 2007).

To summarize, understanding the epidemiology of infectious diseases requires knowledge about the life history strategy of the parasite. In particular, pathogens and typical parasites are modeled differently because it is important to count the number of worms per host to know their effect

on the host. However, a commonality of infectious disease models is that transmission is more efficient at high host densities. As we will discuss on p. XX, such density-dependent transmission allows host-specific parasites to act as regulators of host populations (Tompkins and Begon 1999), and this can have important implications for host communities, making it easier for competitors to coexist (Mordecai 2011). On the other hand, generalist parasites can lead to host extinction because they are not dependent on a single host species. The least host-dependent parasites are opportunistic species that can survive in the environment. Because most free-living species have specific, generalist, and opportunistic parasites, there are many ways that infectious diseases can affect community ecology.

Effects of the Environment on Infectious Diseases

The marine environment affects many aspects of community ecology, including infectious diseases. In this section we start with a theoretical perspective on how environmental factors can affect infectious disease dynamics, focusing on the effects of warming temperatures, acidification, and eutrophication. Our empirical examples include corals and abalones because increases in diseases of these hosts have been linked to changing environmental conditions.

There are many factors to consider when making predictions about the effects of environmental variables on infectious diseases. The first is that effects on individual hosts might or might not scale up to effects on host populations (Lafferty and Holt 2003). As hosts ourselves, we tend to worry about how stress can increase susceptibility to infection. While an individual under stress might be more likely to become infected when exposed, stressed infected hosts might not survive to transmit the parasite. Furthermore, if host density is reduced by stress, density-dependent transmission will be less efficient and infectious diseases less likely to spread. However, as mentioned on p. XX, opportunistic parasites are less sensitive to host density and should therefore do well under stressful environmental conditions. Reviews of empirical studies show that stressors can indeed have positive or negative overall effects on parasitism (Lafferty 1997).

Second, some environmental factors have nonlinear effects on the physiological processes of hosts and parasites. In particular, each species has an optimal range of light, temperature, oxygen, pH, and other aspects of water chemistry. For instance, free-living stages of parasites need warmth to develop, but die faster as temperatures warm (King and Monis 2007), leading to a hump-shaped relationship between temperature and performance. As a result, a shift in a particular environmental variable will disrupt the ability of well-adapted parasites to infect their hosts. These parasites might be able to adapt to changing conditions, or they might be extirpated from the system. Or, other infectious diseases suited to the new conditions could then be favored, resulting in novel host–parasite combinations (Harvell et al. 1999).

Thermal stress and climate warming

Warming is the environmental stressor that has captured the most attention from marine biologists (Chapter 19), and it is a critical factor in many host–parasite interactions (Harvell et al. 2002; Harvell et al. 2009). Although it seems likely that warming will lead to shifts in the latitudinal distribution of infectious diseases, it is far more difficult to determine whether these shifts will result in net increases or decreases in infectious diseases (Lafferty 2009). Because we are most concerned with scenarios in which a shift in climate leads to optimal conditions for a parasite and suboptimal conditions for a host, we highlight some examples in which warming is associated with increases in infectious diseases.

Warmer winters due to climate change can increase the overwinter survival and growth rates of some parasites (Harvell et al. 2009; Weil et al. 2009). Sometimes diseases are correlated with warm conditions. The oyster disease *Perkinsus marinus* is the one example for which enough data exist to tie outbreak cycles to climate cycles (Powell et al. 2012). This protozoan proliferates at high water temperatures and high salinities (seen most often in drought conditions), and it spread northward up the Eastern Seaboard as water temperatures warmed during the 1990s (Ford 1996).

Hosts can suffer stress as the water warms. Reef-building corals, for instance, are near their upper thermal limits (see also Chapter 12). Bleaching occurs when warming disrupts the symbiosis between corals and their associated algal partners (**Figure 5.4**). Bleached corals can more easily

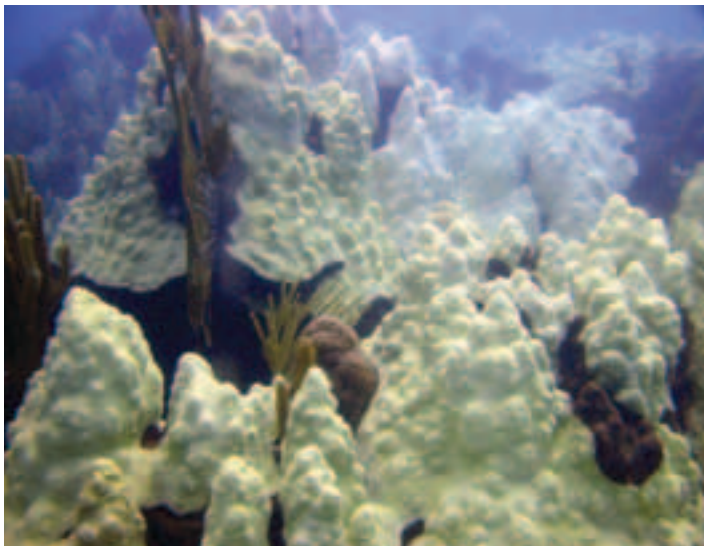


Figure 5.4 In Puerto Rico, the star coral (*Montastraea faveolata*) suffered up to 90% bleaching prevalence during anomalously warm water temperatures in 2005. (Photo courtesy of Ernesto Weil.)

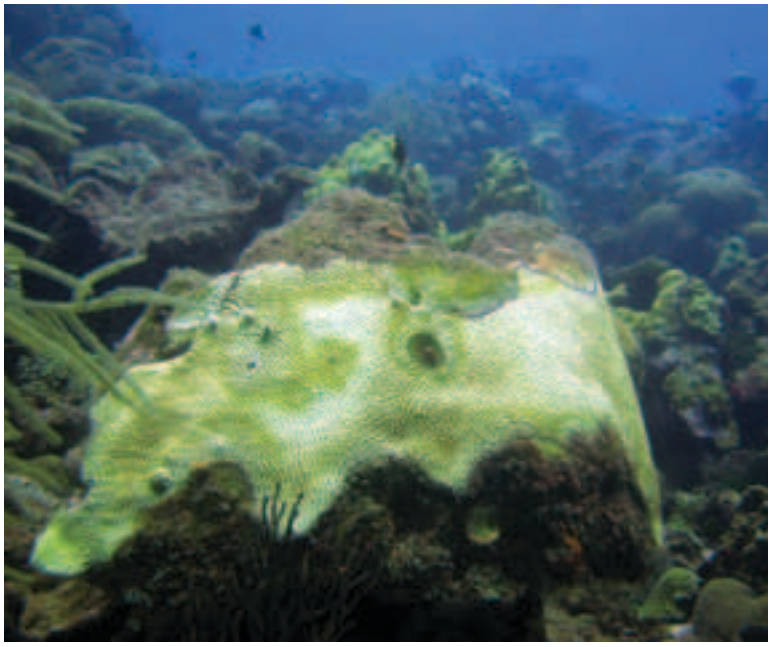


Figure 5.5 Caribbean coral (*Colpophyllia natans*) affected by bleaching and black band disease during the 2010 warm thermal anomaly. (Photo courtesy of Ernesto Weil.)

die or become infected (**Figure 5.5**; Harvell et al. 2009; Weil et al. 2009; Ruiz-Moreno et al. 2012). For example, a long-term study observed that growth rates of disease lesions on the dominant Caribbean reef-building coral, *Montastraea* spp., more than doubled with winter and summer warming from 1996 to 2006 (Weil et al. 2009). Regional warm temperature anomalies were correlated with high coral disease prevalence in the Caribbean and Pacific (Ruiz-Moreno et al. 2012).

Temperature is often implicated in the disease-induced mass mortalities observed in the black abalone. Laboratory studies show that most abalone species can survive infection by a withering-syndrome rickettsia-like organism (WS-RLO) so long as the temperature remains low (Moore et al. 2009). Temperature also influences susceptibility, but in an unusual way: susceptibility does not increase with mean water temperature, but it does increase with variation in water temperature. The variable water temperature of their intertidal habitat makes black abalone more susceptible to WS-RLO infection than are subtidal abalone species; this susceptibility leads to high infection prevalence in black abalone, and to eventual mass mortalities when the mean temperature increases (Ben-Horin et al. 2013). This case study indicates how the thermal stresses of the intertidal zone can make some hosts more susceptible to infectious diseases.

Ocean acidification

Another stressful aspect of greenhouse gas–driven climate change is the direct effect of increased carbon dioxide uptake by water, which leads to ocean acidification (OA; see Chapter 19). In particular, decreased pH can impair the growth of reef-forming corals and shell-forming bivalves (Hofmann et al. 2010). Little thought has been given to the effects of OA on infectious diseases (MacLeod and Poulin 2012). If OA either stresses hosts or helps parasites, individuals might be more susceptible to infections. A situation in which these effects were suspected involves the bacterium *Vibrio tubiashii* (*Vt*), a pathogen of marine shellfish larvae that has been problematic in commercial bivalve hatcheries (Estes et al. 2004). Substantial mortality has occurred in *Crassostrea gigas* (Pacific oyster) since 2005 in larval cultures at two regional hatcheries in Washington State and Oregon coincident with upwelling events. Researchers suspected that an influx of nutrients associated with upwelled water created conditions suitable for bacterial growth once water warmed in the hatchery, but they noted that the nutrient-rich upwelled water was also low in pH, pointing to a potential role of OA (Elston et al. 2008). Subsequent laboratory experiments found that at cool temperatures, growth rates of *Vt* in culture were 13% higher when CO_2 was increased by a factor of five, but OA had no effect on the susceptibility of larvae to *Vt* or on the mortality of infected larvae (Dorfmeier 2012). While most concern has been focused on whether OA will increase disease, it is possible that OA could disrupt parasite life cycles. For instance, fish in acidified freshwater systems have reduced parasite richness due to a lack of intermediate hosts such as snails (Marcogliese and Cone 1996).

Eutrophication

Nutrients can promote infectious diseases (Johnson and Carpenter 2008). One reason is that productivity can lead to higher host densities, which increase transmission efficiency. For instance, phage dynamics in plankton communities are forced by the input of nutrients following upwelling because more hosts are available under productive conditions (Parsons et al. 2012). In addition, snails often benefit from eutrophication, and this benefits the trematode parasites that use snails as intermediate hosts (Johnson and Carpenter 2008). In corals, eutrophication can increase disease risk by stressing the coral host, which is often adapted to oligotrophic conditions, and favoring bacterial and fungal pathogens. For example, adding nutrients near corals can increase lesion progression and doubles the extent of black band disease (Bruno et al. 2003; Voss and Richardson 2006). Eutrophication can also favor algae on reefs, and algal exudates can increase disease risk for corals and act as a reservoir for some pathogens (Szmant 2002; Nugues et al. 2004; Kaczmarky et al. 2005; Haas et al. 2011).

In conclusion, it is already well known that marine communities are sensitive to environmental factors. Parasites are also sensitive to environmental factors, but their responses can be different from those of their hosts. Although we have highlighted cases in which environmental change has been associated with increases in disease, an environmental factor that impairs a free-living species will not necessarily result in an increase in its parasites. Many parasites do ~~best when their hosts are abundant~~, and some aspects of parasite success are themselves constrained by the environment. As a result, parasites have niches, as do free-living species, and changing conditions can put existing parasites outside their niche space, just as it opens up opportunities for new parasites that match the new niche. New host–parasite combinations often fail, but sometimes the new relationship can be detrimental for the host and lead to mass mortalities (Lafferty and Gerber 2002).

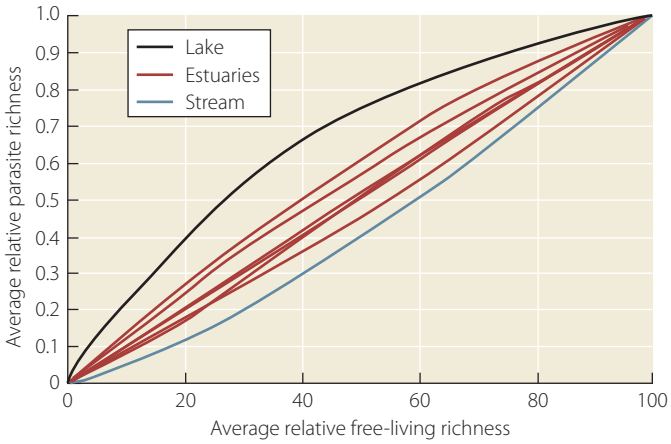


Figure 5.6 Relationship between host and parasite species richness based on simulation modeling of eight aquatic food webs. (After Lafferty 2012.)

Effects of Free-Living Diversity on Infectious Diseases

Parasites depend on the presence of hosts. Thus we should expect that changes to communities of free-living species would lead to changes in infectious diseases. A simple and general hypothesis for the relationship between free-living communities and infectious disease agents is that more complex communities have the potential to support more species of infectious diseases (Hudson et al. 2006). For example, the richness of fish parasites can be higher on pristine reefs (Lafferty et al. 2008b).

One factor that ties parasite diversity to free-living species diversity is life cycle complexity. Parasites are dependent on their hosts just as predators are dependent on their prey. One difference, however, is that parasites tend to be sequential specialists across life stages, which makes them even more sensitive to biodiversity loss than are predators (Rudolf and Lafferty 2011). Individual parasite species vary in their sensitivity to biodiversity loss, but food web models suggest an overall positive linear relationship between free-living and parasite species richness (**Figure 5.6**; Lafferty 2012). In other words, for a 50% reduction in free-living species diversity, we would expect to see, on average, a 50% reduction in parasite diversity. Empirical data support this prediction. A positive relationship between human infectious diseases and vertebrate diversity is seen from country to country (Dunn et al. 2010). In aquatic systems, adding fishes to a community leads to increases in parasite richness (Amundsen et al. 2012). This pattern is also seen at small scales in marine communities. For instance, the diversity of trematode parasites in a sample of snails increases with the diversity of final host birds seen in an estuary (Hechinger and Lafferty 2005; Fredensborg et al. 2006).

Although an increase in free-living species richness often leads to an increase in parasite richness, some parasites can be indirectly affected by reductions in host density that occur in response to species additions. For instance, adding a species to a system could reduce the abundance of its competitors. If high free-living species diversity leads to many rare host species, some host-specific infectious diseases might dip below the host density threshold and be lost from the system, at least on a local scale (Clay et al. 2008). Generalist parasites, on the other hand, will be less affected by the rarity of each host unless there is frequency-dependent transmission (where transmission rate depends on the relative abundance, not the absolute density, of susceptible hosts) and contact among host species is low (Dobson 2004). Similarly, adding a predator to a system could reduce the abundance of its prey. For instance, areas with many sea urchin predators have fewer sea urchins, which leads to fewer bacterial epizootics in sea urchins (Lafferty 2004).

The dilution effect is a ~~recent~~ hypothesis about how free-living communities affect an infectious disease. This hypothesis states that in more diverse communities, a parasite can be "diluted" and even decreased by dead-end hosts (hosts that can become infected, but in which the parasite ~~cannot survive or be transmitted~~), and thus its transmission to target hosts slowed. Dilution has been proposed as an ecosystem service of biodiversity to human health (Keesing et al. 2010), but the evidence has been criticized as lacking empirical support (Randolph and Dobson 2012). Little information exists about the dilution effect in marine systems, so it is premature to evaluate this controversy in the ocean, but one can imagine that the dilution effect could occur there. Trematodes make a good example. These parasites have a swimming larval stage, called a cercaria, that leaves the first intermediate host (a snail) and tries to penetrate a second intermediate (or sometimes, a final) host. Many species, such as fishes, polychaetes, and sea anemones, will eat the undefended, swimming cercariae (Kaplan et al. 2009; Johnson et al. 2010). A cercaria might also penetrate an inappropriate second intermediate host, where it will perish. Labora-

tory and field experiments with freshwater systems have shown that the more predators of cercariae and noncompetent hosts in an aquarium, the harder it will be for cercariae to reach an appropriate host (Johnson et al. 2013).

The addition of invasive species (which often lack parasites) to a community could also lead to a dilution effect and break the transmission of infectious diseases in native species. This effect is seen in San Francisco Bay, which has become a system dominated by invasive species and devoid of parasites (Foster 2012). However, it is not the richness of predators and noncompetent hosts that matters, only their relative abundance. So the dilution effect, unlike the other hypotheses we have discussed, is not a diversity effect unless there is a disproportionate increase in the relative abundance of parasite predators and noncompetent hosts as diversity increases. If this occurs, the prevalence of an infectious disease could indirectly decline with increasing biodiversity. On the other hand, an infectious disease could increase if the relative abundance of competent hosts increases with biodiversity.

In summary, parasites depend on and influence free-living species diversity. Parasites cannot exist without hosts, and a system with few host species should contain few parasite species. Across many different systems, the proposed relationship between parasite diversity and free-living species diversity is positive. However, there are further complexities to this relationship driven by host thresholds and possibly by dilution. Specifically, the addition of some species to a community, while providing resources for some parasites, could have indirect negative effects on the transmission of other parasites. If increased diversity leads to a reduction in the average abundance of each free-living species, the potential for more parasites might not be realized. Instead, species driven to rarity in rich communities might lose their parasites. This would give them an advantage when rare and help maintain them in the community. Alternatively, parasite diversity might not increase linearly with free-living species diversity if most species interfere with the transmission of parasites, either as predators or as dead-end hosts. Though we have ~~no~~ information about the importance of dilution and rarity ~~in~~ marine ~~systems,~~ these are hypotheses in need of rigorous testing. Nonetheless, at this point, the available evidence suggests that parasite richness increases with host richness.

Parasite Communities

A host is a habitat with parasite inhabitants. As such, communities of parasites in hosts have been used as model systems to understand patterns in community ecology (Esch et al. 1990). A key advantage of parasite communities as study systems is that the boundary of the community, the host habitat, is well defined. A host differs somewhat from other habitats, however, in that the host attempts to limit parasite recruitment and is hostile to parasites that do recruit. Another convenient aspect of studying parasite communities is that it is easy to get large numbers of hosts, which leads to replicates of communities for study. Parasite communities are of interest in their own right, particularly when we are concerned with how different species of parasites might interfere with or assist each other in ways that have important outcomes for the health of the host (Lafferty 2010). Although much effort has been devoted to studying communities of parasites, little of this research has crossed over into the general community ecology literature. In this section we begin with an overview of the main theoretical concerns surrounding parasite communities. We then discuss parasite communities in fishes and larval trematode communities in snails, the two systems in which the bulk of work on marine parasite communities has been done. We show how this work provides information about parasite communities as well as general insight into the forces that structure communities.

With respect to the factors that structure parasite communities, researchers often ask about the importance of host biology, ~~parasite recruitment, competition, facilitation, niche partitioning, latitude, isolation, and host body size and age~~. These are the same sorts of questions that marine ecologists ask about free-living communities.

When making predictions about parasite communities, there are many things worth keeping in mind, particularly the life cycle of the parasite. All parasites must recruit to the host from elsewhere. Successful parasite recruitment requires passage through an encounter filter driven by ecology and a compatibility filter driven by evolution (Combes 2001). As mentioned on p. XX, after an infectious stage reaches a host, some parasites build up populations in or on the host by reproducing, whereas others do not. A second issue is the extent to which the host is a limited resource. If parasites deplete available space or nutrition, competitive interactions or niche segregation can result, just as in communities of free-living species. Parasite communities even experience parallels to disturbance or predation. Host death acts like a major disturbance, whereas host birth opens up new habitat for colonization, and the host's immune system sometimes acts in a manner analogous to predation. Overall, there many similarities between parasite and free-living communities.

Parasite communities in fishes

Fishes have a distinctive parasite fauna. For example, helminth communities in fishes differ from those in other vertebrates, having a higher proportion of trematode and acanthocephalan species (Poulin et al. 2011). Across fish species, some have rich parasite communities comprising generalists and specialists, whereas others have just a few generalist species, leading to a "nested" (species-poor communities tend to have common species) pattern in fish-parasite species networks (Bellay et al. 2011). Host phylogeny affects parasite communities such that related fish species share parasite species and have similar parasite communities (Poulin et al. 2011). This effect is due to both

the fishes' shared evolutionary history with host-specific parasites and their similar ecologies, and it is one reason that parasite communities are modular across fish species.

Several ecological traits of hosts have been linked to parasite communities in fishes: size and age, habitat, diet, trophic level, schooling behavior, population size, density, geographic range, latitude, and depth (Luque and Poulin 2008). Age and size affect the amount of time that a host has been able to accumulate parasites, so that large fish tend to have more abundant and diverse parasites than do small fish (Rohde et al. 1995; Timi and Poulin 2003). Large fish are also bigger targets for parasites. The effect of accumulating parasite species richness with size can lead to nested parasite communities within a fish species because young fish are most likely to be parasitized by common parasite species, whereas older fish are more likely to have been exposed to rarer ones (Vidal-Martinez and Poulin 2003). Habitat can also affect fish parasite communities. For instance, pelagic fishes have fewer parasite species than do benthic fishes (Rohde et al. 1995), perhaps because it is easier to find a host while searching in two rather than in three dimensions. Because fish acquire many trophically transmitted parasites (Marcogliese 2002), diet (especially as determined by trophic level) can be an important structuring force for parasite communities, with invertebrate feeders having different parasite communities than piscivores (Timi and Poulin 2003; Jacobson et al. 2012).

Any study of communities must account for sampling artifacts. More parasite species are likely to be found in a larger sample, and common hosts are more sampled or better known. In addition, fishes with broad geographic ranges have more reported parasites, but this is because fishes with larger geographic ranges are more often sampled for parasites (Poulin 1997).

One of the most famous geographic patterns in community ecology is the latitudinal gradient that leads to high diversity near the equator. This pattern holds for ectoparasites of fishes, but not for helminth communities (Rohde and Heap 1998). The only significant latitudinal effect on helminths is a trend toward proportionally more nematode species in fishes at lower latitudes (Poulin et al. 2011). Deep-sea fishes have fewer ectoparasites than do other fishes, perhaps due to lower fish densities in the deep sea (Rohde et al. 1995), though it is hard to distinguish depth and latitude effects from temperature effects.

Overall, fish phylogeny and ecology structure parasite communities in fishes, but the parasites themselves do not appear to interact much. For ectoparasites (Rohde et al. 1995) and helminths (Sasal et al. 1999), there is little evidence of negative competitive interactions among species that are strong enough or frequent enough to structure parasite communities.

Larval trematode communities in snails

Next to those of fishes, the best-studied parasite communities are those of larval trematodes in snails. Trematodes are helminth parasites with complex life cycles, in which a vertebrate is the final host and a mollusc (e.g., a snail) is the first intermediate host. Snails are easy to sample in large numbers, and some snail species support a rich and prevalent trematode assemblage. In this case, we can consider at least two scales of a parasite community (Bush et al. 1997). The *infracommunity* is the assemblage of parasite species within a single host. The *component community* is the assemblage of parasite species within a host population (or a sample of hosts). In this section we describe three factors that structure trematode communities (**Figure 5.7**).

The primary determinant of the trematode community in a snail population is the presence of vertebrate final hosts. Because different trematode species infect different final hosts, it is possible for niche differences among final hosts to lead to negative associations in the recruitment of trematode species to snails (Sousa 1990). However, overall, final host distributions tend to be positively correlated (e.g., a good location for one vertebrate species is also a good place for several other vertebrate species), so that some snail populations are infected by only a few trematodes, while others are infected with a high prevalence of many trematode species (Kuris and Lafferty 1994). For instance, sites with a high abundance and diversity of ~~trematode parasites in the local snail population~~ have a high abundance and diversity of ~~estuarine birds~~ (Hechinger and Lafferty 2005). ~~Manipulating~~ bird perches can lead to foci of infection, with rich and abundant trematode communities in ~~the~~ snails ~~living under bird perches~~ (Smith 2001). Such trematode communities can increase after the restoration of degraded habitats, suggesting that trematode communities can be good indicators of ecosystem integrity (Huspeni and Lafferty 2004).

Another determinant of trematode community structure is snail demography. As indicated on p. XX for fishes, hosts accumulate parasites over time. Older, larger snails tend to have a higher prevalence and richness of trematodes (Sousa 1990). The accumulation of parasites in older snails also aggregates trematodes into a subset of the snail population (as opposed to distributing them randomly or evenly).

Interspecific competition can also structure ~~parasite~~ communities. ~~Although populations of snails can support several trematode species, there is most often one trematode species per individual snail.~~ Most larval trematodes in snails are parasitic castrators. This life history strategy distinguishes larval trematodes from the intestinal helminths and ectoparasites of fishes. ~~The reason for this difference is that a~~ single larval trematode can reproduce asexually until it consumes all the reproductive energy of the ~~snail.~~ Any other species that tries to infect the snail host will have to compete for this energy. As a result, there is strong evidence for competition among larval trematodes within a snail (Kuris 1973). Some trematode species have even evolved soldier castes that engage in intraguild predation on other trematode species

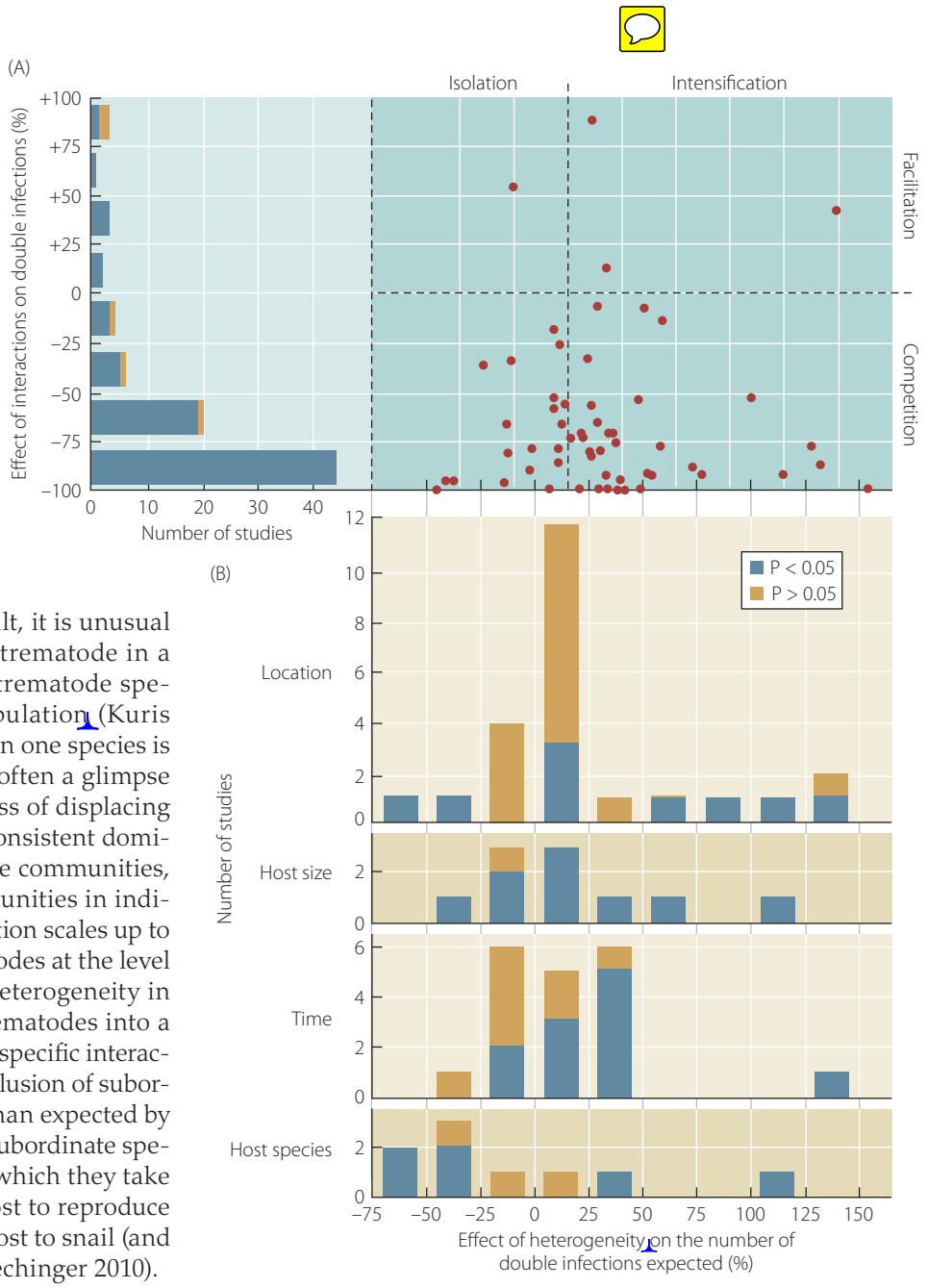


Figure 5.7 Types of interactions among trematode species in snails (A) as well as the effects of heterogeneity in trematode recruitment driven by (B) location, host size, time, and host (snail) species. For instance, variation in bird abundance probably drives much of the heterogeneity in recruitment from place to place, and trematodes accumulate over time in larger snails. Most studies fall in the lower right quadrant, indicating that these outside forces tend to increase species interactions, which are then resolved by competition so that only one species can persist in an infected snail. (After Kuris and Lafferty 1994.)

(Hechinger et al. 2011b). As a result, it is unusual to find more than one species of trematode in a snail, even in areas where many trematode species are common in the snail population (Kuris and Lafferty 1994). When more than one species is seen in an individual snail, this is often a glimpse at a dominant species in the process of displacing a subordinate. ~~Because there are~~ consistent dominance hierarchies within trematode communities, competition structures infracommunities in individual snails. Some of this competition scales up to structure the community of trematodes at the level of the snail population. Because heterogeneity in recruitment tends to aggregate trematodes into a subset of the snail population, interspecific interactions (and resulting competitive exclusion of subordinate species) are more frequent than expected by chance (Kuris and Lafferty 1994). Subordinate species have an altered life history in which they take more resources from their snail host to reproduce early and often, which comes at a cost to snail (and parasite) growth and longevity (Hechinger 2010).

To conclude, parasites can form structured communities in their hosts, host populations, and host communities. Although parasite communities are convenient units for studying general questions about communities, the host is a distinct type of habitat. Parasites must first contact a host (an event driven by host ecology, particularly host density and accumulation with age), and must then infect and develop in that host (an outcome determined by host–parasite coevolution). Older hosts have richer parasite communities with more filled niches. For intestinal parasites of fishes, host diet is an important structuring factor. For ectoparasites, increased host density leads to efficient transmission. Even at high rates of recruitment, fish parasites coexist due to a diversity of niches within a fish. In contrast, larval trematodes in snails try to exploit the same niche (snail reproductive energy), leading to fierce interspecific competition and species-poor infracommunities. Competitive interactions are intensified because transmission from final hosts tends to be aggregated in space, so that trematode recruitment is focused on a subset of the snail population. In short, the diversity of patterns and mechanisms structuring parasite communities matches what occurs in free-living communities.

Host Regulation by Infectious Diseases

We are accustomed to thinking about the effects of disease on an individual host, but less used to thinking about effects on host populations. In this section we focus on examples of population-level effects of infectious diseases on marine species and, where possible, tie our examples back

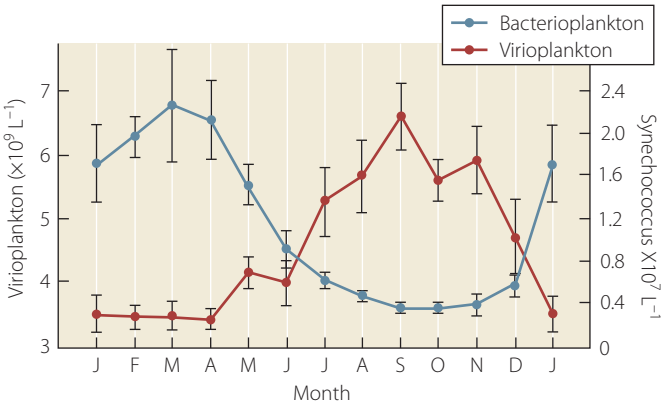


Figure 5.8 Tracking of bacterioplankton by virioplankton over an annual cycle. (After Parsons et al. 2012.)

to theory. A theme will be the conditions under which parasites reduce or even regulate host population density.

Bacteriophages and bacteria

Phage particles are most abundant where their hosts, heterotrophic bacteria and cyanobacteria, are abundant (Cochlan et al. 1993), a pattern consistent with the predictions of density-dependent transmission. And viral infections can be prevalent: about 70% of cyanobacteria are infected with bacteriophages, leading to dramatic declines in the standing stock of phytoplankton (Suttle 2007). These observations suggest that bacteriophages are greater sources of mortality for bacteria than are filter feeders. In the ocean, periods of upwelling lead to blooms of bacterioplankton. These blooms are followed by peaks in virioplankton, which then drive bacterioplankton levels down, leading to viral–host cycles akin to Lotka–Volterra predator–prey cycles (**Figure 5.8**; Parsons et al. 2012). This pattern has important implications for plankton communities, productivity, resources available to herbivores, carbon sequestration, and geochemical cycling. A novel application is the potential use of phages to control bacterial diseases of corals (Efrony et al. 2007; Atad et al. 2012).

Sea urchin epidemics

Reports of sea urchin diseases have increased over time, perhaps due to the increases in sea urchin populations that stem from reductions in sea urchin predators such as crabs, lobsters, and fishes (Ward and Lafferty 2004). In particular, sea urchins sometimes experience mass mortalities associated with bacterial pathogens (Lessios 1988). Whether the bacteria are always the causal agent, or just associated with dying urchins, has been difficult to establish (Gilles and Pearse 1986). Pathogenic amoebae cause similar mortality events in Nova Scotia and the Gulf of Maine (**Figure 5.9**; Scheibling and Hennigar 1997). In years after epizootics, urchin densities decline, suggesting that these infectious diseases can regulate urchin populations where their natural predators are rare—though disease does not appear to reduce urchin densities to the same low levels that predators do (Behrens and Lafferty 2004). We will discuss the indirect effects of these sea urchin diseases on p. XX.

Trematodes and snails

To what extent can nonlethal parasites affect host populations? Larval trematodes don't kill their hosts, but as parasitic castrators, they do block host reproduction. In places where final hosts (e.g., birds) are common, transmission rates to snails are high, so that fewer snails can reproduce (Hechinger and Lafferty 2005). In marine snail species with pelagic larvae it is difficult to see an effect of trematodes on snail density because the size of the adult

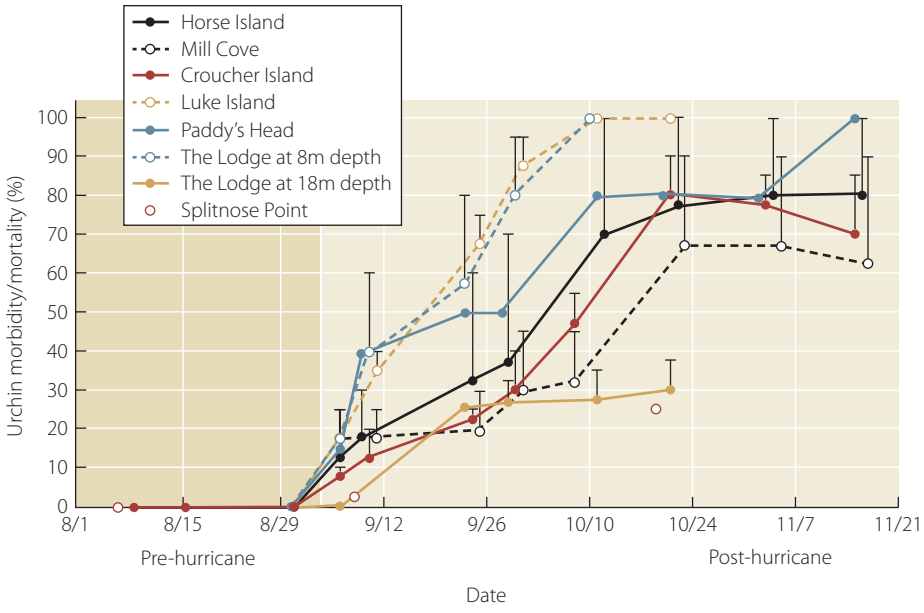


Figure 5.9 Increased morbidity and mortality in green sea urchins at different sites in St. Margaret's Bay, Nova Scotia, associated with epidemics of *Paramoeba invadens* (initiated by a hurricane in late August). (After Feehan et al. 2012.)

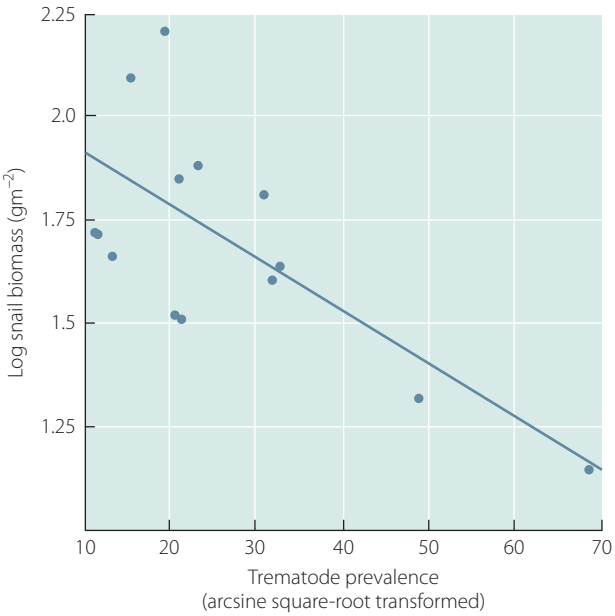


Figure 5.10 Plot of trematode prevalence versus snail density on New Zealand mudflats. (After Fredensborg et al. 2005.)

population may not be limited by its reproductive output (Kuris and Lafferty 1992). For host species with closed recruitment, however, such as estuarine snails with crawl-away larvae, there can be a negative association between the prevalence of parasitism and host density (**Figure 5.10**; Lafferty 1993; Fredensborg et al. 2005). Such population-level effects of parasitic castrators can be common, particularly where parasites reach high prevalence (Lafferty and Kuris 2009b).

Oysters and protozoan parasites in Delaware Bay

There are just a few cases for which we have detailed, long-term data on marine host–parasite cycles that suggest host regulation. Perhaps the best-studied marine parasites are the two Asian protozoan parasites of oysters (*Crassostrea virginica*) in Delaware Bay, *Haplosporidium nelsoni* and *Perkinsus marinus*. Back in 1957, *H. nelsoni* was identified as the causative agent of MSX epizootics in Delaware Bay (Haskin et al. 1966). Subsequently, *P. marinus* was introduced from Chesapeake Bay (Mackin et al. 1950), causing outbreaks of Dermo disease beginning in the 1990s. Current natural mortality rates, driven by *P. marinus*, have exceeded rates of oyster reef formation, leading to a net loss of oyster reefs and their associated ecosystem services (Powell et al. 2012). If hosts can evolve resistance (the ability to block parasite growth or reproduction) or tolerance (reduced injury from an infection) to parasitism, they might be able to rebound after epizootics. The costs of resistance and tolerance can then create opportunities for susceptible hosts to increase in number once a parasite fades. One of the best examples of an evolved response to marine parasites occurred in the 1980s, when survivors of the *H. nelsoni* epizootic developed resistance and their progeny repopulated the bay (Ford and Bushek 2012).

Parasites and sea otters

Even if parasites do not regulate host populations, they can have negative effects. A case in point is the southern sea otter. Sea otters are dead-end or accidental hosts for bacterial infections as well as for three pathogenic parasites that ~~interfere with their recovery~~ (Lafferty and Gerber 2002). One of the latter is the protozoan *Toxoplasma gondii*, an obligate parasite of cats that uses warm-blooded vertebrates as an intermediate host (Dubey and Beattie 1988). Infections of this parasite were once blamed on cat owners flushing pet waste into sewers (Miller et al. 2002). However, better spatial tracking has indicated that an otter's risk of infection is highest near unpopulated areas (Johnson et al. 2009). In addition, the strain of *T. gondii* most often found in otters is common in wildlife (Miller et al. 2008). These two lines of evidence suggest that wild cats (mountain lions and bobcats), not domestic cats, are an important source of infection. Whatever the source, somehow the oocysts of the parasite enter the otter's habitat, where they are ingested. Many otters are infected, and some are intolerant of infection and suffer neurological damage (Thomas and Cole 1996). *Sarcocystis neurona*, a similar protozoan parasite of opossums, also causes pathology in sea otters and is much more associated with periods of freshwater runoff than is *T. gondii* (Shapiro et al. 2012). A third parasite that causes trouble for sea otters is an acanthocephalan worm. This parasite uses sand crabs as intermediate hosts and diving ducks and shorebirds as final hosts (Hennessy and Morejohn 1977; Mayer et al. 2003). Acanthocephalan prevalence in sand crabs is higher on beaches where birds are common (Smith 2007). When otters forage for sand crabs, they ingest the larval worms, which attempt to establish. Because ~~they are~~ not adapted to a sea otter's gut, some bore holes in the otter's intestine, leading to peritonitis and death (Thomas and Cole 1996). ~~Accidental parasitic infections are a prominent source of mortality for California sea otters (Kreuder et al. 2003). In addition, otters suffer secondary bacterial infections from wounds (Miller et al. 2010). High mortality rates appear to be the main factor preventing sea otters from recovering from the brink of extinction (U.S. Fish and Wildlife Service 2003).~~ However, because these parasites don't benefit from parasitizing otters, they cannot act to regulate sea otter populations. To regulate otters, they would need to increase when the otters were common and decrease when otters were rare.

To conclude, there is substantial evidence from terrestrial systems that parasites can regulate host population densities (Tompkins and Begon 1999). This regulation is the basis for modern biological control (Lafferty and Kuris 1996) and is one explanation for why invasive species do better when they escape their parasites (Torchin et al. 2003). Open recruitment makes it harder to see the con-

sequences of nonlethal effects of parasites in the ocean. However, we do have evidence that parasites respond in a density-dependent manner to their hosts and can reduce host density, two of the key requisites for population regulation. Given the ubiquity of parasitism in marine systems, it seems that parasites must play broader regulatory roles than is appreciated. But parasites don't need to regulate host populations to have an effect on them. Sea otters are accidental hosts of parasites of other wildlife, and such parasites can prevent them from recovering from near-extinction.

Mass Mortalities

Mass mortalities are rare and are thought to indicate a change in the ecology of a system (Harvell et al. 1999). There are many examples of mass mortalities in the ocean that are not related to infectious diseases. Perhaps the most common is hypoxia, where sudden bacterial decomposition or an incursion of oxygen-depleted water causes marine organisms to suffocate (Lim et al. 2006). Sudden temperature changes can also cause mass mortalities (Laboy-Nieves et al. 2001). Another common type of mass mortality results from blooms of algae that produce toxins or deplete oxygen (Pitcher and Calder 2000). A final type of mass mortality happens when ocean conditions lead to decreases in nutrients, leading to starvation (Wang and Fiedler 2006). In this section we focus on the rare, but nonetheless dramatic, mass mortalities of marine species induced by diseases, again tying our examples back to theory and using examples from a broad range of host–parasite taxa.

The first observation of a marine mass mortality linked to disease occurred in the 1930s, when there was up to 90% mortality of the eelgrass *Zostera marina* on both coasts of the North Atlantic (Renn 1936). The culprit was eelgrass wasting disease, which is caused by the opportunistic slime mold *Labyrinthula zosterae* (Muehlstein et al. 1988). This wasting disease has also occurred in the Mediterranean and the Pacific Northwest (Short et al. 1987). We will discuss the broad impacts of seagrass wasting disease on p. XX.

The Caribbean has experienced a series of mass mortalities in common invertebrates. In 1983–1984 mass mortalities of the dominant herbivorous sea urchin (*Diadema antillarum*) spread throughout the Caribbean. Urchins dropped their spines and died within days, and populations crashed by 95% within a span of weeks (Lessios et al. 1984). In 1995 an outbreak of aspergillosis in sea fans was observed in the Bahamas, and later throughout the Caribbean (Nagelkerken et al. 1997) and the Florida Keys (Kim and Harvell 2004). The disease was caused by *Aspergillus sydowii*, a common terrestrial fungus. Infected sea fans have necrotic lesions, surrounded by purple halos (**Figure 5.11**; Petes et al. 2003) that indicate an inflammatory response to the fungus. This response includes increased amoebocyte density and production of prophenoloxidase enzymes (Mydlarz et al. 2008). The fungus is a pollutogen in the marine environment, meaning that its dynamics are driven primarily by noninfectious processes (Lafferty and Kuris 2005). Its source is presumed to be sediment runoff (Smith et al. 1996; Rypien et al. 2008), but spatial analysis shows that fan-to-fan infection could also be important (Jolles et al. 2002). Between 1997 and 2003, aspergillosis caused widespread mortality of large sea fans. Around 50% of sea fan tissue was lost, and 20–90% of sea fans died~~,~~ at affected sites. The fall of the epizootic and rise of healthy sea fan populations is attributed to strong selection for increased resistance (Kim and Harvell 2004; Bruno et al. 2011).

Figure 5.11 This sea fan with aspergillosis is mounting an inflammatory immune response in the purple regions surrounding the fungus. (Photo courtesy of Ernesto Weil.)

Although they have been among the most common corals in the Caribbean for the last several interglacial periods (Pandolfi et al. 2002), elkhorn coral and staghorn coral (*Acropora palmata* and *Acropora cervicornis*) were listed for protection under the U.S. Endangered Species Act in 2006 (Hogarth 2006). The Caribbean-wide decline of these two foundation species was largely due to disease (Aronson and Precht 2001a)—primarily white band disease, which appears to be caused by the bacterium *Vibrio carchariae* (Gil-Agudelo et al. 2006), and more recently white pox disease, the prevalence and incidence of which appears to be temperature related (**Figure 5.12**). White pox is also putatively ~~linked to~~ an opportunistic human pathogen, *Serratia marcescens* (Sutherland et al. 2011), which is linked to sewage outflows (Sutherland et al. 2010). Outbreaks of white band disease on *Acropora palmata* and *Acropora cervicornis* in the 1980s caused an estimated 95% reduction in colony density (Bythell and Sheppard 1993; Aronson and Precht 2001a). Longer-term paleontological evidence suggests that the extent of recent losses in *A. cervicornis* because of

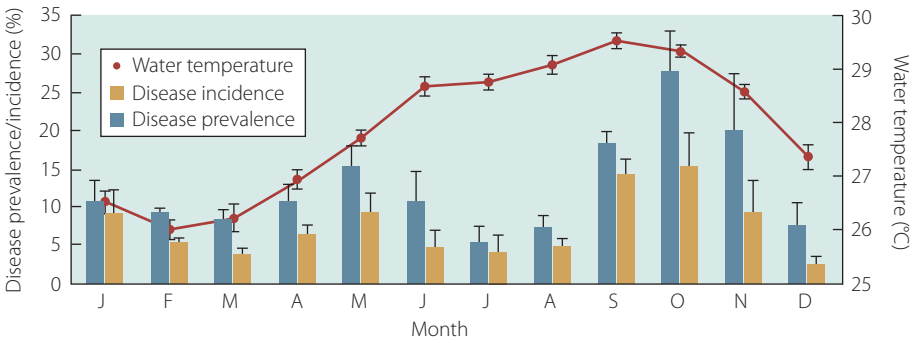


Figure 5.12 Monthly average seawater temperature and white pox incidence and prevalence on *Acropora palmata* in Haulover Bay, Saint John, U.S. Virgin Islands, from 2003 to 2009. (After Rogers and Muller 2012.)

disease is unprecedented on a time scale of at least three millennia (Aronson et al. 2002).

A variety of parasites have affected abalone fisheries and farms. In California and Baja California, black abalone were once used for bait. Although a commercial export fishery depleted subtidal stocks, intertidal stocks were so abundant that animals were stacked on top of one another (probably as a result of the historical removal of sea otters, which are effective abalone predators). In the mid-1980s, marine biologists noticed abalone disappearing from their study plots. The muscular foot was withered, rendering the animals unable to remain attached to the rocks. These die-offs were often rapid and extensive (Richards and Davis 1993). Pathologists identified a rickettsial parasite as the cause (Friedman et al. 2000). The origin of the parasite is unknown, but it spread north and south from the Santa Barbara Channel Islands, proceeding faster in warmer water (Lafferty and Kuris 1993; Altstatt et al. 1996). Currently, the disease occurs throughout most of the range of the black abalone. Even where black abalone have been extirpated by the disease, the parasite persists in other, more tolerant hosts (including infected red abalone in abalone farms). This prevents the parasite from fading out as black abalone become rare. Because of the population collapse, the black abalone has been put on the U.S. endangered species list. There might be hope for its recovery because ~~new recruits show tolerance for infection and~~ pathologists have discovered a novel bacteriophage that infects the parasite (Friedman and Crosson 2012).

In September 1995, a lethal herpesvirus (Crockford et al. 2005) of pilchards (*Sardinops sagax neopilchardus*) swept across New Zealand and Australia (**Figure 5.13**; Whittington et al. 1997). The source of the pathogen was hypothesized to be imported frozen pilchards fed to penned tuna (Griffin et al. 1997). At least 10% of the pilchard population was killed (Gaut 2001). A slower-spreading, but more intense, epizootic (near 70% mortality) occurred in 1998. Social behavior in pilchards did not conform to the assumptions of density-dependent transmission. A consequence of schooling behavior in fish like pilchards is that fish in large schools have local densities equal to those of fish in small schools. There can be many schools in a shoal, so schools might be the more appropriate scale at which to measure contacts between infected and susceptible groups (Murray et al. 2001). Rates of spread were estimated as 21 km per day to the west and 40 km per day to the east, and the spread was counter to prevailing currents, making some speculate that the disease was spread by seabirds foraging on dead and dying fish (Griffin et al. 1997). This and other fast-spreading marine epizootics suggest that epizootics may spread faster in the ocean than on land (McCallum et al. 2004).

As marine mammals have been protected from hunting, standardized reports of infectious diseases have increased (Ward and Lafferty 2004). This increase could be a density-dependent response of infectious diseases to recovering host populations, and it could also be an effect of the bioaccumulation of toxins that can impair pinniped

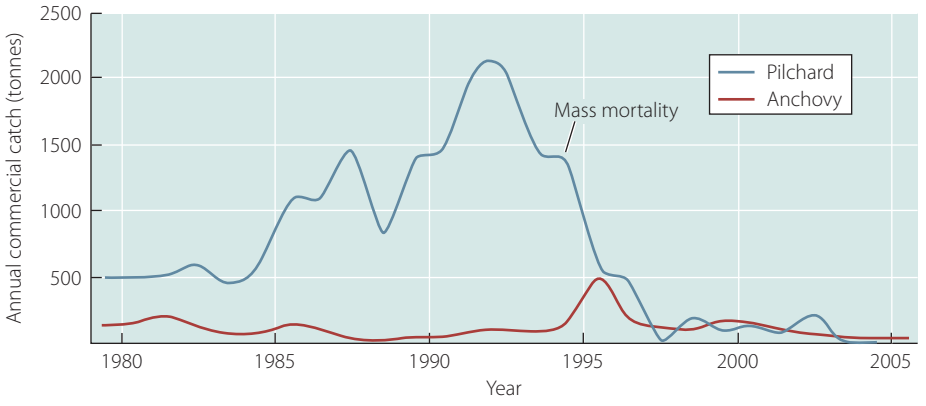


Figure 5.13 Changes in commercial catches of pilchards and anchovies (a potential competitor) from 1[illegible] to 2006 near Australia show the effects of mass mortality of pilchards. (Note: 2006 information not available for pilchards. After Chiaradia et al. 2010.)

immunity (DeStewart et al. 1996). The best-documented case of mass mortality in marine mammals is the 1988 phocine distemper epizootic in North Sea harbor seals. The pathogen might have come with migrating harp seals. Surprisingly, mass mortalities were not more serious in large herds than in small herds of harbor seals, indicating that social aggregation at haulout sites provides enough contact to spread the virus to all seals in a group (Heide-Jorgensen and Harkonen 1992). About a third of the harbor seals in the North Sea died (Dietz et al. 1989), but the remainder became immune, and the pathogen became extinct from the North Sea (Swinton et al. 1998).

To conclude, the drama of mass mortalities suggests a system out of balance. This does not make mass mortalities unnatural, or human-induced, but it is worth considering some commonalities among our examples and contrasting them with the more typical cases in which diseases have either regulatory or inconsequential effects on host populations. Mass mortalities raise many questions: What are the source(s) of the parasites? Are these emergent diseases? What drives their rise and their fall? Is stress leading to immune-compromised hosts and opportunistic infections? Do intact ecosystems play a role in maintaining stability in host–parasite interactions? In several of our examples, the parasites associated with mass mortalities might be novel to a region. Such parasites might be introduced or aided by human activities (as suggested for herpesvirus in pilchards, *Serratia marcescens* in *Acropora* reef-building corals, and perhaps rickettsia in abalones), but it is often difficult to pinpoint the source of an invasion and distinguish it from an unrecognized parasite that somehow became virulent or easier to transmit. Indeed, it is now hypothesized that many new outbreaks of diseases in corals are caused by a change in the environment that makes an existing bacterium pathogenic (Bourne et al. 2009). Novel parasites are often associated with dramatic reductions in host population sizes, particularly if there is a tolerant reservoir host species or parasite input (like sewage) that acts as a reservoir for the parasite as other hosts decline or become extirpated (Lafferty and Gerber 2002). Regardless of their cause, mass mortalities can have large economic costs and put host species in danger of extinction.

Effects of Infectious Diseases on Marine Communities

We have shown how parasites affect individuals and populations of individuals. To what extent can these effects scale up to alter entire communities? There are two ways to consider this question: we can choose to think about the effects of parasites on communities of free-living species, or we can take one step further and consider parasites as members of communities in marine systems. In this section we discuss how parasites can increase or decrease coexistence among competitors. We then give examples of other indirect effects that can result when an infectious disease drives an important host to low densities or changes its behavior or distribution. We end with a review of how parasites can alter food web structure once we consider them equal players with free-living species.

Indirect effects of infectious diseases

The mystery of how similar competing species can coexist is a theme in community ecology and one that has received considerable attention from marine ecologists (being the subject of much of this book). Density-dependent regulation is a much-cited route to species coexistence, albeit more recognized in terrestrial ecosystems. The assumption here is that there is a cost to being common, which leads to a frequency-dependent disadvantage. In other words, strong competitors suffer from their own success, and this makes room for subordinate species. Due to density-dependent transmission, infectious diseases are a form of density-dependent regulation (Mordecai 2011). However, there are few examples of how this regulation works in marine systems. One highly recognized study found that a parasitic plant reduces the abundance of competitively dominant salt marsh plants, facilitating plant diversity in estuaries (Pennings and Callaway 1996). In addition, observations of phytoplankton communities suggest that phage dynamics can be frequency dependent, leading to a disadvantage for common species and to cycles in the phytoplankton community (Parsons et al. 2012). On the other hand, infectious diseases can decrease coexistence when there is variation in tolerance of infection among competing host species. When this occurs, less tolerant species can be at a disadvantage. If a competitive dominant is tolerant, exclusion is hastened, whereas if a subordinate is tolerant, coexistence can occur (Mordecai 2011). As mentioned on p. XX, a key to the mass mortalities of the black abalone in California might be that other abalone species are more tolerant of the disease, creating a reservoir for the pathogen where black abalone populations are extirpated and making it difficult for black abalone to coexist with other abalones.

In addition to affecting interactions among competing species, infectious diseases can have indirect effects up and down the food chain. These effects are most apparent after common species suffer mass mortality. We consider the potential indirect effects of the infectious diseases mentioned in the discussion of host population regulation on p. XX. We will show how the trophic level of the infected host affects the types of indirect effects associated with infectious diseases.

When a disease affects a plant or habitat-forming species, whole ecosystems can change. *Zostera marina* beds provide habitat for invertebrates, fish, and marine birds, stabilize coastal sediments, and filter terrestrial nutrients (de Boer 2007). The loss of eelgrass beds caused by eelgrass wasting disease led to the loss of critical nursery habitat for several fishes and migratory waterfowl (Hughes et al. 2002) and resulted in the presumed secondary ex-

tinction of the eelgrass limpet (*Lottia alveus*; Carlton et al. 1991). Losses of structure-forming species like corals are another example of broad community effects of infectious diseases. The potential damage inflicted by diseases on coral reefs is best exemplified by observations in the Caribbean, where successive disease outbreaks led to a restructuring of Caribbean coral reefs from *Acropora*- to *Agaricia*-dominated communities (Aronson and Precht 2001b). Declines in reef-building corals have in turn led to a flattening of the three-dimensional structure of reefs and a decrease in their capacity to provide shelter and other resources for reef-dependent species of fish and invertebrates (Alvarez-Filip et al. 2009).

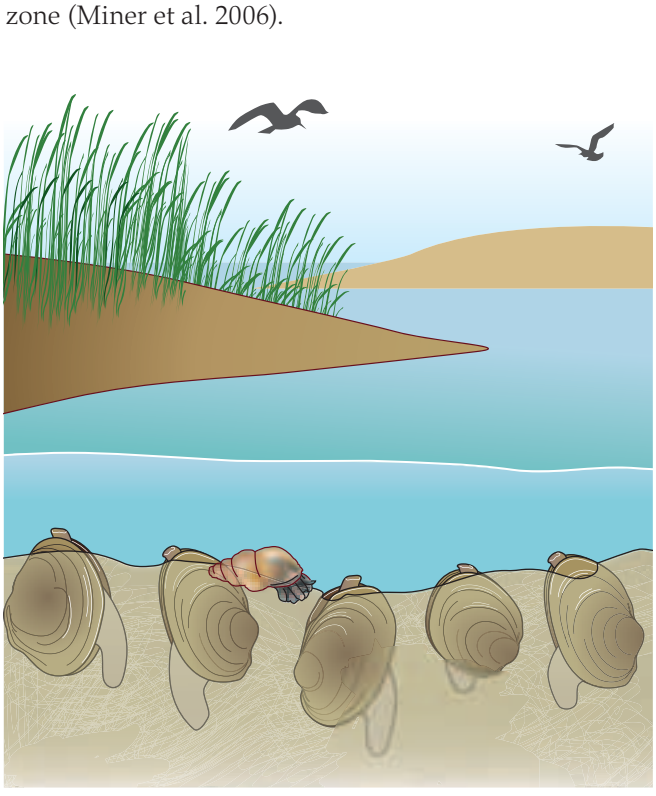
If a disease attacks an important grazer, plants might benefit. Because herbivorous fishes in the Caribbean were already overfished, the *Diadema* mass mortality caused widespread macroalgal blooms, and some even credit the epizootic for causing a phase shift from coral-dominated to alga-dominated reefs, although the influences in this phase shift were many (Hughes et al. 2010). Mass mortalities of the green sea urchin (*Strongylocentrotus droebachiensis*) in Nova Scotia associated with the parasitic amoeba *Paramoeba invadens* led to a phase shift from barrens to a kelp-bed state (Lauzon-Guay et al. 2009). Similarly, bacterial epizootics in California reduced sea urchin densities to allow kelp forests to persist in areas that might otherwise be urchin barrens (Behrens and Lafferty 2004). And mass mortalities of the black abalone released algae and other invertebrates from grazing and competition for space, changing the community structure of the rocky intertidal zone (Miner et al. 2006).

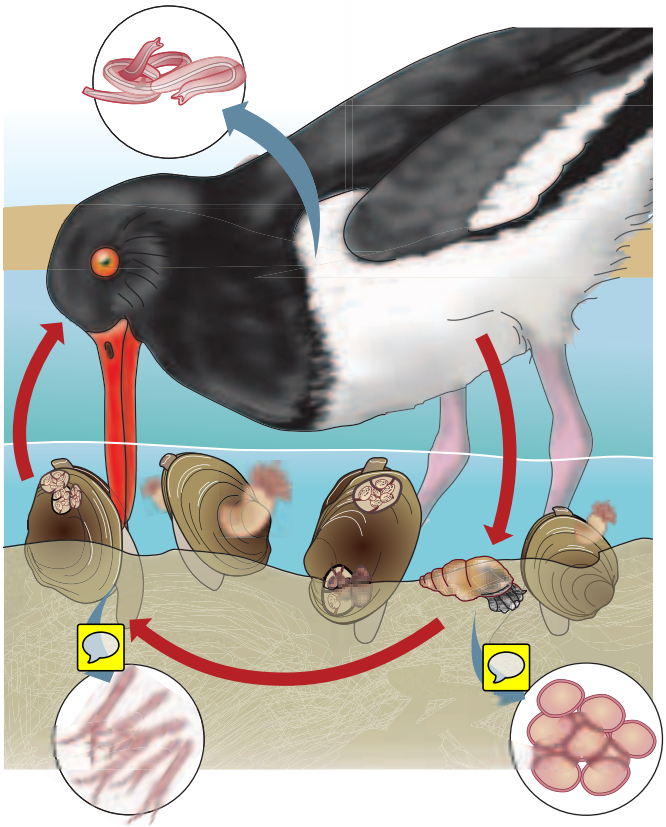
Mass mortalities could also affect predator–prey interactions. When a disease affects a food item, ~~it~~ might compete with upper trophic levels. For instance, mass mortality of pilchards led to reduced forage for little penguins, with correlated increases in mortality and decreases in ~~their~~ breeding success (Dann et al. 2000). Conversely, when a disease affects a top predator, prey might be released, with the potential for trophic cascades. Sea otters are well known as a keystone species because they feed on herbivorous invertebrates, reducing grazing pressure on kelp forests. If parasites impede the recovery of sea otters in California, ~~they~~ could indirectly reduce kelp forests.

Behavior

Parasites don't need to reduce host abundance to affect communities. Sometimes they alter the host's behavior so that it plays a new role in the ecosystem. One of the best examples comes from the mudflats of New Zealand (**Figure 5.14**), where the most noticeable species is a littleneck clam that can dominate the mudflat biomass (Hartill et al. 2005). The shells of dead and live clams may protrude from the sediment, creating a habitat for several epibionts (Thomas et al. 1998). With few alternative hard substrates, the provision of novel habitat makes this clam an ecosystem

Figure 5.14 Effects of a manipulative trematode on the community structure of a New Zealand mudflat. The parasite prevents its host clam from burrowing, facilitating a novel epibiont community. (After Lafferty and Kuris 2012.)





engineer (Thomas et al. 1998). But a trematode parasite is the actual engineer. Pied oystercatchers foraging on the mudflats carry adult trematode worms in their intestines. Two trematode genera use the clams as a second intermediate host, encysting in the tip of the foot (Babirat et al. 2004) and reducing the burrowing ability of the clams, thereby stranding them on the surface, where they become easy prey for oystercatchers (Thomas and Poulin 1998). Stranded shells also make substrate available for epibionts to colonize. In addition, infected clams dig less, which alters infaunal communities (Mouritsen and Poulin 2005). Without these manipulative parasites, clams would remain buried, and New Zealand mudflats would have less biodiversity.

Food webs

The food web is an ecological map for understanding complexity and a formal way to consider the architecture of species interactions in communities. We've mentioned how infectious diseases can affect food webs. What happens when we put parasites on equal par with free-living species in food web analyses? Parasites increase food web complexity. In particular, including parasites in food webs increases species richness and connectance (a measure of complexity). Furthermore, adding parasites means that large predators are no longer at the top of the food chain (**Figure 5.15**; Lafferty et al. 2006). Although we concern ourselves with parasitic stages here, free-living stages of parasites can be important sources of food in food webs (Johnson et al. 2010). Our view of food webs is incomplete without parasites (Lafferty et al. 2008a).

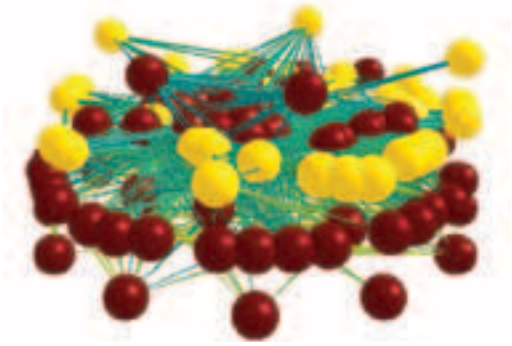


Figure 5.15 Parasites (indicated by yellow balls) are linked with free-living species (indicated by red balls) in an estuarine food web (i.e., Carpinteria Salt Marsh). The addition of parasites to this food web changed it in several ways (Lafferty et al. 2006). Obviously, there were more species. There were also longer trophic chains because parasites tend to be top consumers. The complexity of the web increased because parasites have, on average, more links than do other consumers. The structure of the network also became more nested. However, the food web became less robust to secondary extinctions because parasites were more sensitive to the loss of their hosts than predators were to the loss of their prey (Lafferty and Kuris 2009a). (Image produced with FoodWeb3D, written by R. J. Williams and provided by the Pacific Ecoinformatics and Computational Ecology Lab, www.foodwebs.org, Yoon et al. 2004.)

In summary, parasites affect communities through their effects on host populations and host behavior. Infectious diseases in the marine environment have caused community-wide and ecosystem-wide changes (Harvell et al. 2004; Sutherland et al. 2004). ~~Infectious diseases~~ can promote the coexistence of competitors by putting common species at a disadvantage. However, tolerance of infection by competitive dominants can reduce coexistence. By reducing host density, parasites indirectly affect other species that either depend on or are affected by their host. Therefore, a key to understanding the community-level effects of parasites is to ~~understand~~ the role of their host in the community. ~~Manipulative parasites can have unexpected and idiosyncratic effects on marine communities.~~ Finally, parasites are important to consider as members of communities, not just as forces that affect communities. They alter community complexity through food web interactions, and they are top consumers. Only when analyses put parasites on equal footing with other consumers will we be able to appreciate their role in marine communities.

Applied Aspects of Infectious Diseases

Scientists are trained not to take sides. But humans often value one species over another. For instance, humans eat marine species, and they want to keep catching and eating them. Infectious diseases can limit fisheries yields, cause valued or iconic species to become rare, or impair the recovery of endangered species. In this section we consider ~~infectious~~ diseases that infect marine species of human concern. We begin with a summary of the effects of parasites on fisheries and aquaculture. We then discuss the interactions between parasites and invasive species.

Fisheries and infectious diseases

Parasites can reduce the abundance of fished stocks, thereby competing with fisheries (Kuris and Lafferty 1992). The mass mortality of Australian pilchards caused by herpesvirus is estimated to have cost the fishery $5 million (in U.S. 2001 dollars); (Gaut 2001). Mass mortalities of the black abalone (in conjunction with overfishing) eliminated what could have been a multimillion-dollar industry (Parker et al. 1992). Fisheries models that consider interactions between disease, fishing, and abalone size structure could lead to more sustainable strategies for abalone fishing (Ben-Horin 2013). Herring fisheries are susceptible to environmentally sensitive diseases such as ichthyphoniasis and viral hemorrhagic septicemia (Kocan et al. 2004). Key considerations for managing fisheries subject to infectious diseases are the scales of recruitment of the infectious disease and the fished species~~; selective fishing can impair the spread of diseases and lead to increased yields~~ (Kuris and Lafferty 1992).

Marine protected areas (MPAs) are a ~~new~~ management approach for protecting fisheries from collapse and for protecting biodiversity and ecosystem function. New studies

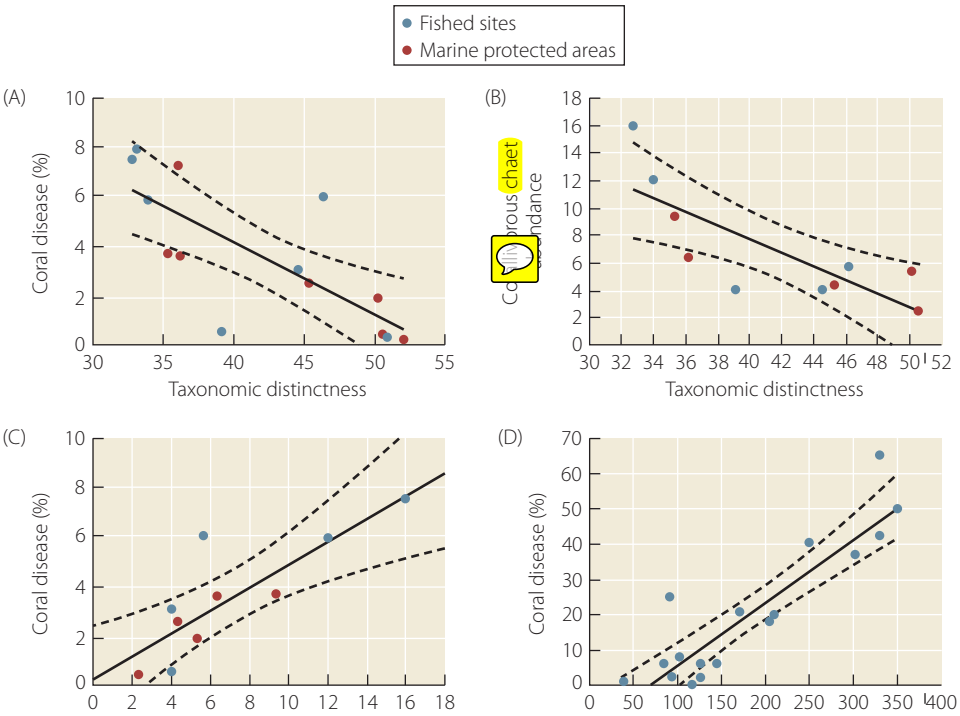


Figure 5.16 Relationships between coral disease and corallivorous fishes in MPAs and at fished sites. (A) Coral disease prevalence versus taxonomic distinctness of fishes across sites. (B) Abundance of corallivorous butterfly fishes versus taxonomic distinctness (which increases where species in a community are more distantly related to each other). (C) Coral disease versus abundance of corallivorous butterfly fishes, (D) Coral disease versus abundance of all butterfly fishes. (After Raymundo et al. 2009.)

show that coral disease can be less frequent in marine protected areas (Page et al. 2009; Raymundo et al. 2009). One hypothesis for the mechanisms at work is that without fishing, predation on corals by invertebrates and small corallivorous fishes is reduced by the presence of apex predators (**Figure 5.16**). Corallivores are both potential disease vectors and a source of wounding that can allow the entry of opportunistic coral diseases. Another hypothesis is that fishing activities outside of reserves damage coral, which can lead to secondary infections (Lamb in prep.).

Protected areas, by increasing host density, might also lead to increases in parasites of fished species (McCallum et al. 2005; Wood et al. 2010). For example, in New Zealand marine reserves, an increase in lobsters after protection led to an increase in bacterial shell disease in lobsters (Wootton et al. 2012). By now, it should not be surprising that the effects of marine reserves can depend on the details. Reserves decrease sea urchin infections in California (Lafferty 2004), but in the Galápagos, due to a difference in the food web, parasitism of sea urchins can be higher in reserves (Sonnenholzner et al. 2011).

In addition to reducing fish stocks, parasites can degrade the sale price of infected fish. Sealworms, a diverse group of larval nematodes that live in the flesh of many fishes and cephalopods, have this effect (**Figure 5.17**; McClelland 2002). These worms, which use seals and porpoises as final hosts, have increased in abundance following marine mammal protection. Sealworms can cause pathology in humans that eat undercooked or brined infected fish. Fortunately, the worms are easy to see and are killed by freezing and cooking. In the Netherlands, sealworm infections in humans were once acquired by eating pickled herring, but laws that require freezing herring before pickling have almost eliminated this disease there (Bouree et al. 1995). The main unavoidable economic impact of sealworms is that nobody wants to eat a piece of fish that has 1–3 cm worms embedded in it. Extracting nematodes from processed filets is time-consuming and drives up the cost of the final product, so the most infected fish stocks are no longer harvested. The fishing industry has advocated culling seals to reduce the exposure of cod and other fishes to sealworms, while conservation groups have opposed culling. Another example is the myxosporean parasite *Kudoa thyrsites* (Moran et al. 1999). This protozoan is not infective to humans, but it infects many marine fishes, forming cysts in their musculature. Heavy infections can damage the flesh, making it soft and unpalatable. The most frustrating aspect of this disease for the fishing industry is that it is difficult to determine flesh quality until after the fish has been killed and processed. Problems with *K. thyrsites* are highest in net pen–reared fish.

In aquaculture, high stocking density increases parasite transmission, and cages can create habitat for intermediate hosts and parasite eggs (Ogawa and Yokoyama 1998). As a result, the aquaculture industry has invested in aquatic health, turning to fish vaccines, antibiotics, and even biological control (cleaner fishes and probiotic bacteria). A key concern that fisheries and conservationists have about aquaculture is the extent to which cultured organisms export infectious diseases to wild stocks. For instance, abalone aquaculturists imported South African abalones whose shells were infested with a sabellid worm, and this worm ended up infesting wild gastropods in California (Culver and Kuris 2004). The abalone culture industry also releas-

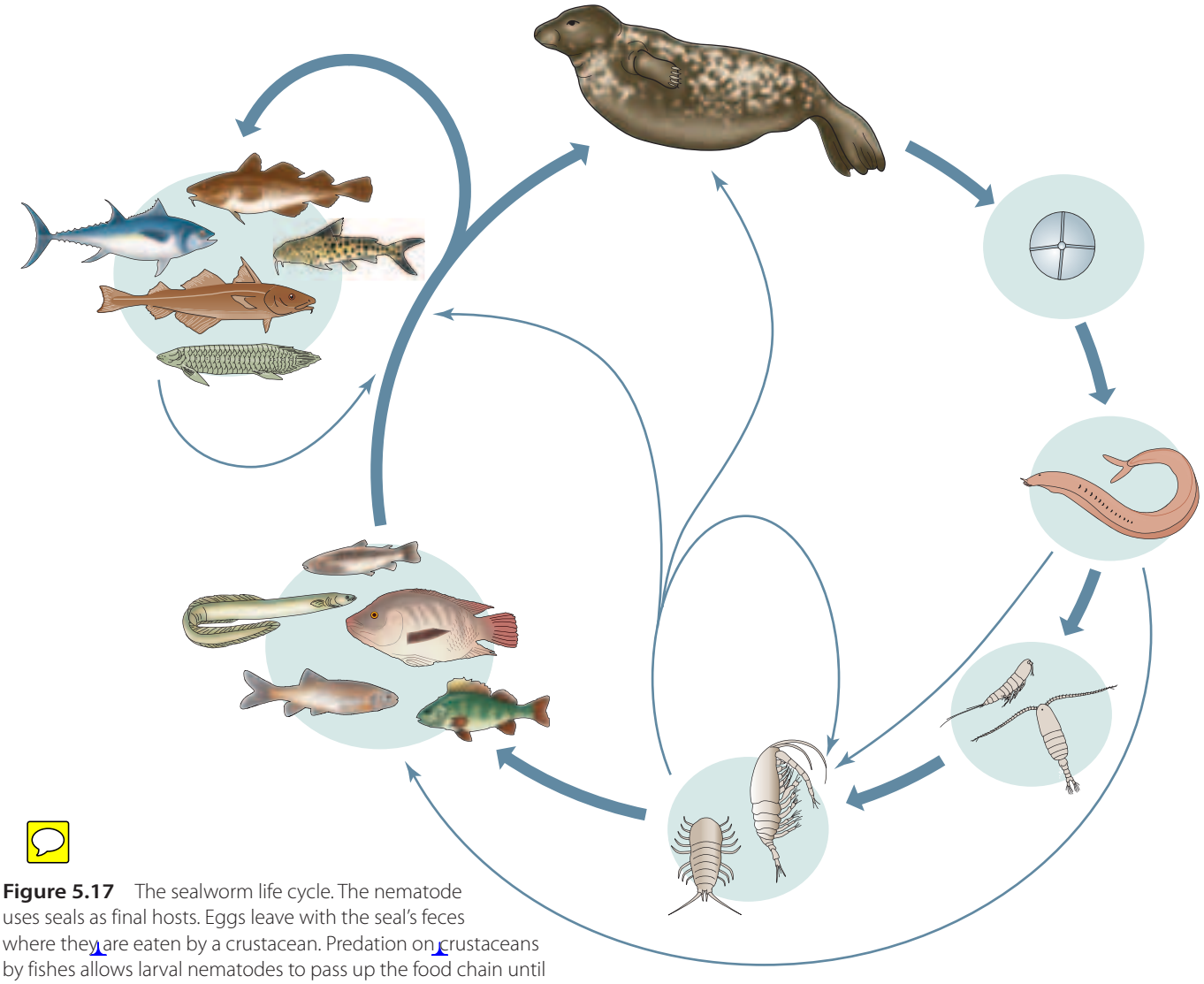


Figure 5.17 The sealworm life cycle. The nematode uses seals as final hosts. Eggs leave with the seal's feces where they are eaten by a crustacean. Predation on crustaceans by fishes allows larval nematodes to pass up the food chain until ~~they are~~ eaten by a seal. (After McClelland 2002.)

es a steady stream of WS-RLO into the wild (Ben-Horin and Lafferty ~~unpublished~~). Another prominent example is infectious salmon anemia virus (an RNA virus), which has caused extensive outbreaks in farmed Atlantic salmon in Norway, Scotland, and Chile and has been detected in British Columbia (Kibenge et al. 2001). The debate over diseases of aquaculture has been strongest for the fish copepod *Lepeophtheirus salmonis*. This species builds up in large numbers in penned salmon, raising concern about spillover to wild stocks (Krkosek et al. 2007) because infected fish suffer 40% higher mortality (Krkosek et al. 2012). Nowhere is the conflict between aquaculture and fisheries more clear (and litigious) than in Australia, where a herpesvirus released by onshore abalone culture spread into the native abalone population, leading to substantial losses (Hooper et al. 2007). In addition to developing technology to reduce the effects of diseases on aquaculture, steps should taken to buffer the spread of diseases from aquaculture facilities to wild populations.

Invasive species

Invasive species are an increasing threat to native ecosystems. In particular, shipping and oyster culture have led to the homogenization of bay and estuarine fauna. The types of natural enemies invasive species bring with them can affect their success in a new location. Invaders that are transported as larvae in ship ballast water are often free of parasites, and the few parasites that do come with them may find their new location unsuitable for completing their life cycle. On average, successful marine invaders bring only a few of their parasite species with them and don't pick up enough new parasite species to make up the difference; on average, marine invaders have only a third of the parasite species that they had in their native regions (Torchin et al. 2002). This is one reason why some marine invaders appear to do so well in introduced regions, sometimes at the expense of their native competitors or prey. Some harbors and bays now have a new dynamic—large numbers of invaders and few infectious disease agents to help limit their abundance (Foster 2012).

If it's bad that invasive species escape most of their parasites, the worse news might be that some marine invaders bring generalist infectious diseases with them. These new diseases can spill over to native species, which don't have the benefit of coevolved defenses. Examples of introduced parasites include rhizocephalan barnacles of crabs, copepods of bivalves, monogeneans of fishes, swim bladder nematodes of fishes, trematodes of snails, various protozoan parasites of oysters (Torchin et al. 2002), and a shell boring sabellid polychaete and herpesvirus of abalones. Of all the anthropogenic factors that can decrease the health of marine species, introductions are the most important to understand and attempt to prevent.

To summarize, marine diseases inconvenience humans when they reduce the abundances of fished species or degrade their market value. In turn, fisheries can reduce infectious diseases by reducing host density. In fact, the strongest trend in marine diseases over the last three decades appears to be a decline in reports of fish parasites (Ward and Lafferty 2004). Marine reserves established to protect fish stocks can therefore restore parasites to their natural level (whether that is higher or lower than in fished areas). Aquaculture, on the other hand, keeps hosts under conditions ideal for disease transmission and also provides routes for invasive parasites to enter natural systems.

CONCLUSION

Parasites are diverse elements of marine communities, and they affect marine communities. In return, infectious diseases are affected by the community ecology of their hosts. Community ecology can best further our understanding of infectious diseases by being aware of the variety of infectious disease agents in the ocean and the various ways in which they can affect and be affected by marine communities. New technology can help us to identify and study infectious diseases, and with better understanding, we might enact legislation, or provide therapeutics, that will better protect marine systems from introduced infectious diseases agents.

ACKNOWLEDGMENTS

We thank the editors for including a chapter on infections diseases in this textbook. Several of our colleagues read and commented on earlier drafts of this chapter. In particular, we thank the editors, anonymous reviewers, and C. Burge, C. Couch, C. Fong, A. Garcia, C. Kim, A. Kuris, and D. Morton. Any use of trade, product, or firm names in this publication is for descriptive purposes only and does not imply endorsement by the U.S. government.

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