CHAPTER 7

Parasitism and environmental disturbances

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Several new diseases have gained celebrity status in recent years, fostering a paradigm that links environmental stress to increased emergence of disease. Habitat alteration, biodiversity loss, pollution, climate change, and introduced species are increasing threats to the environment that are postulated to lead to emerging diseases. However, theoretical predictions and empirical evidence indicate environmental disturbances may increase some infectious diseases but will reduce others.

7.1 Introduction

To build a predictive framework for how environmental disturbances can affect parasitic diseases, we limit our scope to those environmental disturbances that result from human activities. Anthropogenic change that may affect parasite communities can be divided into five broad types: habitat alteration, biodiversity loss, pollution, climate change, and introduced species. We do not limit ourselves to the facile prediction that environmental change will lead to increases in parasitism. As we will make clear, there are substantial theoretical and empirical reasons to expect the opposite will also often result from such changes.

With the possible exception of invasive species, environmental disturbances can collectively be considered as stressors (Lafferty and Kuris 1999). Perhaps the first thing that comes to mind when one thinks about the effect of stress on disease is our own health. Studies link stress to reduced immune function and various associated maladies of the modern age (Yang and Glaser 2002). Immune systems are costly to maintain and stressed individuals may lack sufficient energy to mount an effective defence (Rigby and Moret 2000), making them more susceptible to opportunistic infections (Scott 1988; Holmes 1996). But stress is not just fretting about how to make an unreasonable deadline or frustration over being late for an appointment while sitting in stalled traffic. Toxic chemicals (Khan 1990), malnutrition (Beck and Levander 2000), and thermal stress (Harvell *et al.* 1999) are all examples of stressors hypothesized to increase individual susceptibility to infectious diseases. This line of thought suggests that environmental stress should aggravate infectious disease. An opposing prediction emerges if one considers population dynamics. Abundant species have more parasites (Arneberg *et al.* 1998*a*). The likelihood and impact of an epidemic increases with host density because density determines contact rates between infected and uninfected individuals (Stiven 1964; Anderson and May 1986). Infectious agents require a threshold host density for transmission (McKendrick 1940). Outside stressors that reduce host vital rates will depress host population density, thereby reducing the chance of an epidemic process, or even the ability of a parasite to persist at all in a declining or low density population.

Stressors may also induce a more negative impact on parasites than on their hosts. This should

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increase recovery rates of infected individuals and mitigate the population-level impacts of the disease. In addition, infected hosts might experience differentially high mortality when under stress. This would remove parasites more rapidly from the host population than would occur without the stressor. While this increases the impact of disease on infected individuals, it simultaneously decreases the spread of an epidemic through the host population. Such a relationship underscores the point that population effects of stress and infectious disease cannot necessarily be predicted from their effects on individuals.

It is more likely that stress will have multiple effects on hosts and parasites such as increasing host susceptibility to disease while impairing host vital rates. This makes it unclear how a particular stressor should affect disease in a host population. Although stressed individuals should be more susceptible to infection if exposed, the stressor will likely also reduce the contact rate between infected and uninfected individuals to the extent that the stressor reduces host density. Simulation models help resolve the opposing predictions stemming from these alternative effects. Stress is most likely to reduce the impact of closed system, host-specific infectious diseases, and increase the impact of other types of disease (Lafferty and Holt 2003).

7.2 Habitat alteration

Humans have altered nature in ways that can affect diseases (Lafferty and Kuris 1999) (see also Chapter 10). Conversion of forest to agricultural land dramatically changes the environment for parasites and their hosts; and this has raised concerns for human health (Patz *et al.* 2000). In particular, deforestation, damming, road construction, fish farming, and rice farming increase malaria transmission by creating mosquito breeding habitat (Smith 1981; Desowitz 1991). In addition, domestic animals may provide new food sources for mosquitoes, leading to increased malarial transmission in the associated human population (Giglioli 1963).

Habitat alteration has also created conditions conducive for the transmission of trematodes. For instance, dumps and fish farms attract seagulls which fuel trematode life cycles (Kristoffersen 1991; Bustnes *et al.* 2000). Increases in trematodes are of particular concern for those trematode species that cause human disease. Deforestation reduces acidic leaf litter and increases algal growth in ponds and streams, creating conditions suitable for snails that serve as intermediate hosts for schistosomes (Southgate 1997). The Aswan Dam that created Lake Nasser also created excellent habitat for the snails serving as the intermediate host for the trematodes that cause human schistosomiasis (Heyneman 1979). Construction of other large impoundments throughout Africa (e.g. Paperna 1969) has substantially increased schistosome transmission, resulting in increased human morbidity and mortality (Gryseels *et al.* 1994).

Due to concerns for human health, the literature tends to focus on the types of habitat changes that increase disease. However, there are many ways that habitat alteration, through its effects on biodiversity loss, should decrease infectious disease (as discussed below). In particular, the wholesale draining and conversion of wetlands has dramatically reduced the transmission of various infectious diseases (Lafferty and Kuris 1999; Reiter 2000). Management of water sources for breeding mosquitoes, through drainage and controlled water levels, was instrumental in the successful malaria control campaigns in the southern United States and Israel/Palestine (Kitron 1987).

7.3 Biodiversity loss

Although authors disagree on the present rate of extinction associated with human induced environmental degradation, there is no denying that it is orders of magnitude above background levels (Regan 2001). None of these estimates considers extinctions of parasites which, for some host groups, may exceed the extinction rate of host species (Sprent 1992). Few will lose sleep over the notion of parasites going extinct but one only need imagine the diversity of now extinct parasites specializing on dinosaurs (Kuris 1996) to realize that parasite extinction has been a vast, but hidden, component of evolutionary history (see also Chapter 6). In addition, given the possible role of parasites in stabilizing ecosystems (Freeland and Boulton 1992) conservation biologists may one day come to appreciate the

potential need to protect parasites (Combes 2001, see also Chapter 8). Two caveats are: (1) many parasites are not strictly host specific and the fates of these parasites are not tied to the extinction or persistence of single host species; and (2) parasites, due to the nature of density-dependent transmission dynamics, are likely to go extinct well before their hosts (Lyles and Dobson 1993). For this reason, host extinction may not be the key for understanding parasite extinction. Reduced host species densities and host species ranges are more likely to be good predictors of parasite losses.

As mentioned previously, a decline in host density below a transmission threshold can cause host specific infectious diseases to go locally extinct. The number of species put on endangered lists is a good example of cases where host densities have been reduced to such low levels that parasite extinction seems likely. Sometimes, we have enough evidence for wide-scale declines in whole groups of taxa or habitats. For instance, amphibians (Houlahan *et al.* 2001) and British birds (Balmford *et al.* 2003) are now thought to be at substantially lower densities than during prior decades. Populations of monitored species from a wide range of taxa have declined appreciably in marine, estuarine, and freshwater ecosystems; this is likely because aquatic habitats are particularly susceptible to the sort of degradation that leads to reductions in host densities (Balmford *et al.* 2003).

About half of the primordial terrestrial habitats have been cleared or converted to human use (Balmford *et al.* 2003). However, habitat loss does not necessarily translate into reductions in host density if the remaining habitats are not degraded. Under this condition, disease transmission would be maintained. Transmission might even increase, at least temporarily, if habitat loss leads to crowding in the fragmented remaining habitats (Holmes 1996). Despite this potential maintenance of transmission on a local scale, habitat contraction and fragmentation reduces the geographic range of host species. Since most parasite species exploit a host only over a subset of the host's range, we predict that host range contraction will eliminate a proportion of the parasite species from a host species. A better understanding of the rate at which parasite communities change over the landscape would provide more insight into this potentially large effect.

Parasites, particularly those with complex life cycles, should generally decline with a decrease in biodiversity (Robson and Williams 1970; Pohley 1976; Hughes and Answer 1982; Hudson *et al.* 1998). Digenetic trematodes are a good example. Trematode communities can vary considerably within a wetland (Lafferty *et al.* 1994; Stevens 1996; unpublished thesis) and among wetlands (Lafferty *et al.* 1994; Huspeni 2000, unpublished thesis). This is likely a direct consequence of the biodiversity of final hosts that use a particular area. A healthy marsh ecosystem provides rich feeding grounds and habitat for dozens of species of birds that act as definitive hosts for 20+ species of trematodes. The primary first intermediate host for the trematodes, the California horn snail, occurs throughout the marshes (Lafferty 1993*a*,*b*). Huspeni and Lafferty (in press) found that degraded sites in an estuary had fewer trematode infections and lower species richness relative to undisturbed control sites. This seems most likely because estuarine birds, the definitive hosts, should be less abundant and diverse in disturbed areas (Kuris and Lafferty 1994; Lafferty 1997). Correlations between trematode species richness and bird species richness at different sites in an estuary (Hechinger and Lafferty in review) and demonstration that the addition of bird perches to an estuary leads to increased prevalence of trematodes in snails (Smith 2001), further support the hypothesis that functioning ecosystems facilitate parasite communities.

Deforestation, particularly clear cutting, along lakes and streams, also leads to a significant decrease in the prevalence of trematodes and other parasites with complex life cycles. In the most heavily cut watersheds, rates of fish parasitization declined to the extent that only unparasitized fishes were present in those lakes (Marcogliese *et al.* 2001). Since other parasites with direct life cycles (copepods and monogenes) actually increased in the most impacted lakes, this supports the hypothesis that biodiversity maintains parasites with complex life cycles in ecosystems.

These effects can be seen over time as well. A decline in trematode species richness at Douglas

Lake, Michigan, is postulated to result from half a century of increasing human disturbance, and an associated reduction in shorebirds (Cort *et al.* 1937; Keas and Blankespoor 1997). Habitat restoration can generate the same pattern, but in reverse. Restoration of degraded salt marsh was followed by an increase in trematode prevalence and species richness so that after 7 years trematode communities at restored sites were comparable to control sites (Huspeni and Lafferty, in press). In addition to providing substantial evidence for the link between biodiversity and parasites, these studies indicate how parasites can be used to monitor changes in the environment over time (Lafferty 1997).

The cessation of hunting and other protections has favoured many marine mammal species. In the United States and elsewhere, regulations such as the Marine Mammal Protection Act of 1972 fully protect pinniped populations and these have soared (Stewart and Yochem 2000). Not surprisingly, the prevalence and intensity of larval anasakid nematodes in fish that use marine mammals as final hosts increased when and where seals became common (Chandra and Khan 1988). The combination of increased susceptibility due to stressors, and increased population density due to marine mammal protection regulations suggests that marine mammals are one group in which host specific diseases will increase.

In contrast, fishing and hunting can reduce populations of targeted species, even to extinction. Reduction in seal populations that are still hunted is expected to reduce the intestinal nematode parasites of seals by reducing host-density thresholds (Des Clers and Wootten 1990). Recent studies show how fishing has dramatically reduced populations of many species across the globe (Jackson *et al.* 2001; Myers and Worm 2003). In depleting a stock, a fishery can 'fish out' a parasite. This is possible if the fishery takes the population below the host density threshold for the parasite and can even be profitable if the host threshold density is higher than the density for Maximum Sustainable Yield (Dobson and May 1987). Fishing out a parasite at a local scale is most probable for host-parasite interactions where the parasite has a recruitment system that is relatively closed compared to the recruitment of its host (Kuris and Lafferty 1992). For example, in the Alaskan red king crab (*Paralithodes camtschatica*) fishery, nemertean worms can consume nearly all crab eggs in some areas. Nemerteans develop rapidly, are probably transmitted locally and king crab larvae disperse widely. Hence, fishing king crabs intensively (including females) in certain fjords has the potential to extirpate the nemertean locally in those fjords (Kuris *et al.* 1991).

Several examples illustrate the potential to fish out parasites. Fishing reduces the prevalence of the tapeworm, *Triaenophorus crassus*, in whitefish, *Coregonus lavaretus,* (Amundsen and Kristoffersen 1990) and has apparently extirpated a swim bladder nematode from native trout in the Great Lakes (Black 1983). Similarly, the prevalence of a bucephalid trematode in scallops declined from 50–70% (Sanders 1966) to 1–2% (Sanders and Lester 1981) following intensive fishing of scallops and of the final host, the leatherjacket filefish. These examples suggest that parasites of fished species should be declining over time. In contrast, a fishery may be inadvertently managed to increase parasite populations (Lafferty and Kuris 1993). In some cases, as happened with bitter crab disease, fisheries can spread parasites by releasing infected animals because they cannot be marketed (Petrushevski and Schulman 1958). Further, inadvertent management, by targeting unparasitized stocks, can also protect parasites in the unharvested infected stocks. This may be able to sustain parasite populations that might otherwise collapse as host abundance is greatly reduced in efficient fisheries. For example, some fishermen avoid areas where fish have high intensities of sealworm, because this reduces the value of the catch (Young 1972). Fishing practices may unintentionally protect parasites. Crab fisheries often protect reproductive output by releasing trapped females. This protects parasites of females or parasites that feminize males (nemertean worms, rhizocephalan barnacles) (Kuris and Lafferty 1992).

While removal of top predators may break transmission of parasites with complex life cycles it can also have indirect positive effects on some diseases of prey populations (Hochachka and Dhondt 2000; Jackson *et al.* 2001). At the California Channel Islands, lobsters historically kept urchin populations

at low levels and kelp forests developed in a community-level trophic cascade (Tegner and Levin 1983). Where lobsters were fished, urchin populations increased and they overgrazed kelps (Lafferty and Kushner 2000). In 1992, an urchin-specific bacterial disease entered the area where urchin densities well exceeded the host-threshold density for epidemics (Lafferty in press). This study found that epidemics were more probable and led to higher mortality in dense urchin populations. Hence, this bacterial disease acted as a density-dependent mortality source. Another example may be the removal of sea otters and Native Americans as black abalone predators on the Channel Islands in the 1800s. This facilitated an increase in black abalone populations to great abundance which then enabled a previously unknown rickettsial disease to cause a catastrophic collapse of the black abalone populations (Lafferty and Kuris 1993). These examples show how fishing top predators can favour disease transmission in prey populations (Hochachka and Dhondt 2000). Indeed, this may be the major cause of increased diseases in marine organisms at lower trophic levels, rather than climate change (Jackson *et al.* 2001). Predator removal is a management strategy sometimes used to protect livestock or increase wild prey populations of conservation concern or (because they are endangered or hunted for sport) (Packer *et al.* 2003). Mathematical models find that this practice can inadvertently increase the incidence of parasitic infections, reduce the number of healthy individuals in the prey population and decrease the overall size of the prey population, particularly when the parasite is highly virulent, highly aggregated in the prey, hosts are long-lived, and predators formerly selected infected prey (Packer *et al.* 2003).

7.4 Pollution

Pollution interacts with parasitism in complex ways, making it difficult to generalize broadly about its effects on disease (Lafferty 1997). This is most clear in reviews of parasites of fishes (MacKenzie *et al.* 1995). Some pollutants are toxins and these can impair host immune systems and host vital rates. Pollutants may also impair parasite vital rates and some may even preferentially concentrate in parasite tissues (Sures *et al.* 1997). However, sometimes parasites have reduced levels of toxicants in their tissues (Bergey *et al.* 2002). These possibilities lead to a diverse set of predictions about the effect of toxic pollutants on parasites (Overstreet and Howse 1977). However, specific predictions for some parasite–pollution pairs are possible.

Perhaps the best case for a link between toxic pollution and an increase in infectious disease is from parasitic gill ciliates and monogenes of fishes (Khan and Thulin 1991). Intensities and prevalences of ciliates increase with a wide range of pollutants (Lafferty 1997). This appears to be due to an increase in host susceptibility. Toxins somehow impair mucus production which is a fish's main defence against gill parasites (Khan 1990).

Marine mammals have the potential for interactions between pollutants and increased susceptibility to parasites. As top predators, marine mammals bioaccumulate lipophillic toxins that can be broadly pathogenic (O'Shea 1999). These contaminants can affect the mammalian immune system (Swart *et al.* 1994); for example, harbour seals fed fish from polluted areas have lower killer cell activity, decreased responses to T and B cell mitogens and depressed antibody responses (DeStewart *et al.* 1996). In seals, such immunosuppression may be a cofactor in the pathology associated with morbillivirus (Van Loveren *et al.* 2000), Phocine Distemper (Harder *et al.* 1992), Leptospirosis and calicivirus (Gilmartin *et al.* 1976). Similarly, marine contaminants may increase sea otter susceptibility to infectious diseases (see Lafferty and Gerber 2002).

Toxic chemicals have a consistent negative effect on helminths (Lafferty 1997). For example, selenium is more toxic to tapeworms than to fish hosts (Riggs *et al.* 1987). Free-living stages of parasites may be particularly sensitive to toxins (Evans 1982). Trace metals kill free-living trematode cercariae and miracidia, reducing infection rates of snails in polluted waters (Siddall and Clers 1994). This can help otherwise heavily infected snail species compete with other species, greatly altering snail communities (Lefcort *et al.* 2002). Additionally, if infected hosts are differentially killed by pollution, the parasite population

will decline (Guth *et al.* 1977, Stadnichenko *et al.* 1995), further reducing prevalence. For instance, cadmium kills amphipods infected with larval acanthocephalans more readily than it kills uninfected amphipods (Brown and Pascoe 1989). In addition, pollution can negatively affect fish vital rates. For example, oil pollution causes liver disease and reduces reproduction and growth (Johnson 2001). Such effects should reduce density and contact rates, further reducing parasitism.

In contrast to toxic pollution, eutrophication and thermal effluent often raise rates of parasitism in aquatic systems because the associated increased productivity can increase the abundance of intermediate hosts. Parasites that increase under eutrophic conditions tend to be host generalists and have local recruitment; cestodes with short life cycles and trematodes seem to be particularly favoured (Marcogliese 2001). The most dramatic examples include parasites whose intermediate hosts favour enriched habitats. These include some species of tubificid oligochaetes and snails. Myxozoan parasites of fishes, which require oligochaete hosts, are frequently more prevalent at sites polluted by sewage (having high coliform counts) (Marcogliese and Cone 2001). Beer and German (1993) described how eutrophication improved conditions for snails that serve as first intermediate host for the digene, *Trichobilharzia ocellata*. Similarly, Valtonen *et al.* (1997) found that eutrophication correlates positively with greater overall parasite species richness in two fish species. An increase in frog deformities has been linked to eutrophication of ponds which increases the density of snails infected with *Ribeiroia ondatrae*, a trematode known to cause abnormal growth in second intermediate hosts (Johnson *et al.* 2002). The association between eutrophication and pollution is not likely to be linear. At high nutrient inputs, toxic effects may occur and parasitism can decline (Overstreet and Howse 1977). The influence of pollutant stressors, must be analysed in the context of natural history. Some tubificids require clean water and will not be present at enriched sites (Kalavati and Anuradha 1992).

Evaluating the changes in the fish parasitofauna of oligotrophic and eutrophic lakes in Michigan, Esch (1971) recognized that eutrophication opens up the scale of interactions in an aquatic ecosystem. As biomass increases due to increased productivity, birds and mammals increasingly feed at enriched sites. Hence, snails and fishes acquire increasing numbers of larval parasites that will be trophically transmitted to the non-piscine top predators. In oligotrophic lakes, some of these same fishes are the top trophic level and harbour mostly adult parasites. Since larval parasites are more pathogenic than adult parasites there will be a further cascade of disease effects on a eutrophic ecosystem.

Acid precipitation associated with air pollution can negatively effect parasites in waters with poor buffering capacity. Marcogliese and Cone (1996) found that yellow eels (*Anguilla rostrata*) from Nova Scotia have an average of 4 parasite species at buffered sites, about 2.5 parasite species at moderately acidified sites, and 2 parasite species at acidified sites. This decline in parasite richness with acidity is due to drops in the prevalence of monogenes and digenes. The latter require molluscs as intermediate hosts and these cannot survive in acidified conditions. Parasites that use freshwater crustaceans as intermediate hosts may be similarly impacted by reduced access to calcium ions.

7.5 Climate change

The most notable prediction of anthropogenic global change is widespread increases in average temperatures (Houghton *et al.* 1996). This is particularly troubling to most parasitologists from temperate climes because many of the most deadly human parasitic diseases we teach about, but are not at direct personal risk to, are tropical (Rogers and Randolph 2000). The fear is that if our world becomes more tropical, tropical diseases will go hand in hand with the more benign benefits of pleasant weather. This is a bit simplistic; forecasts of climate change do not predict that the weather in Milwaukee will necessarily resemble that in Manaus. Still, there is a general expectation that temperatures will rise and precipitation patterns will change. The distributions of parasites, as for all species, are bounded by suitable climatic conditions. Thus, climate change should alter the future distribution of parasitic disease (Marcogliese 2001).

Some parasites should be more sensitive than others to warming. Temperature would seem particularly important when hosts are ectotherms that do not actively regulate their temperature. In addition, parasites with free living stages should have more opportunity to interact with climatic conditions (Overstreet 1993). For example, trematodes of littorine snails that have free swimming cercarial stages are not able to persist in arctic regions, presumably due to the effect of harsh weather (Galaktionov 1993).

Moderate increases in temperature are likely to alter birth, death, and development rates in ways that could conceivably favour parasites or intermediate hosts. For example, if individuals are infectious for longer time periods under warmer conditions, then disease will increase with temperature. The impact of parasites on their hosts may increase with temperature if parasites are, as a result, able to grow more or mature more rapidly (Chubb 1980). More complicated situations arise in vector-borne diseases where increased temperature may simultaneously increase pathogen development and vector mortality rates (Dye 1992). Much of the research on the effects of temperature on disease concerns fungal pathogens of plants. In general, fungal pathogens induce most damage to their plant hosts at warm (but not too warm) temperatures and at high humidities.

Studies of seasonal variation in parasites provide insight into the effect of temperature. Direct life cycle parasites (such as some monogenes) may be able to reduce generation times in warm water, leading to increases in these parasites (Pojmanska *et al.* 1980). However, aquatic helminths vary in their optimal temperature (Chubb 1979), making it impossible to make a general prediction about the effect of warming. The cestode *Cyathocephalus truncatus* has poor establishment success in trout if the water is warmer than 10 °C (Awachie 1966), presumably because host resistance is stronger at warm temperatures (Leong 1975). Other parasites with complex life cycles may be favoured by warming. Trematode cercariae are released from snails only when the water is warm (Chubb 1979), suggesting that the season for completion of trematode life cycles will be prolonged under global warming scenarios, a prediction borne out by observations of parasite communities in a thermal effluent (Sankurathri and Holmes 1976). Nonetheless, it is hard to predict the effect of warming on the parasite community as a whole. In one case where this has been studied, parasite communities in turtles declined with increasing thermal pollution (Esch *et al.* 1979).

Most fitness traits for hosts and their parasites will exhibit a peak performance at a thermal optimum. If the relationship between performance and temperature differs between host and parasite, the resulting gene by gene by environment interaction will either increase or decrease disease at a given temperature, at least on the level of the individual host (Elliot *et al.* 2002). For example, the optimal temperature of a fungal pathogen is higher than the optimal temperature of its sea fan host, placing the sea fan at risk to global warming (Alker *et al.* 2001). But the evidence does not always suggest that warming will increase parasitism. Insect hosts gain several advantages with moderate increases in temperature. Haemocyte production increases and this promotes general defences (Ouedraogo *et al.* 2003). The ability to encapsulate parasitoid eggs (and presumably other foreign bodies) increases (Blumberg 1991). Pathogenic fungal cells lyse at high temperatures, enabling insect host recovery (Blanford *et al.* 2003). So, in contrast to the general assumption that parasitism should increase with temperature, there is a general trend for less parasitism at higher temperatures, at least for insect hosts (Thomas and Blanford 2003). Some hosts use this to their advantage by changing their behaviour to increase body temperature in an effort to fight infections (Elliot *et al.* 2002). This suggests that warming may release some insect pests from their parasitic natural enemies, potentially leading to a variety of economic and ecological impacts. If climate change increases the abundance of insects that transmit diseases, there may be a subsequent increase in the spread of diseases such as malaria (see below).

Precipitation is another aspect of climate that may change with environmental degradation. Increased precipitation should favour parasites (e.g. trematodes) that have an aquatic phase. Outbreaks of water-borne diseases may increase with climate change (Shope 1991), as these are linked to periods of

increased rainfall (Curriero *et al.* 2001; Pascual *et al.* 2002). This should also result in increases in parasites that require vectoring by biting arthopods with juvenile aquatic stages (particularly mosquitoes, but also black flies). Despite these direct effects of precipitation, some scenarios do not predict increases in aquatic habitat with increased precipitation because increased temperature may increase evaporation even more than precipitation (Schindler 2001). Although, in some areas, humidity associated with increased precipitation should favour some parasites, especially nematodes transmitted by eggs or with free-living juvenile stages, elsewhere, higher temperatures will dessicate soil (Kattenberg *et al.* 1996). Increased aridity should impair the transmission of parasites with stages that live in soil.

There are important differences in the effect of climate change on aquatic and terrestrial systems. The first obvious difference is that atmospheric humidity is irrelevant in aquatic systems. This means that free-living stages of fully aquatic parasites are less likely to be affected by some aspects of climate change. The second difference has to do with respiration. Because the ability of gas to dissolve in liquid decreases with temperature, warmer water contains less oxygen. This, coupled with the fact that ectotherms have increased metabolic demands at high temperature, suggests that increases in temperature can place aquatic species under respiratory stress. The extent to which hosts or parasites are differentially sensitive to such stress has not been studied to our knowledge but we suspect that hosts, particularly infected hosts, will, on average, be at a greater disadvantage as temperatures rise and less oxygen is available. For example, high temperatures promote rapid reproduction of gill parasites that impair respiration at a time when oxygen is limited (Pojmanska *et al.* 1980). Also, marine snails, infected with larval trematodes, had elevated mortalities under reduced oxygen conditions (Sousa and Gleason 1989). Once again, the ecological consequence of this interaction may be to decrease or eliminate parasites from such populations by increasing parasite mortality.

Global warming could shift ranges of parasites poleward. For example, along the Atlantic coast of the United States, northward expansion of the protozoan *Perkinsus marinus*, which causes Dermo disease in oysters, is associated with increases in winter water temperatures, greatly expanding the economic impact of this disease (Cook *et al.* 1998). One likely ramification of increased temperature and precipitation is a shift in the distribution, and a probable expansion of the geographic range of mosquitoes and other haematophagous insects that serve as vectors for infectious disease (Shope 1991; Dobson and Carper 1993). The potential for malaria to expand is probably the most feared health consequence of climate change (Patz *et al.* 2000). The present distribution of malaria in tropical areas and reports of increasing outbreaks of malaria (Mouchet and Manguin 1999; Guarda *et al.* 1999; Keystone 2001; Hay *et al.* 2003), in conjunction with concern over warming, has prompted fear that current and future warming will expand malaria's distribution. This hypothesis recognizes that variation in malaria transmission is associated with climate. In Venezuela and Colombia, malaria mortality and morbidity predictably increase following El Niño events (Bouma and Dye 1997; Poveda *et al.* 2001). Modellers have used the associations between climate and mosquito distributions along with predicted patterns of climate change to further predict that the potential for malaria transmission will greatly expand in the future (Martens *et al.* 1999). This concern has attracted widespread public attention. However, other models using multivariate approaches to consider a range of factors find that the distribution of malaria is unlikely to expand as a result of global climate change (Rogers and Randolph 2000). In this regard, recall that malaria was once endemic in relatively temperate areas of the Americas and Europe (Reiter 2000), suggesting that climate, *per se*, is not the best predictor of future malaria distribution (Dye and Reiter 2000). Instead, the abandonment of vector control programmes coupled with the evolution of drug resistance by the parasite and pesticide resistance by the vectors are much more likely reasons for the current and future spread of malaria (Hay *et al.* 2002).

7.6 Introduced species

Humans import animals and plants for pets and agriculture. Many of these are raised near wild

species or have escaped to form feral populations. In addition, humans intentionally release species for hunting and fishing, plant them for dune stabilization and use them for biological control. Global trade and travel accidentally introduced many additional species (Ruiz *et al.* 2000). When exotics bring infectious agents with them, they may expose similar native hosts that have no evolved defences to new diseases. Some species have invaded or were introduced without their parasites and are apparently not susceptible to local parasites (Torchin *et al.* 2002, 2003), while others may bring with them a subset of their native parasite fauna (e.g. Lyles and Dobson 1993; Lafferty and Page 1997) (see also Chapter 3). Lafferty and Gerber (2002) recently reviewed published records of infectious diseases of conservation concern. For common native species that were decimated by an epidemic, the source of the disease was usually novel and was first recognized as a pathogen of the species during the epidemic. Sources for these epidemics were usually intentionally introduced species. Most of these diseases have broad host specificity and are less severely pathogenic in their original and abundant (exotic) hosts (McCallum and Dobson 1995; Woodroffe 1999; Gog *et al.* 2002). Relatively low virulence in their coevolved hosts has contributed to poor management decisions concerning the spread of an avian malaria with introduced wild turkeys (Castle and Christensen 1990). Chestnut blight (a fungus introduced with Chinese chestnut trees) is infamous for killing nearly every American chestnut tree. Infectious diseases from domestic sheep have extirpated populations of bighorn sheep (Goodson 1982) and rinderpest (brought to East Africa with cattle) has devastated native ungulates (Dobson 1995*a*,*b*; see also Chapter 8). A monogene was introduced into the Aral sea along with the Caspian stellate sturgeon; this parasite infected the gills of the native spiny sturgeon, leading to mass mortalities of this naïve host (Dogiel and Lutta 1937). Whirling disease, presumed to have originated with introduced European trout, has spread from stocked trout to native trout in North America, with severe consequences for native populations (Bergersen and Anderson 1997; Gilbert and Granath 2003). Canine distemper virus (originating from domestic dogs) led to the death of 35% of the lions in the Serengeti (Roelke-Parker *et al.* 1996) and has created problems for several other species at risk (Lafferty and Gerber 2002). Similarly, parapox virus may play a crucial role in the replacement of red squirrels by grey squirrels in Great Britain (Tompkins *et al.* 2003). Perhaps the most tragic example of an introduced vector is the night mosquito in Hawaii which permitted avian malaria to exterminate several malaria-sensitive endemic bird species in the lower altitudes where the mosquito lives (Warner 1968). Finally, an introduced tachinid parasitoid uses abundant exotic gypsy moths as hosts without sufficiently controlling those forest pests. Spillover from the gypsy moth reservoir has led to substantial declines of native North American moths (Boettner *et al.* 2000).

Non-indigenous species are an increasingly common component of estuarine systems (Cohen and Carlton 1998). One of these invaders, the European green crab, *Carcinus maenas*, and its parasites have been well studied. Torchin *et al.* (2001) found that the catch per unit effort of green crabs in their native range (Norway to Gibraltar), decreases with the prevalence of parasitic castrators (rhizocephalan barnacles and entoniscid isopods), supporting the hypothesis that these infectious agents control green crab populations. In addition, samples from introduced regions indicated that parasites are strikingly less common or absent where *C. maenas* is introduced compared to where it is native. Additional analyses indicate that reduced parasitism is a principle reason that green crabs perform better in introduced locations. This is not to say that introduced species remain completely unparasitized. As an example, a native nemertean worm was able to colonize *C. maenas* in California by transferring from the native shore crab, *Hemigrapsus oregonensis* (Torchin *et al.* 1996).

Averaging across several taxa, introduced animals leave an average of 84% of their parasite species behind; in addition, native parasites do not sufficiently colonize introduced species to make up for this release from natural enemies, leaving introduced animals with fewer than half the parasites species they have in their native range (Torchin *et al.* 2003).

The same pattern is true for plant pathogens (Mitchell and Power 2003). Such a release from natural enemies could greatly facilitate subsequent impacts of an introduced species.

Introduced species may indirectly impact native species if they help maintain transmission of native diseases (Daszak *et al.* 2000). On average, about four species of native parasites occur in introduced hosts (Torchin *et al.* 2003) and these, by gaining a wider host base, could increase in prevalence, intensity, and geographic range. This is particularly problematic if the disease has little impact on the invader and a big impact on native species.

7.7 Pollutogens

A distinctive class of infectious agents appears to be increasing in prevalence and ecological impact. We define pollutogens as infective agents that have a source exogenous to the ecosystem, but are able to develop within a host in that ecosystem yet do not require that host for reproduction. Two diseases of California sea otters are good examples of pollutogens; Valley Fever is caused by a fungus that enters the marine environment from eroded soil and Toxoplasmosis is caused by a protozoan that enters the ocean along with faces from domestic cats (see Lafferty and Gerber 2002). Another example under extensive investigation is *Aspergillus sydowii*. This is a terrestrial fungus that has appeared across the Caribbean Sea as a severely pathogenic parasite of several species of sea fans (Garzón-Ferreira and Zea 1992). It is believed to have arrived in the Caribbean from a terrestrial source and that secondary infection occurs only when prevalence is high (Jolles *et al.* 2002). Like other classes of infectious agents, pollutogens have an internal physiological dynamic within their hosts. They may also elicit defensive responses. However, unlike other parasites, they have little or no infectious dynamics within the host population. Hence, neither macroparasite nor microparasite models are relevant (no feedback occurs). Pollutogens have no threshold for transmission, no virulence tradeoff consequences, and no coevolution (the host can evolve resistance, but the pollutogen cannot selectively respond because its reproductive success is very low or nil in those hosts). In a sense, this new class of emerging infectious disease is an extreme form of spillover from a reservoir host (even if they are not actually or primarily parasitic in their evolved habitat).

7.8 Concluding remarks

Given the diversity of interactions between environmental disturbance and infectious disease, is it possible to generalize about whether these diseases are increasing or decreasing in association with environmental degradation? Recent attention has been given to mass mortalities in marine systems (e.g. Caribbean sea urchins, Lessios 1988), phocine distemper virus (Heide-Jorgensen *et al.* 1992), pilchard mortalities (Jones *et al.* 1997), and infectious coral bleaching (Hoegh-Guldberg 1999). This has led several authors to speculate that disease outbreaks in marine organisms have increased in recent years (Williams Jr. and Bunkley-Willimas 1990; Epstein *et al.* 1998; Harvell *et al.* 1999; Hayes *et al.* 2001). Unfortunately, a lack of baseline data precludes a direct evaluation of this hypothesis.

Ward and Lafferty (2004) developed a proxy method to evaluate a prediction of the increasing disease hypothesis: that the proportion of scientific publications reporting marine disease has increased in recent decades. Reports of parasites and disease, normalized for research effort, have increased in turtles, corals, mammals, sea urchins, and molluscs. There are no significant trends for reports of disease in sea grasses, decapods, and sharks/rays (though disease occurs in these groups). Consistent with the expectation that fishing reduces parasites, disease reports have significantly decreased in teleost fishes. The increase in reports of coral disease is notable, but this is driven by reports of non-infectious coral bleaching, not reports of infectious disease. These latter results are consistent with the general theory that environmental degradation should increase non-infectious and generalist diseases and parasites (Lafferty and Holt 2003). Increasing host populations, such as seen in many marine mammals, should see increases in most types of infectious disease, while decreasing populations, such as recently experienced by many commercially fished species of fin fish, crabs, lobsters,

and shrimps, should result in decreased prevalences and intensities, and may even prevent transmission of inefficiently transmitted infectious diseases with high host-threshold densities. So, although environmental degradation is occurring at an alarming rate, an increase in infectious disease is not a necessary outcome of these changes. Some parasites will increase, but we expect that many more will decrease, even to the point of extinction. This may seem a blessing amidst otherwise sobering expectations for the future. However, before we count loss of parasites as something to look forward to, we should consider that parasites play important roles in ecosystems. Fungal pathogens (Gilbert *et al.* 1994) and specialized herbivorous insects (Barone 1998), for example, are thought to be responsible for maintaining the high diversity of tropical forest trees through density dependent mortality of seedlings close to parents (Janzen 1970; Connell 1971, see Chapter 8). Although their roles are generally unseen and little appreciated, the loss of parasites may create more problems for us than it solves.

While the evidence for global warming is strong, its ecological effects are not obvious. We are faced with a difficult confound. Other major factors with strong effects on infectious disease dynamics are changing in temporal concert. These certainly include population increases of humans and some other anthropophillic species, invasive species that are now so pervasive in some regions that a parasite-diminished homogicene has been established; economic pressures reducing or eliminating programmes to decrease transmission of diseases; loss of top predators—mostly long gone from terrestrial systems and now severely depleted in aquatic ecosystems; eutrophication; expanded use of pesticides, antibiotics, anthelminthics; and herbicides in agriculture; and evolution of drug resistance by malaria, tuberculosis, and other important infectious diseases. Interpreting changes over time simply with climate change will hinder comprehension of the interactions between disease and the environment. Analysing these specific effects is now an important task for ecologists, parasitologists, and public health investigators.

Given that there are no simple answers to the questions about how environmental disturbances will affect parasitic diseases, substantial research effort will be needed to unravel the complex linkages between these two forces. Until recently, this has been sparsely supported. The US National Institutes of Health (NIH) has traditionally funded few studies that consider the relationship between environmental degradation and infectious disease because its mission focuses on human health. Ironically, the National Science Foundation, which traditionally funds ecological research, has shied away from issues related to infectious disease (as these are perceived to be within the mission of the NIH). Emerging diseases such as Lyme Disease, West Nile Virus, and SARS have forced health professionals to consider the ecological context of infectious disease in a changing world (Aguirre *et al.* 2002) (see Chapter 10). Now, both agencies are aware that an ecological perspective seems necessary to meet these challenges and have recently combined to fund research through their joint Ecology of Infectious Diseases programme in the context of anthropogenic changes. These new research efforts should considerably expand our understanding of how environmental disturbances interact with infectious diseases.