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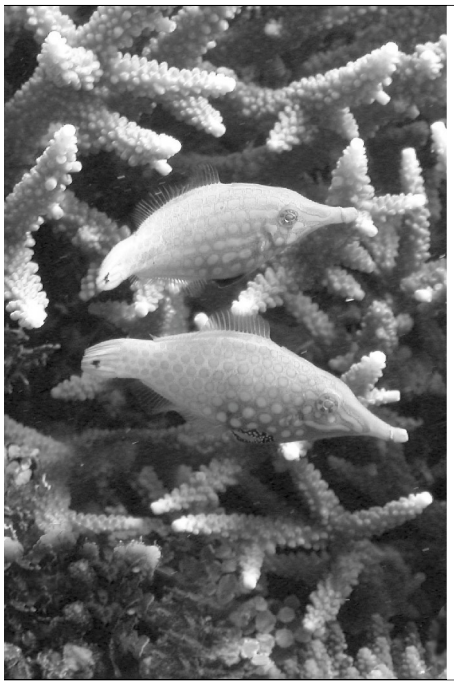
CHAPTER 4

Pathogens and Parasites in a Changing Climate

Andy Dobson, Susan Kutz, Mercedes Pascual, and Rachael Winfree



Corals are more susceptible to disease when they have been damaged by coral bleaching due to thermal stress, as has this reef in Japan.

The world is changing. It has always changed. Without long-term evolutionary change, it would not be possible to write (or read) this article. However, current rates of change are unprecedented. The increase in the size of the human population and the huge increases in our demand for natural resources have led to significant changes in a wide number of ecological processes. Climate is warming. Natural habitat is eroding into agricultural land while also being invaded by alien species. The stocks of most exploited fish species have declined, and huge increases have

occurred in the levels of nitrogen, phosphorus, and other elements in both our rivers and oceans, as well as in the atmosphere. Elucidating the long-term societal and environmental impact of these changes is arguably the greatest scientific and economic challenge facing humans. Paradoxically, most of the world's political agenda is currently focused on resolving conflicts that are emergent epiphenomena of this larger economic crisis.

This chapter focuses on two aspects of global change: what role do pathogens and invasive species play in ecological processes and how will these roles be modified by global climate change? This focus on parasites is warranted by the fact that more than half of biodiversity is parasitic upon the remaining half. Invasive species have similar population dynamics to many pathogens; such species are one of the greatest and fastest growing threats to biodiversity. The insights derived from comparative studies of parasitic and invaded systems shed important light on future threats to biodiversity in a changing world. Many interesting parallels and interactions exist between these two groups of organisms. For example, emerging diseases can be considered as a specific class of invasive species. They are usually pathogens that have recently expanded their host range, which in turn may allow them to expand their geographical range. For example, HIV, the human immunodeficiency virus which causes AIDS, was previously restricted to chimpanzees (HIV1) and Sooty mangabees (HIV2) in West and Central Africa. Close human interactions with these species, probably through close exposure to infected tissue in the bush meat trade, allowed the pathogen to cross the species barrier and invade humans. The relative ease with which the virus spreads as a sexually transmitted disease (STD) allowed it to quickly colonize humans throughout the world. In thirty years its distribution has gone from local to global.

All parasites, though, share some interesting similarities with invasive species. First, both are highly dependent on dispersal. The spread of both pathogens and invading species is facilitated by the creation of new resources. For parasites and pathogens of humans, the resource is more humans, crop plants, or domestic livestock. For many invasive species, it is human-disturbed habitats.

Invasive species are often present for years at low densities ("lag period") before becoming super-abundant. Similar patterns are seen for some emerging pathogens. In both cases, this may reflect an initial period of time when the pathogen or invasive species persists as a metapopulation colonizing an ephemeral sequence of patches of host resources. For example, HIV can persist for 50 to 100 years in a population divided into many small towns and villages with occasional human contact (May and Anderson 1990). Eventually, a large, permanent patch of resources is colonized (or a large urban area), enabling continued persistence and the frequent colonization of surrounding local resources. Finally, emerging diseases and invasive species are both subject to new selection regimes (in new hosts or new habitats). Initial invasion ability can be strain-specific (Jousson *et al.* 2000) or, in the case of pathogens, the initial invasion may be dominated by the limited genetic diversity associated with founder effects. This may be followed by a period of rapid evolutionary change (Allendorf 2003).

Furthermore, many invasive species owe their success to escaping from their natural enemies (Torchin *et al.* 2003). This similarity leads to a further ironic parallel: many invasive species are ones that escape from their natural enemies by dispersing away from the pathogens that may regulate them in their native range. Important exceptions here are the small subset of species that introduce a new pathogen when they colonize a new habitat. Many important fungal and viral pathogens of domestic crops were introduced in this way. Whirling disease of salmonid fish in western North America was a consequence of the introduction of European trout species for game fishing.

EMERGING DISEASES

New pests and pathogens have appeared throughout human history. Ironically, the appearance of new pests and pathogens is usually a consequence of human modification of the environment. Agriculture and the domestication of other species created multiple opportunities for pathogens to invade humans and for pests to invade monocultures of host crops. Simultaneously, the disturbance and subsequent abandonment of agricultural land (and the creation of roads between patches of disturbed land) created large-scale patches and networks of habitat that could readily be colonized by invading alien species, many of which had lifestyles adapted to invade smaller scale, more ephemeral disturbances.

One of the "central theorems" of epidemiology states that pathogens require a threshold number of hosts if they are to establish and persist in a host population. As human populations increased over the last 10,000 years they were sequentially colonized by pathogens that could not previously persist in the smaller, more isolated groups of hosts. Although each emergence occurred by chance, usually following domestication and more intimate contact with livestock, the conditions for establishment and persistence were rather sharply defined and are known to be a mix of deterministic, stochastic, and spatial forces. Much of mathematical epidemiology is involved with understanding how the interaction of these forces allows pathogens to persist, coexist, and ultimately coevolve with their hosts and attempted control measures. Much of this theory also applies to invading species that also need to colonize a patch of resource and then spread from this to other available patches. In both cases this requires the population to be able to increase when rare.

CLIMATE CHANGE AND PATHOGEN ESTABLISHMENT

Climate plays a fundamental role in determining the conditions for disease outbreaks in a wide variety of pathogens. In particular, vector-borne pathogens and those with free-living infective stages are highly dependent upon local temperature and humidity conditions to complete their life cycles. In contrast, pathogens that are directly transmitted by physical contact, such as many respiratory pathogens or STDs, are less directly dependent upon climate, and more subtlety dependent

upon the mechanisms that cause populations to aggregate and mix in different ways. In order for any pathogen to establish and persist in a population, each infected individual has to pass his or her infection on to one or more susceptible hosts before recovering. More formally, the basic reproductive number of the pathogen has to exceed unity (Anderson & May 1982; Diekmann, Metz *et al.* 1990). Deriving mathematical expressions for the basic reproductive rate of a pathogen allows us to examine the sensitivity of the pathogen to variations and changes in climate conditions. Detailed examples of this are provided in a large number of publications (Grenfell *et al.* 1987; Dobson & Carper 1993; Pascual *et al.* 2002). Rather than repeat these derivations, we focus here on some important results. Let us consider the general case of a pathogen that infects a host living in an environment where climate varies seasonally. Assume that the climate conditions under which the pathogen can be transmitted are at a maximum at some time of the year (usually the summer) and that transmission falls off over the colder winter months. Such conditions would apply to mosquito, or tick-transmitted diseases, such as malaria, West Nile virus, or Lyme disease. In an environment where temperature and humidity vary on a roughly sinusoidal annual period, there would be a block of time when the conditions for transmission are initially just permissible (the time when the basic reproductive number, R_0, roughly equals unity). As conditions become warmer (and more humid), transmission increases and we may see large numbers of new infectives (R_0 is now larger than 1). Eventually the weather cools and the number of new cases declines, perhaps to zero (R_0 is now less than one). Figure 4.1 illustrates a sketch of this effect and then considers what happens if the average climate becomes warmer (Harvell *et al.* 2002). The crucial point to note here is that an increase in average temperature leads to an increase in the time period for which the pathogen can increase, while it is also likely to attain a higher maximum rate of increase. Furthermore, this figure assumes there is no change in the variability of the system as the mean increases. In many climate systems, an increase in the mean may be matched by an increase in the variability, allowing the peak of transmission to reach even higher levels. Let us add some details to this by considering three different systems in more detail.

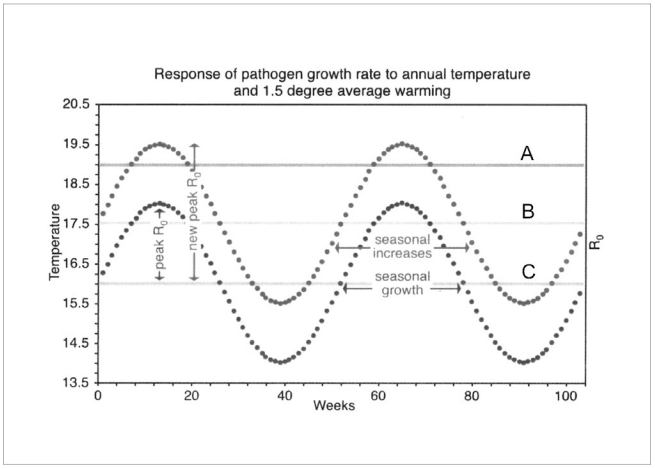


Figure 4.1. The influence of an average 1.5-degree rise in temperature on the basic reproductive number of a hypothetical pathogen. The bottom curve illustrates the average annual temperature prior to climate change; the top curve illustrates average weekly temperature following an average 1.5 degree temperature increase. A simple linear relationship is assumed between temperature and basic reproductive number, R_0. A more complicated model structure could create situations where R_0 increases non-linearly, or faster, or more slowly with temperature change. This illustrates a general case which only considers when the threshold at which the parasite population will grow (Line C corresponds to R_0=1). Below this temperature, the pathogen declines in abundance. The pathogen increases at temperatures above this and we assume that disease problems become severe when temperature exceeds Line B and epidemic above Line A. This figure illustrates that increases in temperature not only allow the peak value of R_0 to increase, but the annual duration of time for when the pathogen is a problem will also increase.

Cholera in Bangladesh

Two components of climate change are central to our understanding and prediction of temporal disease patterns; the first concerns long-term changes in underlying average temperature or rainfall. Much of the climate debate has focused on whether we can distinguish these changes given the inherent variability within climate data sets. However, this variability is also of central importance to the dynamics of infectious disease. Thus, the second component concerns changes in the variability itself, both in seasonal patterns and in longer-term cycles such as ENSO, with changes in the underlying mean. These changes will create more extreme conditions upon which pathogens may capitalize via the crossing of environmental thresholds and the longer seasons for the population growth and persistence of vectors (Harvell *et al.* 2002).

Historical and recent patterns of cholera in endemic regions provide evidence that climate variability, specifically ENSO, plays a role in driving multiyear cycles of disease. Recent patterns show a clear disease cycle of close to four years in Dhaka, Bangladesh, and an association of this cycle to ENSO (Pascual *et al.* 2000). Time series models, allowing for the alternative hypothesis that these cycles are generated intrinsically from the interaction of seasonality and disease dynamics, have demonstrated that ENSO plays a role in the interannual variability of cholera. Comparison of the association between ENSO and cholera prevalence in Bangladesh (former Bengal) across two different periods shows an increased role of climate variability in recent decades. A strong and consistent signature of ENSO is apparent in the last two decades (1980–2001), while it is weaker and eventually undetectable during the first part of the last century (1893–1920 and 1920–1940, respectively) (Rodó *et al.* 2002). Concomitant with these changes, an index of ENSO (the Southern Oscillation Index) undergoes changes in its interannual variability, including a more intense contribution of the four-year period (Rodó *et al.* 2002). These findings provide quantitative evidence for an increased role of climate variability on the temporal dynamics of an infectious disease. Furthermore, the recent data show a strong but discontinuous association in time between an ENSO index and cholera prevalence, which suggests the existence of a threshold in the effect of climate variability (Rodó *et al.* 2002). The existence of such a threshold and the regional variables that mediate the ENSO effect are the subject of ongoing studies.

Nematode parasites in high latitude ecosystems

Climate warming is predicted to occur most rapidly at high latitudes and may have a profound impact on the diversity of parasites and stability of host-parasite systems in the north (Hoberg 1997; Kutz *et al.* 2002). The diversity of helminth parasites in northern wildlife species tends to decrease at high latitudes. Reasons for this decreased diversity include climatic limitations, reduced host species diversity and density, historical isolation, and disruption of ecological continuity. Here the focus is on climate as a limiting factor. Most helminths of mammals have a life stage that develops free in the environment or in an invertebrate intermediate host. These life stages typically require a minimum amount of heating to progress to the next stage of development and, therefore, to maintain themselves in the host population. The short and cool summers of the Arctic and Subarctic restrict the development of the free-living stages of many parasites, and only those that have adapted strategies to maintain themselves in such a climate persist (e.g., rapid development, intermediate hosts, long life spans, resistance to environmental extremes, and ability to over-winter).

Protostrongylid nematodes are one component of the parasite fauna that has been maintained at high latitudes. These are primarily parasites of ungulates, but also infect lagomorphs (Anderson 2000). They live as adults in the lungs, musculature, or central nervous system of their definitive hosts. Eggs are either laid in the lungs (lung dwelling adult parasites) or in/near blood vessels (adult parasites living outside of the lungs) and then are transported via the blood stream to the lungs. Once there they hatch to first-stage larvae, break into the airways, and are moved up the trachea, swallowed, and pass eventually to the feces. The life cycle is heteroxenous, requiring gastropod intermediate hosts for the L1 (larvae) to develop to the infective third-stage larvae (L3). Development rates are temperature-dependent, occurring more rapidly at warmer temperatures, and they differ both among protostrongylid species as well as for the same parasite species in different intermediate host species.

The establishment and persistence of protostrongylids in a host population requires suitable climatic conditions, a sufficient density of appropriate gastropod intermediate host species, and sufficient spatial and temporal overlap between gastropod and final hosts to ensure transmission. Climate, primarily temperature and precipitation, is a key variable in the maintenance of protostrongylids in host populations. Changes in climate may alter the abundance, geographic distribution, and patterns of disease associated with these parasites (Hoberg *et al.* 2001). At Arctic and Sub-arctic latitudes larval development is restricted by short summers and cool temperatures; thus, it typically requires two years for parasites to complete development from L1 to L3. Because over-winter mortality of gastropods and larvae can be substantial, the availability of infective L3 in subsequent years may be low to moderate (Kutz *et al.* 2002). Nevertheless, the ability of larvae to over-winter, the longevity of the adult parasites, the high larval output, and the resistance of larvae to environmental extremes act synergistically to ensure the persistence of protostrongylids in these northern systems. Disease outbreaks, characterized by significant amplification of the parasite in the host populations, occur when the environmental conditions change to the benefit of the parasite. For example, outbreaks of clinical disease in reindeer caused by *Elaphostrongylus rangiferi* in Norway are associated with summer temperatures that exceed 1.5° C above normal. In these years, larval development starts earlier and occurs more rapidly, allowing larvae to complete development in one summer. The mass exposure to these L3 by the naive young of the year results in clinical disease (Handeland & Slettbakk 1994).

At temperate latitudes, conditions are usually sufficient for protostrongylids to complete development and be transmitted within a single season. Increases in parasite abundance have been associated with longer periods of transmission (Samson & Holmes 1985; Samuel *et al.* 1985; Ball *et al.* 2001). For example, for *E. rangiferi* in Newfoundland, Canada, the transmission period is considered to be the snow-free period when the gastropod intermediate hosts are active (temperatures above 0° C). Abundance of *E. rangiferi* in these caribou populations has been most closely associated with the number of days above 0° C. In this system, summer temperatures are not limiting since larvae consistently complete development within a single year. Rather, it is the period of time for which they can be ingested that appears to limit the parasite abundance.

Climate models for northern latitudes forecast milder winters, earlier, warmer springs, and later autumns (see IPCC 2001). Clearly these changes will alter patterns of parasite abundance and disease occurrence in both temperate and northern protostrongylid systems, allowing parasites to begin development earlier, develop faster, and continue later into the fall. Retrospective and experimental studies demonstrate that in "abnormally" warm years parasite amplification in the host population occurs (Handeland & Slettbakk 1994; Kutz *et al.* 2002). These years can serve as indicators for the effects of climate change in the future.

A case study: Umingmakstrongylus pallikuukensis in the Arctic

A combination of field and laboratory studies on *U. pallikuukensis,* a recently discovered protostrongylid lungworm of muskoxen, provides further insight into the epidemiology of protostrongylids at high latitudes as well as tools for assessing and predicting the effects of climate change on these host-parasite systems. *U. pallikuukensis* is a large (up to 65 cm in length) lungworm that forms cysts 0.5–4 cm in diameter in the lungs of muskoxen. Cysts typically contain 2–7 adult nematodes, and infection intensities in excess of 250 cysts have been reported in older bulls. The population effects are not well understood, but reports of infected individuals exhibiting exercise intolerance and epistaxis (bleeding from the lungs) suggest that the parasites may have effects on respiratory function. There is speculation that such compromise may result in increased predation by the barren-ground grizzly bear. While *U. pallikuukensis* is well established in the infected muskox population (100% prevalence in adult animals), its geographic distribution is limited to a region of the west-central mainland Arctic and Subarctic. This limited geographic distribution is

probably a result of a combination of factors, two of the most important being (1) the historical distribution of muskoxen and (2) limiting climatic conditions (Hoberg *et al.* 1995).

A series of field trials with experimentally infected slugs (*D. laeve*) in tundra enclosures were used to describe "natural" parasite development and abundance in gastropod intermediate hosts on the tundra (Kutz *et al.* 2002). Because the trials took place during an exceptionally warm year (fifth warmest on record, with summer temperatures 2.5° C above normal), the results may reflect the patterns expected under climate warming conditions. Larval development was rapid, with those infecting slugs by the middle of July completing development to L3 in a single summer. L1 infecting slugs at the end of July or later did not develop to L3, but over-wintered and completed development the following year. The peak abundance of L3 in slugs occurred in mid-late August of the first year. Interestingly, while all larval stages were able to over-winter in slugs, the subsequent abundance of L3 was significantly lower than it had been the preceding August. The pattern of larval development observed during this study parallels that predicted for *E. rangiferi* in Norway during the abnormally warm years preceding disease outbreaks. It is likely that this was a year where the parasite was amplified in the muskox populations; however, because of the remoteness of the infected muskox population and absence of baseline data, changes in prevalence, intensity, or disease occurrence could not be detected. In years with normal temperatures, larval development probably requires two years and the subsequent abundance of L3 is predicted to be considerably lower.

CORAL DISEASES: WHITE POX DISEASE IN THE FLORIDA KEYS

Coral reefs are of particular relevance to the question of emerging diseases in the ocean and as indicators of the health of marine coastal environments. They harbor the most diverse assemblage of species on Earth, and are at the same time especially susceptible to environmental change given the narrow range of their physical requirements in warm sea surface temperatures, high water clarity, and extremely low nutrient concentrations (Porter & Tougas 2001). An extensive monitoring program in the Florida Keys (Porter *et al.* 2001) demonstrates significant declines in coral cover. In particular, the elkhorn coral *Acropora palmata*, an important Caribbean species responsible for the complex three-dimensional structure of shallow water reefs, has declined in cover by 70 percent since 1996 in the Florida Keys (Patterson *et al.* 2002). Parallel to such losses, there has also been an increase in reports of coral disease incidence (Harvell *et al.* 1999; Porter *et al.* 2001). Although the links between these two trends are only starting to be addressed, and the causes of coral decline are known to be multiple, recent findings provide support for coral diseases as an emerging cause. This raises important concerns regarding the consequences of climate change-induced changes in ocean temperatures.

One striking recent finding establishes that the pathogen responsible for white pox, a lethal disease of *A. palmata,* is a common fecal enterobacterium *Serratia marcescens* (Patterson *et al.* 2002). For the first time, a bacterial species associated with the human gut has been shown to cause disease in an invertebrate marine organism. This discovery is consistent with one common property of emerging diseases—their origin as a spillover from one host to another as the effective contact between species increases with environmental deterioration and/or changes in population abundances and distributions. Interestingly, another disease of corals appears also to have crossed the boundary between terrestrial and aquatic environments. Aspergillosis of the soft corals *Gorgonia ventalina* and *G. flabellum* in the Caribbean is caused by the terrestrial fungus *Aspergillus* sydowi (Harvell *et al.* 1999).

Evidence is also accumulating for the potential detrimental effects of future scenarios of rising average ocean temperatures for the next century. Elevated temperature is known to cause bleaching, a major stress in corals due to the loss of their symbiotic algae (Porter & Tougas 2001). This breakdown appears to accelerate the growth of pathogens and reduce the effectiveness of the host's immune system (Kushmaro *et al.* 1996; Alker *et al.* 2001; Carlton & Richardson 1995). These findings are consistent with the described association between the rate of tissue loss due to white pox disease in *A. palmata* and seasonal patterns of elevated water temperature (Patterson *et al.* 2002).

CONSERVATION LESSONS

Climate change will have significant impact on the transmission dynamics of many pathogens of animals, plants, and humans. In particular, pathogens that are vector-transmitted and those with long-lived infective stages are highly sensitive to climate variation. A warmer world will allow these pathogens to extend their geographical range. In areas where they are currently endemic, a warmer climate will allow them to expand the annual period of time during which they can be effectively transmitted. There may also be an increase in the maximum level of transmission at the peak times of the year. The last 20 years have seen a huge increase in our understanding of the role that parasites and pathogens play in the population dynamics of animals and plants (Anderson & May 1991; Grenfell & Dobson 1995). This quantitative understanding of the dynamics of host pathogen biology now needs to be expanded to examine the role that climate and seasonality play in mediating interactions between host and parasite species.

This chapter has presented three examples of systems in which there is a strong link between climate and disease transmission dynamics. In each case the role of climate was subtle and often non-linear; furthermore, detailed fieldwork and long-term data analysis were required to tease out the relationship between the underlying climate signal and its resultant effect. A few other disease systems have been examined in similar detail, but considerable need still exists for more studies of this type to be undertaken. Comparisons have also been drawn between the dynamics of infectious diseases and the dynamics of free-living invasive alien species. These species are the world's least endangered species. Unfortunately, they

rarely replace, or replicate, the full suite of ecosystem goods and services of the species they replace. Controlling them and returning natural ecosystems to their previous undisturbed state remains as large a challenge to ecologists and conservation biologists as the control of infectious diseases is to the medical community.

There is increasing evidence that biological diversity plays an important role in buffering the magnitude of pathogen outbreaks (Ostfeld & Keesing 2000; Ostfeld & Keesing 2000; Dobson & Foufopoulos 2001). This is particularly important for vector-transmitted pathogens, where the level of infection in a vulnerable host species may be considerably reduced when mosquitoes, or ticks, feed upon nonviable host species that fail to develop the infection. Each one of these "wasted bites" reduces the chance that a viable host is bitten and thus reduces the rate at which infections are amplified. This creates a worrying irony: the greatest diversity of pathogens occurs in the tropics where warm, humid climates often allow year-round transmission of many vector-transmitted and water-borne infections. The large biological diversity of these regions probably plays a significant role in buffering the magnitude of these outbreaks. As anthropogenic activity reduces the tropical biodiversity, it will diminish this buffering effect and likely enhance the impact of pathogens on humans and their domestic livestock and crops. Furthermore, as the range of many vector-transmitted pathogens expands into the temperate zone, the lower levels of biodiversity in these regions will allow the impact of these pathogens to focus on the commonest potential host species, which again is likely to be humans and their domestic livestock and crops. It is hard to think of a more cogent argument for protecting biological diversity; in a warmer world, human health may well be highly dependent upon a diversity of other species to protect us from the bites of the vectors of infectious diseases.