

2012

# Climate Change and Infectious Disease Dynamics

Raina K. Plowright  
*Pennsylvania State University*

Paul C. Cross  
*U.S. Geological Survey Northern Rocky Mountain Science Center*

Gary M. Tabor  
*Center for Large Landscape Conservation*

Emily Almberg  
*Pennsylvania State University*

Leslie Bienen  
*Center for Large Landscape Conservation*

*See next page for additional authors*

Follow this and additional works at: <http://digitalcommons.unl.edu/usgsstaffpub>

 Part of the [Geology Commons](#), [Oceanography and Atmospheric Sciences and Meteorology Commons](#), [Other Earth Sciences Commons](#), and the [Other Environmental Sciences Commons](#)

---

Plowright, Raina K.; Cross, Paul C.; Tabor, Gary M.; Almberg, Emily; Bienen, Leslie; and Hudson, Peter J., "Climate Change and Infectious Disease Dynamics" (2012). *USGS Staff -- Published Research*. 970.  
<http://digitalcommons.unl.edu/usgsstaffpub/970>

This Article is brought to you for free and open access by the US Geological Survey at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in USGS Staff -- Published Research by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

---

**Authors**

Raina K. Plowright, Paul C. Cross, Gary M. Tabor, Emily Almberg, Leslie Bienen, and Peter J. Hudson

Published in *New Directions in Conservation Medicine: Applied Cases in Ecological Health*, edited by A. Alonso Aguirre, Richard S. Ostfield, and Peter Daszak (New York: Oxford University Press, 2012).

### *Authors*

Raina K. Plowright, B.V.Sc., M.S., Ph.D.  
David H. Smith Conservation Research Fellow  
Center for Infectious Disease Dynamics  
Pennsylvania State University  
University Park, Pennsylvania

Paul C. Cross, Ph.D.  
U.S. Geological Survey Northern Rocky Mountain Science Center  
Bozeman, Montana

Gary M. Tabor, V.M.D., M.E.S.  
Director  
Center for Large Landscape Conservation  
Bozeman, Montana

Emily AlMBERG, M.S.  
Department of Biology  
Pennsylvania State University  
University Park, Pennsylvania

Leslie Bienen, D.V.M., M.F.A.  
Center for Large Landscape Conservation  
Bozeman, Montana

Peter J. Hudson, F.R.S.  
Willaman Professor of Biology  
Director of the Huck Institutes of Life Sciences  
Pennsylvania State University  
University Park, Pennsylvania

# 8

## CLIMATE CHANGE AND INFECTIOUS DISEASE DYNAMICS

---

Raina K. Plowright, Paul C. Cross, Gary M. Tabor,  
Emily Almborg, Leslie Bienen, and Peter J. Hudson

The International Panel on Climate Change has made an unequivocal case that the earth's climate is changing in profound ways, and that human activities are contributing significantly to climate disruption (IPCC 2007). The weight of evidence demonstrates warming global temperatures, changing patterns of precipitation, and increasing climate variability, with more extreme events. Thus, the physical underpinnings of ecology are changing, with pervasive effects on disease dynamics. Interactions among environment, hosts, and pathogens drive disease processes, and climate change will influence every interaction in this triad, directly and indirectly.

Direct effects of climate change on hosts include the health consequences of diminishing water availability and increasing thermal stress. Some host species will succumb to the indirect effects of climate disruption through an array of mechanisms, including increased competition from invasive species, decreasing food availability, changes in parasite ranges and/or virulence, and many others. Some species will flourish under changed climate scenarios; ultimately, survival of each species will depend on its ability to adapt to climate change. Species adaptation will take on various forms, some unpredictable, such as through behavioral changes that may lead to different patterns of aggregation; changes in timing of life-history events to follow changing phenology or other climate signals;

changes to physiology to allow for survival in different climatic regimes; and development of coping mechanisms based on phenotypic plasticity. Under worst-case scenarios of potential future climate change, many species may survive at their ecological limits and face narrowing climatic and ecological constraints, including the rise of novel ecological conditions.

Of course, species adaptation to climate change will not happen in a vacuum, isolated from other environmental stressors; existing environmental threats create constraints for species and ecosystems that will affect their ability to adapt to climate change. For example, large-scale habitat fragmentation hinders species adaptation capacity by impeding animal movement necessary for reproduction, dispersal, seasonal migration, and tracking of new environmental conditions. Climate change will likely exacerbate the impacts of existing environmental threats, and this may include disease.

Extracting the effects of climate change on disease from the effects of other environmental changes is challenging, in part because we have only recently begun to understand the complexity of the interactions between climate change and disease dynamics. To date, some of the strongest system-specific evidence elucidating the effects of climate change on disease dynamics comes from vector-borne disease systems and from diseases that are environmentally

transmitted and have obviously climate-dependent processes. For example, many insect vectors are highly sensitive to both temperature and precipitation, with climate change predicted to influence life-cycle completion times, biting rates, and overwintering survival, among other factors (Harvell et al. 2002; Ostfeld 2009). For parasites with free-living stages, temperature affects their development times and transmission windows (see Chapter 9 in this volume). Nevertheless, very little work on climate change and disease has focused on how changes in host population structure, dynamics, and phenology will influence disease dynamics under global warming scenarios—a key element of research as we begin considering how to integrate climate-change adaptation into conservation efforts. The links between climate change and host-population response—and how those links are manifested in disease ecology—are highlighted in this chapter.

#### DENSITY, CONNECTIVITY, AND COMPOSITION: EFFECTS OF CLIMATE CHANGE ON HOST-POPULATION ECOLOGY AND SUBSEQUENT IMPACT ON DISEASE DYNAMICS

##### Population Density

As climate warms, vegetation communities shift in composition and distribution, and the availability of surface water changes. Wildlife population structures and aggregation patterns will be sensitive to these changes as animals adapt to new conditions or seek alternative habitats, especially if temperature and precipitation change very quickly. In many regions water and/or snow accumulation are major drivers of host aggregation, but the direction and strength of the climatic effects will vary between species, region, and time. For example, diminishing water availability may concentrate wildlife around remaining water sources in the short term, while reducing population densities over the long term and on broader spatial scales. Elevational migration is another climate-driven process that may concentrate wildlife populations locally on high-elevation “islands” while wildlife populations decline globally (Parmesan and Yohe 2003). Some species, or populations, may experience

increased densities due to increased population size. For example, elk (*Cervus elaphus*) from Montana are predicted to increase in density as snow accumulation decreases (Creel and Creel 2009). Finally, rapid ecological changes brought about by climate change, such as the massive outbreak of mountain pine beetle (*Dendroctonus ponderosa*) across Canada and the United States, will have varying effects on different species; moose (*Alces alces*) may gain increased foraging resources and hence increase in density, while caribou (*Rangifer tarandus caribou*), fisher (*Martes pennanti*), and marten (*Martes americana*) will be adversely affected by the loss of coniferous trees critical for forage, cover, and denning (Ritchie 2008).

These changes in host density and spatial structure will have cascading impacts on pathogen transmission rates and alter thresholds for pathogen establishment and persistence. Models predict that pathogens with density-dependent transmission are unable to persist when host density is reduced below some threshold (Kermack and McKendrick 1927; Getz and Pickering 1983), a result well supported by classic studies of measles (Bartlett 1957; Bjornstad et al. 2002; Grenfell et al. 2002). These studies provide an understanding to influence policy, for example school closures to control human pandemics (Glass and Barnes 2007; Cauchemez et al. 2008; Halloran et al. 2008), as well as for using hunting or culling as a disease-control measure in wildlife populations (Caley et al. 1999; Lloyd-Smith et al. 2005; Conner et al. 2007).

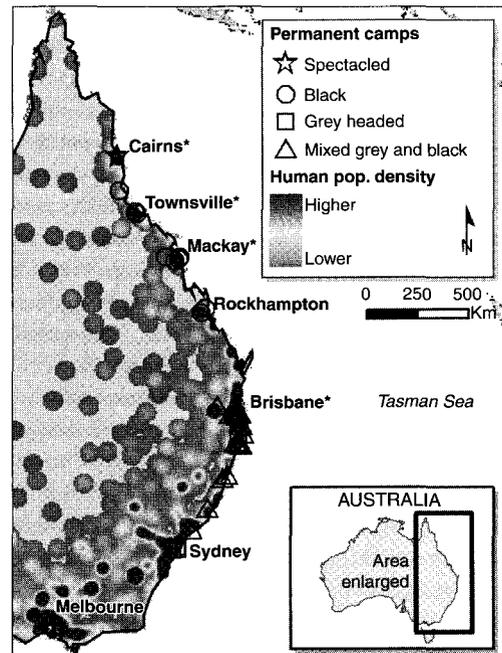
For systems in which transmission is correlated with host density, if climate change aggregates hosts, transmission rates may increase and contribute to larger disease outbreaks locally, but the relationship between host density and disease transmission is complex and not always possible to predict. Several studies suggest that host density is unassociated with transmission or parasitism, even for directly transmitted parasites (Bouma et al. 1995; Rogers et al. 1999; Delahay et al. 2000; Joly and Messier 2004), or even that decreasing density can increase transmission. For example, culling badgers in the United Kingdom counter-intuitively increased the incidence of tuberculosis on adjacent cattle properties, perhaps due to culling resulting in increased movement rates of badgers (Donnelly et al. 2006). Such contradictory results may also be explained by the fact that the density-transmission relationship is complicated by parasite life history (Côté and Poulin 1995), by seasonal

fluctuations in both host density and transmission (Altizer et al. 2006; Cross et al. 2007), by movement among groups (Ball et al. 1997; Cross et al. 2005), by the effects of alternative hosts (Dobson 2004; Craft et al. 2008), and by interactions with other pathogens (Jolles et al. 2008).

Human diseases are also influenced by changing patterns of aggregation; Ferrari et al. (2008) hypothesized that the large seasonal variation in measles transmission in Niger was driven by human migrations from agricultural to urban areas during the dry season. Drought in combination with habitat loss may have driven a similar phenomenon of urban aggregations of Australian flying foxes (*Pteropus* spp.) with consequences for disease emergence into humans. Though flying foxes were historically nomadic, they increasingly prefer well-watered urban landscapes where food is abundant and reliable year-round (Parry-Jones and Augee 1991; Birt 2004; Markus and Hall 2004; McDonald-Madden et al. 2005). Concomitant with an increase in the number and size of urban flying fox camps was the emergence of Hendra virus, a fatal zoonotic disease that has repeatedly emerged from flying foxes into horses and humans since 1994. Many hypotheses exist to explain the cause of emergence (Plowright et al. 2008a), including higher rates of intra-specific transmission within urban flying fox colonies—consistent with density-dependent transmission, and closer and more frequent contact between flying foxes and high-density human and horse populations (Fig. 8.1) (Plowright et al. 2008a). The above examples highlight the complexity of the relationship between host density and disease dynamics. Although there are special cases where increasing host density does not increase disease transmission, the default hypothesis is for a positive association. More data are needed to explore these relationships, and generalize predictions across systems.

### Population Connectivity and Metapopulation Structure

Climate change will affect connectivity among populations and species by affecting the spatial distribution of habitat; dispersal and migration rates; and species distributions. One mechanism predicted to have profound effects on connectivity is range contraction, particularly affecting species in arid or montane regions, which are predicted to shift to areas



**Figure 8.1:**

Permanently occupied flying fox colonies (known as camps) and their relation to urban areas in Australia (depicted by high human population density). Spectacled = spectacled flying fox (*Pteropus conspicillatus*); Black = black flying fox (*P. alecto*); Grey Headed = grey headed flying fox (*P. poliocephalus*). Modified from Plowright et al. (2011).

of higher elevation and greater precipitation. For example, elevation shifts in distribution have already been observed for the Edith checkerspot butterfly in California (Parmesan 1996), for montane woodlands plant assemblages (Fisher 1997), for tropical species in Costa Rica (Pounds et al. 1999), and for desert bighorn sheep (*Ovis canadensis nelsoni*) (Epps et al. 2004). In the short term, reduced resource availability may concentrate animals on remaining habitat, thus increasing local densities and intensifying disease transmission. However, at the same time, densities and connectivity at a broader scale may decrease, effectively isolating local populations and decreasing metapopulation function. Thus, climate change may lead to the loss of parasites and pathogens, and the potential release of hosts from this source of regulation.

Epps et al. (2004) use desert bighorn sheep as the basis for a convincing case study to show how

climate-induced changes to population structure may impose conditions that both decrease population viability while, theoretically, also decreasing the threat of infectious diseases. The study demonstrates that climate change has altered the metapopulation structure of desert bighorn sheep, which occupy isolated mountain ranges in the Sonoran, Mojave, and Great Basin deserts of the southwestern United States, where recent trends in warming have been particularly severe, annual precipitation has declined by approximately 20% over the past century, and mean annual temperatures have simultaneously increased 0.12°C per decade. Epps et al. (2004) demonstrated that populations inhabiting lower, drier mountain ranges were more likely to be extirpated, essentially contracting the metapopulation into isolated high-elevation islands. Long-term persistence of desert bighorn sheep metapopulations depends on some connectivity for gene flow and demographic rescue, but immigration also promotes the transmission and persistence of infectious diseases (Hess 1996; McCallum and Dobson 2002). Thus, desert bighorn sheep are an excellent example of the conundrum facing wildlife-disease managers: as climate change decreases population sizes and connectivity, isolation and decreased connectivity diminish long-term population viability; however, these conditions may also reduce disease threats. Many pathogens, particularly those with short infectious periods, no environmental reservoir, and no alternative hosts have a difficult time persisting in isolated, spatially structured systems (Cross et al. 2005; Lloyd-Smith et al. 2005; Cross et al. 2007). Climate change may drive such pathogens to extinction.

Geographic isolation is not the only mechanism that may reduce animal migrations as climate changes. Climate change may reduce migration in areas where seasonal ranges become hospitable year-round (Harvell et al. 2009). For example, increasingly urbanized Australian flying foxes (*Pteropus* spp.) are choosing a resident lifestyle because food supplies in well-watered urban gardens reduce their need for energy-expensive long-distance migrations (Parry-Jones and Augee 1991; Birt 2004; Markus and Hall 2004; McDonald-Madden et al. 2005). It has been hypothesized that decreasing flying fox migration, and subsequent declining population connectivity, may lead to a reduction in herd immunity. When virus is reintroduced into populations where herd immunity has declined, more intense outbreaks may result—a

mechanism that potentially explains the recent emergence of Hendra virus from flying foxes into humans and domestic animals (Plowright et al. 2011).

Migration rates may also decline when some seasonal ranges become inhospitable, rather than more hospitable. For example, a complex ecological scenario may lead to increasing year-round resident populations of elk, while migratory populations decline. Some populations of elk on the eastern side of Yellowstone National Park migrate to higher elevations during the summer and return to lower-elevation areas in winter, while non-migratory elk remain at lower-elevation sites year-round. Over the past 20 years, pregnancy and recruitment rates in migratory elk have been declining relative to non-migratory populations (Middleton et al., in review). These declines appear to be the result of increasing grizzly bear (*Ursus arctos*) and wolf (*Canis lupus*) populations at higher elevations, as well as of shorter growing seasons at those elevations. This pattern of increased resident behavior may be a recurring one in northern latitudes, where higher-elevation areas are more likely to still have intact predator communities, with cascading impacts upon herbivore migrations, population dynamics, and diseases. Harvell et al. (2002) describe increasing resident behavior as a growing phenomenon, where higher population densities increase host contact, parasite transmission, and the concentration of parasites in the environment. Additionally, increasing resident behavior may reduce the opportunity for migration to “weed out” weak or infected animals, leading to a higher prevalence of parasites in resident populations compared to migratory populations (Bradley and Altizer 2005).

### Changing Community Composition

Climate change has been projected to change the composition of host communities, not only through new species assemblages, but also via the exchange of parasite communities (Dobson and Carper 1992), thus providing a mechanism for increased interspecies connectivity. Many species are responding to climate change by shifting geographic ranges towards the poles or higher altitudes (Parmesan and Yohe 2003), which is a pattern reinforced by habitat conversion. Species do not move as communities, but rather at individual directions and rates (Lovejoy 2008). Therefore, we anticipate that differential rates in

range shifts and survival will lead to new species assemblages (Williams et al. 2007). As new species come together, we anticipate greater opportunities for pathogen spillover, which may in some cases result in severe decline of novel susceptible hosts. For example, elk and white-tailed deer have expanded their ranges northward, in part due to warming temperatures, and consequently have introduced novel pathogens to caribou and musk ox (Dobson et al. 2003; Kutz et al. 2005). By rearranging communities we are also potentially breaking coevolved host-pathogen life cycles, a disruption that could potentially outpace spillover and reduce the disease burden in some species. Parasites that play an important role in mediating competitive interactions among hosts may also experience shifts in abundance and distribution associated with changes to the larger parasite community, potentially triggering cascading changes in host communities (Dobson and Carper 1992). Feedback loops between changing host assemblages and parasite communities may be most pronounced in temperate regions, where the buffering effects of biodiversity are absent (Harvell et al. 2002).

#### TIMING AND SYNCHRONY: CHANGING THE PHENOLOGY OF HOST BEHAVIORS IMPORTANT FOR PATHOGEN TRANSMISSION

##### Host Behavior

Climate change also has potential to change the timing of host behaviors that are fundamental to pathogen transmission. For example, climate-mediated shifts in phenology have been documented across a wide range of taxa and may act to decouple the spatial and temporal overlap between hosts and their parasites (Hoberg et al. 2008). We may see changes in the timing, intensity, and location of seasonal aggregations, and in migrations of hosts in response to changes in temperature or to the timing of food availability on the landscape (Buskirk et al. 2009). These shifts may increase, or decrease, the likelihood of pathogen transmission if they alter the timing of host contacts with infective stages of parasites in the environment.

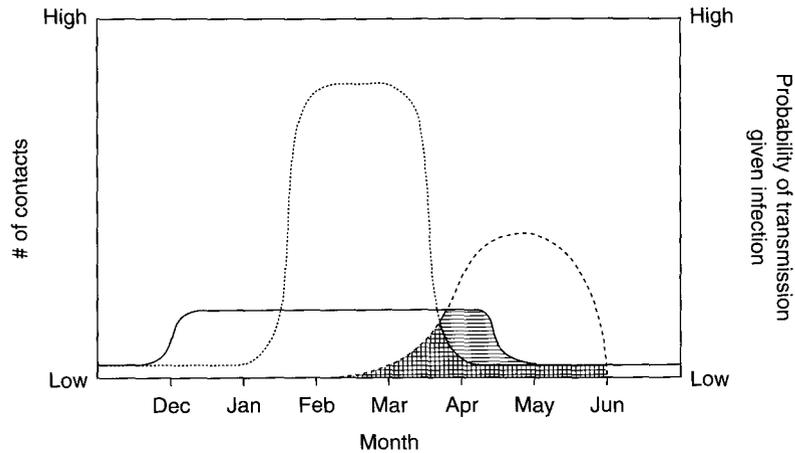
Research on brucellosis in elk highlights connections between climate, timing of host aggregation, and disease. In this system, both elk density and

disease transmission have strong seasonal patterns. The largest elk aggregations occur in late winter prior to snowmelt and spring migration (Cross et al. 2007), and can be particularly large (several hundred to several thousand individuals per group) in areas of Wyoming where elk receive supplementary feed from managers. Brucellosis is typically transmitted by abortion events, which tend to occur in winter and spring (Cheville et al. 1998). The overlap between the timing of high densities of animals and the timing of disease-transmission windows is critical (Fig. 8.2).

The duration of the supplemental feeding season explained almost 60% of variation in elk seroprevalence for brucellosis across numerous feeding areas. Meanwhile, there was no relationship between elk seroprevalence and peak winter elk densities. Brucellosis exposure was determined by the length of time animals were aggregated during the transmission window; low-density elk aggregations that occurred during the transmission window may have similar levels of pathogen exposure to sites with short aggregations but high densities (Cross et al. 2007) (Fig. 8.2). Further, the duration that elk were aggregated each winter was highly correlated with snowpack, because elk were concentrated for longer during years with more snow. Climate change is likely to alter the timing and duration of winter elk aggregations. Over the past 50 years, snowpack in areas of the southern Greater Yellowstone Ecosystem has fallen as much as 50%, though the declines are stronger in early winter (Fig. 8.3). As a result of earlier snowmelt we might expect brucellosis transmission to decline. However, the timing of birth and abortion (and thus brucellosis transmission) may also shift to earlier in the year.

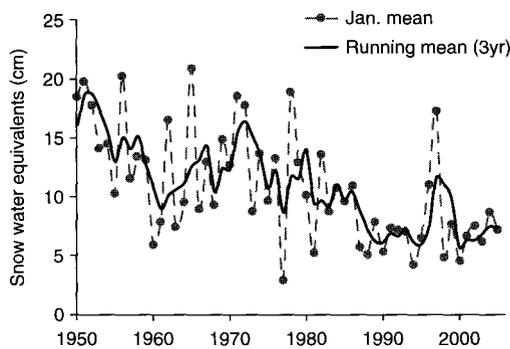
##### Synchrony of Host Population Dynamics and Parasite Life Cycles

An important component of climate disruption is an increase in the frequency of extreme climatic events, which may act to synchronize disease outbreaks over large spatial areas. More than 50 years ago, Moran (1953) proposed that independent populations that have the same underlying density-dependent structure will fluctuate in synchrony when their climatic conditions are correlated. The extent of this synchrony between populations will depend on the correlation of these climatic events between localities,



**Figure 8.2:**

Conceptual illustration of the interaction between contact and transmission rates when both are varying over time in the elk brucellosis system. Some elk populations are aggregated in smaller groups for long periods during winter (*solid line*) while others are in large groups for shorter periods (*dotted line*). Brucellosis transmission is associated with abortion events, which may be more likely in late spring (*dashed line*). The cumulative annual transmission should be more correlated with the amount overlap between high contact rates and brucellosis transmission than with maximum density.



**Figure 8.3:**

The average snow water equivalent (cm) measured on January 1 at 16 SNOwpack TELEmetry sites in the Jackson and Pinedale regions of Wyoming. Data source: USDA Natural Resources Conservation Service.

able to identify the years when populations were unpredictably brought into synchrony. Analyses of climatic conditions together with levels of parasitism by the nematode *Trichostrongylus tenuis* in these years identified the weather variables that were associated with these changes. Subsequent model fitting provided evidence to support that climatic conditions acted on parasite transmission, either to (1) accelerate transmission, leading to a common, large-scale disease outbreak and a decline in the host population or (2) reduce transmission, and this would result in low parasite intensity and an increase in the host population. In effect, extreme climatic events may bring populations into synchrony by acting on the transmission stages. This is important since it provides good evidence to support that an increase in the frequency of extreme events could lead to spatially synchronized disease outbreaks.

and this correlation would increase with increased frequency of large-scale extreme events, as expected with climate change. Cattadori et al. (2005) examined the spatial synchrony of grouse in northern England and found that spatially independent grouse populations exhibit unstable cyclic fluctuations, and they were

The spatial scale of these disturbances in relation to species distributions will obviously influence the impact on infectious diseases. For example, small-scale disturbances such as tornadoes will not have the same effect as widespread drought. Large-scale impacts are more likely to reduce the host population across large areas, rendering these populations

vulnerable to all effects. Large-scale extreme climatic events could also synchronize resource availability and so influence disease transmission by changing host susceptibility over large scales. For example, nutritional stress increases Hendra virus seroprevalence in flying foxes (Plowright et al. 2008b), suggesting that processes that alter flying fox food sources—such as climate-change-associated drought—could both increase and synchronize the risk of Hendra virus spillover. Temporal correlation of some Hendra virus outbreaks supports this hypothesis of “stress synchrony” and is worth further exploration (Plowright et al. 2011).

### Environmental Variation and Parasite Development Rate

Climate may influence transmission by killing the infective stage faster with directly transmitted pathogens; however, with helminths and vector-borne infections, the free-living stages are subject to temperature variations that may influence development. Generally, there is a minimum temperature below which free-living stages of parasites do not develop, and as temperature increases, the parasite development rate increases linearly. This is a pattern well documented in a wide range of nematode species where the free-living stages were incubated at set temperatures and development rates were tracked (Anderson 2002). As such, development rate can be modeled using a degree-day approach, based on the assumption that a fixed amount of thermal energy is required by a parasite to develop, and this accumulates daily according to the local temperature (Grenfell and Smith 1983). Implicit in this assumption is that variation in temperature is not important but development is achieved after the thermal energy is accumulated; this assumption has indeed been confirmed in some systems. Interestingly, there is mounting evidence that variation in temperature may accelerate development faster than the degree-day models predict. In a study of the development rate of eggs of the caecal nematode *Heterakis gallinarum*, Saunders et al. (2000) found that an increase in temperature conditions resulted in a linear increase in the development rate, which could be described by a simple degree-day model. However, when the parasite was placed in a daily temperature cycle, development started significantly earlier than that expected from the linear

model. Furthermore, when eggs were placed in stochastic fluctuations (with temperatures giving the same thermal energy as the temperature cycle) they developed significantly earlier, indicating that fluctuations in temperature, and particularly increased variation, could accelerate parasite development rates (Saunders et al. 2000). An aspect of climate disruption may well be increased variability in temperature, which could have a profound effect on the development of free-living stages of parasites. It is important to note that increasing temperatures also increase parasite mortality rates for non-feeding organisms, and therefore the relationship between temperature and an organism's performance may follow a convex function (Lafferty 2009).

### STRESS: CLIMATE-CHANGE IMPACTS ON HOST SUSCEPTIBILITY TO DISEASE

A substantial body of evidence suggests that climate change has the potential to mediate stress, and hence host susceptibility to infectious disease. Thermal stress has been documented among numerous ectotherms, ranging from amphibians to corals and fish (Marcogliese 2008; Harvell et al. 2009). Increasing temperatures have raised the metabolic rates of common toads in the UK during hibernation, resulting in their spring emergence in a reduced physiological state (Reading 2007). Corals exhibit stress responses to increased water temperatures, and recent increases in pathogen loads and distributions have been, in part, attributed to increased coral susceptibility due to thermal stress (Bruno et al. 2007; Mydlarz et al. 2010). Changes in behaviors due to increased environmental temperatures may also result in sub-optimal foraging, and thus in compromised body condition and immune function. Endotherms with narrow temperature envelopes are also expected to be susceptible to thermal stress—moose are known to have extremely inflexible temperature thresholds, above which their metabolism rapidly speeds up (Renecker and Hudson 1986). Thermal stress in moose is thought to contribute, in part, to reduced immune function and ability to handle parasite loads (Murray et al. 2006). However, increasing temperatures may not always equate to negative effects on immune function. In some cases, increased

temperatures may increase the productivity of primary producers (although decreased primary productivity may be just as likely), providing additional nutritional resources to hosts, and thereby increasing body condition and immune function. For example, on St. Kilda, the site of the long-term study on Soay sheep (*Ovis aries*), primary productivity is related to sheep body condition and winter survival in the face of parasites (Crawley et al. 2004; Wilson et al. 2004). If the trends of increasing temperature and less severe winters documented on St. Kilda result in increased primary productivity (Ozgul et al. 2009), there may be a suite of climate-mediated effects on host-parasite dynamics.

The flying fox scenario may also illustrate how climate can serve as an ecological vise, with water and temperature stress influencing disease dynamics. While water stress may have altered flying fox colonization ecology, the associated temperature increases in urban environments due to climate-related urban heat island effects have pushed some flying foxes to their maximum physiological thresholds of heat tolerance (Welbergen et al. 2008). Temperature-related stress is, therefore, one more potential causal factor to consider when understanding Hendra virus emergence (Plowright et al. 2008a,b).

Other forms of climate-mediated stress may also have immunosuppressive effects. Host crowding, as individuals respond to shifting habitats, may increase intra-specific aggression and stress, resulting in increased pathogen and parasite susceptibility. Climate change may also affect the accumulation and persistence of pollutants in the Arctic, for example, via changes to air and ocean currents (Burek et al. 2008). If these changes result in increased exposure of marine life to toxins, we may see increased susceptibility to disease among these organisms (Bustnes et al. 2004).

## CONCLUSIONS

In this chapter we have discussed several modalities of host population response to climate change and the disease implications of these responses. Density, connectivity, metapopulation structure, phenology, and stress responses are presented as examples of host population parameters involved in species adaptation and reaction to climate change. A key challenge now for disease ecologists is to develop tools and

methods necessary to accurately detect the potential effects of climate change within already-complex disease dynamics.

A second challenge will be to anticipate and understand the unforeseen consequences of host responses to climate change on disease dynamics, such as through species shifting to new ranges and transporting their diseases into novel situations and hosts. The consequences range from amplification of disease impacts, to changes in geographic disease distribution, to potential pathogen extinction (Lafferty 2009; Randolph 2009).

Because it is difficult to predict precisely what will happen in any individual ecological scenario, conservationists must be as prepared as possible to make rapid assessments and potentially intervene in worst-case scenarios as they arise. However, a more preemptive role for conservation managers and researchers—and one that conservation medicine practitioners can prepare for by following the precepts of this discipline—would be to actively facilitate ecological resilience, an emerging concept often defined as the ability of a system to buffer disruption (Holling 1996). Examples of strategies that promote ecological resilience include maintaining biodiversity and preserving large landscapes for conservation purposes. To date, strategies linking the concept of ecological resilience with ecological health are lacking, and ecological health benchmarks to guide preventive actions do not exist. However, we can presume that strategies that enhance ecological resilience will also buffer the effects of changing infectious disease dynamics. Furthermore, a sound management concept is that once disease is introduced into a susceptible system it is very difficult to control, and almost always impossible to eradicate. Thus, a crucial strategy is to take measures to limit new disease introductions. The most important tool in this endeavor is surveillance and monitoring and, fortunately, climate-change monitoring efforts are increasing exponentially worldwide, offering opportunities to incorporate disease monitoring within these efforts. The growing body of data from these surveillance efforts should form the basis of adaptive management responses for disease interventions (Plowright et al. 2008a). Understanding the interactions between climate change, species responses, and disease is an emerging area within ecological health investigation, and a vital one if we are to succeed in limiting the damaging effects of

climate change on species and ecosystems already under stress.

## ACKNOWLEDGMENTS

Thanks to Kevin Lafferty and Greg Pederson for their insightful reviews, which significantly improved this manuscript.

## REFERENCES

- Altizer, S., A. Dobson, P. Hosseini, P. Hudson, M. Pascual and P. Rohani. 2006. Seasonality and the dynamics of infectious diseases. *Ecol Lett* 9:467–484.
- Anderson, R.C. 2002. Nematode parasites of vertebrates: Their development and transmission. CABI Publishing, London.
- Ball, F., D. Mollison and G. Scalia-Tomba. 1997. Epidemics with two levels of mixing. *Ann App Probab* 7:46–89.
- Bartlett, M.S. 1957. Measles periodicity and community size. *J R Stat Soc* 120:48–71.
- Birt, P. (2004). Mutualistic interactions between the nectar-feeding little red flying fox *Pteropus scapulatus* (Chiroptera: Pteropodidae) and flowering eucalypts (Myrtaceae): habitat utilisation and pollination. University of Queensland PhD Thesis, Brisbane.
- Bjornstad, O.N., B.F. Finkenstadt and B.T. Grenfell. 2002. Dynamics of measles epidemics: Estimating scaling of transmission rates using a Time series SIR model. *Ecol Monogr* 72:169–184.
- Bouma, A., M.C.M. de Jong and T.G. Kimman. 1995. Transmission of pseudorabies virus within pig-populations is independent of the size of the population. *Prev Vet Med* 23:163–172.
- Bradley, C., and S. Altizer. 2005. Parasites hinder monarch butterfly flight ability: implications for disease spread in migratory hosts. *Ecol Lett* 8:290–300.
- Bruno, J.F., E.R. Selig, K.S. Casey, C.A. Page, B.L. Willis, C.D. Harvell, H. Sweatman and A.M. Melendy. 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. *PLoS Biol* 5:e124.
- Burek, K.A., F.M.D. Gulland and T.M. O'Hara. 2008. Effects of climate change on Arctic marine mammal health. *Ecol Appl* 18:S126–S134.
- Buskirk, J.V., R.S. Mulvihill and R.C. Leberman. 2009. Variable shifts in spring and autumn migration phenology in North American songbirds associated with climate change. *Glob Change Biol* 15:760–771.
- Bustnes, J.O., S.A. Hanssen, I. Folstad, K.E. Erikstad, D. Hasselquist and J. U. Skaare. 2004. Immune function and organochlorine pollutants in arctic breeding glaucous gulls. *Arch Environ Con Tox* 47:530–541.
- Caley, P., G.J. Hickling, P.E. Cowan and D.U. Pfeiffer. 1999. Effects of sustained control of brushtail possums on levels of *Mycobacterium bovis* infection in cattle and brushtail possum populations from Hohotaka, New Zealand. *N Z Vet J* 47:133–142.
- Cattadori, I.M., D.T. Haydon and P.J. Hudson. 2005. Parasites and climate synchronize red grouse populations. *Nature* 433:737–741.
- Cauchemez, S., A. Valleron, P. Boëlle, A. Flahault and N.M. Ferguson. 2008. Estimating the impact of school closure on influenza transmission from sentinel data. *Nature* 452:750–754.
- Cheville, N.F., D.R. McCullough and L.R. Paulson. 1998. Brucellosis in the Greater Yellowstone Area. National Academy Press, Washington, D.C.
- Conner, M.M., M.W. Miller, M.R. Ebinger and K.P. Burnham. 2007. A meta-BACI approach for evaluating management intervention on chronic wasting disease in mule deer. *Ecol Appl* 17:140–153.
- Côté, I.M., and R. Poulin. 1995. Parasitism and group-size in social animals—a metaanalysis. *Behav Ecol* 6:159–165.
- Craft, M.E., P.L. Hawthorne, C. Packer and A.P. Dobson. 2008. Dynamics of a multihost pathogen in a carnivore community. *J Anim Ecol* 77:1257–1264.
- Crawley, M.J., S.D. Albon, D.R. Bazely, J.M. Milner, J.G. Pilkington and A.L. Tuke. 2004. Vegetation and sheep population dynamics. In Clutton-Brock, T.H. and J.M. Pemberton, eds. *Soay Sheep: Dynamics and Selection in an Island Population*, pp. 89–112. Cambridge University Press, Cambridge.
- Creel, S., and M. Creel. 2009. Density dependence and climate effects in Rocky Mountain elk: an application of regression with instrumental variables for population time series with sampling error. *J Anim Ecol* 78: 1291–1297.
- Cross, P.C., W.H. Edwards, B.M. Scurlock, E.J. Maichak and J.D. Rogerson. 2007. Effects of management and climate on elk brucellosis in the Greater Yellowstone Ecosystem. *Ecol Appl* 17:957–964.
- Cross, P.C., P.L.F. Johnson, J.O. Lloyd-Smith and W.M. Getz. 2007. Utility of  $R_0$  as a predictor of disease invasion in structured populations. *J R Soc Interface* 4:315–324.
- Cross, P.C., J.O. Lloyd-Smith, P.L.F. Johnson and W.M. Getz. 2005. Duelling timescales of host mixing and disease recovery determine disease invasion in structured populations. *Ecol Lett* 8:587–595.
- Delahay, R.J., S. Langton, G.C. Smith, R.S. Clifton-Hadley and C.I. Cheeseman. 2000. The spatio-temporal distribution of *Mycobacterium bovis* (bovine

- tuberculosis) infection in a high-density badger population. *J Anim Ecol* 69:428–441.
- Dobson, A. 2004. Population dynamics of pathogens with multiple host species. *Am Nat* 164:S64–S78.
- Dobson, A., S. Kutz, M. Pascual and R. Winfree. 2003. Pathogens and parasites in a changing climate. In Hannah, L., & T. Lovejoy, eds. *Climate change and biodiversity: synergistic impacts*, pp. 33–38. Yale University Press, New Haven.
- Dobson, A.P., and R.J. Carper. 1992. Global warming and potential changes in host–parasite and disease vector relationships. In Peters, R., & T. Lovejoy, eds. *Global warming and biodiversity*, pp. 201–217. Yale University Press, New Haven.
- Donnelly, C.A., R. Woodroffe, D.R. Cox, F.J. Bourne, C.L. Cheeseman, R.S. Clifton-Hadley, G. Wei, G. Gettinby, P. Gilks, H. Jenkins, W.T. Johnston, A.M. Le Febvre, J.P. McInerney and W.I. Morrison. 2006. Positive and negative effects of widespread badger culling on tuberculosis in cattle. *Nature* 439:843–846.
- Epps, C.W., D.R. McCullough, J.D. Wehausen, V.C. Bleich and J.L. Reche. 2004. Effects of climate change on population persistence of desert-dwelling mountain sheep in California. *Conserv Biol* 18:102–113.
- Ferrari, M.J., R.F. Grais, N. Bharti, A.J.K. Conlan, O.N. Bjornstad, L.J. Wolfson, P.J. Guerin, A. Djibo and B.T. Grenfell. 2008. The dynamics of measles in sub-Saharan Africa. *Nature* 451:679–684.
- Fisher, M. 1997. Decline in the juniper woodlands of Raydah Reserve in southwestern Saudi Arabia: a response to climate changes? *Global Ecol Biogeogr* 6:379–386.
- Getz, W.M. and J. Pickering. 1983. Epidemic models: thresholds and population regulation. *Am Nat* 121:892–898.
- Glass, K. and B. Barnes. 2007. How much would closing schools reduce transmission during an influenza pandemic? *Epidemiology* 18:623–628.
- Grenfell, B.T., O.N. Bjornstad and B.F. Finkenstadt. 2002. Dynamics of measles epidemics: Scaling noise, determinism, and predictability with the TSIR model. *Ecol Monogr* 72:185–202.
- Grenfell, B.T. and G. Smith. 1983. Population biology and control of ostertagiasis in first-year grazing calves. *P Vet Epid Preventative Med*: 54–61.
- Halloran, M.E., N.M. Ferguson, S. Eubank, I.M. Longini, D.A. Cummings, B. Lewis, S. Xu, C. Fraser, A. Vullikanti, T.C. Germann, D. Wagener, R. Beckman, K. Kadau, C. Barrett, C.A. Macken, D.S. Burke and P. Cooley. 2008. Modeling targeted layered containment of an influenza pandemic in the United States. *P Acad Nat Sci Phila* 105:4639–4644.
- Harvell, C.D., C.E. Mitchell, J.R. Ward, S. Altizer, A.P. Dobson, R.S. Ostfeld and M.D. Samuel. 2002. Ecology—Climate warming and disease risks for terrestrial and marine biota. *Science* 296:2158–2162.
- Harvell, D., S. Altizer, I.M. Cattadori, L. Harrington and E. Weil. 2009. Climate change and wildlife diseases: when does the host matter the most? *Ecology* 90:912–920.
- Hess, G. 1996. Disease in metapopulation models: Implications for conservation. *Ecology* 77:1617–1632.
- Hoberg, E.P., L. Polley, E.J. Jenkins and S.J. Kutz. 2008. Pathogens of domestic and free-ranging ungulates: global climate change in temperate to boreal latitudes across North America. *Rev Sci Tech* 27:511–528.
- Holling, C.S. 1996. Engineering resilience versus ecological resilience. In Schulze, P., ed. *Engineering within ecological constraints*, pp. 31–44. National Academy, Washington, D.C.
- IPCC. 2007. Fourth Assessment Report of the Intergovernmental Panel on Climate Change.
- Jolles, A.E., V.O. Ezenwa, R.S. Etienne, W.C. Turner and H. Olf. 2008. Interactions between macroparasites and microparasites drive infection patterns in free-ranging African buffalo. *Ecology* 89:2239–2250.
- Joly, D.O. and F. Messier. 2004. Factors affecting apparent prevalence of tuberculosis and brucellosis in wood bison. *J Anim Ecol* 73:623–631.
- Kermack, W.O. and A.G. McKendrick. 1927. Contributions to the mathematical theory of epidemics. *P Royal Soc Edin* 115:700–721.
- Kutz, S.J., E.P. Hoberg, L. Polley and E.J. Jenkins. 2005. Global warming is changing the dynamics of Arctic host-parasite systems. *P Biol Sci* 272:2571–2576.
- Lafferty, K.D. 2009. The ecology of climate change and infectious diseases. *Ecology* 90:888–900.
- Lloyd-Smith, J.O., P.C. Cross, C.J. Briggs, M. Daugherty, W.M. Getz, J. Latto, M.S. Sanchez, A.B. Smith and A. Swei. 2005. Should we expect population thresholds for wildlife disease? *Trends Ecol Evol* 20:511–519.
- Lovejoy, T. 2008. Climate change and biodiversity. *Rev Sci Tech* 27:331–338.
- Marcogliese, D.J. 2008. The impact of climate change on the parasites and infectious diseases of aquatic animals. *Rev Sci Tech* 27:467–484.
- Markus, N. and L. Hall. 2004. Foraging behaviour of the black flying-fox (*Pteropus alecto*) in the urban landscape of Brisbane, Queensland. *Wildlife Res* 31:345–355.
- McCallum, H. and A. Dobson. 2002. Disease, habitat fragmentation and conservation. *P Roy Soc Lond B Biol* 269:2041–2049.
- McDonald-Madden, E., E.S.G. Schreiber, D.M. Forsyth, D. Choquenot and T.F. Clancy. 2005. Factors affecting grey-headed flying-fox (*Pteropus poliocephalus* :

- Pteropodidae) foraging in the Melbourne metropolitan area, Australia. *Austral Ecol* 30:600–608.
- Middleton A.D., M.J. Kauffman, D.E. McWhirter, J.G. Cook R.C. Cook, A.A. Nelson, M.D. Jimenez, and R.W. Klaver (in review) Animal migration amid shifting patterns of phenology and predation: lessons from a Yellowstone elk herd.
- Moran, P.A.P. 1953. The statistical analysis of the Canadian Lynx cycle II. Synchronization and meteorology. *Aust J Zool* 1:291–298.
- Murray, D.L., E.W. Cox, W.B. Ballard, H.A. Whitlaw, M.S. Lenarz, T.W. Custer, T. Barnett and T.K. Fuller. 2006. Pathogens, nutritional deficiency, and climate influences on a declining moose population. *Wildlife Monog* 1–29.
- Mydlarz, L.D., E.S. McGinty and C.D. Harvell. 2010. What are the physiological and immunological responses of coral to climate warming and disease? *J Exp Biol* 213:934–945.
- Ostfeld, R.S. 2009. Climate change and the distribution and intensity of infectious diseases. *Ecology* 90:903–905.
- Ozgul, A., S. Tuljapurkar, T.G. Benton, J.M. Pemberton, T.H. Clutton-Brock and T. Coulson. 2009. The dynamics of phenotypic change and the shrinking sheep of St. Kilda. *Science* 325:464–467.
- Parnesan, C. 1996. Climate and species' range. *Nature* 382:765–766.
- Parnesan, C. and G. Yohe. 2003. A globally coherent fingerprint of climate change impacts across natural systems. *Nature* 421:37–42.
- Parry-Jones, K.A. and M.L. Augée. 1991. Food selection by gray-headed flying foxes (*Pteropus poliocephalus*) occupying a summer colony site near Gosford, New South Wales. *Wildl Res* 18:111–124.
- Plowright, R.K., H.E. Field, C. Smith, A. Divljan, C. Palmer, G.M. Tabor, P. Daszak and J.E. Foley. 2008b. Reproduction and nutritional stress are risk factors for Hendra virus infection in little red flying foxes (*Pteropus scapulatus*). *Proc R Soc B* 275:861–869.
- Plowright, R.K., P. Foley, H.E. Field, A.P. Dobson, J.E. Foley, P. Eby and P. Daszak. 2011. Urban habituation, ecological connectivity and epidemic dampening: the emergence of Hendra virus from flying foxes (*Pteropus* spp.). *P R Soc B* doi:10.1098/rspb.(2011).0522.
- Plowright, R.K., S.H. Sokolow, M.E. Gorman, P. Daszak and J.E. Foley. 2008a. Causal inference in disease ecology: investigating ecological drivers of disease emergence. *Front Ecol Environ* DOI: 101890/070086.
- Pounds, J.A., M.P.L. Fogden and J.H. Campbell. 1999. Biological responses to climate change on a tropical mountain. *Nature* 398:611–615.
- Randolph, S.E. 2009. Perspectives on climate change impacts on infectious diseases. *Ecology* 90:927–931.
- Reading, C. 2007. Linking global warming to amphibian declines through its effects on female body condition and survivorship. *Oecologia* 151:125–131.
- Renecker, L.A. and R.J. Hudson. 1986. Seasonal energy expenditures and thermoregulatory responses of moose. *Can J Zool* 64:322–327.
- Ritchie, C. 2008. Management and challenges of the mountain pine beetle infestation in British Columbia. *Alces* 44:127–135.
- Rogers, L.M., R.J. Delahay, C.L. Cheeseman, G.C. Smith and R.S. Clifton-Hadley. 1999. The increase in badger (*Meles meles*) density at Woodchester Park, south-west England: a review of the implications for disease (*Mycobacterium bovis*) prevalence. *Mammalia* 63:183–192.
- Saunders, L.M., D.M. Tompkins and P.J. Hudson. 2000. The role of oxygen availability in the embryonation of *Heterakis gallinarum* eggs. *Int J Parasitol* 30:1481–1485.
- Welbergen, J.A., S.M. Klose, N. Markus and P. Eby. 2008. Climate change and the effects of temperature extremes on Australian flying-foxes. *Proc Biol Sci* 275:419–425.
- Williams, J.W., S.T. Jackson and J.E. Kutzbach. 2007. Projected distributions of novel and disappearing climates by 2100 AD. *Proc Natl Acad Sci USA* 104:5738–5742.
- Wilson, K., B.T. Grenfell, J.G. Pilkington, H.E.G. Boyd and F.M.D. Gulland. 2004. Parasites and their impact. *In* Clutton-Brock, T.H., & J.M. Pemberton, eds. *Soay Sheep: Dynamics and selection in an island population*, pp. 113–165. Cambridge University Press, Cambridge.