Mathur, K. K., G. Harpalani, N. L. Kalra, G. G. K. Murthy, and M. V. V. L. Narasimham. 1992. Epidemic of malaria in Barmer district (Thar desert) of Rajasthan during 1990. Indian Journal of Malariology 2:1–10.

McMichael, A., and M. J. Bouma. 2000. Global change, invasive species and human health. Pages 191–210 *in* H. Mooney and R. Hobbs, editors. The impact of global change on invasive species. Island Press, Covelo, California, USA.

Molineaux, L. 1988. The epidemiology of human malaria as an explanation of its distribution, including some implications for its control. Pages 913–998 *in* W. H. Wernsdorfer and I. McGregor, editors. Malaria. Churchill Livingstone, Edinburgh, UK.

Mouchet, J., S. Manguin, J. Sircoulon, S. Laventure, O. Faye, A. W. Onapa, P. Carnevale, J. Julvez, and D. Fontanille. 1998. Evolution of malaria in the last 40 years: impact of climate and human factors. Journal of the American Mosquito Association 14:121–130.

Negash, K., A. Kebede, A. Medhin, D. Argaw, O. Babaniyi, J. O. Guintran, and C. Delacollette. 2005. Malaria epidemics in the highlands of Ethiopia. East African Medical Journal 82:186–192.

Pascual, M., J. Ahumada, L. F. Chaves, X. Rodó, and M. Bouma. 2006. Malaria resurgence in East African Highlands: temperature trends revisited. Proceedings of the National Academy of Sciences (USA) 103:5829–5834.

Pascual, M., L. F. Chaves, X. Rodo, B. Cash, and M. Yunus. 2008*a*. Predictability of endemic cholera: the role of climate variability and disease dynamics. Climate Research 36:131–140.

Pascual, M., B. Gazelles, M. J. Bouma, L. F. Chaves, and K. Koelle. 2008*b*. Shifting patterns: malaria dynamics and rainfall variability in an East African Highland. Proceedings of the Royal Society Series B 275:123–132.

Rogers, D. J., S. E. Randolph, R. W. Snow, and S. I. Hay. 2002. Satellite imagery in the study and forecast of malaria. Nature 415:710–715.

Shanks, G. D., S. I. Hay, J. A. Omumbo, and R. W. Snow. 2005. Malaria in Kenya's western highlands. Emerging Infectious Diseases 11:1425–1432.

Sissoko, M. S., A. Dicko, O. J. Briët, M. Sissoko, I. Sagara, H. D. Keita, M. Sogoba, C. Rogier, Y. T. Touré, and O. K. Doumbo. 2004. Malaria incidence in relation to rice cultivation in the irrigated Sahel of Mali. Acta Tropica 89:161–170.

Tanser, F. C., B. Sharp, and D. le Sueur. 2003. Potential effect of climate change on malaria transmission in Africa. Lancet 362:1792–1798.

Thomson, M. C., F. J. Doblas-Reyes, S. J. Mason, R. Hagedorn, S. J. Connor, T. Phindela, A. P. Morse, and T. N. Palmer. 2006. Malaria early warnings based on seasonal climate forecasts from multi-model ensembles. Nature 439(2):576–579.

Tulu, A. N. 1996. Determinants of malaria transmission in the highlands of Ethiopia: impact of global warming on morbidity and mortality ascribed to malaria. Dissertation. London School of Hygiene and Tropical Medicine, London, UK.

United Nations. 2004. Focus on Ethiopia. UN Country team Ethiopia. August 2004. United Nations, New York, New York, USA.

Climate change and wildlife diseases: When does the host matter the most?

Drew Harvell,[1,7] Sonia Altizer,[2] Isabella M. Cattadori,[3,4] Laura Harrington,[5] and Ernesto Weil[6]

[1]*Department of Ecology and Evolutionary Biology, Cornell University, Ithaca, New York 14853 USA*
[2]*Odum School of Ecology, University of Georgia, Athens, Georgia 30602 USA*
[3]*Division of Animal Production and Public Health, Faculty of Veterinary Medicine, University of Glasgow, Glasgow G611QH United Kingdom*
[4]*Center for Infectious Disease Dynamics, Department of Biology, The Pennsylvania State University, University Park, Pennsylvania 16802 USA*
[5]*Department of Entomology, Cornell University, Ithaca, New York 14853 USA*
[6]*Department of Marine Sciences, University of Puerto Rico, P.O. Box 9013, Mayagüez, Puerto Rico 00681-9013 USA*

Introduction

The paper by Lafferty (2009) injects careful detail and new science into the question of how climate warming will affect the prevalence and distribution of vector-borne diseases of humans. This has been an area of

intense controversy, in part because of implications for human health and disease prevention strategies. Six years ago, one hypothesis about climate change and infectious diseases suggested that increased temperature and rainfall could facilitate the emergence and persistence of many infectious microorganisms, i.e., a warmer world would be a sicker world (Harvell et al. 2002). Of all the infectious diseases, it seemed likely that human vector-borne diseases would respond most promptly to localized warming events. Specifically, in temperate climates, we might expect the range and activity of

mosquitoes and the pathogens they vector, such as malaria and dengue, to increase with warmer temperatures (Martens 1999; but see Rogers and Randolph 2000). From a later vantage point in 2009, the evidence outlined by Lafferty considers new complexities and uses long-term data sets to re-evaluate previous predictions. Surprisingly, insect-vectored diseases resoundingly do not show a net expansion in range or increase in prevalence, based on evidence and predictions from GIS analyses and mathematical modeling studies. Lafferty (2009) lays out three explanations: (1) anthropogenic activities directly influence the distributions of vectors and infectious disease in ways unrelated to climate, (2) vectors and pathogens are limited by thermal maxima, so that temperature changes lead to shifts rather than expansions in distribution, and (3) other factors such as host acquired immunity and vector or parasite life history traits are linked to habitat suitability in addition to climate.

As highlighted throughout Lafferty's paper, the complexity of malaria ecology (as well as that of other mosquito-borne infections) requires that models adequately consider the effects of climate on vector population biology, biting activity and vector–parasite interactions, each of which can have a significant impact on the final predictions (Garrett-Jones 1964, Styer et al. 2007, Harrington et al. 2008). Human malaria is, in fact, caused by four biologically distinct species of *Plasmodium*. Each species has characteristic rates of development within the human host and invertebrate vector, intricate relationships with the immune systems of both host and vector, and biological compatibility with certain species of mosquito. Not all vectors are the same, just as the course of malaria pathology is not the same in all human hosts. Additionally, the impact of factors such as partial immunity and multiple infections are difficult to estimate. Therefore, the alarming predictions of malaria returning to temperate regions should be critically assessed.

One reason why the threat of malaria returning to Europe and North America is not as dire as some would predict is because humans are the only vertebrate reservoir for infection, and large numbers of infected people would be required to sustain a malaria outbreak in England and many other countries where malaria has been eradicated. Competent mosquito vectors still thrive in England, other European countries and also in the United States, but the quality of health care and standards of living have changed tremendously in these countries since malaria was considered endemic. Indeed, the potential for positive feedback between human poverty and the threat of an expansion of vector borne diseases was highlighted by Lafferty in his review, with particular reference to the difficulty in teasing apart interactions between anthropogenic environmental changes, drug resistance, lack of health care, and vector control. Given our limited scientific knowledge, Lafferty (2009) has presented a balanced overview, demonstrat-

ing little evidence for net range expansions and increases in human cases based on climate warming alone. Instead, a clearer relationship between poverty, lack of effective control strategies and malaria infections represents a more certain cause of future increases in incidence of human cases of malaria.

Lafferty's focus on vector-borne human infectious diseases provides a useful test case against which to compare our expectations for the effect of global changes on pathogens affecting wildlife populations. New questions that are raised include: do simple predictions that many infections will increase with climate warming hold up where pathogens and vectors are not controlled by humans? What happens when both host and pathogen are sensitive to temperature changes? If climate–disease relationships are not observed for some pathogens, why and when is the modulating effect of climatic variables on the interaction between hosts and infectious diseases lost? In this perspective we detail a few examples of wildlife-pathogen interactions in both marine and terrestrial animal systems that emphasize how host biology can be crucially important for climate-disease relationships. Specifically, we argue that (1) correlations between climate warming and pathogen outbreaks have contributed to dramatic declines or extinctions in several groups of ectothermic hosts, (2) animal migrations and range shifts can be affected by climate change and this can potentially increase or decrease disease transmission, and (3) host acquired immunity can alter the expected direct effect of climate on disease dynamics.

Pathogen Expansions and Emergence in Ectothermic Hosts

Interactions between climate warming and pathogen outbreaks have caused serious declines or extinctions in several groups of ectothermic hosts. In terrestrial ecosystems, entire communities of amphibians have been driven to the edge of extinction and beyond owing to chytridiomycosis outbreaks, with the timing of die-offs linked with warmer temperatures. Amphibians on the whole are extremely threatened, with nearly half of all species declining in their range of distribution (Stuart et al. 2004). Infectious diseases have caused some of the most rapid and severe declines, in some cases wiping out entire communities in a matter of months (Lips et al. 2006). A correlational analysis on the amphibian community in Costa Rica provided support for warmer years favoring the growth of the pathogenic fungus *Batrachochytrium dendrobatidis* at high altitudes (Pounds et al. 2006). Specifically, it has been suggested that climate warming shifted nighttime temperature minima towards the pathogen's growth optimum, and simultaneously prevented daytime maxima from exceeding the pathogen's thermal limits through increased cloud cover. This hypothesis helps to explain the die-offs of numerous species of harlequin frogs in Central and South America following warm years, and the spread of

the pathogenic fungus into amphibian populations at mid-to-high elevations. However, the strength of this climate-disease association was recently questioned by Lips et al. (2008); specifically, a reanalysis of mortality data suggested that multiple introductions of *B. dendrobatidis*, coupled with its spatial spread along mountain ranges, better explained the timing of harlequin frog die-offs (Lips et al. 2008). It is important to note that warmer temperatures have also been linked with the occurrence of chytrid-related disease in montane regions in Spain (Bosch et al. 2007), and that the effects of climate, disease and other factors causing amphibian declines are not mutually exclusive and could interact in ecologically significant ways.

In the ocean, several species of California abalones have experienced major population declines caused by a combination of warming water and infectious disease. Elevated seawater temperatures have been shown to play a key role driving both transmission of the rickettsial agent and development of associated clinical disease (withering syndrome, WS) in red abalone, *Haliotis rufescens*, one of the five California abalones that were commercially fished prior to closure of the fishery nearly a decade ago (Moore et al. 2000, Braid et al. 2005, Vilchis et al. 2005). Similarly, black abalone (*H. cracherodii*) has been almost extirpated from southern California by its temperature-dependent susceptibility to WFS (Altstatt et al. 1996, Moore et al. 2000). On the Atlantic coast, infections of oysters (*Crassostrea virginica*), by the protozoan parasite *Perkinsus marinus* represent the best documented case of a marine parasite range expansion driven by climate warming. This parasite expanded from its range in the southeast up through Maryland to a new 500 km northeastern range, coincident with winter warming in the 1990s (Cook et al. 1998). The 1990–1991 expansion into the northeast has continued in coincidence with the anomalously warm temperatures recorded in the last decade (Ford and Smolowitz 2007). Epizootics of *P. marinus* are impressively seasonal at all locations, with prevalence peaking in late summer and dying back in winter (Ford and Smolowitz 2007). However, careful studies highlight the potential for multiple factors to be involved in warming-associated range expansion (Ford and Smolowitz 2007). For example, there is evidence that the level of aggressiveness and transmission of *P. marinus* has increased throughout its range, and hence recent epizootics are more quickly established in new areas. This raises the now familiar question of whether the new range expansion is caused by change in temperature directly, or by evolutionary changes in properties of the host or pathogen. The evolutionary hypothesis was recently rejected by experimental studies which showed that *P. marinus* had not differentiated genetically in northern oyster populations that had experienced more recent increases in disease; the parsimonious conclusion is that warming winter temperatures alone have allowed

the northward parasite range expansion (Ford and Chintala 2006, Ford and Smalowitz 2007).

Coral reefs represent an entire marine ecosystem that is threatened by the encroachment of temperature-sensitive disease outbreaks; indeed, coral reefs are the ecosystem the most impacted by small increases in temperature causing bleaching and mortality and facilitating disease outbreaks (Hoegh-Guldberg et al. 2007). For example, 2005 was the warmest summer/fall season recorded for the eastern Caribbean in 100 years. At its peak in late October, the warm temperature anomaly that developed reached 14 degree-heating weeks (i.e., temperatures of 1°C above the typical seasonal maximum for 14 successive weeks; Wilkinson and Souter 2008). In the Virgin Islands, the primary reef building coral species bleached during this warming event, eventually recovered its pigments, but then developed lethal white plague disease (Miller et al. 2006). As a result, coral cover was reduced by 50% in this one event alone. Again in the Virgin Islands, a disease of the federally listed endangered coral species *Acropora palmata* also increased during the 2005 temperature anomaly event (Muller et al. 2008).

In many reef areas of the Caribbean, Caribbean yellow band disease (CYBD) prevalence and virulence increased significantly from the late 1990s to the mid 2000s, and this is now the most damaging bacterial disease that affects all four coral species of the genus *Montastraea*, the most important reef-building species for this area (Fig. 1A; Bruckner and Bruckner 2006, Weil et al. 2006, Croquer and Weil 2009, Weil and Croquer 2009). Disease prevalence in *Montastraea* populations has markedly increased over the last eight years and significantly covaried ($r^2 = 0.787$, $P \leq 0.01$) with the increase in the annual mean sea water temperature from 1999 to 2007 (Fig. 1A). Likewise, the within-colony dynamics of the disease (based on visual disease signs and lesion growth rates) have changed dramatically in the last eight years. In fact, the seasonal pattern of high growth rates of lesions in summer and significantly lower growth rates or complete disappearance in winter has now been replaced by a significant increase in the growth rate of the lesions throughout the year, and no sign of disease decline (Fig. 1B). A significant positive relationship was observed between the lesion growth rate and mean water temperatures ($r^2 = 0.54$, $P \leq 0.05$), providing evidence that water temperature affects the development of CYBD on colonies of the star coral *Montastraea faveolata* (Fig. 1B). Winter average water temperatures used to drop below 25.5°C in the area, however, following the summer of 2002, the average winter temperatures have not dropped below 26.5°C, and a steady increase in the mean winter temperatures has been recorded throughout the last nine years. Warmer winters seem to favor CYBD growth and pathogenicity, which now remain high even during the winter seasons,
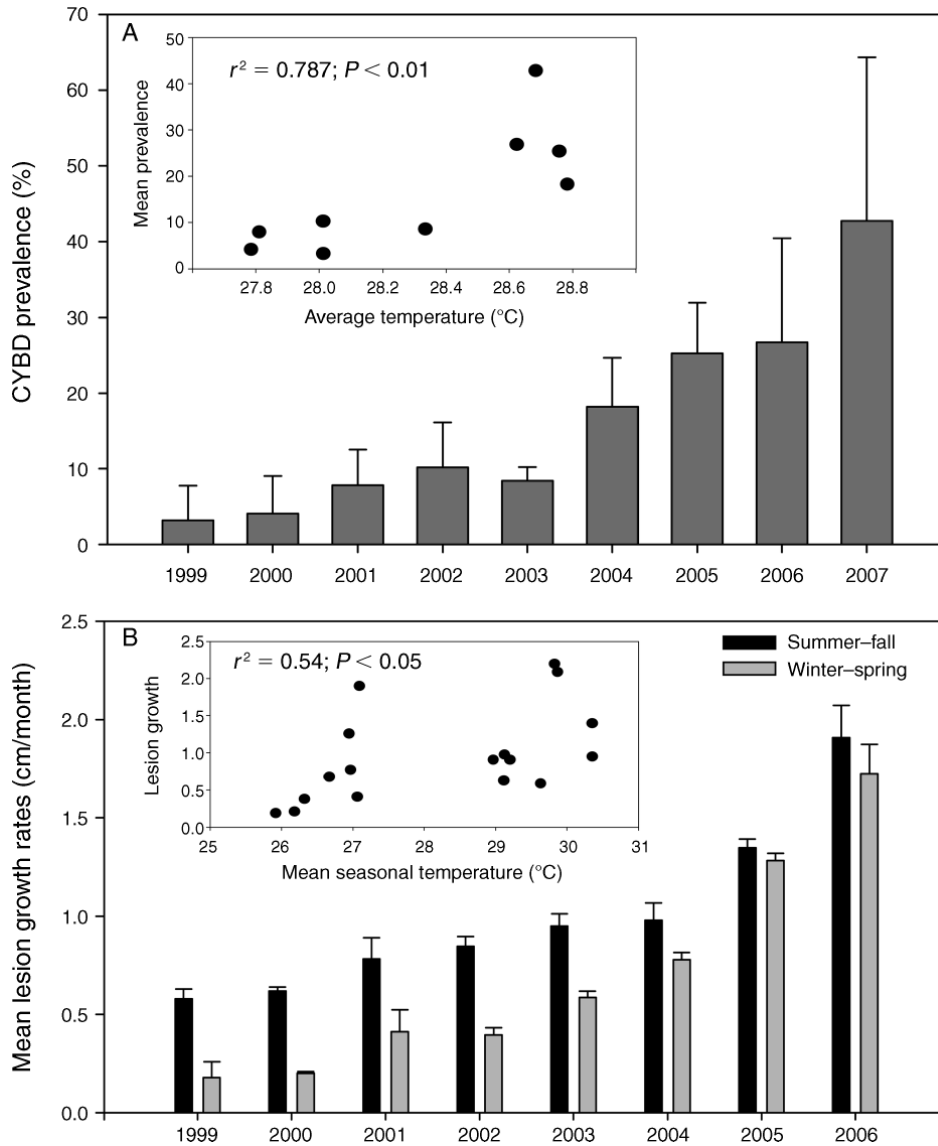
FIG. 1. Temporal changes in Caribbean yellow band disease (CYBD). (A) Increase in CYBD prevalence in the Caribbean coral genus *Montastraea* in reefs off the southwest coast of Puerto Rico from 1999 to 2007. The inset shows the positive and significant correlation between the yearly increase in mean prevalence and the average yearly surface water temperature. (B) Seasonal variability in CYBD lesion growth rates measured in over 100 tagged colonies of *Montastraea faveolata* in La Parguera, Puerto Rico from 1999 to 2006. The inset shows the significant positive covariation between the linear lesion growth rates and the average seasonal surface seawater temperature for the same period. Error bars show +SE.

causing epidemic levels of disease in populations of the *Montastraea* species in many reefs across the Caribbean.

The mechanisms of climate sensitivity for many different coral syndromes are not fully understood (Harvell et al. 2007). In the few cases where a coral pathogen was cultured, experiments showed a positive relationship between temperature and pathogen growth rate (Harvell et al. 2007). One prevailing hypothesis is that many new coral pathogens are opportunists that become more virulent (grow faster or become more infectious) at warmer temperatures. Because warmer temperatures can also directly immune compromise the hosts, this combined impact of virulent pathogens and immuno-compromised hosts could underlie many dis-

ease outbreaks (Harvell et al. 2007). Although an appealing hypothesis, in the case where immunity and environment were studied in gorgonian corals using short term laboratory experiments and data from sustained field monitoring, results showed that enzymes and cellular immunity were actually activated (and not suppressed) by both pathogen inoculation and temperature increase (Ward et al. 2006, Mydlarz et al. 2008). This observation raises questions concerning whether some clades of cnidarians like gorgonians corals show more immune resilience in the face of warming than others, like scleractinian corals.

As in most ecosystems, the question remains open about which is the more important driver of changing

pathogen distributions: increasing pathogen growth, transmission and virulence with temperature, or changes in host immunity (Ellner et al. 2007). The tension between a direct positive effect of temperature on host defenses, combined with the potential for temperature stress to compromise host immunity, has been often highlighted in ectothermic hosts. For example, a recent study by Raffel et al. (2006) on red-spotted newts showed that between-season (longer-term) environmental temperature was positively correlated with the numbers of circulating lymphocytes and eosinophils in the individuals. Moreover, within-season effects and responses of newts to climate variability showed that these animals could be more susceptible to infectious diseases during autumn or following fluctuations in temperature. As pointed out by Fisher (2007), these considerations are important for predicting (1) how well amphibians and other ectotherms can mount an immune response against infectious agents outside of their typical climate envelope, (2) how temperature affects different types of host defenses (which in turn respond to different types of infectious diseases), and (3) the role of climate variability, in addition to average changes in temperature, on host susceptibility.

ANIMAL MOVEMENTS, HOST RANGE SHIFTS, AND INFECTIOUS DISEASES

Another mechanism relevant to the emergence or outbreaks of infectious diseases is the effect of climate change on the movements of animal populations to enhance their exposure to infectious diseases. Migratory species can be particularly sensitive to climate change (Collingham and Huntley 2000, Hickling et al. 2006) and some migratory species have already responded to altered thermal regimes by shifting their ranges of distribution and migration routes (Parmesan and Yohe 2003, Perry et al. 2005). In other cases, climate warming could cause a gradual break down in the migration itself by making environmental conditions in breeding grounds more favorable for the year-round survival of host populations (i.e., replacing migratory populations with year-round resident populations; Lusseau et al. 2004, Bradshaw and Holzapfel 2007). Migrations can confer multiple benefits by allowing hosts to escape the continual build-up of pathogens in the environment (Loehle 1995, Altizer et al. 2004) or by weeding infected animals out of the population during strenuous migratory journeys (e.g., Gylfe et al. 2000, Bradley and Altizer 2005). Several host–parasite systems offer evidence that seasonal host migrations reduce parasite prevalence, including studies of warble flies affecting reindeer (Folstad et al. 1991) and nematodes affecting fall armyworm moths (Simmons and Rogers 1991).

A case study of monarch butterflies and the protozoan parasite *Ophryocystis elektroscirrha* provides support for both the role of migration in lowering parasite prevalence (Altizer et al. 2000) and the vulnerability of migration to climate warming (Oberhauser and Peterson 2003). Globally, all monarch populations examined to date are parasitized by *O. elektroscirrha*; prevalence is highest in monarchs from tropical locations that breed year-round (i.e., nonmigratory populations) and lowest in the population in eastern North America that migrates the longest distances annually. Climate warming, combined with increased planting of tropical milkweed species that do not die back seasonally, has enabled the persistence of small winter breeding populations in the southeastern United States in recent years. These nonmigratory populations are likely to suffer from heavy parasite burdens and can also harbor more virulent parasite strains (i.e., strains that replicate faster and cause greater harm to hosts; De Roode et al. 2008). Thus, reductions in migratory tendencies among animals that breed in traditionally seasonal temperate regions could favor the transmission of infectious diseases and negatively impact wildlife populations.

Altered migration routes could also result in migratory animals encountering and transferring pathogens to previously unexposed host populations, or becoming exposed themselves to novel infectious diseases. More generally, many terrestrial species are expected to undergo range shifts to higher elevations or latitudes as a result of climate warming, a phenomenon already observed for some taxonomic groups (Hickling et al. 2006). A largely overlooked phenomenon is the degree to which these range shifts could bring novel groups of species into contact and facilitate cross-species transmission of infectious disease (e.g., Morgan et al. 2004, Brooker et al. 2007). Pathogens introduced into previously unexposed host populations can spread quickly, cause high case fatality rates and lead to stunning reductions in host abundance. For example, two major outbreaks of phocine distemper virus among harbor seals in the North Sea are thought to have been triggered by viral introduction from harp seals migrating far outside of their range (Osterhaus and Vedder 1988, Jensen et al. 2002). Although the hypothesis is that aberrant harp seal migrations were probably caused by over-fishing rather than by climate change, future introductions of novel pathogens are likely to occur as animal movement routes and geographic ranges shift in response to climate warming.

ACQUIRED IMMUNITY AND CLIMATE-DISEASE SYNERGISMS

Host immunity can play a major role in driving large scale and long term disease emergence and persistence. For parasites with direct life cycles that are not regulated by an effective immune response, we expect climate warming to increase the force of infection (the rate at which a host gets infected), leading to an increase in the prevalence and/or intensity of infection in adult hosts. On the other hand, for parasites that are regulated by inducible and lasting host defenses, climate warming could still increase the force of infection and parasite intensity in younger hosts, but older individuals will be more efficient at clearing the infection because of a faster

activation of the immune response (Cattadori et al. 2005a, Hudson et al. 2006). Therefore, if host–parasite interactions are immune-modulated, we expect that climate changes will be less powerful in driving disease dynamics, and the direct effect of climate on host–parasite interactions will be less apparent.

Climate change, immunity, and the pattern of infection of two common gastrointestinal nematodes (*Graphidium strigosum* and *Trichostrongilus retortaefor-mis*) have been investigated in a population of European rabbits (*Oryctolagus cuniculus*) in Scotland. Parasite intensity has changed in the rabbit population over 26 years, but in different ways for each of the two parasite species: *T. retortaeformis* showed large multi-annual variations but no long term trend, whereas *G. strigosum* increased more than two-fold over the same time period (Hudson et al. 2006). These patterns were consistent among hosts of different age classes, suggesting that changes in parasite intensity were not caused by a variation in the age structure of the host population over the years. Previous studies suggested that *T. retortae-formis*, but not *G. strigosum*, elicits a strong acquired immune response (Cattadori et al. 2005a, 2007, 2008, Cornell et al. 2008). Adult rabbits are able to clear almost completely *T. retortaeformis* infections, whereas no obvious regulation seems to occur in *G. strigosum*, and parasite intensity increases exponentially with host age.

Both parasite species have a free-living stage spent on the pasture, and the warm and moist environment of the grass micro-climate has a fundamental role in the survival, development, and behavior of the parasites (Crofton 1948a, b, Anderson 2000). Climatic data from the rabbit study showed an increase of 1°C in the mean air temperature and a weak positive trend in rainfall between 1980 and 2002 (Hudson et al. 2006; I. M. Cattadori, *unpublished data*). These data sets were also characterized by large multi-annual variations (coefficient of variation [CV]: 4% and 16%, respectively). An analysis of the relationship between climatic variables and parasite intensity revealed a significant and positive association between *G. strigosum* intensity and mean air temperature ($P = 0.004$, Fig. 2) but no association with total rainfall ($P = 0.241$). As expected, no relationships between climatic variables and *T. retortaeformis* were observed ($P = 0.860$ and $P = 0.174$, for mean air temperature and total rainfall, respectively, Fig. 2). These findings suggest that *G. strigosum*, which elicits no apparent immune response in its host, has been affected by temperature warming, and this probably caused the increase in parasite intensity in the adult rabbit population over two decades. Although *T. retortaefor-mis* free living stages are exposed to similar environmental conditions, the damping effect of host immunity has probably prevented this parasite from showing clear associations with climate records.

This example of climate change influencing a host–nematode interaction is not an exception. The intensity of infection of the gastrointestinal nematode *Tricho-*
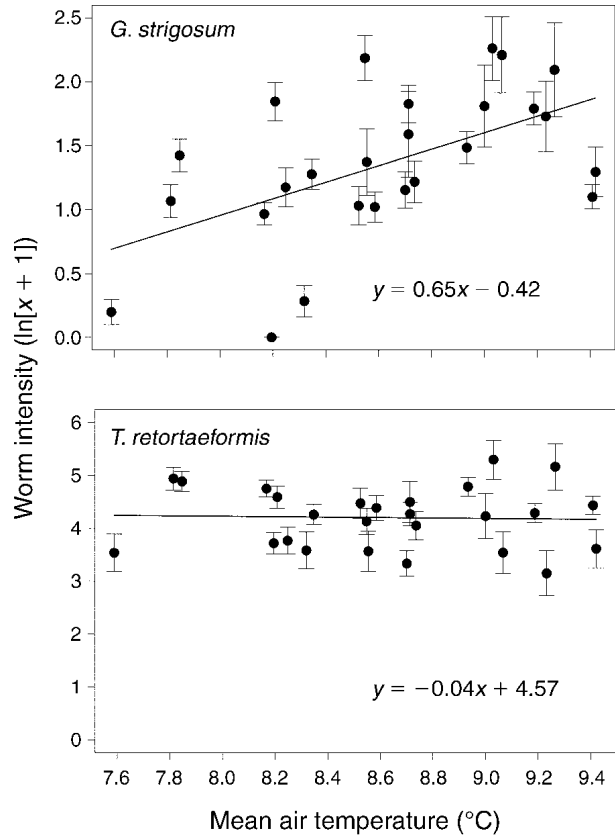

FIG. 2. Relationship between the mean intensity for two parasite species of the European rabbit and mean air temperature between 1977 and 2002 in Scotland (UK). Standard errors of the mean annual parasite intensity are reported.

strongylus tenuis has increased in its common host, the Red Grouse (*Lagopus lagopus scoticus*), over the last 30 years in England (Hudson et al. 2006). *T. tenuis* has a direct life cycle with a free living stage, parasite intensity is mainly regulated by birth–death demographic processes, and acquired immunity does not appear to play a relevant role in clearing host infections (Hudson and Dobson 1997, Hudson et al. 2006). Interestingly, the long term pattern of infection of this nematode very much resembles *G. strigosum*, such that intensity has consistently increased over the last three decades (Hudson et al. 2006). A detailed look at this pattern revealed that mean rainfall between April and July has slowly increased in the Red Grouse in English moorlands since 1979 and a very weak but significant positive relationship has been observed between *T. tenuis* intensity and April–July rainfall ($P < 0.024$). The Red Grouse breed mainly between April and July, and during this time of the year the parasite has the most detrimental effects on host reproduction and annual growth rate (Hudson et al. 1992). April to July mean rainfall also exhibited large multi-annual fluctuations since 1979 (CV: 29.5%). A large scale study on the effect of climate fluctuations and nematode transmission on spatial synchrony among Red Grouse populations

revealed that unexpected climatic events could cause dramatic and synchronous crashes or peaks in Red Grouse abundance. These stochastic climate anomalies were suggested to have a major effect on the transmission of *T. tenuis* infective stages during the host breeding period, and consequently caused large scale synchrony in the dynamics of Red Grouse populations (Cattadori et al. 2005b).

SUMMARY AND CONCLUSIONS

In conclusion, Lafferty (2009) is a strong summary of climate change effects on mosquito-transmitted human infectious diseases. The goal of our commentary was to highlight examples from wildlife diseases that span many types of animal hosts and for which human management has not yet affected our ability to detect climate drivers. Importantly, introduced diseases are driving some amphibian and coral hosts to extinction; evidence of climate-linked disease epidemics in amphibians has been recently questioned, but for corals, the evidence linking disease prevalence and severity with warmer temperatures is stronger. We also point out that the migrations of animals are recognized as being especially sensitive to climate warming, and changes in host migratory patterns will likely have important consequences for infectious disease in ways that have not yet been fully explored. Finally, as with malaria, parasites affecting vertebrate animals can be driven by host immunity, and this can dampen the effect of climate change on disease dynamics. Some invertebrate hosts, particularly in marine ecosystems, appear to lack this buffering effect and are currently experiencing strong declines by infectious and opportunistic diseases. Collectively, examples discussed here suggest that ecologists need to consider how host biology, including movement behavior and acquired immunity, can mediate the impacts of global change on parasite/pathogen dynamics and disease severity. At present, many of these mechanisms are poorly known. Immune-mediated changes in host susceptibility and resistance as well as climate-related changes in parasite transmission may alter the interaction between wildlife hosts and their communities of parasites (Cattadori et al. 2007). Therefore, it is important to identify how climate changes modulate, both directly and indirectly, host–parasite interaction and disease persistence.

ACKNOWLEDGMENTS

LITERATURE CITED

Altizer, S. M., K. O. Oberhauser, and L. P. Brower. 2000. Associations between host migration and the prevalence of a protozoan parasite in natural populations of adult monarch butterflies. Ecological Entomology 25:125–139.
Altizer, S. M., K. O. Oberhauser, and K. A. Geurts. 2004. Transmission of the protozoan parasite, *Ophryocystis elektroscirrha*, in monarch butterfly populations. Pages 213–218 in K. Oberhauser and M. Solensky, editors. The monarch butterfly: biology and conservation. Cornell University Press, Ithaca, New York, USA.
Altstatt, J. M., R. F. Ambrose, J. M. Engle, P. L. Haaker, K. D. Lafferty, and P. T. Raimondi. 1996. Recent declines of black abalone *Haliotis cracherodii* on the mainland coast of central California. Marine Ecology Progress Series 142:185–192.
Anderson, R. C. 2000. Nematode parasites of vertebrates: their development and transmission. Oxford University Press, CABI Publishing, Wallingford, UK.
Bosch, J., L. M. Carrascal, L. Duran, S. Walker, and M. C. Fisher. 2007. Climate change and outbreaks of amphibian chytridiomycosis in a montane area of Central Spain; is there a link? Proceedings of the Royal Society Series B 274:253–260.
Bradley, C., and S. Altizer. 2005. Parasites hinder monarch butterfly flight ability: implications for disease spread in migratory hosts. Ecology Letters 8:290–300.
Bradshaw, W. E., and C. M. Holzapfel. 2007. Evolution of animal photoperiodism. Annual Review of Ecology, Evolution and Systematics 38:1–25.
Braid, B. A., R. P. Hedrick, R. S. Tjeerdema, and C. S. Friedman. 2005. Health and survival of red abalone, *Haliotis rufescens*, under varying temperature, food supply and exposure to the agent of withering syndrome. Journal of Invertebrate Pathology 89:219–231.
Brooker, R. W., J. M. T. Travis, E. J. Clark, and C. Dytham. 2007. Modelling species' range shifts in a changing climate: the impacts of biotic interactions, dispersal distance and the rate of climate change. Journal of Theoretical Biology 245:59–65.
Bruckner, A. W., and R. Bruckner. 2006. Consequences of YBS on *Montastraea annularis* (species complex) populations on remote reefs off Mona Island, Puerto Rico. Diseases of Aquatic Organisms 69:67–73.
Cattadori, I. M., R. Albert, and B. Boag. 2007. Variation in host susceptibility and infectiousness generated by co-infection: the myxoma-*Trichostrongylus retortaeformis* case in wild rabbits. Journal of the Royal Society Interface 4:831–840.
Cattadori, I. M., B. Boag, O. N. Bjørnstad, S. Cornell, and P. J. Hudson. 2005a. Immuno-epidemiology and peak shift in a seasonal host-nematode system. Proceeding of the Royal Society B 272:1163–1169.
Cattadori, I. M., B. Boag, and P. J. Hudson. 2008. Parasite co-infection and interaction as drivers of host heterogeneity. Journal of International Parasitology 38:371–380.
Cattadori, I. M., D. T. Haydon, and P. J. Hudson. 2005b. Parasites and climate synchronize red grouse populations. Nature 433:737–741.
Collingham, Y. C., and B. Huntley. 2000. Impacts of habitat fragmentation and patch size upon migration rates. Ecological Applications 10:131–144.
Cook, T., M. Folli, J. Klinck, S. Ford, and J. Miller. 1998. The relationship between increasing sea surface temperature and the northward spread of *Perkinsus marinus* (Dermo) disease epizootics in oysters. Estuarine Coastal Shelf Science 40:587–597.
Cornell, S., O. N. Bjørnstad, I. M. Cattadori, B. Boag, and P. J. Hudson. 2008. Seasonality, cohort-dependence and the development of immunity in a natural host–nematode system. Proceedings of the Royal Society B 275:473–591.
Crofton, H. D. 1948a. The ecology of immature phases of Trichostrongyle nematodes: I. The vertical distribution of

infective larvae of *Trichostrongylus retortaeformis* in relation to their habitat. Parasitology 39:17–25.

Crofton, H. D. 1948*b*. The ecology of immature phases of Trichostrongyle nematodes: II. The effect of climatic factors on the availability of the infective larvae of *Trichostrongylus retortaeformis* to the host. Parasitology 39:26–38.

Croquer, A., and E. Weil. 2009. Local and geographic variability in distribution and prevalence of coral and octocord diseases in the Caribbean II: genera-level analyses. Diseases of Aquatic Organisms, *in press*.

De Roode, J. C., A. Yates, and S. Altizer. 2008. Virulence–transmission tradeoffs and population divergence in virulence in a naturally occurring butterfly parasite. Proceedings of the National Academy of Sciences (USA) 105:7489–7494.

Ellner, S., L. E. Jones, L. Mydlarz, and D. Harvell. 2007. Within host disease ecology in the sea fan *Gorgonia ventalina*: modeling the spatial immunodynamics of a coral–pathogen interaction. American Naturalist 170:1–21.

Folstad, I., F. I. Nilssen, A. C. Halvorsen, and O. Andersen. 1991. Parasite avoidance: the cause of post-calving migrations in *Rangifer*? Canadian Journal of Zoology 69:2423–2429.

Ford, S. E., and M. M. Chintala. 2006. Northward expansion of a marine parasite: testing the role of cold adaptation. Journal of Experimental Marine Biology and Ecology 339:226–235.

Ford, S. E., and R. Smolowitz. 2007. Infection dynamics of an oyster parasite in its newly expanded range. Marine Biology 151:119–133.

Garrett-Jones, C. 1964. Prognosis for the interruption of malaria transmission through assessment of a mosquito's vectorial capacity. Nature 204:1173–1175.

Gylfe, A., S. Bergstrom, and J. Lundstrom. 2000. Reactivation of *Borrelia* infection in birds. Nature 403:724–725.

Harrington, L. C., F. Vermeylen, J. J. Jones, S. Kitthawee, R. Sithiprasasna, J. D. Edman, and T. W. Scott. 2008. Age-dependent survival of the dengue vector, *Ae. aegypti*, demonstrated by simultaneous release and recapture of different age cohorts. Journal of Medical Entomology 45:307–313.

Harvell, C. D., E. Jordan-Dahlgren, S. Merkel, L. Raymundo, E. Rosenberg, G. Smith, E. Weil, and B. Willis. 2007. Coral disease, environmental drivers and the balance between coral and microbial associates. Oceanography 20:58–81.

Harvell, C. D., C. E. Mitchell, J. R. Ward, S. Altizer, A. P. Dobson, R. S. Ostfeld, and M. D. Samuel. 2002. Climate warming and disease risks for terrestrial and marine biota. Science 296:2158–2162.

Hickling, R., D. R. Roy, J. K. Hill, R. Foz, and C. D. Thomas. 2006. The distributions of a wide range of taxonomic groups are expanding polewards. Global Change Biology 12:450–455.

Hoegh-Guldberg, O., et al. 2007. The carbon crisis: coral reefs under rapid climate change and ocean acidification. Science 318:1737.

Hudson, P. J., I. M. Cattadori, B. Boag, and A. P. Dobson. 2006. Climate disruption and parasite–host dynamics: patterns and processes associated with warming and the frequency of extreme climatic events. Journal of Helminthology 80:1–9.

Hudson, P. J., and A. P. Dobson. 1997. Transmission dynamics and host–parasite interactions of *Trichostrongylus tenuis* in Red Grouse (*Lagopus lagopus scoticus*). Journal of Parasitology 83:194–202.

Jensen, T., M. van de Bildt, H. H. Dietz, T. H. Andersen, A. S. Hammer, T. Kuiken, and A. Osterhaus. 2002. Another phocine distemper outbreak in Europe. Science 297:209.

Lafferty, K. D. 2009. The ecology of climate change and infectious diseases. Ecology 90:888–900.

Lips, K. R., F. Brem, R. Brenes, J. D. Reeve, R. A. Alford, J. Voyles, C. Carey, L. Livo, A. P. Pessier, and J. P. Collins. 2006. Emerging infectious disease and the loss of biodiversity in a Neotropical amphibian community. Proceedings of the National Academy of Sciences (USA) 103:3165–3170.

Lips, K. R., J. Diffendorfer, J. R. Mendelson, and M. W. Sears. 2008. Riding the wave: reconciling the roles of disease and climate change in amphibian declines. PLoS Biology 6(3):e72.

Loehle, C. 1995. Social barriers to pathogen transmission in wild animal populations. Ecology 76:326–335.

Lusseau, D., R. Williams, B. Wilson, K. Grellier, T. R. Barton, P. S. Hammond, and P. M. Thompson. 2004. Parallel influence of climate on the behaviour of Pacific killer whales and Atlantic bottlenose dolphins. Ecology Letters 7:1068–1076.

Martens, P. 1999. Climate change impacts on vector-borne disease transmission in Europe. Pages 45–54 *in* A. Haines and A. J. McMichael, editors. Climate change and human health. Royal Society, London, UK.

Miller, J., R. Waara, E. Muller, and C. Rogers. 2006. Coral bleaching and disease combine to cause extensive mortality on corals reefs in US Virgin Islands. Coral Reefs 25:418.

Moore, J. D., T. T. Robbins, and C. S. Friedman. 2000. Withering syndrome in farmed red abalone *Haliotis rufescens*: thermal induction and association with a gastrointestinal Rickettsiales-like prokaryote. Journal of Aquatic Animal Health 12:26–34.

Morgan, E. R., E. J. Milner-Gulland, P. R. Torgerson, and G. F. Medley. 2004. Ruminating on complexity: macroparasites of wildlife and livestock. Trends in Ecology and Evolution 19:181–188.

Muller, E. M., C. S. Rogers, A. S. Spitzack, and R. van Woesik. 2008. Bleaching increases likelihood of disease on *Acropora palmata* (Lamarck) in Hawksnest Bay, St. John, U.S. Virgin Islands. Coral Reefs 27:191–195.

Mydlarz, L., S. Holthouse, E. Peters, and D. Harvell. 2008. Cellular responses in sea fan corals: granular amoebocytes react to pathogen and climate stress. PLos One 3:1811.

Oberhauser, K., and A. T. Peterson. 2003. Modeling current and future potential wintering distributions of eastern North American monarch butterflies. Proceedings of the National Academy of Sciences (USA) 100:14063–14068.

Osterhaus, A. D. M. E., and E. J. Vedder. 1988. Identification of a virus causing recent seal deaths. Nature 335:20.

Parmesan, C., and G. Yohe. 2003. A globally coherent fingerprint of climate change impacts across natural systems. Nature 42:37–42.

Perry, A. L., P. J. Low, J. R. Ellis, and J. D. Reynolds. 2005. Climate change and distribution shifts in marine fishes. Science 308:1912–1915.

Pounds, J. A., et al. 2006. Widespread amphibian extinctions from epidemic disease driven by global warming. Nature 439:161–167.

Raffel, T. R., J. R. Rohr, J. M. Kiesecker, and P. J. Hudson. 2006. Negative effects of changing temperature on amphibian immunity under field conditions. Functional Ecology 20:819–828.

Rogers, D. J., and S. E. Randolph. 2000. Fragile transmission cycles of tick-borne encephalitis virus may be disrupted by predicted climate change. Proceedings of the Royal Society B 267:1741–1744.

Simmons, A. M., and C. E. Rogers. 1991. Dispersal and seasonal occurrence of *Noctuidonema guyanense*, an ectoparasitic nematode of adult fall armyworm (Lepidoptera, Noctuidae), in the United States. Journal of Entomological Science 26:136–148.

Styer, L. M., J. R. Carey, J. L. Wang, and T. W. Scott. 2007. Mosquitoes do senesce: departure from the paradigm of constant mortality. American Journal of Tropical Medicine and Hygiene 76:111–117.

FORUM

Vilchis, L. I., M. J. Tegner, J. D. Moore, C. S. Friedman, K. L. Riser, T. T. Robbins, and P. K. Dayton. 2005. Ocean warming effects on growth, reproduction and survivorship of southern California abalone. Ecological Applications 15:469–480.

Ward, J. R., K. Kim, and C. D. Harvell. 2006. Temperature affects coral disease resistance and pathogen growth. Marine Ecology Progress Series 329:115–121.

Weil, E., and A. Croquer. 2009. Local and geographic variability in distribution and prevalence of coral and octocoral diseases in the Caribbean I: community-level analyses. Diseases of Aquatic Organisms, in press.

Weil, E., G. W. Smith, and D. L. Gil-Agudelo. 2006. Status and progress in coral reef disease research. Diseases of Aquatic Organisms 69:1–7.

Wilkinson, C., and D. Souter. 2008. Status of Caribbean coral reefs after bleaching and hurricanes in 2005. Global Coral Reef Monitoring Network and Reef and Rainforest Research Centre, Townsville, Australia.

Climate variability, global change, immunity, and the dynamics of infectious diseases

Andy Dobson[1]

Ecology and Evolutionary Biology, Guyot Hall, Princeton University, Princeton, New Jersey 08544 USA

Global climate change will have a diversity of impacts on ecological systems. The paper by Lafferty (Lafferty 2009) provides a number of important insights into the ways in which climate change will modify the dynamics of infectious diseases. Here I will focus on three aspects that pick up on his theme that any simple links between climate change and rates of disease transmission will be complicated by the web of ecological interactions within which host–parasite relationships are intimately embedded (Lafferty et al. 2006). Much of the history of mathematical modeling of infectious diseases has focused upon systems where a single pathogen infects a single host species; this approach has provided critical insights into the dynamics of many human pathogens and vital insights into how increased efficiency can be achieved in their control (Anderson and May 1991, Smith et al. 2005). These approaches now need to be extended to consider systems where transmission is dependent upon climate variability and also upon systems with multiple host species and potentially multiple pathogens (Dobson 2004). This will be particularly important if climate warming allows vector-transmitted pathogens to spread from the species rich tropics, into the temperate and Mediterranean countries where reduced host diversity will focus vector activity and potentially increase the proportion of bites that go to humans and domestic livestock. Ironically, the complexities of studying infectious disease dynamics in the tropics will be confounded by a diversity of other interacting variables (insecticide resistance, host species diversity, land conversion and the emergence of new pathogens such as HIV); thus I will conclude with a plea to also study host–pathogen systems in the Arctic where there are less confounding variables and a stronger signal of climate change. The lessons learned in these less diverse systems will be crucial in interpreting the more complex patterns we will certainly see in the tropics.

Global change and climate variability

A variety of different approaches have been used to examine the relationship between climate and disease; they can be broadly grouped into two different approaches: static dynamics and time-series approaches. Static approaches focus on global climate change and use expressions for R_0 (basic reproductive number), or "entomological potential" to examine how potential changes in climate will modify the potential range of specific human pathogens. Most interest here has focused on malaria and other vector transmitted diseases as there is a considerable body of empirical work that has quantified the temperature dependence of pathogen development in the poikilothermic insect and tick species that act as vectors. While there is naturally an emphasis on expansion of the pathogen into new areas, this will always be a relatively weak prediction of these models as it assumes the vectors and pathogen can readily disperse into areas where the climate has become favorable. In contrast, much stronger predictions can be made about the reduction in malaria incidence, and other insect-vectored pathogens, that will occur in areas that are no longer viable for transmission (Dobson and Carper 1992). While I partly agree with Kevin Lafferty that some of the costs of potential infectious disease expansion will be balanced by pathogen extinctions in