ResearchGate

See discussions, stats, and author profiles for this publication at: https://www.researchgate.net/publication/254278078

Climate Change and Infectious Diseases: From Evidence to a Predictive Framework

Article *in* Science · August 2013

DOI: 10.1126/science.1239401 · Source: PubMed

CITATIONS
556

READS
3,970

5 authors, including:

Richard Ostfeld
Cary Institute of Ecosystem Studies
324 PUBLICATIONS **21,153** CITATIONS
SEE PROFILE

Pieter Johnson
University of Colorado Boulder
207 PUBLICATIONS **9,015** CITATIONS
SEE PROFILE

Susan J Kutz
The University of Calgary
225 PUBLICATIONS **4,677** CITATIONS
SEE PROFILE

Catherine Drew Harvell
Cornell University
176 PUBLICATIONS **21,318** CITATIONS
SEE PROFILE

Some of the authors of this publication are also working on these related projects:

Project Muskox View project

Project OCEAN OUTBREAKS View project

All content following this page was uploaded by Richard Ostfeld on 10 June 2014.

The user has requested enhancement of the downloaded file.

EXTENDED PDF FORMAT
SPONSORED BY
Stem Cell
Marker Antibodies
>Learn More
R&D SYSTEMS
www.rndsystems.com

Climate Change and Infectious Diseases: From Evidence to a Predictive Framework

Sonia Altizer *et al.*

Science **341**, 514 (2013);
DOI: 10.1126/science.1239401

This copy is for your personal, non-commercial use only.

If you wish to distribute this article to others, you can order high-quality copies for your colleagues, clients, or customers by clicking here.

Permission to republish or repurpose articles or portions of articles can be obtained by following the guidelines here.

The following resources related to this article are available online at www.sciencemag.org (this information is current as of August 5, 2013):

Updated information and services, including high-resolution figures, can be found in the online version of this article at:
http://www.sciencemag.org/content/341/6145/514.full.html

Supporting Online Material can be found at:
http://www.sciencemag.org/content/suppl/2013/07/31/341.6145.514.DC1.html
http://www.sciencemag.org/content/suppl/2013/08/01/341.6145.514.DC2.html

A list of selected additional articles on the Science Web sites **related to this article** can be found at:
http://www.sciencemag.org/content/341/6145/514.full.html#related

This article **cites 89 articles**, 19 of which can be accessed free:
http://www.sciencemag.org/content/341/6145/514.full.html#ref-list-1

Science (print ISSN 0036-8075; online ISSN 1095-9203) is published weekly, except the last week in December, by the American Association for the Advancement of Science, 1200 New York Avenue NW, Washington, DC 20005. Copyright 2013 by the American Association for the Advancement of Science; all rights reserved. The title *Science* is a registered trademark of AAAS.

REVIEW

Climate Change and Infectious Diseases: From Evidence to a Predictive Framework

Sonia Altizer,[1]* Richard S. Ostfeld,[2] Pieter T. J. Johnson,[3] Susan Kutz,[4] C. Drew Harvell[5]

Scientists have long predicted large-scale responses of infectious diseases to climate change, giving rise to a polarizing debate, especially concerning human pathogens for which socioeconomic drivers and control measures can limit the detection of climate-mediated changes. Climate change has already increased the occurrence of diseases in some natural and agricultural systems, but in many cases, outcomes depend on the form of climate change and details of the host-pathogen system. In this review, we highlight research progress and gaps that have emerged during the past decade and develop a predictive framework that integrates knowledge from ecophysiology and community ecology with modeling approaches. Future work must continue to anticipate and monitor pathogen biodiversity and disease trends in natural ecosystems and identify opportunities to mitigate the impacts of climate-driven disease emergence.

The life cycles and transmission of many infectious agents—including those causing disease in humans, agricultural systems, and free-living animals and plants—are inextricably tied to climate (1, 2). Over the past decade, climate warming has already caused profound and often complex changes in the prevalence or severity of some infectious diseases (Fig. 1) (2–5). For human diseases, vector-control, antimicrobial treatments, and infrastructural changes can dampen or mask climate effects. Wildlife and plant diseases are generally less influenced by these control measures, making the climate signal easier to detect (4). For example, although the effects of climate warming on the dynamics of human malaria are debated, climate warming is consistently shown to increase the intensity and/or latitudinal and altitudinal range of avian malaria in wild birds (6, 7).

Predicting the consequences of climate change for infectious disease severity and distributions remains a persistent challenge surrounded by much controversy, particularly for vector-borne infections of humans [boxes S1 and S2 (8)]. Work using climate-based envelope models has predicted that modest climate-induced range expansions of human malaria in some areas will be offset by range contractions in other locations (9). An alternative approach, based on mechanistic models of physiological and demographic processes of vectors and pathogens (10), predicts large geographic range expansions of human malaria into higher latitudes (11). Both approaches have their limitations (2), and the challenge remains to accurately capture the contributions of multiple, interacting, and often nonlinear underlying responses of host, pathogen, and vector to climate. This challenge is further exacerbated by variation in the climate responses among host-pathogen systems arising from different life history characteristics and thermal niches (12).

A decade ago, Harvell *et al.* (1) reviewed the potential for infectious diseases to increase with climate warming. Since then, the frequency of studies examining climate-disease interactions has continued to increase (Fig. 2), producing clear evidence that changes in mean temperature or climate variability can alter disease risk. Some of the best examples of climate responses of infectious diseases to date are from ectothermic hosts and from parasites with environmental transmission stages that can persist outside the host (Fig. 1). Indeed, first principles suggest that the rates of replication, development, and transmission of these pathogens should depend more strongly on temperature relative to other host-pathogen interactions. The next challenges require integrating theoretical, observational, and experimental approaches to better predict the direction and magnitude of changes in disease risk. Identifying the contribution of other environmental variables, such as precipitation, humidity, and climate variability remains a challenge (13, 14).

Here, we review the individual, community, and landscape-level mechanisms behind climate-induced changes in infectious disease risk and illustrate how a quantitative, ecophysiological framework can predict the response of different host-pathogen relations to climate warming. We mainly focus on changes in temperature, which have been more thoroughly explored both empirically and theoretically, relative to other environmental variables. We consider impacts of climate change on human diseases and on pathogens affecting species of conservation or economic concern, including agroecosystems [box S3 (8)]. A crucial need remains for long-term ecological studies that examine the consequences of climate-disease interactions for entire communities and ecosystems, as well as for efforts that couple effective disease forecasting models with mitigation and solutions.

Ecophysiology of Host-Pathogen Interactions

More than a century of research has firmly established that temperature and other climatic variables strongly affect the physiology and demography of free-living and parasitic species [e.g., (15)], with effects on behavior, development, fecundity, and mortality (16). Because these effects can be nonlinear and sometimes conflicting, such as warmer temperatures accelerating invertebrate development but reducing life span, a central challenge has been to identify the net outcomes for fitness (1). For infectious diseases, this challenge is compounded by the interactions between at least two species—a host and a pathogen—and often vectors or intermediate hosts, which make the cumulative influence of climate on disease outcomes elusive [e.g., (17, 18)].

Immune defenses are physiological processes crucial for predicting changes in disease dynamics. Warmer temperatures can increase immune enzyme activity and bacterial resistance for insects, such as the cricket *Gryllus texensis* (19). Positive effects of temperature on parasite growth and replication, however, might outweigh greater immune function of the host. In gorgonian corals, for example, warmer temperatures increase cellular and humoral defenses (20), but because coral pathogens also replicate faster under these conditions, disease outbreaks have coincided with warmer sea temperatures in the Caribbean (Fig. 1) (4, 5). Warm temperatures also can lower host immunity; for example, melanization and phagocytic cell activity in mosquitoes are depressed at higher temperatures (21). In addition, increased climate variability can interfere with host immunity, as illustrated by decreased frog resistance to the chytrid fungus *Batrachochytrium dendrobatidis* (*Bd*) in response to temperature fluctuations (14). Even though *Bd* grows best in culture at cooler temperatures, which suggests that warming should reduce disease, incorporating variability-induced changes in host resistance suggests a more complex relation between climate change and *Bd*-induced amphibian declines (22). These issues are important for predicting the immunological efficiency of ectotherms outside of their typical climate envelope.

One promising approach for predicting how host-pathogen interactions respond to climate

---

[1] Odum School of Ecology, University of Georgia, Athens, GA 30602, USA. [2] Cary Institute of Ecosystem Studies, 2801 Sharon Turnpike, or Post Office Box AB, Millbrook, NY 12545–0129, USA. [3] Ecology and Evolutionary Biology, N122, CB334, University of Colorado, Boulder, CO 80309–0334, USA. [4] Department of Ecosystem and Public Health, Faculty of Veterinary Medicine, University of Calgary, and Canadian Cooperative Wildlife Health Centre, Alberta Node, 3280 Hospital Drive, NW, Calgary, Alberta T2N 4Z6, Canada. [5] Ecology and Evolutionary Biology, E321 Corson Hall, Cornell University, Ithaca, NY 14853, USA.

*Corresponding author. E-mail: saltizer@uga.edu

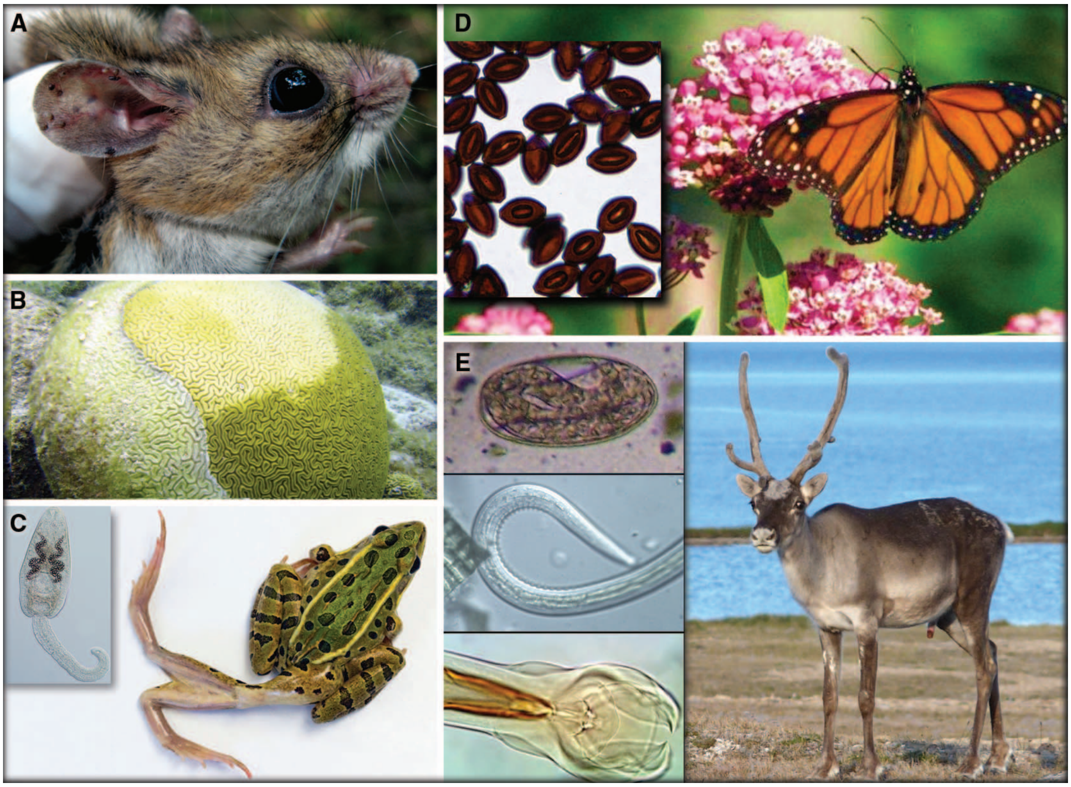

Fig. 1. Animal-parasite interactions for which field or experimental studies have linked climate change to altered disease risk. (**A**) Black-legged ticks, *Ixodes scapularis*, vectors of Lyme disease, attached to the ears of a white-footed mouse, *Peromyscus leucopus*, show greater synchrony in larval and nymphal feeding in response to milder climates, leading to more rapid Lyme transmission. (**B**) Caribbean coral (*Diploria labyrinthiformis*) was affected by loss of symbionts, white plague disease, and ciliate infection during the 2010 warm thermal anomaly in Curaçao. (**C**) Malformed leopard frog (*Lithobates pipiens*) as a result of infection by the cercarial stage (inset) of the multihost trematode *R. ondatrae*; warming causes nonlinear changes in both host and parasite that lead to marked shifts in the timing of interactions. (**D**) Infection of monarchs (*D. plexippus*) by the protozoan *O. elektroscirrha* (inset) increases in parts of the United States where monarchs breed year-round as a result of the establishment of exotic milkweed species and milder winter climates. (**E**) Infection risk with *O. gruehneri* (inset shows eggs and larvae) the common abomasal nematode of caribou and reindeer (*R. tarandus*), may be reduced during the hottest part of the Arctic summer as a result of warming, which leads to two annual transmission peaks rather than one (e.g., Fig. 3C). Photo credits (A to E): J. Brunner, E. Weil, D. Herasimtschuk, S. Altizer, P. Davis, S. Kutz.

warming involves infusing epidemiological models with relations derived from the metabolic theory of ecology (MTE). This approach circumvents the need for detailed species-specific development and survival parameters by using established relations between metabolism, ambient temperature, and body size to predict responses to climate warming (*23*). One breakthrough study (*12*) used MTE coupled with traditional host-parasite transmission models to examine how changes in seasonal and annual temperature affected the basic reproduction number (R_0) of strongylid nematodes with direct life cycles and transmission stages that are shed into the environment. By casting R_0 in terms of temperature-induced tradeoffs between parasite development and mortality, this approach facilitated both general predictions about how infection patterns change with warming and, when parameterized for *Ostertagia gruehneri,* a nematode of caribou and reindeer (Fig. 1), specific projections that corresponded with the observed temperature dependence of parasite stages. Moreover, this model predicted a shift from one to two peaks in nematode transmission each year under warming conditions (Fig. 3C), a result consistent with field observations (*12*, *24*).

In some cases, ecophysiological approaches must consider multiple host species and parasite developmental stages that could show differential sensitivity to warming. Such differential responses can complicate prediction of net effects, especially for ectothermic hosts with more pronounced responses to temperature. For instance, because both infectivity of a trematode parasite (*Ribeiroia ondatrae*) and defenses of an amphibian host (*Pseudacris regilla*) increase with temperature, maximal pathology (limb malformations) (Fig. 1) occurs at intermediate temperatures (*25*). Other work showed that the virulence of both a coral fungus (*Aspergillus sydowii*) and protozoan (*Aplanochytrium* sp.) increased with temperature, probably because pathogen development rate continued to increase in a temperature range where coral defenses were less potent (*26*). Thus, the ideal approach will be an iterative one that combines metabolic and epidemiological modeling to predict general responses and to identify knowledge gaps, followed by application of models to specific host-pathogen interactions.

Community Ecology, Biodiversity, and Climate Change

Host-pathogen interactions are embedded in diverse communities, with climate change likely leading to the loss of some host-pathogen interactions and the gain of novel species pairings. In some cases, pathogen extinction and the loss of endemic parasites could follow from climate change, potentially reducing disease or conversely releasing more pathogenic organisms from competition. In other cases, multiple pathogens can put entire host communities at risk of extinction. Although ecosystems of low biodiversity, such as occur in polar regions, can be particularly sensitive to emerging parasitic diseases (*27*), most knowledge of community-wide responses stems from tropical marine systems. For example, the wider Caribbean region is a "disease hot spot" characterized by the rapid, warming-induced emergence of multiple new pathogens that have caused precipitous coral declines with ecosystem-wide repercussions (*28*, *29*). Impacts of climate-induced changes in disease can be especially large when the host is a dominant or keystone species. For example, near extinction of the once-dominant, herbivorous abalone (genus *Haliotis*) by warming-driven rickettsial disease caused pervasive community shifts across multiple trophic levels (*5*). Similarly, seagrass (*Zostera marina*) declines caused by infection with the protist *Labyrinthula zosterae,* which correlates positively with warming, have degraded nursery habitats for fish and migratory waterfowl and caused the extinction of the eelgrass limpet (*30*).

Microbial communities, which are often part of the extended phenotype of host defenses, are also likely to respond to climate changes. For instance, warming sea-surface temperatures in coral reefs can inhibit the growth of antibiotic-producing bacteria, sometimes causing microbial communities to shift from mutualistic to pathogenic (*31*). In agroecosystems, higher temperatures can suppress entomopathogenic fungi and antibiotic production by bacterial mutualists in plants (*32*). Warming also underlies bacterial shifts from endosymbiotic to lytic within host amoebas that live in human nasal passages, increasing the potential risk of respiratory disease (*33*). Thus, effects of warmer temperatures on the diversity and function of commensal or mutualist microbes could promote pathogen growth and pest outbreaks.

From a broader perspective, biodiversity loss is a well-established consequence of climate change (*16*, *34*) and can have its own impact on infectious diseases. For many diseases of humans, wildlife, and plants, biodiversity loss at local or regional scales can increase rates of pathogen transmission (*35*–*37*). This pattern can result from several mechanisms, including the loss of the dilution effect (*36*). For example, lower parasite diversity could allow more pathogenic species to proliferate when endemic and competing parasites are lost from a system (*36*). Climate warming can also weaken biotic regulation of disease vectors by inhibiting their predators (*38*) and competitors (*39*). Interactions between biodiversity and infectious disease underscore the need to put climate-disease interactions into the broader context of other forms of global change, such as land-use change and habitat loss, when extending predictions from focused host-pathogen interactions to larger spatial and taxonomic scales.

Shifts in Behavior, Movement, and Phenology of Hosts and Parasites

Changes in climate are already affecting the phenology of interactions between plants and pollinators, predators and prey, and plants and herbivores (*16*). Climate-induced shifts in phenology and species movements (*40*) will likely affect disease dynamics. Many species are already moving toward higher elevations or latitudes (*41*), and an open question is whether these shifts could disrupt established interactions or bring novel groups of hosts and pathogens into contact (*42*). For instance, the range expansion of the Asian tiger mosquito (*Aedes albopictus*) across Europe and the Americas has created the potential for novel viral diseases such as Chikungunya to invade (*10*); this pathogen is already expanding in geographic range, and a recent outbreak in Europe emphasizes the need for surveillance and preparedness. Along eastern North America, warming sea temperatures and changes in host resistance facilitated a northward shift of two oyster diseases into previously unexposed populations (*5*).

Migratory species in particular can be sensitive to climate change (*41*), with the routes and timing of some species' migrations already shifting with climate warming (*16*). Long-distance migrations can lower parasite transmission by allowing hosts to escape pathogens that accumulate in the environment or by strenuous journeys that cull sick animals (*43*). In some cases, milder winters can allow previously migratory host populations to persist year-round in temperate regions (*44*); this residency fosters the build-up of environmental transmission stages, and mild winters further enhance parasite over-winter survival (*2*). A case study of monarch butterflies (*Danaus plexippus*) and the protozoan parasite *Ophyrocystis elektroscirrha* (Fig. 1) provides support for climate-warming shifts in migration and disease. Monarchs typically leave their northern breeding grounds in early fall and fly to Mexican wintering sites. Milder winters, combined with increased planting of exotic host plants, now allow monarch populations to breed year-round in parts of the United States (*45*). Relative to migratory monarchs, winter-breeding monarchs suffer from higher rates of infection (*43*). Similarly, migration is considered an important parasite avoidance strategy for barren-ground caribou (*24*), but the loss of sea ice with climate warming will likely inhibit migrations and prevent them from seasonally escaping parasites (*46*). Thus, diminishing migration behaviors among animals that use seasonal habitats can increase the transmission of infectious diseases.

Changes in the timing of vector life stages and feeding behavior can also arise from interactions between climate and photoperiod. For several tick-borne infections (Fig. 1), pathogens are sequentially transmitted from infected vertebrate hosts to naïve larval tick vectors, and from infected nymphal ticks to naïve vertebrate hosts. Asynchrony in the seasonal activity of larval and nymphal ticks can delay transmission and select for less virulent strains of the Lyme bacterium *Borrelia burgdorferi* (*47*), whereas synchrony allows for more rapid transmission and the persistence of virulent strains. In the case of tick-borne encephalitis (TBE), viral transmission occurs directly between cofeeding ticks; thus, viral maintenance requires synchronous larval and nymphal feeding (*48*). Because synchrony of larval and nymphal ticks characterizes milder winter climates, climate change could increase tick synchrony and the transmission and virulence of several tick-borne infections.

Changes in the timing of shedding or development of environmental transmission stages could result from climate warming. Some parasites could experience earlier hatching, exposure to hosts earlier in the season, and encounters with earlier (and often more sensitive) life stages of hosts. For example, a long-term data set of lake plankton showed that warming shifted fungal prevalence patterns in diatom hosts from acute epidemics to chronic persistence, in part because of faster transmission and more widespread host population suppression under warmer temperatures (*49*). In contrast, Brown and Rohani (*50*) argued for the opposite outcome with respect to avian influenza (AI) in reservoir bird hosts. Climate-driven mismatch in the timing of bird migration and their prey resources (e.g., horseshoe crab eggs) amplified variability in epidemiological outcomes: Although mismatch increased the likelihood of AI extinction, infection prevalence and spillover potential both increased in cases where the virus persisted.

Plasticity in parasite traits could allow parasites with environmental transmission stages to

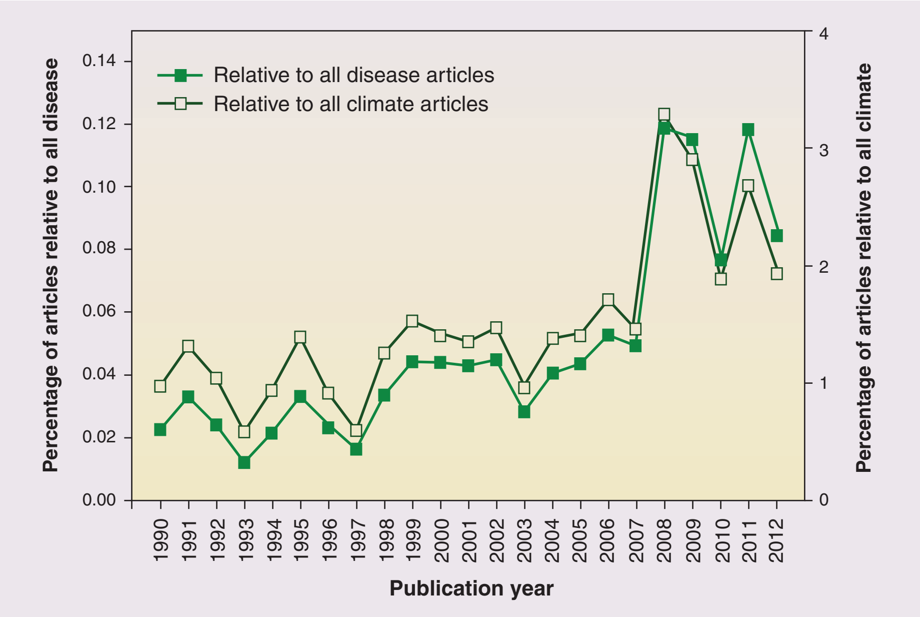

Fig. 2. Rising interest in climate-disease interactions. Research focused on associations between infectious disease and climate change has increased steadily over the past 20 years. After correcting for total research interest in climate change (open symbols) or infectious disease (closed symbols), the frequency of papers referencing a climate-disease link in the title has nearly doubled over this period, based on long-term publication trends following a Web of Science search of article titles (1990 to 2012). Search criteria and statistical analyses are provided in the supplementary materials, and the total number of climate change–infectious disease papers identified by our search criteria ranged from 5 to 117 publications per year.

respond more rapidly to climate warming. For example, arrested development (hypobiosis) of the nematode *O. gruehneri* within its caribou host is a plastic trait more commonly expressed in areas with harsher winters as compared with milder climates (*51*). This arrested state prevents wasted reproductive effort for the parasites, because eggs produced in late summer in colder regions are unlikely to develop to infective-stage larvae by fall. Ultimately, plasticity in life history traits could speed parasite responses to changing environments and allow parasites to deal with climate instabilities (e.g., a series of severe winters interspersed by mild), relative to the case where selection must act on genetically variable traits (*52*). For example, if climate warming extends the transmission season for *O. gruehneri* on tundra, a rapid decrease in the frequency of nematode hypobiosis could shorten the life cycle and increase infection rates.

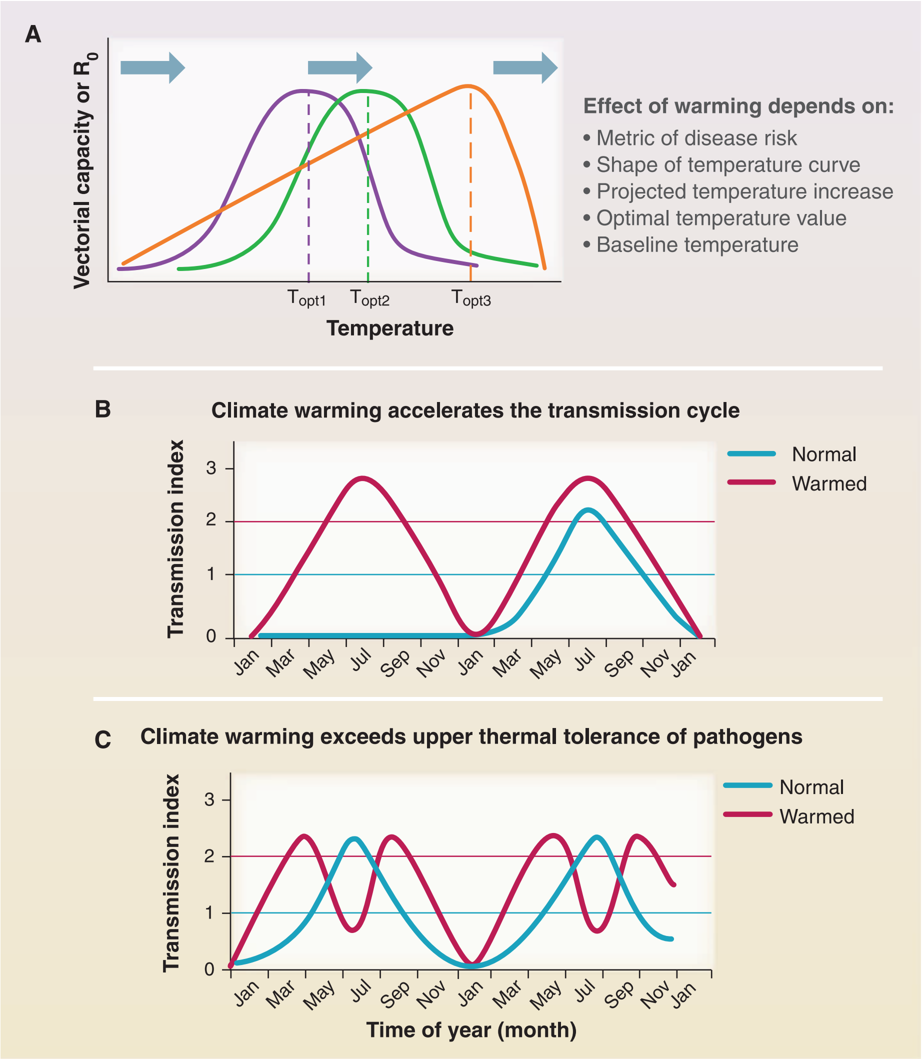

Fig. 3. Theoretical underpinnings and categorization of disease responses to climate change. Pathogen responses to climate change depend on thermal tolerance relative to current and projected conditions across an annual cycle. (**A**) Gaussian curves relating temperature to a metric of disease risk suggest symmetrical temperature zones over which warming will increase and decrease transmission, whereas left-skewing [a common response for many terrestrial ectotherms, including arthropod vectors (*74*)] indicates greater potential for pathogen transmission to increase with warming [box S2 (*8*)]. Bold arrows represent geographic gradients that span cool, warm, and hot mean temperatures, which indicate that the net effect of warming (at point of arrows) depends on whether temperatures grow to exceed the optimum temperature (T_{opt}) for disease transmission. Projected changes in disease will further depend on the starting temperature relative to T_{opt}, the magnitude of warming, measurement error, adaptation, and acclimation. (**B**) Pathogens at their northern or altitudinal limits might show range expansion and nonlinear shifts in their life cycle in response to warmer temperatures (red) relative to baseline (blue). For example, a shift from 2- to 1-year cycles of transmission has occurred for the muskox lungworm (*27*). This outcome could generate sporadic disease emergence in a naïve population (if extremes in temperature allow only occasional invasion and/or establishment), or could gradually increase prevalence and establishment. (**C**) At the low-latitude or low-altitude extent of a pathogen's range, where temperature increases could exceed the pathogen's thermal optimum, transmission might be reduced, or we might see the emergence of a bimodal pattern whereby R_0 peaks both early and late in the season, but decreases during the midsummer [as in the case of the arctic *O. gruehneri*–reindeer example (*12*)]. In (B) and (C), the lower blue line represents $R_0 = 1$, above which the pathogen can increase; values above the pink line represent severe disease problems owing to a higher peak of R_0 and a greater duration of time during which $R_0 > 1$.

Consequences for Conservation and Human Health

Climate change is already contributing to species extinctions, both directly and through interactions with infectious disease (*53*). Roughly one-third of all coral species and the sustainability of coral reef ecosystems are threatened by human activities, including climate warming and infectious diseases (*5*). In contrast to tropical marine systems, the Arctic is a less diverse and minimally redundant system that is warming at least twice as fast as the global average (*54*) and simultaneously experiencing drastic landscape changes from an expanding human footprint. Altered transmission dynamics of parasites, poleward range expansion of hosts and parasites, and disease emergence coincident with climate warming or extremes have all been reported in the Arctic (*27*, *55*). Together, these phenomena are altering host-parasite dynamics and causing endemic Arctic species—unable to compete or adapt rapidly enough—to decline (*56*). Changes in wildlife health can also compromise the livelihoods and health of indigenous people who depend on wildlife for food and cultural activities (*57*).

In humans, exposure to diarrheal diseases has been linked to warmer temperatures and heavy rainfall (*58*). Human infections of cholera, typically acquired through ingestion of contaminated water (in developing countries) or undercooked seafood (in the developed world), affect millions of people annually with a high case-fatality rate. Coastal *Vibrio* infections are associated with zooplankton blooms, warmer water, and severe storms (*3*). For example, in the Baltic Sea, long-term warming and temperature anomalies have been linked to increased disease from *Vibrio vulnificus*, which was first reported in 1994 along the German coast after an unusually warm summer (*3*). Long-term sea surface warming can increase the geographic range, concentration, and seasonal duration of *Vibrio* infections, as seen in coastal Chile, Israel, and the U.S. Pacific Northwest. Modeling approaches indicate that *Vibrio* illnesses from the Baltic region could increase nearly twofold for every 1°C increase in annual maximum water temperature (*3*).

Human mosquito-borne diseases, such as malaria and dengue fever, are frequently proposed as cases where vector and disease expansion into the temperate zone could follow from climate warming (*59*). However, some researchers have argued that ranges will shift with warming, rather than expand, and that the best predictors of infection risk are economic and social factors, especially poverty (*17*, *60*). Controversy has also arisen over which climatic variables are most important in delimiting the distributions of these diseases [boxes S1 and S2 (*8*)]. Detecting impacts of climate change on human vector-borne diseases remains difficult, in part, because active mitigations, such as vector-control, antimicrobials, and improved infrastructure can complicate detection of a climate signal. Several unresolved issues include identifying conditions under which climate warming will cause range expansions versus contractions, understanding the impact of increasing variability in precipitation, and determining the additional economic costs associated with increased disease risk caused by warming.

Ultimately, the societal implications of climate-driven shifts in diseases of humans, crops, and natural systems will demand solutions and mitigation, including early-warning programs. Recently, a forecasting system linking global coupled ocean-atmosphere climate models to malaria risk in Botswana allowed anomalously high risk to be predicted and anticipatory mitigations to be initiated (*61*). Forecasting is well-established in crop disease management and leads to improved timing of pesticide application and deployment of planting strategies to lower disease risk [box S3 (*8*)]. Modeling efforts to better predict crop loss events are also tied to improved insurance returns against losses (*2*). Similarly, accurate forecasting programs for coral bleaching have become a mainstay of marine climate resilience programs (*62*) and are leading to the development of coral disease–forecasting algorithms (*63*). Appropriate management actions under outbreak conditions include reef closures to reduce stress and transmission, culling of diseased parts of some colonies, and increased surveillance (*64*). In the ocean, efforts are also under way to increase the resilience of marine ecosystems to disease, including developing no-fishing zones and reducing land-based pollution that can introduce new pathogens (*5*).

Outlook and Future Challenges

Climate change will continue to limit the transmission of some pathogens and create opportunities for others. To improve predictions and responses we need to deepen our understanding of mechanistic factors. Although the initial climatic drivers to be explored should be temperature variables (both mean and variability), because the data are available and we understand the mechanisms at work, future work must concurrently explore the effects of precipitation, relative humidity, and extreme events. In particular, models are needed that combine the principles of ecophysiology and MTE (*23*) with epidemiological response variables, such as R_0 or outbreak size, and that are designed to accommodate distinct pathogen types (e.g., vector-borne, directly transmitted, or complex life cycle) and host types (ectotherm versus endotherm) (*12*). These models should be applied, by using climate-change projections, to evaluate how broad classes of pathogens might respond to climate change. Building from this foundation, the next step is to extend such general models to specific pathogens of concern for human health, food supply, or wildlife conservation, which will require empirical parameterization, with attention to the on-the-ground conditions. Modeling efforts should be integrated with experiments to test model predictions under realistic conditions, and with retrospective studies to detect the "fingerprint" of climate-induced changes in infection.

Scientists still know relatively little about the conditions under which evolution will shape host and pathogen responses to climate change. Although evolutionary change in response to climate warming has been reported for some free-living animals and plants, the evidence remains limited (*52*). Even less is known about how climate change will drive host-pathogen evolution. Corals have multiple levels of adaptation to intense selection by thermal stress that could also affect resistance to pathogens, including symbiont shuffling of both algae and bacteria, and natural selection against thermally intolerant individuals (*65*). In oysters (*Crassostrea virginica*), warming might have contributed to increased resistance to the protozoan multinucleated sphere X (MSX) disease (*66*), but climate variability might also slow the evolution of oyster resistance (*67*). In cases where increased rates of transmission follow from warming, selection could favor higher pathogen virulence, although examples are now unknown.

A persistent challenge involves the ability to detect changes in disease risk with climate across different systems. In the oceans, for example, impacts of disease on sessile hosts like corals, abalones, and oysters are readily apparent, and for terrestrial systems, clear impacts are seen for plant diseases and some wildlife-helminth interactions. But for highly mobile species and many human diseases, detecting signals of climate change remains problematic. For these less tractable systems, long-term ecological studies that examine the past distributions of pathogens, important hosts, and severity of diseases are indispensable. Permanent repositories of intact physical specimens, as well as their DNA, can provide records of diversity that will be critical resources as new methodologies become available (*68*, *69*). Moreover, new technologies can detect variability in physiological processes and gene expression and can improve climate projections from global circulation models. Sophisticated experimental designs conducted under appropriate ranges of environmental conditions and retrospective studies to identify past climatic effects on disease (*5*, *70*) will help advance predictive power.

An additional key challenge is predicting the impacts of climate-disease interactions for human societies and gauging how these compare with other components of climate change, such as the loss of arable land. By affecting food yields and nutrition, water quality and quantity, social disorder, population displacement, and conflict, past climate changes have long influenced the burden of infectious disease in many human societies (*71*, *72*). Predicting the regions where humans and natural systems are most vulnerable to pressures from infectious disease and how these pressures will translate to changes in global health and security constitute critical research priorities (*73*). Building a mechanistic understanding of climate-disease interactions will allow public health interventions to be proactive and will facilitate effective responses to new or expanding health threats. Surveillance programs capable of detecting pathogen or disease emergence are essential and, in many instances, predicting and detecting local-scale impacts might be more important than predicting global-scale changes. To this end, the value of engaging local communities in disease surveillance is increasingly recognized, with the goal of advancing science on climate-disease linkages for practical solutions to protecting human and wildlife health.

References and Notes

1. C. D. Harvell *et al.*, *Science* **296**, 2158–2162 (2002).
2. K. Garrett *et al.*, *Agric. For. Meteorol.* **170**, 216–227 (2013).
3. C. Baker-Austin *et al.*, *Nat. Clim. Change* **3**, 73 (2013).
4. D. Harvell, S. Altizer, I. M. Cattadori, L. Harrington, E. Weil, *Ecology* **90**, 912–920 (2009).
5. C. Burge *et al.*, *Annu. Rev. Mar. Sci.* **6**, (2014).
6. L. Z. Garamszegi, *Glob. Change Biol.* **17**, 1751–1759 (2011).
7. I. Zamora-Vilchis *et al.*, *PLoS ONE* **7**, e39208 (2012).
8. Boxes S1, S2, and S3 are available as supplementary materials on *Science* Online.
9. D. J. Rogers, S. E. Randolph, *Science* **289**, 1763–1766 (2000).
10. D. Ruiz-Moreno *et al.*, *PLoS Negl. Trop. Dis.* **6**, e1918 (2012).
11. W. J. Martens *et al.*, *Environ. Health Perspect.* **103**, 458–464 (1995).
12. P. K. Molnár, S. J. Kutz, B. M. Hoar, A. P. Dobson, *Ecol. Lett.* **16**, 9–21 (2013).
13. K. P. Paaijmans, A. F. Read, M. B. Thomas, *Proc. Natl. Acad. Sci. U.S.A.* **106**, 13844–13849 (2009).
14. T. R. Raffel *et al.*, *Nat. Clim. Change* **3**, 146–151 (2013).
15. G. R. Walther *et al.*, *Nature* **416**, 389–395 (2002).
16. C. Parmesan, G. Yohe, *Nature* **421**, 37–42 (2003).
17. K. D. Lafferty, *Ecology* **90**, 932–933 (2009).
18. J. R. Rohr *et al.*, *Trends Ecol. Evol.* **26**, 270–277 (2011).
19. S. A. Adamo, M. M. Lovett, *J. Exp. Biol.* **214**, 1997–2004 (2011).
20. L. D. Mydlarz, L. E. Jones, C. D. Harvell, *Annu. Rev. Ecol. Evol. Syst.* **37**, 251–288 (2006).
21. C. C. Murdock *et al.*, *Proc. Biol. Sci.* **279**, 3357–3366 (2012).
22. J. R. Rohr, T. R. Raffel, *Proc. Natl. Acad. Sci. U.S.A.* **107**, 8269–8274 (2010).
23. J. H. Brown *et al.*, *Ecology* **85**, 1771–1789 (2004).
24. B. M. Hoar *et al.*, *Parasitology* **139**, 1093–1100 (2012).
25. S. H. Paull, B. E. LaFonte, P. T. J. Johnson, *Glob. Change Biol.* **18**, 3558–3567 (2012).
26. C. A. Burge *et al.*, *Microb. Ecol.* **65**, 869–879 (2013).
27. S. J. Kutz *et al.*, *Vet. Parasitol.* **163**, 217–228 (2009).
28. C. Rogers, E. Muller, *Coral Reefs* **31**, 807–819 (2012).
29. D. Ruiz-Moreno *et al.*, *Dis. Aquat. Organ.* **100**, 249–261 (2012).
30. J. E. Hughes *et al.*, *Estuaries* **25**, 235–249 (2002).

31. K. B. Ritchie, *Mar. Ecol. Prog. Ser.* **322**, 1–14 (2006).
32. B. Humair *et al.*, *ISME J.* **3**, 955–965 (2009).
33. D. Corsaro, G. Greub, *Clin. Microbiol. Rev.* **19**, 283–297 (2006).
34. W. Jetz *et al.*, *PLoS Biol.* **5**, e157 (2007).
35. B. J. Cardinale *et al.*, *Nature* **486**, 59–67 (2012).
36. P. T. J. Johnson, J. T. Hoverman, *Proc. Natl. Acad. Sci. U.S.A.* **109**, 9006–9011 (2012).
37. F. Keesing *et al.*, *Nature* **468**, 647–652 (2010).
38. P. H. Hobbelen, M. D. Samuel, D. Foote, L. Tango, D. A. LaPointe, *Theor. Ecol.* **6**, 31–44 (2013).
39. T. Farjana, N. Tuno, Y. Higa, *Med. Vet. Entomol.* **26**, 210–217 (2012).
40. I.-C. Chen, J. K. Hill, R. Ohlemüller, D. B. Roy, C. D. Thomas, *Science* **333**, 1024–1026 (2011).
41. R. Hickling *et al.*, *Glob. Change Biol.* **12**, 450–455 (2006).
42. E. R. Morgan, E. J. Milner-Gulland, P. R. Torgerson, G. F. Medley, *Trends Ecol. Evol.* **19**, 181–188 (2004).
43. S. Altizer *et al.*, *Science* **331**, 296–302 (2011).
44. W. E. Bradshaw, C. M. Holzapfel, *Annu. Rev. Ecol. Evol. Syst.* **38**, 1–25 (2007).
45. E. Howard *et al.*, *Psyche (Camb. Mass.)* **2010**, 1–6 (2010).
46. E. Post *et al.*, *Science* **341**, 519–524 (2013)
47. K. Kurtenbach *et al.*, *Nat. Rev. Microbiol.* **4**, 660–669 (2006).
48. S. E. Randolph *et al.*, *Parasitology* **118**, 177–186 (1999).
49. B. W. Ibelings *et al.*, *Freshw. Biol.* **56**, 754–766 (2011).
50. V. L. Brown, P. Rohani, *Biol. Lett.* **8**, 1036–1039 (2012).
51. B. M. Hoar, A. G. Eberhardt, S. J. Kutz, *Parasitology* **139**, 1339–1345 (2012).
52. C. Moritz, R. Agudo, *Science* **341**, 504–508 (2013).
53. C. D. Thomas *et al.*, *Nature* **427**, 145–148 (2004).
54. Intergovernmental Panel on Climate Change, *Summary for Policymakers. Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change* (Cambridge Univ. Press, New York, 2007).
55. S. Laaksonen *et al.*, *EcoHealth* **7**, 7–13 (2010).
56. O. Gilg *et al.*, *Ann. N. Y. Acad. Sci.* **1249**, 166–190 (2012).
57. S. Meakin, T. Kurvits, *Assessing the Impacts of Climate Change on Food Security in the Canadian Arctic* (GRID-Arendal, Arendal, Norway, 2009).
58. M. Pascual *et al.*, *Microbes Infect.* **4**, 237–245 (2002).
59. J. N. Mills *et al.*, *Environ. Health Perspect.* **118**, 1507–1514 (2010).
60. S. E. Randolph, *Vet. Parasitol.* **167**, 92–94 (2010).
61. M. C. Thomson *et al.*, *Nature* **439**, 576–579 (2006).
62. C. M. Eakin *et al.*, *PLoS ONE* **5**, e13969 (2010).
63. J. Maynard *et al.*, *Coral Reefs* **30**, 485–495 (2011).
64. R. Beeden *et al.*, *Environ. Manage.* **49**, 1–13 (2012).
65. E. Howells *et al.*, *Nat. Clim. Change* **2**, 116–120 (2011).
66. S. E. Ford, D. Bushek, *J. Mar. Res.* **70**, 205–223 (2012).
67. E. N. Powell *et al.*, *J. Mar. Res.* **70**, 309 (2012).
68. E. P. Hoberg, *Rev. Sci. Tech.* **29**, 255–272 (2010).
69. J. Fernandez-Triana *et al.*, *PLoS ONE* **6**, e23719 (2011).
70. J. T. Hoverman, S. H. Paull, P. T. J. Johnson, in *Climate Vulnerability: Understanding and Addressing Threats to Essential Resources*, P. Roger, Ed. (Academic Press, Oxford, 2013), vol. 4, pp. 61–70.
71. A. J. McMichael, *Proc. Natl. Acad. Sci. U.S.A.* **109**, 4730–4737 (2012).
72. T. Wheeler, J. von Braun, *Science* **341**, 508–513 (2013).
73. S. S. Myers, J. A. Patz, *Annu. Rev. Environ. Resour.* **34**, 223–252 (2009).
74. C. A. Deutsch *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **105**, 6668–6672 (2008).

Acknowledgments: This work was supported in part by an NSF grant (DEB-0643831) to S.A., a Fellowship from the David and Lucile Packard Foundation and NSF grant (IOS-1121529) to P.T.J.J., an NSF Research Coordination Network grant on the Ecology of Infectious Marine Diseases, NSF Ecology and Evolution of Infectious Diseases grant (OCE-1215977) to C.D.H., and by the Atkinson Center for a Sustainable Future at Cornell University. S.K. thanks the Natural Sciences and Engineering Council of Canada, the Nasivvik Centre for Inuit Health; the governments of the Northwest Territories, Nunavut, and Yukon; and the government of Canada International Polar Year Program.

Supplementary Materials
www.sciencemag.org/cgi/content/full/341/6145/514/DC1
Materials and Methods
Supplementary Text
Fig. S1
Boxes S2 to S3
References (*75*–*95*)

10.1126/science.1239401

REVIEW

Ecological Consequences of Sea-Ice Decline

Eric Post,[1]* Uma S. Bhatt,[2] Cecilia M. Bitz,[3] Jedediah F. Brodie,[4] Tara L. Fulton,[5] Mark Hebblewhite,[6] Jeffrey Kerby,[1] Susan J. Kutz,[7] Ian Stirling,[8] Donald A. Walker[9]

After a decade with nine of the lowest arctic sea-ice minima on record, including the historically low minimum in 2012, we synthesize recent developments in the study of ecological responses to sea-ice decline. Sea-ice loss emerges as an important driver of marine and terrestrial ecological dynamics, influencing productivity, species interactions, population mixing, gene flow, and pathogen and disease transmission. Major challenges in the near future include assigning clearer attribution to sea ice as a primary driver of such dynamics, especially in terrestrial systems, and addressing pressures arising from human use of arctic coastal and near-shore areas as sea ice diminishes.

As one of Earth's major biomes, sea ice not only comprises unique ecosystems in, on, and under the ice itself but also strongly influences patterns and processes in adjacent terrestrial ecosystems (*1*, *2*) (Fig. 1). Sea ice harbors an array of microorganisms, provides critical habitat for vertebrates, and influences terrestrial productivity and diversity in the Arctic, where 80% of low-lying tundra lies within 100 km of seasonally ice-covered ocean (*3*–*5*). Ice-loss-driven amplification of arctic warming is a potentially important driver of ecological dynamics in the region, where seasonal temperature limitation is an important constraint on productivity (*6*). Here, we synthesize recent developments in the study of ecological responses to arctic sea-ice decline and highlight the importance of sea-ice loss as a driver of ecological dynamics in both marine and terrestrial systems.

Record of Recent Sea-Ice Loss

One of the most conspicuous consequences of recent anthropogenic warming has been declining annual minimum extent of arctic sea ice (*7*). Over the past several decades, the Arctic has warmed at twice the global rate, with sea-ice loss accelerating (*8*) (Fig. 2A), especially along the coasts of Russia, Alaska, and the Canadian Archipelago (Fig. 2B). The sea ice's annual minimum reached a record low in 2012. Arctic sea-ice loss has exceeded most model projections (*9*) and is unprecedented in the past 1.5 millennia (*10*).

Sea-ice loss is most commonly discussed as an indicator of arctic warming (*11*), but it is also a major factor in amplification of warming in the Arctic through feedback deriving from declining surface albedo (*6*). In 2007, the year of second-lowest arctic sea-ice extent on record, sea ice loss accounted for a large portion of warming over land north of 60° (*12*). Further, much of arctic near-surface warming over the past three decades is attributable to declining sea ice concentration (*13*), and land-surface warming is linked to summer sea-ice loss in global climate models (*14*).

---

[1] The Polar Center, and Department of Biology, Pennsylvania State University, 208 Mueller Laboratory, University Park, PA 16802, USA. [2] Department of Atmospheric Sciences, Geophysical Institute, University of Alaska Fairbanks, Fairbanks, AK 99775, USA. [3] Department of Atmospheric Sciences, University of Washington, 408 ATG Building, Box 351640, Seattle, WA 98195, USA. [4] Departments of Botany and Zoology, Biodiversity Research Centre, University of British Columbia, Vancouver, BC V6T 1Z4, Canada. [5] Department of Ecology and Evolutionary Biology, University of California Santa Cruz, 1156 High Street, Santa Cruz, CA 95064, USA. [6] Wildlife Biology Program, Department of Ecosystem and Conservation Sciences, College of Forestry and Conservation, University of Montana, Missoula, MT 59812, USA. [7] Department of Ecosystem Public Health, University of Calgary Veterinary Medicine, TRW 2D01, 3280 Hospital Drive NW, Calgary, AB T2N 4Z6, Canada. [8] Wildlife Research Division, Environment Canada, c/o Department of Biological Sciences, University of Alberta, Edmonton; and Department of Biological Sciences, University of Alberta, Edmonton AB T6G 2E9, Canada. [9] Alaska Geobotany Center, Institute of Arctic Biology, and Department of Biology and Wildlife, University of Alaska Fairbanks, Fairbanks, AK 99775, USA.

*Corresponding author. E-mail: esp10@psu.edu

View publication stats