Predicted Wildlife Disease-Related Climate Change Impacts of Specific Concern to USDA APHIS Wildlife Services

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ABSTRACT: USDA APHIS Wildlife Services plans for and responds to a variety of exigencies such as wildlife hazards to aircraft, disease emergence from wildlife translocations, oral rabies vaccine barrier compromises, and extreme weather events. These are often collaborative efforts with state and federal agencies and others. Climate change based in part on fossil fuel use and methane gas emissions has predictable as well as unknown consequences. As a federal leader in wildlife disease research and management, it is incumbent upon Wildlife Services to be current with the scientific literature; assess potential impacts and wildlife disease management intervention needs from predicted climate change scenarios; and outline a plan of preparedness to meet a variety of potential exigencies.

KEY WORDS: climate change, disease, epizootic, USDA Wildlife Services, vector-borne disease, zoonotic disease

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INTRODUCTION

USDA APHIS Wildlife Services (WS) plans for a variety of exigencies such as aircraft incidents, disease/ vector translocations, vaccine barrier breaches, and extreme weather events. Climate change based on fossil fuel use, methane gas emissions, and other greenhouse gas emission sources is accepted by most scientists, but the predictions are varied in terms of impact and expected outcomes. As a leader in wildlife disease research and management, it is incumbent upon WS to assess potential impacts of predicted climate change scenarios on wildlife disease ecology, and outline a plan of preparedness that will assist the program in effectively meeting future management needs and the environmental changes expected. We provide examples of wildlife and zoonotic diseases likely to be affected by climate change, and consider the current and potential future roles of our agencies' programs relative to climate-related challenges.

CLIMATE CHANGE AND INFECTIOUS DISEASES

Predicted climate change is a leading contemporary issue of national and international importance. Temperature increases of 0.2°C per decade are predicted by the Intergovernmental Panel on Climate Change (IPCC 2007), and effects to public health are expected (Hales et al. 1997). Temperature, rainfall, and humidity affect both vector abundance and pathogen transmission (Reiter 2001). Examples of these that may consequently be impacted by climate change include certain mosquitoes of the genus *Anopheles*, which transmit malaria caused by protozoa belonging to the genus *Plasmodium* spp. (Afrane et al. 2012); the genus *Culex*, which transmit the causative agents of Japanese encephalitis and West Nile virus (Hongoh et al. 2012); the genus *Aedes*, which transmit viruses that cause yellow fever (Reiter 2010) and equine encephalitis virus (Rochlin et al. 2013); and certain rodent-borne zoonoses such as the Hantaviruses (Gubler et al. 2001, Patz and Olson 2006). Other zoonotic infectious disease distributions that could be altered under potential climate change scenarios include others with wild vertebrate reservoirs such as avian influenza, rabies, tularemia, and plague.

Climate change-based models predict potentially significant increases in risk from certain diseases. For example, González et al. (2010) predict a doubling of the number of North Americans with leishmaniasis (caused by a protozoan parasite) by 2080 under even a relatively conservative IPCC climate change scenario. Parkinson and Butler (2005) also suggest considerable potential for climate induced change in Arctic disease epidemiology. In some cases, contemporary vector-borne diseases appear to already be responding, as in the case of dengue fever in the state of Veracruz in Mexico during 1995-2003, where for each degree (C) increase in sea-surface temperature, weekly minimum temperature, and rainfall, accompanying significant increases in dengue cases were noted (Hurtado-Díaz et al. 2007).

While climate change is predicted to be a chief factor in the reemergence of certain infectious disease pathogens in areas currently not affected (Cutler et al. 2010), caution is warranted in assessing the potential role of climate change without consideration of other factors (e.g., land use changes, pollution, socio-economics and lifestyle), which may also be important determinants of disease spread (Reiter 2001, Harvell et al. 2002). For example, Langlois et al. (2001) found that landscape structure was more important in determining Hantavirus incidence than was climate. In some cases (e.g., plague), certain predicted climate change scenarios are expected to result in

reduced incidence of disease (Holt et al. 2009, Snäll et al. 2009). Questions of scale are also worth considering, as global models may not accurately reflect local conditions or provide adequate predictions of disease dynamics at the local level (Snäll et al. 2009).

We examined the literature on selected zoonotic diseases and climate change and provide overviews for predicted changes to the epizootiology of certain zoonoses which WS currently addresses, as well as some for which WS may have a future role in managing. We also assessed WS in terms of potential impacts to the rabies (NRMP) and wildlife disease (NWDP) programs relative to predicted climate change.

WILDLIFE DISEASES, CLIMATE CHANGE, AND WILDLIFE SERVICES

Wildlife Diseases Caused by Fungi

***White Nose Syndrome* (WNS)** – is an emerging disease associated with the fungus *Geomyces destructans* among little brown Myotis (*Myotis lucifugus*) bats and others in the eastern United States and Canada (Gargas et al. 2009a,b; Frick et al. 2010a), which may potentially lead to their extirpation from the region. An expectation of additional population impacts from climate change to little brown Myotis abundance is based on predictions for drier summers in the northeast, which lead to reduced female survival (Frick et al. 2010b).

WS is currently not involved in the management or investigation of WNS on a broad scale but has conducted or assisted with WNS surveillance in certain states. However, in the event that treatment or preventive measures for WNS were made available, WS would be well-positioned to assist in control efforts, with numerous personnel within the current range of WNS. In addition, WS provides guidance through technical assistance to citizens requesting assistance in managing bat problems with consideration for the potential for compounding WNS impacts, and seeks to avoid this.

Aspergillosis – is a disease caused by *Aspergillus* sp. fungi that can infect humans and wildlife, but which is more problematic among birds (Tell 2005). Climate effects to the distribution of *Aspergillus* are thought to be related to its propensity for warmer, wetter environments (Phalen 2000). WS currently engages in, or assists with, Aspergillosis surveillance in a very small number of locations but has no operational activities related to this disease. Shifts in the range of this fungus, coupled with climate-driven increases in its incidence, might necessitate its consideration for action by WS in localized campaigns at some future time.

Wildlife Diseases Caused by Bacteria

Plague – is a bacterial (*Yersinia pestis*) infection vectored by fleas (Siphonaptera**)** to primarily rodents, but with incidental zoonotic spillover into humans. WS has been engaged with partner agencies in plague eradication control programs aimed at prairie dog (*Cynomys* sp.) colonies in the western United States, and plans to continue with these efforts. Climate appears to affect the distribution of plague directly (environmental conditions) and indirectly (through impacts to rodent abundance) (Holt et al. 2009). Plague dynamics models suggest generally reduced plague on the American prairies and in California, overall reduced plague incidence with shifts of plague outbreaks to higher latitudes and higher elevations under some scenarios of climate change due to limitations to the flea vector (Holt et al. 2009, Snäll et al. 2009). Consequently, plague control activities may potentially be less intense.

Lyme Disease – is caused by the bacterium *Borrelia burgdorferi* resulting from the bite of infected Ixodid ticks (CDC 2014a). Reservoirs and vectors vary between locations worldwide. In the eastern U.S., *I. scapularis* ticks serve as the Lyme disease vector while white-footed mice (*Peromyscus leucopus*) serve as reservoirs and white-tailed deer (*Odocoileus virginianus*) serve as Ixodid tick hosts in complex, multi-year cycles (CDC 2014a). In the Pacific west, *I. pacificus* is the primary Lyme disease vector involved in a similar enzootic cycle with an apparently more diverse assemblage of reservoir species, none of which seems to dominate that cycle in the manner of white-footed mice in the east. These include deer mice (*P. maniculatus*) (Lane and Loye 1991, Castro and Wright 2007); dusky-footed wood rats (*Neotoma fuscipes*) and California kangaroo rats (*Dipodomys californicus*) (Lane and Brown 1991); and western gray squirrels (*Sciurus griseus*) (Salkeld et al. 2008). The Lyme disease vectors *I. scapularis* and *I. pacificus* caused 248,074 reported cases of human infection during 1992-2006 (Bacon et al. 2008).

The range of the Lyme disease vector *I. scapularis* has expanded considerably. However, non-occupied, suitable habitats exist (Brownstein et al. 2003), and the range of *I. scapularis* is expected to increase further in Canada as a consequence of predicted climate change scenarios (Ogden et al. 2006). For example, using altitude as a proxy for climate change, Gilbert (2010) determined that *I. ricinus* (a known vector of Lyme disease northern Europe, including the British Isles) abundance was negatively related to altitude, suggesting that climate change may increase its range. However, climate may not be the only factor contributing to increasing Lyme disease. For example, high incidence rates of Lyme disease in the northeastern United States may be a function of reduced biodiversity-driven low alternate host densities (LoGiudice et al. 2003). In addition, the incidence of Lyme disease may be ameliorated in the presence of certain species not currently widespread. For example, Kuo et al. (2000) reported that nymphal *I. scapularis* in the southeastern U.S. tend to prey on lizards, which are not competent reservoirs of Lyme disease, and this likely accounts for relatively low incidence rates of the disease there (Steere et al. 2004). Increased ranges for certain lizard (Squamata) species under warming conditions may consequently reduce the incidence of Lyme disease (Gage et al. 2008). These factors can be assumed to complicate predictions for Lyme disease under climate change scenarios.

No current WS programs are aimed at ameliorating the impacts from Lyme disease on humans and domestic animals. However, since *I. scapularis* abundance has been related to white-tailed deer abundance (Rand et al. 2003), several options for control of which WS is well-suited do exist. In addition, Acaricide self-treatment of

deer through the use of experimental applicators is underway (Pound et al. 2000) and may gain wider use.

Wildlife Diseases Caused by Prions

Chronic Wasting Disease **(CWD)** – is a prion-caused, spongiform encephalopathy of importance to wild cervid conservation, and the captive cervid industry. CWD is distributed among elk (*Cervus elaphus*), mule deer (*O. hemionus*), and white-tailed deer. While most cases of CWD have occurred in the rocky Mountain and western Great Plains states of the U.S., scattered foci have occurred as far east as New York State. Although current medical opinion is that CWD poses no risk to humans, long-term ongoing research into exposure to cervids and Creutzfeldt–Jakob disease in humans will be important for providing a more complete risk assessment (CDC 2013a).

Direct contact between captive and wild cervid populations is of concern for its potential for disease transmission, although contact rates between captive and wild white-tailed deer (VerCauteren et al. 2007a), and between mule deer and captive elk (VerCauteren et al. 2007b) are relatively low. Even captive elk-wild elk contact, which occurs with somewhat greater frequency, can probably be mitigated by certain types of fencing (VerCauteren et al. 2007b). Although snow cover limits the distribution of white-tailed deer, they are sympatric with moose (*Alces alces*) populations at the northern extent of their range. It can further be presumed that under various climate change scenarios that white-tailed deer range may shift further north, with the potential for regular contact with woodland caribou (*Rangifer tarandus caribou*) at their southern-most distribution in locations such as the Gaspe Peninsula of Quebec and in the Selkirk Mountains of the northwestern United States and British Columbia. The potential for white-tailed deer to transmit CWD to these cervids is unknown.

Current and potential operational roles for WS relative to CWD include population reductions, sampling, and fence construction. Given that WS frequently conducts, and has considerable expertise in, cervid direct control activities for protecting agriculture and human health and safety (e.g., airport damage mitigation), the WS CWD role may be easily expanded as needed.

Wildlife Diseases Caused by Parasites

Chagas Disease (aka American trypanosomiasis) – is caused by the parasite *Trypanosoma cruzi*, which is acquired from contact with the feces of infected triatomine bugs (aka assassin/conenose/kissing bugs, genus *Triatoma*). Most of the more than 300,000 documented U.S. human cases of Chagas disease have been acquired in endemic areas of Latin America, and very few cases of Chagas among people living in the U.S. are thought to result from infection here (Bern et al. 2011, CDC 2013b). However, predicted climate change may affect the distribution of the disease. While the southern U.S. is currently considered to be at risk for the establishment of Chagas disease, under certain climate change predictions the central U.S. may be at risk as well (Click Lambert et al. 2008). The low incidence of endemic Chagas disease in the U.S. compared with Latin America is probably due, at least partially, to differences in housing conditions (Bern et al. 2011, CDC 2013b). Regardless, Click Lambert et al. (2008) consider that given that triatomine bug activity levels are related to temperature, possible future temperature increases and other factors may lead to the establishment of Chagas disease in the U.S. To date, WS efforts directed at Chagas disease have been restricted to the provision of serological sampling of raccoons (*Procyon lotor*) captured as part of routine ORV-related activities in Tennessee as sentinels for the disease in collaboration with the Tennessee Department of Health (K. Wehner, USDA APHIS WS, pers. comm.).

Leishmaniasis – is caused by parasites from the genus *Leishmania*, transmitted by Phlebotomine sandflies (Family Psychodidae) (WHO 2014). Both wild and domestic animals are involved in the ecology of leishmania as reservoirs and hosts, primarily in parts of Latin America, Asia, Africa, southern Europe, and the Middle East (CDC 2013c). Leishmania antibodies have been detected in wild canids in the southeastern United States (Duprey et al. 2006). This observation, taken with potential range expansion of the vectors and the leishmaniasis parasite in the future (Dereure et al. 2009, CDC 2013c), suggest a possible wildlife reservoir management role for WS in areas of the U.S. currently, or predicted to be endemic in the future.

Wildlife Diseases Caused by Viruses

Rabies – is a fatal zoonotic disease caused by the bite of mammals infected with the rabies virus, a member of the family Rhabdoviridae, genus *Lyssavirus*, which has a nearly worldwide distribution. Primary vectors of rabies are dogs (*Canis familiaris*), especially in tropical regions. Of importance, the canine variant of rabies is prevalent in Mexico. However, as a consequence of an active oral rabies vaccination (ORV) program in Texas, the canine variant of the virus has been eliminated from the U.S. (Velasco-Villa et al. 2008) and prevented from returning. At present, rabies in the U.S. is found only in wildlife reservoirs. Of special concern is the raccoon variant of the virus, which is distributed throughout the eastern seaboard, but which has been prevented from moving west of the Appalachian Ridge by an ongoing ORV program (Slate et al. 2005, Slate et al. 2009).

Climate change may influence the distribution and intensity of rabies epizootics. For example, climate change may have already released raccoons from restriction to their historical range in conjunction with land use changes (Lariviere 2004), which would potentially bring the threat of raccoon variant rabies to higher latitudes. Increasing temperatures, as a consequence of climate change, could result in changes to red fox (*Vulpes vulpes*) and arctic fox (*Alopex lagopus*) population dynamics, with consequential changes to Arctic fox variant rabies epizootiology (Kim et al. 2013). Under these circumstances, likely results might include increased spending related to responses to sick or strange-acting wildlife incidents, costs and stress related to rabies prophylaxis, and an increase in the risk of mortality to humans and domestic animals. In addition, Charron (2002) suggests that potential climate change may foster rabies virus spillover

into, and epidemics within, non-traditional reservoirs such as ungulates.

Hantavirus Pulmonary Syndrome (HPS) – in the Americas is caused by contact with *Hantavirus* (family Bunyaviridae) particles in the excreta of members of the murid rodents (family Muridae). The primary host of HPS is the deer mouse (*Peromyscus maniculatus*). A related rodent-borne Hantavirus-mediated hemorrhagic fever with renal syndrome is known from Asia, Russia, and parts of Europe (CDC 2014b). HPS is traditionally associated with the southwestern U.S.; however, cases have occurred across the nation, although >95% have been in western states (CDC 2014b). HPS in the American southwest has been related to El Niño-driven deer mouse population irruptions (Engelthaler et al. 1999, Yates et al. 2002, Mills 2005). However, the ecological drivers of Hantavirus in other parts of the nation are not as well studied or understood. Deer mice infected with Hantaviruses are found in a wide range of habitat types (CDC 2014b), but Glass et al. (2000) found that sites above 2,094 m in elevation were associated with a greater HPS risk. If elevation is considered a suitable proxy for climate, it could be assumed that increasing temperatures under predicted climate scenarios might result in a somewhat reduced range of high incident HPS. However, caution in oversimplified interpretations of these results is warranted given that Langlois et al. (2001) found that landscape structure was more important in determining Hantavirus incidence than were climate variables.

WS has assisted in epidemiological investigations of HPS on only rare occasions (Craig et al. 2001). Given the nature of the capture and animal handling procedures required for collecting HPS samples, specialized training and preparation before embarking on such projects is warranted.

SUMMARY

With WS remaining at the forefront of wildlife disease management, becoming and remaining well-versed as an institution in current and potential future impacts from climate change to the distribution and intensity of wildlife diseases is necessary. Consideration for the need to possibly shift programmatic focus and increase specialized training should be given regular attention, and participation in interagency and professional society working groups focused on tracking of and planning for potential new situations as a consequence of climate change or other anthropogenic or natural influences is crucial to full preparedness.

LITERATURE CITED

Afrane, Y. A., A. K. Githeko, and G. Yan. 2012. The ecology of Anopheles mosquitoes under climate change: Case studies from the effects of deforestation in East African highlands. Annals N.Y. Acad. Sci. 1249:204-210.

Bacon, R. M., K. J. Kugeler, and P. S. Mead. 2008. Surveillance for Lyme Disease --- United States, 1992-2006. Morbidity and Mortality Weekly Report 57:1-9.

Bern, C., S. Kjos, M. J. Yabsley, and S. P. Montgomery. 2011. *Trypanosoma cruzi* and Chagas' Disease in the United States. Clin. Microbiol. Rev. 24:655-681.

Brownstein, J. S., T. R. Holford, and D. Fish. 2003. A climate-based model predicts the spatial distribution of the Lyme disease vector *Ixodes scapularis* in the United States. Envir. Health Perspect. 111:1152-1157.

Castro, M. B., and S. A. Wright. 2007. Vertebrate hosts of Ixodes pacificus (Acari: Ixodidae) in California. J. Vector Ecol. 32:140-149.

CDC (Centers for Disease Control). 2013a. Chronic Wasting Disease. Centers for Disease Control. Website.

CDC (Centers for Disease Control). 2013b. Chagas Disease. Volume 71. Centers for Disease Control. Website.

CDC (Centers for Disease Control). 2013c. Leishmaniasis. Centers for Disease Control. Website.

CDC (Centers for Disease Control). 2014a. Lyme Disease Home Page. Centers for Disease Control. Website.

CDC (Centers for Disease Control). 2014b. Hantavirus. Centers for Disease Control. Website.

Charron, D. F. 2002. Potential impacts of global warming and climate change on the epidemiology of zoonotic diseases in Canada. Can. J. Publ. Health 93:334-335.

Click Lambert, R., K. N. Kolivras, L. M. Resler, C. C. Brewster, and S. L. Paulson. 2008. The potential for emergence of Chagas disease in the United States. Geospat. Health 2:227-239.

Craig, W., K. Cook, J. Carney, S. Schoenfeld, B. Wilcke, and T. Algeo. 2001. Hantavirus pulmonary syndrome--Vermont, 2000. Morbidity and Mortality Weekly Report 50:603-605.

Cutler, S. J., A. R. Fooks, and W. H. M. van der Poel. 2010. Public health threat of new, reemerging, and neglected zoonoses in the industrialized world. Emerg. Infect. Dis. 15:1-7.

Dereure, J., S. O. Vanwambeke, P. Malé, S. Martinez, F. Pratlong, Y. Balard, and J.-P. Dedet. 2009. The potential effects of global warming on changes in canine leishmaniasis in a focus outside the classical area of the disease in southern France. Vector Borne Zoon. Dis. 9:687-694.

Duprey, Z. H., F. J. Steurer, J. A. Rooney, L. V Kirchhoff, J. E. Jackson, E. D. Rowton, and P. M. Schantz. 2006. Canine Visceral Leishmaniasis, 2000-2003. Emerg. Infect. Dis. 12: 440-446.

Engelthaler, D. M., D. G. Mosley, J. E. Cheek, C. E. Levy, K. K. Komatsu, P. Ettestad, T. Davis, D. T. Tanda, L. Miller, J. W. Frampton, R. Porter, and R. T. Bryan. 1999. Climatic and environmental patterns associated with Hantavirus pulmonary syndrome, Four Corners region, United States. Emerg. Infect. Dis. 5:87-94.

Frick, W. F., J. F. Pollock, A. C. Hicks, K. E. Langwig, D. S. Reynolds, G. G. Turner, C. M. Butchkoski, and T. H. Kunz. 2010a. An emerging disease causes regional population collapse of a common North American bat species. Science 329:679-682.

Frick, W. F., D. S. Reynolds, and T. H. Kunz. 2010b. Influence of climate and reproductive timing on demography of little brown myotis *Myotis lucifugus*. J. Anim. Ecol. 79:128-136.

Gage, K. L., T. R. Burkot, R. J. Eisen, and E. B. Hayes. 2008. Climate and vectorborne diseases. Amer. J. Prevent. Med. 35:436-450.

Gargas, A., M. T. Trest, M. Christensen, T. J. Volk, and D. S. Blehert. 2009. *Geomyces destructans*. Mycotaxon 108: 147-154.

Gargas, A., M. T. Trest, M. Christensen, T. J. Volk, and D. S. Blehert. 2009. *Geomyces destructans* sp. nov. associated with bat white-nose syndrome. Mycotaxon 108:147-154.

Gilbert, L. 2010. Altitudinal patterns of tick and host abundance: A potential role for climate change in regulating tick-borne diseases? Oecologia 162:217-225.

Glass, G. E., J. E. Cheek, J. A. Patz, T. M. Shields, T. J. Doyle, D. A. Thoroughman, D. K. Hunt, R. E. Enscore, K. L. Gage, C. Irland, C. J. Peters, and R. Bryan. 2000. Using remotely sensed data to identify areas at risk for hantavirus pulmonary syndrome. Emerg. Infect. Dis. 6:238-247.

González, C., O. Wang, S. E. Strutz, C. González-Salazar, V. Sánchez-Cordero, and S. Sarkar. 2010. Climate change and risk of leishmaniasis in North America: Predictions from ecological niche models of vector and reservoir species. PLoS Neglected Tropical Diseases 4:e585.

Gubler, D. J., P. Reiter, K. L. Ebi, W. Yap, R. Nasci, and J. A. Patz. 2001. Climate variability and change in the United States: Potential impacts on vector- and rodent-borne diseases. Environ. Health Perspect. 109 (Suppl.):223-233.

Hales, S., P. Weinstein, and A. Woodward. 1997. Public health impacts of global climate change: Reviews on environmental health. Rev. Environ. Health 12:191-199.

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.

Holt, A. C., D. J. Salkeld, C. L. Fritz, J. R. Tucker, and P. Gong. 2009. Spatial analysis of plague in California: Niche modeling predictions of the current distribution and potential response to climate change. Intl. J. Health Geogr. 8:38.

Hongoh, V., L. Berrang-Ford, M. E. Scott, and L. R. Lindsay. 2012. Expanding geographical distribution of the mosquito, *Culex pipiens*, in Canada under climate change. Appl. Geogr. 33:53-62.

Hurtado-Díaz, M., H. Riojas-Rodríguez, S. J. Rothenberg, H. Gomez-Dantés, and E. Cifuentes. 2007. Short communication: impact of climate variability on the incidence of dengue in Mexico. Trop. Med. Intl. Health 12: 1327-1337.

IPCC (Intergovernmental Panel on Climate Change). 2007. Summary for policymakers. P.18 *in*: M. M. Solomon, S., D. Qin, M. Manning, Z. Chen, M. Marquis, K. B. Averyt, M. Tignor, and H. L. Miller (Eds.), 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, Cambridge, UK and New York, NY.

Kim, B. I., J. D. Blanton, A. Gilbert, L. Castrodale, K. Hueffer, D. Slate, and C. E. Rupprecht. 2014. A conceptual model for the impact of climate change on fox rabies in Alaska, 1980-2010. Zoonos. Publ. Health 61(1):72-80.

Kuo, M. M., R. S. Lane, and P. C. Giclas. 2000. A comparative study of mammalian and reptilian alternative pathway of complement-mediated killing of the Lyme disease spirochete (*Borrelia burgdorferi*). J. Parasitol. 86: 1223-1228.

Lane, R. S., and R. N. Brown. 1991. Wood rats and kangaroo rats: Potential reservoirs of the Lyme disease spirochete in California. J. Med. Entomol. 28:299-302.

Lane, R. S., and J. E. Loye. 1991. Lyme disease in California: Interrelationship of Ixodid ticks (Acari), rodents, and *Borrelia burgdorferi*. J. Med. Entomol. 28:719-725.

Langlois, J. P., L. Fahrig, G. Merriam, and H. Artsob. 2001. Landscape structure influences continental distribution of hantavirus in deer mice. Landscape Ecol. 16:255-266.

Lariviere, S. 2004. Range expansion of raccoons in the Canadian prairies: Review of hypotheses. Wildl. Soc. Bull. 32:955-963.

LoGiudice, K., R. S. Ostfeld, K. A. Schmidt, and F. Keesing. 2003. The ecology of infectious disease: Effects of host diversity and community composition on Lyme disease risk. Proc. Nat. Acad. Sci. USA 100:567-571.

Mills, J. N. 2005. Regulation of rodent-borne viruses in the natural host: Implications for human disease. Arch. Virol. Suppl. 2005(19)45-57.

Ogden, N. H., A. Maarouf, I. K. Barker, M. Bigras-Poulin, L. R. Lindsay, M. G. Morshed, C. J. O'Callaghan, F. Ramay, D. Waltner-Toews, and D. F. Charron. 2006. Climate change and the potential for range expansion of the Lyme disease vector *Ixodes scapularis* in Canada. Intl. J. Parasitol. 36:63-70.

Parkinson, A. J., and J. C. Butler. 2005. Potential impacts of climate change on infectious diseases in the Arctic. Intl. J. Circumpol. Health 64:478-486.

Patz, J. A., and S. H. Olson. 2006. Climate change and health: Global to local influences on disease risk. Ann. Trop. Med. Parasitol. 100:535-549.

Phalen, D. N. 2000. Respiratory medicine of cage and aviary birds. Vet. Clin. N. Amer. Exot. Anim. Prac. 3:147-154.

Pound, J. M., J. A. Miller, J. E. George, and C. A. Lemeilleur. 2000. The "4-Poster" passive topical treatment device to apply Acaricide for controlling ticks (Acari: Ixodidae) feeding on white-tailed deer. J. Med. Entomol. 37:588-594.

Rand, P. W., C. Lubelczyk, G. R. Lavigne, S. Elias, S. Holman, E. H. Lacombe, R. P. S. Jr, M. S. Holman, and R. P. Smith. 2003. Deer density and the abundance of *Ixodes scapularis* (Acari: Ixodidae). J. Med. Entomol. 40:179-184.

Reiter, P. 2001. Climate change and mosquito-borne disease. Envir. Health Perspect. 109:141-161.

Reiter, P. 2010. Yellow fever and dengue: A threat to Europe? Eur. Commun. Dis. Bull. 15:1-7.

Rochlin, I., D. V Ninivaggi, M. L. Hutchinson, and A. Farajollahi. 2013. Climate change and range expansion of the Asian Tiger Mosquito (*Aedes albopictus*) in northeastern USA: Implications for public health practitioners. PloS One 8:e60874.

Salkeld, D. J., S. Leonhard, Y. A. Girard, N. Hahn, J. Mun, K. A. Padgett, and R. S. Lane. 2008. Identifying the reservoir hosts of the Lyme disease spirochete *Borrelia burgdorferi* in California: The role of the western gray squirrel (*Sciurus griseus*). Am. J. Trop. Med. Hyg. 79:535-540.

Slate, D., C. E. Rupprecht, J. A. Rooney, D. Donovan, D. H. Lein, and R. B. Chipman. 2005. Status of oral rabies vaccination in wild carnivores in the United States. Virus Res. 111:68-76.

Slate, D., T. P. Algeo, K. M. Nelson, R. B. Chipman, D. Donovan, J. D. Blanton, M. Niezgoda, and C. E. Rupprecht. 2009. Oral rabies vaccination in North America:

Opportunities, complexities, and challenges. PLoS Neglected Trop. Dis. 3:e549.
Snäll, T., R. Benestad, and N. Stenseth. 2009. Expected future plague levels in a wildlife host under different scenarios of climate change. Global Change Biol. 15:500-507.
Steere, A. C., J. Coburn, and L. Glickstein. 2004. The emergence of Lyme disease. J. Clin. Investig. 113:1093-1101.
Tell, L. 2005. Aspergillosis in mammals and birds: Impact on veterinary medicine. Med. Mycol. 43:71-73.
Velasco-Villa, A., S. A. Reeder, L. A. Orciari, P. A. Yager, R. Franka, J. D. Blanton, L. Zuckero, P. Hunt, E. H. Oertli, L. E. Robinson, and C. E. Rupprecht. 2008. Enzootic rabies elimination from dogs and reemergence in wild terrestrial carnivores, United States. Emerg. Infect. Dis. 14:1849-1854.
VerCauteren, K. C., M. J. Lavelle, N. W. Seward, J. W. Fischer, and G. E. Phillips. 2007a. Fence-line contact between wild and farmed white-tailed deer in Michigan: Potential for disease transmission. J. Wildl. Manage. 71:1603-1606.
VerCauteren, K. C., M. J. Lavelle, N. W. Seward, J. W. Fischer, and G. E. Phillips. 2007b. Fence-line contact between wild and farmed cervids in Colorado: Potential for disease transmission. J. Wildl. Manage. 71:1594-1602.
WHO (World Health Organization). 2014. Leishmaniasis. World Health Organization. Website.
Yates, T. L., J. N. Mills, C. A. Parmenter, T. G. Ksiazek, R. R. Parmenter, J. R. Vande Castle, C. H. Calisher, S. T. Nichol, K. D. Abbott, J. C. Young, M. L. Morrison, B. J. Beaty, J. L. Dunnum, R. J. Baker, J. Slazar-Bravo, and C. J. Peters. 2002. The ecology and evolutionary history of an emergent disease: Hantavirus pulmonary syndrome. BioScience 52: 989-998.