

# Changing Patterns of Wildlife Diseases

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The gradual warming trend throughout many regions of North America during the last several decades has been linked to global warming caused by increases in atmospheric carbon dioxide (National Assessment Synthesis Team 2000). Increasing average temperatures of a few degrees could have direct and indirect effects on the intensity, distribution, and impact of some diseases of wildlife. Disease agents are transmitted between animals and to humans from animals (zoonotic diseases) either directly through contact or aerosols (such as with rabies and hantavirus) or indirectly by vector species (such as mosquitoes with West Nile virus (WNV) or ticks with Lyme disease). It is difficult to confirm a direct causal relationship between climate variability like gradual warming and diseases of wildlife, especially for those disease agents that are transmitted between animals by a vector species like a mosquito. However, components in disease transmission cycles could be influenced by increased ambient temperatures and the dynamics of transmission and factors associated with it could be affected enough to promote increased frequency of disease. Data analyses have not been performed to examine any causal relationships and possibly insufficient data exists now, but some of the components and factors involved in the transmission of mosquito-borne, avian viral diseases can be used to illustrate potential effects (Figure 1). Ambient temperatures can influence the population dynamics and interactions of vector mosquitoes, vertebrate hosts, and viruses in temperate climates (Moore et al. 1993). Milder winters improve the survival rate of adult mosquitoes (*Culex* spp.) and birds to provide an increased population to start the breeding season. Warmer spring weather allows more efficient early blood feeding by adult mosquitoes, increased egg laying, and increased survival of early hatches of mosquito larvae in warmer waters. The warmer water also decreases the developmental time for the water stages of mosquitoes. All of these factors favor an increase in the number of mosquito generations and their abundance during the transmission season. Bird populations can be expanded by warmer temperatures through earlier first broods in some species (e.g. Rock dove, *Columba livia*), by increased survival of nestlings and fledglings, by the

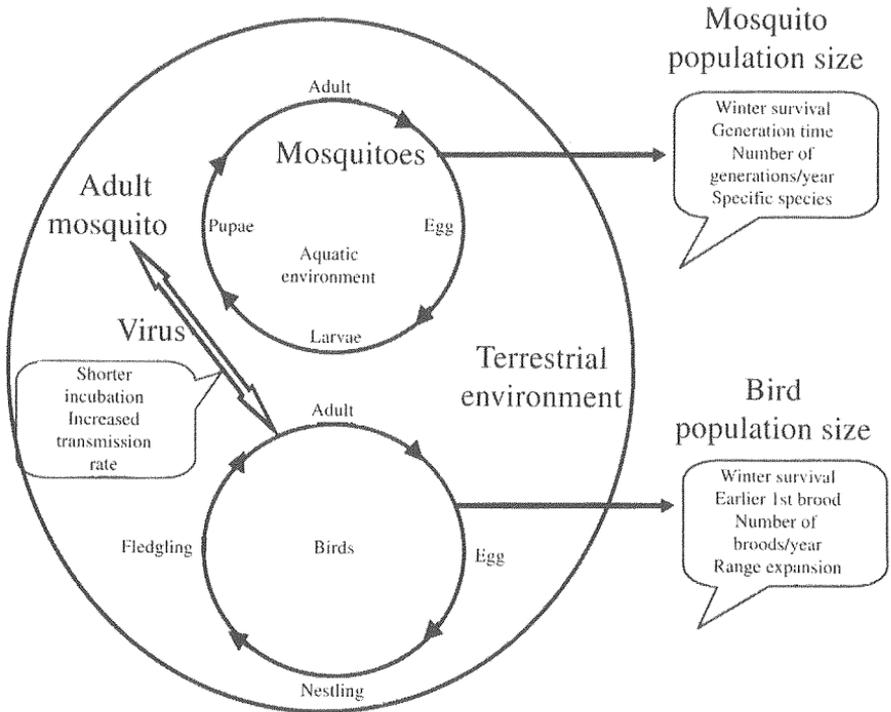


Figure 1. Transmission dynamics of mosquito-borne viral diseases of birds

number of successful broods per year, and by enlarged ranges. If these expanded populations of competent vectors and hosts are aligned temporally and spatially, then the frequency of disease transmission can be increased if the virus is present. Warmer temperatures can also influence the magnitude of transmission by shortening the incubation time of the virus in the mosquito vector following ingestion of virus-infected bird blood thus increasing the transmission rate between birds. However, sustained warming temperatures could have a different impact on mosquito-transmitted diseases as habitats and available water sources change.

In addition, indirect effects may influence changes in disease patterns. Warmer temperatures and associated precipitation changes may gradually alter natural and human-modified wildlife habitats. The modified habitats alone could promote local increases in the abundance of vector species or regional increases in population densities and geographical expansions of some wildlife species. I would like to discuss how warming could affect the rate of transmission and the magnitude of some selected diseases.

## Selected Wildlife Diseases

The disease patterns of some selected wildlife diseases that could be changed by warming trends are presented (Table 1) to encourage further investigation of the interacting physical and biological factors in the transmission cycles, the transmission dynamics, and the components of the transmission chain that could be targeted for prevention and control. Each of these diseases will be discussed in relation to the potential effects of gradual warming temperatures.

Table 1. Examples of wildlife diseases that could be influenced by global warming.

Disease	Pathogen	Wildlife species	Route of transmission	Morbidity/mortality
Avian botulism	Bacterial toxin ( <i>Clostridium botulinum</i> )	Waterfowl, some songbirds	Ingestion of bacterial toxin	>1,000,000 birds died
Lyme disease	Spirochete ( <i>Borrelia burgdorferi</i> )	Mice, chipmunks, songbirds, deer	Tick vector ( <i>Ixodes scapularis</i> )	± 125,000 humans ill, no wildlife effect
West Nile virus	<i>Flavivirus</i>	Birds, particularly Corvidae	Mosquito vector ( <i>Culex</i> spp.)	81 humans ill, 9 died, ±50,000 birds died
Hantavirus pulmonary syndrome	Sin nombre virus ( <i>Hantavirus</i> )	<i>Peromyscus maniculatus</i> , other rodents	Direct by aerosol	277 humans ill, 100 died, no wildlife effect
Rabies	<i>Lyssavirus</i>	Raccoon, other wild carnivores, bats	Direct by bite of infected animal	1-2 humans/year, 6,000-7,000 wildlife/year

## Mosquito-borne Viral Diseases

Expanded transmission of mosquito-borne diseases in the U.S. occurred only once during the last two decades until the introduction of the exotic West Nile virus in 1999 (Lanciotti 1999). This invasive pathogen would have infected our naïve avian species regardless, but warmer temperatures may have influenced the establishment of the introduced virus and its rapid expansion. West Nile virus, like the related St. Louis encephalitis virus, is a bird virus transmitted between birds by selected species of mosquito species. Humans are an incidental, dead-end host for the virus. As discussed above, warmer weather certainly enhances survival and production of mosquitoes and certain bird species in temperature climates that would

provide larger populations of the host and vector species for increased transmission. The expansion and persistence of WNV in northern latitudes of North America may be dependent upon continuing warm temperatures.

### ***Avian Botulism***

The predicted reduction in wetland habitats in the prairie pothole regions of the Northern Great Plains due to climatic change (National Assessment Synthesis Team 2000) may not only gradually suppress waterfowl production, but could facilitate more frequent and larger outbreaks of certain waterfowl diseases associated with these habitats. Increased risk of waterfowl mortality from avian botulism caused by the toxin produced by *Clostridium botulinum* bacteria is strongly associated with a delicate balance of environmental conditions of the wetlands and with waterfowl populations (Rocke and Friend 1999). The severity of the outbreaks is also associated with the density of the waterfowl populations. As wetland habitats are reduced by climatic change, waterfowl are forced to crowd into smaller and smaller habitats and to further increase the mixing of species. Both situations of overcrowding and smaller, more polluted habitats provide the conditions for dramatic increases in transmission of avian botulism and other avian diseases. An example of the effect of reduced waterfowl habitat on avian diseases is the loss of many wetlands of the Central Valley of California by conversion for agricultural purposes and by the diversion of shrinking water supplies for human use. These wetlands were crucial stopover and wintering habitats for migratory water birds that have been forced to move to much smaller and less optimal habitats such as the Salton Sea in southern California. The mass concentration of these birds on the remaining wetlands for prolonged periods of time degrades the habitats and promotes transmission of disease agents that may be present. Massive die-offs of water birds at the Salton Sea from avian botulism have occurred during the last few years (USGS National Wildlife Health Center unpublished data: 1997)

### ***Lyme Disease***

The transmission of tick-borne, zoonotic diseases involving wildlife may be affected by warmer climates. Lyme disease has emerged dramatically during the last two decades in the Northeastern and upper midwestern states. The incidence and geographical distribution of human cases have increased significantly and illness from Lyme disease now exceeds all other zoonotic diseases combined (Dennis 1998). The Lyme disease spirochete,

*Borrelia burgdorferi*, is transmitted between the primary rodent reservoir species, white-footed mouse (*Peromyscus leucopus*) and eastern chipmunk (*Tamias striatus*, by the insidious tick vector (*Ixodes scapularis*). Other small mammals and birds contribute to the transmission and dissemination of the spirochete as well. The white-tailed deer (*Odocoileus virginianus*) serves an important role as a host for the tick vector and in locally amplifying tick populations. There is a multiplicity of animal species involved in the transmission cycle and factors influencing the dynamics of the transmission; however, increasing warmer temperatures alone could indirectly enhance transmission. The increasing survival and expansion of populations of white-tailed deer, small mammals, and ticks were likely induced by warmer climates in combination with other changes such as regional land use shifts away from agriculture and toward re-growth of secondary forests and subsequent fragmentation of the available habitats by suburban development. The optimal habitat for Lyme disease transmission was created with expanded white-tailed deer and small mammal populations in close proximity to an expanding human population mixed with a tick species that feeds on all three.

### ***Hantavirus***

A human outbreak of Hantavirus pulmonary syndrome (HPS) that occurred in 1993 may have been caused by El Nino events in the southwestern United States (Centers for Disease Control and Prevention El Nino Special Report 2000) that resulted in increased rainfall and milder winters for the region for several years. These weather changes to a region that is normally very dry greatly improved the rodent habitat quality. Population densities of the primary rodent reservoir, the deer mouse (*Peromyscus maniculatus*), for the *Hantavirus* causing HPS (Sin Nombre Virus) expanded dramatically during 1991-1993 in response to the habitat changes. High rodent abundance facilitated increased transmission of the virus among rodents and increased encounters with humans living in rural environments; therefore, the risk of an outbreak was elevated. Dramatic changes in environmental conditions that favor rodent-borne diseases could continue if climate variability persists.

### ***Wild Animal Rabies***

Natural and human-modified wildlife habitats gradually modified by warmer temperatures and associated precipitation changes could promote regional increases in population densities and geographical expansions of some carnivore species that are reservoirs for rabies virus. Cases of wild

animal rabies reported in the United States have doubled during the last two decades, mostly due to the expansion of raccoon rabies in the northeastern states (Hanlon and Rupprecht 1998). The introduction of the raccoon strain of rabies virus into naïve populations in this region was largely responsible for this dramatic expansion in such a short period of time, but more abundant populations of carnivores also contributed to the magnitude and persistence of rabies transmission. Warmer winters that make winter food sources and shelter more available and expanded human-modified habitats enable wildlife species like raccoons to increase their populations in northern latitudes. An increase in transmission and distribution of wild animal rabies would elevate the disease risk to humans.

## Conclusion

The purpose of this paper was not to analyze the effects of global warming on wildlife disease patterns, but to serve as a springboard for future efforts to identify those wildlife diseases, including zoonotic diseases, that could be influenced the most by warming climates and to encourage the development of models to examine the potential effects. Hales et al. (1999) examined the relationship of the incidence of a vector-borne human disease, Dengue fever, and El Nino southern oscillations for South Pacific Island nations. The development of similar models on specific wildlife diseases which have environmental factors strongly associated with transmission would provide information and options for the future management of our wildlife resources.

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