CD Summary

December 9, 2008
Vol. 57, No. 25

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OREGON PUBLIC HEALTH DIVISION • DEPARTMENT OF HUMAN SERVICES

CLIMATE CHANGE AND COMMUNICABLE DISEASE

The past generation has seen the emergence of omnifarious infectious diseases — viral, bacterial, fungal, protozoan, even helminthic. In its 2003 treatise on *Microbial Threats to Health*, the Institute of Medicine identified 13 factors that lead to such emergence, one of which was "climate and weather."[1] In the Pacific Northwest, models predict an average increase in temperature of 3°–4°C year-round and, in non-summer months, increasing precipitation, more of which will be delivered as rain rather than snow.[2] This *CD Summmary* illustrates mechanisms and explores potential effects of changes in climate and weather on communicable diseases in the Pacific Northwest.

Climate and weather may influence the emergence and epidemiology of infectious diseases through several mechanisms.[3]

Direct Effects

- microbial multiplication
- microbial movement
- movement and replication of vectors and animal hosts

Indirect Effects

- effects operating through ecological changes
- effects operating through changes in human activities

MICROBIAL MULTIPLICATION

Many arthropod vector-borne pathogens are sensitive to temperature. West Nile virus (WNv) appears not to replicate within female *Culex* mosquitoes at temperatures lower than about 14°C. Titers of WNv within infected female *Culex* mosquitoes are a function of time and temperature, and there seems to be a threshold viral titer within the mosquito, below which transmission doesn't occur.[4] WNv infection already exhibits a strong seasonality, with peak transmission in late summer in the Northwest; longer summers with higher temperatures may substantially increase the incidence of WNv fever and encephalitis in human beings.

Vibrio parahaemolyticus causes gastroenteritis in persons who eat raw oysters. This bacterium may require water temperatures ≥17°C to thrive and to reach quantities in oysters sufficient to infect humans.[5] Oysters harvested in Oregon, Washington, and British Columbia during summer months have caused outbreaks of *V. parahaemolyticus* infection;[6] but in 2004, an outbreak aboard a cruise ship implicated oysters harvested from Prince William Sound, Alaska — 1,000 km north of the pathogen's previously recognized northern outpost.[7] Warming waters in the Pacific Northwest would lead to higher concentrations of *Vibrio* spp. in shellfish beds and more prolonged periods of summer risk.

MICROBIAL MOVEMENT

Pathogenic microbes may be passively transported by air or water. One relevant example is *Cryptosporidium parvum*, a protozoan agent of diarrhea that is enzootic in cattle. The predicted increasing rain in the Northwest, with flooding effects multiplied by rain-on-snow events in the Cascades, may lead to the washing of C. *parvum* and other animal intestinal indwellers into drinking water reservoirs.[3, 8] Rivers swelled by spring rains and snow runoff may well have been the cause of contamination of the Milwaukee, Wisconsin, drinking water supply and the massive outbreak of cryptosporidiosis that occurred there in 1993.[9] Indeed, a review of waterborne disease outbreaks in the United States during 1948–1994 found a significant correlation with "extreme precipitation events" in the two months before the outbreaks.[10]

VECTORS AND ANIMAL HOSTS

A review of infectious diseases in 2001 identified 175 that were caused by pathogens considered to be "emerging"; of these diseases, 132 (75%) were zoonoses — i.e., transmitted to human beings from animals.[11] We should expect that animal hosts for zoonotic pathogens will be affected by changes in climate. The 1993 outbreak of hantavirus pulmonary syndrome in New Mexico was preceded by increasing densities of *Peromyscus* mice, the definitive host for Sin Nombre virus (SNv). *Peromyscus* populations were higher in areas where precipitation had increased with the El Niño event of 1992. Further studies of precipitation, *Peromyscus* densities, and SNv seropositivity among *Peromyscus* mice in the Southwest corroborated the association of higher rates of hantavirus pulmonary syndrome with periods of higher-than-usual rainfall, after a lag time that allowed the mouse reservoir populations to increase and to become infected with SNv.[12]

ECOLOGICAL CHANGES

The fungus *Cryptococcus neoformans* is an environmental saprophyte whose niche is dead or rotting trees. *C. neoformans* has been notorious as a cause of meningitis in patients with organ transplants or AIDS, but one variety has shown a particular ability to infect even immunocompetent hosts.[13] This variety, known as *gattii*, was thought to have been restricted to tropical and subtropical areas.[14] However, the pathogen emerged on the east coast of Vancouver Island as the cause of an outbreak beginning in 1999, and environmental sampling in a provincial park uncovered an ecological berth among several tree species there, notably our beloved Douglas Fir. The researchers hypothesized that the establishment of the fungus in this area may have been due to climatic changes.[15]

CHANGES IN HUMAN ACTIVITIES

Places and processes whereby people eat, drink, dwell and recreate will probably have more effect

The **CD Summary** (ISSN 0744-7035) is published biweekly, free of charge, by the Oregon Dept. of Human Services, Office of Communicable Disease and Epidemiology, 800 NE Oregon St., Portland, OR 97232
Periodicals postage paid at Portland, Oregon.
Postmaster—send address changes to:
CD Summary, 800 NE Oregon St., Suite 730, Portland, OR 97232

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on the epidemiology of infectious diseases than any of the factors listed above. Legionnaire's disease has been uncommon in the Northwest, but should summers become warmer and longer, increased use of air conditioning may lead to higher rates of this infection. Fluvial flooding or rising sea levels could lead to displacement and concentrations of populations, with consequent increases in diseases transmitted directly from person to person. In-migration of populations from a hotter and drier U.S. Southwest could lead to changes in land use in our part of the continent — e.g., increased settlement east of the Cascade Mountains, with increases in diseases, like hantavirus pulmonary syndrome or relapsing fever, more commonly seen there;[16] or increased housing adjacent to forested areas west of the Cascades, leading to more contact with the *Ixodes* tick vectors of Lyme disease.

PERSPECTIVE

We can't predict what the net effects of climate change will be. We expressed concern about the possibility of more *Cryptosporidium* or *Giardia* infections if the climate gets wetter. The flip side is that the survival of their cysts in water is shortened by warmer water temperatures.[17] Expected increases in vector-borne and other diseases might be more than offset by reduced incidence of diseases typically associated with cold weather like influenza or norovirus infection.

That said, given the dynamic interplay among reservoirs, vectors, human hosts, and the environment, we can predict that communicable disease patterns will change. The best means of fending off any changes for the worse are the very ones advocated ten years ago for addressing emerging infectious diseases: ensuring that we can detect changes in disease patterns, investigate as needed and mount an appropriate public health response.[18]

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