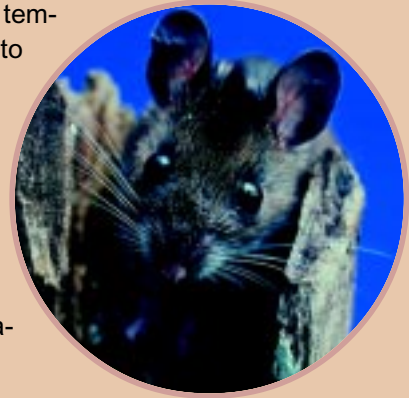


Human health can be linked to climatic conditions. For example, warmer temperatures can lead to heat-induced illnesses. Drier conditions can lead to breathing difficulties, compounded by increases in the amount of airborne dust in some areas. Two very specific climate-related diseases illustrate how changes in climate and climate variability can affect human health.

Valley fever is endemic to the Southwestern United States. The disease is caused by a climate-sensitive fungus that grows in the soil. The timing and magnitude of seasonal climate variations affect the release and wind dispersal of the spores that cause the disease. Precipitation and temperature appear to be important climatic controls on the fungus.

Hantavirus pulmonary syndrome was first recognized as a major threat to human health in the Western Hemisphere in 1993, following an outbreak of disease in the Southwest. The origins of the disease were traced to a virus found in populations of deer mice. The incidence of Hantavirus may be linked to seasonal and interannual patterns of rainfall, which affect rodent food sources and changes in rodent population.



Chapter

8

## HUMAN HEALTH

### Climate and Valley Fever

#### Historical Importance and Current Stresses

Coccidioidomycosis, commonly known as valley fever or “cocci,” is caused by *Coccidioides immitis* (*C. immitis*), a fungus that grows in the soil of limited regions in the United States, as well as portions of Central and South America. Humans and other mammals, such as dogs and cattle, are susceptible to the disease. Historically, endemic regions within the United States (Figure 30) have included Kern County in the San Joaquin Valley of California; Maricopa, Pima, and Pinal counties of Arizona; and a small portion of Texas which runs east from the southeast corner of New Mexico to slightly beyond Laredo (Maddy 1965). These areas are still at risk in modern times.

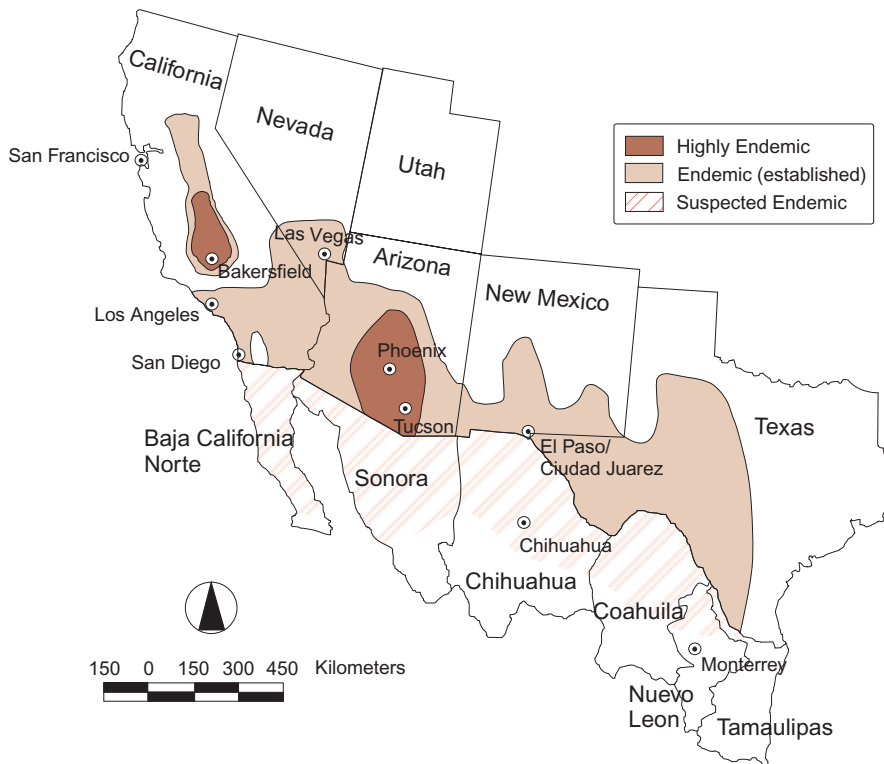
There is documented evidence relating outbreaks of valley fever and climatic conditions. *C. immitis* is sensitive to climate variability, and responds to changes in moisture and temperature. Previous studies have suggested a relationship between the incidence of valley fever and climatic conditions. Temperature, precipitation, humidity, wind, and the occurrence of dust storms have been shown to affect either the growth of *C. immitis* and/or the distribution of the fungus. It is expected that the distribution of *C. immitis* and outbreaks of valley fever will be affected by climate change.

Valley fever cannot be spread from person to person, and people who have been infected with valley fever gain, in most cases, lifelong immunity to the disease (Pappagianis 1988).

The majority of the people infected (60%) either present no symptoms, or experience mild, cold-like conditions. Some may endure a variety of flu-like symptoms that usually appear after an incubation period of one to three weeks. Of those infected by *C. immitis*, about one percent experience a disseminated form of the disease when the fungus enters the bloodstream and spreads beyond the lungs (Einstein and Johnson 1992). Disseminated valley fever can express itself with a wide variety of conditions. Lesions may occur on organs outside of the pulmonary system, as well as on the skin; bones and joints may be damaged. The most severe form of the disseminated disease is coccidioidal meningitis, the mortality of which is essentially one hundred percent when produced by valley fever (Fiese 1958).

Valley fever is a regional disease that is likely to increase in prominence. The population of Southwestern United States is growing at a rapid pace; in 1998, Arizona was the second fastest growing state in the nation. Many of the people who have migrated to this region are primarily from non-endemic areas, thus it is unlikely they have previously been exposed to valley fever and therefore do not have immunity to the disease. This is of particular concern in the tourist areas. For example, winter visitors flock to Phoenix and Tucson. If they were to contract a serious case of valley fever and then return home, their physicians may misdiagnose the disease.

Although most people infected with valley fever do not need to seek medical care, treatment of serious cases can be costly, both directly through medical care and indirectly, through lost



**Figure 30.** Areas of the United States and northern Mexico considered endemic to valley fever. Source: Adapted from Kirkland and Fierer, 1996

worker-hours. On average, valley fever treatment in the United States costs \$9 million annually, and results in a loss of almost one million person-days of labor (Pappagianis 1980). A 1977 outbreak in California cost approximately \$2 million. Another outbreak in California, which lasted from 1991 to 1994, cost an estimated \$66 million in treatment, hospitalization, and lost wages (Jinadu 1995).

### Potential Impacts of Climate Change

Precipitation and temperature appear to be the most important climatic controls on valley fever. During prolonged periods of hot, dry conditions, the surface of the soil is partially sterilized and *C. immitis* and many competing microorganisms are removed. However, *C. immitis* remains viable below the surface. When rain falls, conditions in the surface soil eventually approach the ideal for the growth of the fungus. *C. immitis* returns to the surface layer, which contains few competing organisms (Maddy 1957).

Precipitation is a significant control on the distribution of *C. immitis* and incidence of valley fever. Very low rainfall, as well as annual rainfall in excess of 500 mm (20 inches), decreases the prevalence of *C. immitis* in the soil (Reed 1960). The Mohave and Sonoran Deserts of California receive approximately 76 mm (3 inches) of rain annually, too dry for *C. immitis* (Maddy 1958). Coincidentally, increased rainfall at the eastern limits of the endemic zone in Texas enables competitive species to thrive.

Previous studies, conducted several decades ago, have concluded that *C. immitis* is most often found in the soil following rainy seasons, and less frequently during hot, dry periods (Egeberg and Ely 1956; Plunkett and Swatek 1957; Maddy 1965). Likewise, studies have shown that the incidence of human infections of valley fever is highest during dry periods following the rainy season when the soil is dry and the fungus can be distributed by winds (Smith et al. 1946a; Maddy 1965; Stevens 1995).

An epidemic of valley fever in the early 1990s in California was linked to variability in precipitation. Jinadu (1995) reported that the epidemic followed five years of drought in California. February and March of 1991 through 1994 had approximately double the normal amount of rainfall, and Jinadu commented that these “intense rains caused an abundant growth” of the fungus in the soil. After drying, the soil was disturbed by winds, and *C. immitis* spores were released into the air causing a much greater number of cases than normal, particularly in Kern County, California.

A study by Hugenholtz (1957) concerned the relationship between the incidence of valley fever and several climatic variables in Arizona, namely temperature, rainfall, and dust storms. An examination of hospital admissions records at Williams Air Force Base in Maricopa County from 1952 to 1956, for example, showed two annual peaks in valley fever incidence, one in July and a second in October or November (Hugenholtz 1957). Months with highest incidence coincided

with months having the lowest rainfall. Hugenholtz employed quantitative techniques in the study, correlating temperature, dust storm incidence, or total rainfall, with valley fever incidence. The study did not find a strong relationship between rainfall and incidence, but found stronger relationships with temperature and dust storms. Based on the findings however, Hugenholtz concluded that it is possible to predict lower infection rates during a season if the preceding wet period was drier than normal. For example, infection rates should be lower in the spring and summer following a relatively dry winter, and a drier than usual July and August should be followed by fewer infections in fall. Hugenholtz commented that his “remarks have been largely theoretical and based on an incomplete study, but they may serve to stimulate studies by other investigators.”

### Future Information and Research Needs

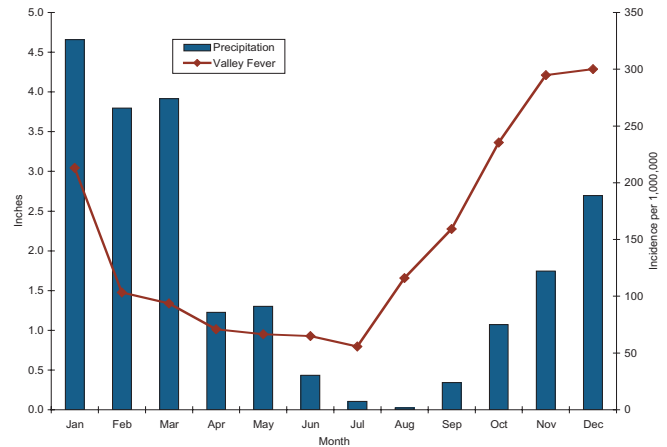
Little research examining the role of climate variability in the occurrence of valley fever has been performed since the 1950s and 1960s. Of the studies during that period, only a few compared climate and incidence data. In particular, the study by Hugenholtz in 1957 looked for a correlation between such information, and analyzed 14 years of data for a specific area. Although there is a general understanding of the climatic characteristics of the endemic region, the specific conditions that may result in an outbreak of valley fever are not well understood.

Although the data are in some ways problematic (given different reporting techniques and a varying incubation period), long records of valley fever incidence are available. We recommend quantitative analysis of incidence data in conjunction with climate data, such as temperature, precipitation, wind speed, and relative humidity. An analysis of the entire endemic region in the United States will allow comparisons of different climatic regimes. An analysis of climate and valley fever incidence in California can, for example, be compared to the Arizona/New Mexico region. California receives the majority of its precipitation in winter (see Figure 31), while the Southwest experiences a bimodal precipitation pattern (see Figure 32). The two regions therefore have differing patterns of valley fever incidence, which may provide insight into the distribution of the fungus. Analysis of multivariate climate data and valley fever incidence data can then be used to develop models of *C. immitis*’ response to climate. A predictive model will be particularly useful to health care providers and government health services.

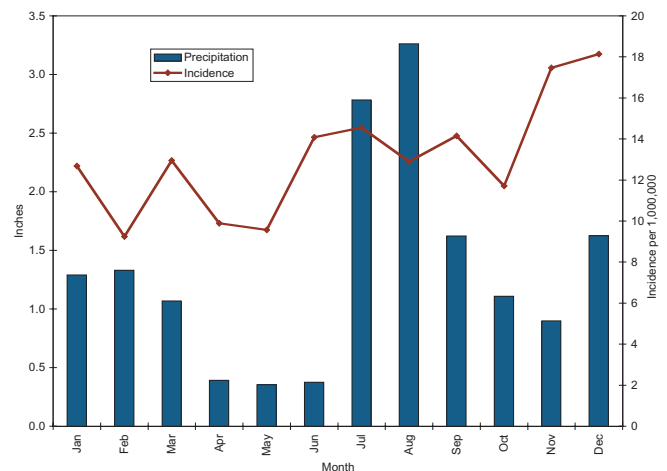
## Hantavirus

### Historical Importance and Current Stresses

Hantavirus pulmonary syndrome (HPS) was first recognized as a major threat to human health in the western hemisphere in the spring and summer of 1993, with an outbreak of disease

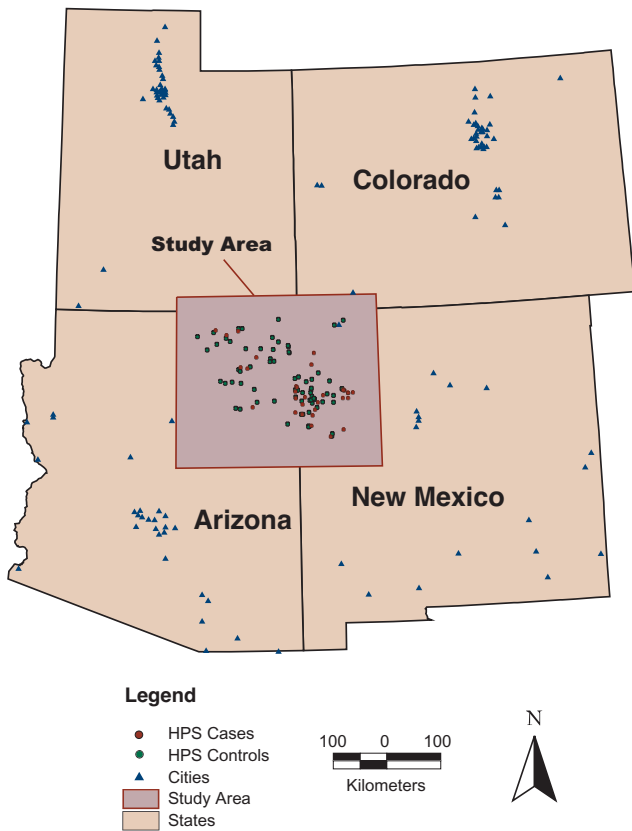


**Figure 31.** Precipitation pattern and incidence of valley fever, Kern County California, 1989–1998. Source: Kolivras et al., in press



**Figure 32.** Bimodal precipitation pattern and incidence of valley fever, Pima County Arizona, 1980–1998. Source: Kolivras et al., in press

among previously healthy populations in the Southwestern United States. Medical professionals were first warned of the disease by the deaths of a young Navajo woman and her fiancé, both healthy individuals, who died within five days of each other (Sternberg 1994). Three additional victims of the disease were quickly identified, all of whom had died within a few days of each other of an unidentified “rapidly progressive” respiratory illness (Warner 1996). Local health officials quickly realized a potential linkage between the five deaths, and alerted medical officers at the Centers for Disease Control and Prevention (CDC) of a possible epidemic. Intensive research and testing by the CDC and other local medical agencies traced the disease to a previously unrecognized group of New World Hantaviruses (genus Hantavirus, family Bunyaviridae) (Schmaljohn and Hjelle 1997). Hantavirus infection is characterized by acute respiratory distress and associated symptoms including fever, chills, myalgias, and nausea (Warner 1996), with an overall case fatality rate of approximately 50% (Bryan et al. 1997).



**Figure 33.** Hantavirus pulmonary syndrome (HPS) cases in the Four Corners region. Source: Rachel Loehman, Sandia National Laboratories

Early cases of HPS were clustered in and around the Navajo Reservation in the Four Corners area of northwestern New Mexico and northeastern Arizona (see Figures 33 and 34). Although not all of the victims of HPS were American Indians, fear of disease resulted in prejudicial behavior, including prohibitions against serving American Indians in restaurants and stores, unfavorable media attention, and a reference to HPS as “Navajo Flu.” In reality, this clustering of HPS cases was the result of favorable conditions for human contact with the disease and its carrier. The specific viral agent responsible for the 1993 outbreak of HPS was found to be a previously undescribed Hantavirus, Sin Nombre Virus (SNV). SNV, like other Hantaviruses, is spread by rodents. Extensive testing of rodent species in the Four Corners region (convergence of New Mexico, Arizona, Utah, and Colorado) revealed the deer mouse, *Peromyscus maniculatus*, as the primary host for SNV. Sin Nombre Virus is transmitted to humans through inhalation of aerosolized rodent urine, feces, and saliva (Bryan et al. 1997). Activities such as gardening, hand plowing, and cleaning, especially in the rural and semirural areas where deer mouse populations are greatest, can result in increased risk of exposure to the Sin Nombre Virus. Conditions in the Four Corners region in the spring and summer of 1993 proved to be very favorable for an increase in rodent population density, bringing these carriers of HPS into close contact with human populations.



**Figure 34.** Study area and Indian reservations in the Four Corners region of northwestern New Mexico and northeastern Arizona. Source: Rachel Loehman, Sandia National Laboratories

One of the early breakthroughs in understanding the 1993 disease outbreak came when CDC Epidemiological Investigative Service (EIS) members conferred with Navajo elders, to ask for permission to perform autopsies on and conduct interviews about Navajo HPS victims. Although both practices violate traditional Navajo cultural and religious beliefs, the elders complied with the requests. In addition they provided information suggesting that HPS is not a new occurrence in the Southwest, or to American Indian populations. Elders informed the CDC that the Navajos had long ago matched periods of higher rainfall with increased piñon nut production, increases in rodent densities, and subsequent outbreaks of an HPS-like disease. Navajo traditions have historically supported practices that reduce contact with rodents and rodent feces and saliva, and reduce risk of infection. These include burning articles of clothing if a mouse has run over them, and removing all vegetation and other debris from around the hogan, or dwelling area. When Navajo elders were asked to recall earlier episodes of deaths from HPS-like symptoms, they named several dates, passed down as oral history, that coincide with recorded instances of higher than average precipitation. Specifically, these dates match with several of the recorded cycles of El Niño events.

### Potential Impacts of Climate Change

The El Niño Southern Oscillation (ENSO; commonly called El Niño) is a cyclical climatic phenomenon that results in a

large-scale weakening of the trade winds, warming of the surface layers of the tropical Pacific Ocean, and increased rainfall across the southern regions of the United States. El Niño events occur irregularly at intervals of 2 to 7 years. In 1991 and 1992 the Southwestern United States experienced warmer than average winters and atypically heavy rainfall as a result of ENSO. The effects of these environmental fluctuations were especially pronounced, because the Southwest had for the previous six years experienced drought conditions (Stephenson 1997). It has been proposed that the result of the El Niño phenomenon was to increase vegetation growth in the Four Corners region, causing a dramatic explosion in rodent populations in response to the greater availability of food resources. Specifically, rodent populations reacted to an abundance of piñon seeds in the winter season and grass seeds in the spring season (Yates 1997). The ENSO event amplified the risk of zoonotic (animal to human) transmission of HPS by bringing host populations into closer contact with humans. Estimates of the abundance of deer mice following the ENSO event suggest that rodent populations were 10 to 15 times higher than average (Glass et al. in press), and that the origins of the 1993 outbreak of HPS may be traced to this abundance (Zeitz et al. 1995).

### Coping Strategies

A total of 229 cases of HPS have been reported in the United States as of October 25, 1999. Almost half of the confirmed cases have been reported from areas within the Four Corners region (CDC data). Much of the area within this region is rural or semirural, placing inhabitants at significant risk for contact with rodents, and contraction of HPS. Rural health clinics, rather than full-service hospitals, provide health care for much of the region, and many people live significant distances from any source of medical treatment. Early treatment of HPS symptoms is critical because patients experiencing full distress are less likely to recover than patients treated earlier in the course of the disease (CDC data). One method for HPS prevention is to increase public awareness about the disease, its symptoms, and types of treatment. Another method, adopted by some researchers and institutions, is to examine potential “risk areas” for HPS by modeling the factors that influence the presence of Hantaviruses among rodent populations, and increase probability of zoonotic transmission. Delineation of local HPS risk areas is vital for identification of at-risk human populations. Medical professionals, once made aware of the locations of HPS risk areas, may use the information as a method of triage, or treatment prioritization.

### Future Information and Research Needs

The relationship between climatic variability and incidence of HPS can be examined through analysis of factors that may influence fluctuations in rodent populations, such as vegetation density, precipitation, temperature, and elevation. These data may be obtained from satellite-based remote sensors, which collect data at regular intervals over large areas. Other sources of data include ground-based precipitation and weather monitoring stations, global positioning systems, and vegetation surveys (Engelthaler et al. 1999).

In a recent analysis, the significance of vegetation density as a predictor of risk of HPS was tested in a probabilistic model. Time-series vegetation density data from within a study region in the immediate Four Corners area of the Southwestern United States were analyzed in a logistic regression model as potential predictor variables for HPS incidence. Parameters from the fitted logistic model were applied to a linear sum expression, which was used to determine estimated HPS risk across the study region. Estimated risk areas, created for twelve biweekly periods in 1993, were used to develop a possible explanation for the relationship between vegetation density and HPS incidence. Spatial variation in these risk areas across the study region suggests that risk of HPS is higher in areas where vegetation is more dense and green, and is minimized in sparsely vegetated areas. Variation in estimated risk areas through time may be attributed to changes in the distribution and density of vegetation, in response to cyclical or season climatic patterns. In general, risk of HPS is maximized during periods of intense vegetative green-up, when rodent populations are most abundant, and is minimized during periods of vegetative senescence.

The successful implementation of a predictive model for HPS risk areas provides a useful tool for monitoring and forecasting effects of climatic variability on human health. Such models can be expanded to fit a wider geographic region, and used as an early-warning system of potential disease outbreaks. The application of spatially and temporally relevant data to disease-related problems of risk assessment and change detection is relatively unexplored in epidemiological and biological research. Incorporation of such datasets into a predictive modeling context will aid in bringing better and more expedient treatment and prevention to potentially affected human populations, and increase our understanding of the interactions between climate, environment, and emerging diseases. ●