Human Health and the Environment

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Human Health and the Environment

Environmental conditions are relatively safe for North Americans: for the most part, the air is relatively safe to breathe, potable water is supplied to most of the population, and ample food of good quality is available. Many human-made chemicals used in agriculture, industry, and medicine have improved health and environmental quality. But the same beneficial substances may become pollutants when found in the wrong place, at the wrong time, and in the wrong quantity. Two opposite approaches have guided efforts to identify individual pollutants: one promoting the assumption that a substance is not harmful until evidence proves otherwise, and the other espousing precautionary consideration of a substance as potentially harmful until found otherwise (UN 1972).

In the past, the more obvious environmental health threats, such as asbestos, benzene, and formaldehyde, were investigated and their links to the environment understood. The fact that hundreds of children are poisoned each year by accidentally ingesting toxic household products was clear and therefore many substances were removed from use (Grier 2001). Early decision-making in North America banned some very hazardous substances–witness the EPA ban on the manufacture of polychlorinated biphenyls (PCBs) in 1977 (Kaplan and Morris 2000). By the late 1970s, risk assessment, which enabled continued use of toxic chemicals at scientifically 'acceptable' levels, was the generally recognized regulatory tool (Cooper, Vanderlinden, and others 2000).

More than 35,000 substances are currently in commercial use in Canada and over 62,000 in the United States. Some 1,500 new chemicals are introduced each year in the United States alone, but complete data on the health and environmental effects are only available for 7 percent of those produced in high volume (EC 1998a, Cooper, Vanderlinden, and others 2000). Many of these chemicals become pollutants when they are released to the air, water, and land,

and North Americans are exposed to countless kinds of them in their immediate environments.

Although the linkages between health and the environment are often difficult to determine, knowledge of the more subtle human health effects of environmental pollution is now coming to light. For example, as a result of risk assessments for many chemicals for a range of subpopulations over the years, it has become clear that children and other vulnerable populations are more susceptible than the average adult to the harmful health effects of most pollutants. (See Box 54). Children's environmental health is an issue of high priority in North America.

Air pollution is emerging as a key contributor to some respiratory and cardiovascular diseases that are impairing health and killing vulnerable people. About 80 million US citizens are exposed to levels of air pollution that can impair health, and more than 2 percent of all deaths annually in that country can be attributed to air pollution (WRI, UNEP, UNDP, and World Bank 1998). Air pollution is also linked to an alarming rise over the past two decades in the prevalence of asthma among children and young adults worldwide, mostly in affluent countries such as Canada and the United States. Ozone, a principal component of smog, is thought to exacerbate asthma symptoms. There is also clear evidence that acidic air pollutants affect the health of sensitive individuals, especially the young, the elderly, and those with respiratory ailments.

Another priority issue is the recent emergence or resurgence of some vector-borne disease. It is

Box 54: Children's Special Vulnerablility

Children are more susceptible than adults to the harmful health effects of most pollutants because of their developmental, physiological and behavioral characteristics. For instance, in some circumstances, children cannot excrete toxins as well as adults, and so are more vulnerable to them. The ratio of skin surface area to body weight is also higher in children than in adults, which makes it possible for an excessive portion of toxins to enter their bodies. And because their airways are narrower, children breathe more rapidly and inhale more air per kg of body weight. Children also drink more fluids and eat more food per unit of body weight compared to adults. In addition, crawling and a tendency to put their hands in their mouths provides more opportunities for chemical or heavy metal residues in dust, soil, and heavy vapors to be ingested or to contact the skin (EPA 1997; PMRA 2002; CEHN n.d.).

Children (like all people and animals) may be exposed to toxic or hazardous chemicals through chemically treated building materials, furnishings, appliances, cleaning products, lawn and farm chemicals, and other materials as well as some foods—such as fruits and vegetables—that may have minute residues of pesticides. Children may also consume chemical residues in breast milk and formula (CEC 2000a; Kaplan and Morris 2000; PMRA 2002). Pollutants in the air indoors and out—cigarette smoke and vehicle exhaust, for example—and the sun's damaging rays can also affect children more than adults.

believed that climate change and human-induced land use change are important factors in the emergence of diseases new to North America and the resurgence of some organisms that have heretofore been held in check by public health controls.

Children's Health and Environmental Contamination

Recent estimates suggest that one in every 200 US children suffer developmental or neurological deficits due to exposure to known toxic substances (NET, PSR, and NDAA 2000). Scientific understanding of the trend is still limited, especially since genetic, social, economic, and cultural factors affect the risk determinant and the statistics. For example, data bias can be related to better detection, inherited susceptibility, improved access to health care, medications, and the presence of allergies, among other factors. Economic and social elements are also significant variables. Poor and aboriginal children are more often at greater risk of environment-related health problems for a number of reasons (Alpert 1999; Cooper, Vanderlinden, and others 2000). With better understanding of children's environmental health, it has become clear that children are not just 'little adults' in terms of their susceptibility to the health effects of exposure to environmental contaminants (see Box 54). The story of lead illustrates how exposures and standards that were once thought to be safe require continued revision to protect children's health (see Box 55).

Exposure to a wide range of chemicals during pregnancy or after birth may also be implicated in neurodevelopmental effects in children. Exposure to PCBs has been tied to lack of coordination, diminished IQ, and poor memory in some children. It is estimated that problems such as autism, aggression, dyslexia, and attention deficit hyperactivity disorder affect one out of every six children in the United States, and increasing evidence suggests that environmental contaminants and nutrition are primary causative factors (CEC 2000a; Kaplan and Morris 2000).

Mounting evidence implicates exposure to pesticides in adverse health effects, including cancer, birth defects, reproductive harm, neurological and developmental toxicity, immunotoxicity, and disruption of the endocrine system (NRDC 1997; Cooper, Vanderlinden, and

Box 55: The Phase-out of Lead

In the 1970s, lead in gasoline, paints, solders, and cans was recognized as causing grave neurobehavioral health risks to children. Attention deficits, lower IQ scores, hyperactivity, and juvenile delinquency are among these potential effects (Cooper, Vanderlinden, and others 2000; CEHN n.d.). In response, mandatory childhood blood-lead testing was instituted in the late 1980s and early 1990s in the United States. A 1981 survey found that one in every 25 US preschool children and one of every five inner-city black preschool children had dangerous lead levels in their blood (Miller Jr. 1985). Lack of definitive proof of the harmful nature of lead in gasoline, abetted by commercial interests in the additive, sparked a lengthy debate while environmental contamination and the threats to children's health continued. But thanks to a strong lobby of health and children's advocates, lead additives to gasoline were banned in the late 1980s in the United States and in 1990 in Canada. The situation improved dramatically as lead emissions declined (see Figure 48), and as acceptable blood lead levels were revised downward in the early 1990s (EC 1996; Cooper, Vanderlinden, and others 2000; Cooper 2002). Today, the average gasoline lead content in North America is among the lowest in the world and the average blood lead level in North American children has declined steadily (UNEP, UNICEF, and UNITAR 1998).

Lead Emissions in the United States, 1970-1989, and Canda, 1970-1995

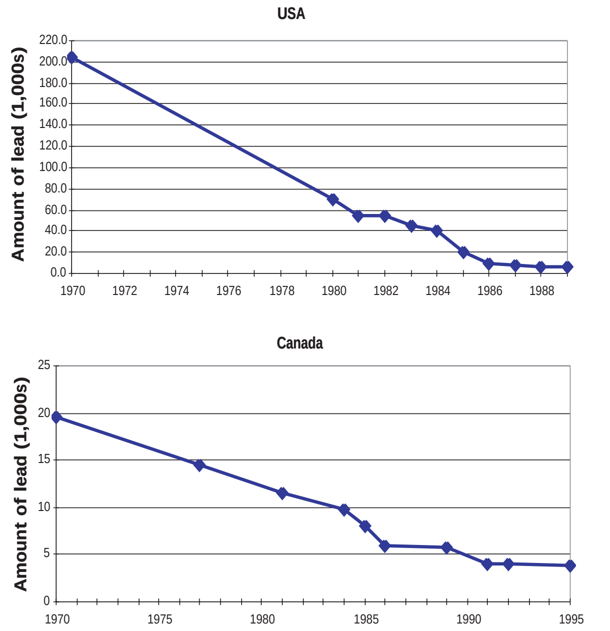

Figure 48 *Source: EC 1996*

Despite all this progress, mid-1990 estimates indicated that some 1.6 million North American children still had blood-lead levels in the range known to have health effects (GBPSR 1999; Cooper, Vanderlinden, and others 2000). Lead in paint is now considered to be the most common high-dose source of lead exposure for pre-school children in the United States. Children are more likely to ingest lead-containing paint chips and dust and so are particularly vulnerable to lead poisoning. Paint with levels higher than 600 parts per million (ppm) was banned in the United States in 1978, while the same low limit on lead-based exterior paint was introduced in Canada by voluntary agreement in 1991 (LDAC 2000). Thus, many older homes in North America still contain paint with levels of lead that are dangerous to children's health. Poor children are at greater risk since a larger percentage of low-income families live in older homes. It is now thought that there is likely no acceptable threshold for health effects in children from lead exposure (UNEP, UNICEF, and UNITAR 1998).

More recently, there is evidence that children are exposed to new and unexpected sources of lead, like that found in some cheap jewellery, candles with lead wicks and PVC (polyvinyl chloride) mini-blinds. So the risk assessment for this toxic metal remains crucial (Cooper 2002). From the lessons learned with lead, the North American governments are now supporting more rigorous research into childhood risks from environmental contaminants, are issuing warnings and advisories, and implementing stronger standards.

others 2000). Pesticides are used on the farm and elsewhere to control indoor and yard pests where children are present. When pesticides run off fields into waterways or groundwater or become airborne, drinking water can be contaminated. Pesticides can be ingested in food, and most fruit and fruit juices, as well as breast milk.(NRDC 1997; NRDC 1998).

Another environmental risk to children is air pollution, both outdoor and indoor, which is among the most significant triggers for asthma symptoms. Childhood asthma is on the rise, affecting over 5 and a half million children in North America and accounting for more than 60 percent of all their hospital visits (PSR 1997; Health Canada 1998). In Canada, childhood asthma increased fourfold between 1978-79 and 1994-95, with environmental change a possible contributor to the increase (Kidder, Stein, and Fraser 2000). Asthma is now the number one childhood illness in the United States (PEHC 2000). Scientific understanding of the trend is still limited and must account for the influence of hereditary characteristics, social and cultural variables, and the fact that medications for treating the illness have greatly improved (Alpert 1999).

The links between air pollution and human health in general also require more study. On average, air quality has improved in North America, although it remains true that more than half the US population lives in areas that exceed federal standards for many pollutants. These include those associated with asthma such as ozone, sulphur dioxide, and particulates (PSR 1997). Almost every major Canadian city exceeded the nation's stringent ozone guidelines at least once between 1984 and 1991. Researchers now conclude that even ozone levels below federal standards (see the atmosphere section) cause ill effects that contribute to asthma (PSR 1997). There is also clear evidence that acidic air pollutants affect children's cardio-respiratory health (Burnett, Dales, and others 1995).

With the increase in asthma prevalence and concerns over the links to environmental pollutants, more research money is being dedicated to studying children's health, particularly to what is being called a childhood asthma epidemic (PEHC 2000).

In recent years, there has been a dramatic surge in the recognition of children's environmental health issues, and as a result stricter regulations have been introduced that take children's vulnerabilities into account. In the mid-1980s, the EPA began to consider developmental risks in its assessment protocol (Cooper, Vanderlinden, and others 2000). A key event was a 1993 National Academy of Science study showing that pesticide regulation did not allow for the special risks faced by children. It prompted the 1996 US Food Quality Protection Act (FQPA) to apply a 10-fold child-protective safety factor as well as requirements to aggregate chemical exposures and to assess groups of chemicals with common mechanisms of toxicity. It also stipulated the review of some 9,000 pesticides to determine whether they pose a health risk for children. The FQPA sets standards for exposure to hazardous substances through risk assessment, a practice that has evolved over the years from focusing disproportionately on adult cancer, to considering developmental risks, including those that affect children (EPA 1999; Cooper 2002).

In Canada, the Pest Management Regulatory Agency's (PMRA) consideration of the increased sensitivity to a pesticide experienced by the young and other vulnerable populations, as well as the exposure of infants, children, and pregnant women, is consistent with FQPA practice. Also in keeping with the EPA, it uses a case-specific determination regarding the size of the additional safety factor where reliable scientific data are available. Canada's Food and Drugs Act now also takes into account the dietary patterns and special needs of children (PMRA 2002).

Both federal governments recently initiated special bodies or actions to deal with the issue of children's health. In 1996, the EPA adopted a National Agenda to Protect Children's Health From Environmental Threats. In 1997, the Office of Children's Health Protection (OCHP) and the President's Task Force on Environmental Health Risks and Safety Risks in Children were formed to identify, assess, and address children's disproportionate environmental health and safety risks. In April 1998, the Task Force identified childhood asthma, accidental injury, developmental disorders, and childhood cancer as priority areas for immediate attention (EPA 2001). Beginning in 1997, Health Canada strove to strengthen departmental and interdepartmental linkages among legislation, policies, and programs aimed at children and contributed to the 1997 National Children's Agenda, which includes the need for safe and secure environments and communities for healthy childhood development (Health Canada 1999).

Canada and the United States are party to the 1989 UN Convention on the Rights of the Child and also support Agenda 21, which acknowledges the dangers and risks of environmental pollution to children. Agenda 21 urges governments to develop programs to protect children from the effects of environmental and occupational toxic compounds. In addition, the Declaration of the Environment Leaders of the Eight on Children's Environmental Health (G7 countries plus Russia 1997) acknowledges that current levels of protection may not provide children with adequate protection and provides a framework for national, bilateral, and international efforts to improve the protection of children's health from environmental threats such as pesticides (CEC 2000a). And at the Health and Environment Ministers

Box 56: Bilateral Cooperation

Canada and the United States, together with Mexico, are working cooperatively in the field of children's environmental health. In 2000, the Commission for Environmental Cooperation for North America's (CEC) Council of Ministers resolved to apply the perspective of children's health to their work, including increasing information exchange and public awareness and education about environmental threats to children's health and how to prevent them. Building on the outcomes of its 2000 Symposium on Children's Health and the Environment in North America, the CEC Council called for cooperative efforts to address asthma and other respiratory diseases, and the effects of lead and other toxic substances as a matter of priority. With the involvement of health and environment officials from the three countries, an Expert Advisory Board and members of the public, the CEC has developed a Cooperative Agenda for Children's Health and the Environment in North America which will serve as the blueprint for trilateral action on issues of common concern (CEC 2000a; CEC 2000b).

The Canadian PMRA and the US EPA also work cooperatively under the umbrella of the NAFTA Technical Working Group on Pesticides to provide expert guidance to other scientists generating data to refine residential exposure estimates for infants and children (PMRA 2002). In addition, 10 US Pediatric Environmental Health Specialty Units, forming a network joined by one Canadian counterpart, now receive US public funding. US researchers are currently initiating a pilot Longitudinal Cohort Study to track environmental exposures and health effects on children (Cooper 2002).

of the Americas meeting in 2002, it was agreed to develop a set of indicators for children's health and the environment (EC 2002). The two countries also work together on a number of joint projects (see Box 56).

At the grassroots, people are invoking 'The Right to Know' and local initiatives are being taken to protect children's health in many communities. In both countries, a strong preventive and precautionary approach to risk assessment is slow to emerge, however. People still tend to require positive evidence of health and exposure risks before taking action (Cooper 2002). Governments are under increasing pressure from scientists, pediatricians, academics, and parents for more rigorous tests for neurotoxic effects, prenatal and early postnatal exposures, and cumulative health effects from exposure to environmental contaminants, as well as regulations for shifting onus from the public to the chemical manufacturers by requiring the latter to prove that their products are safe instead of leaving it to governments to report on their toxicity (Cooper, Vanderlinden, and others 2000; Kaplan and Morris 2000).

Emergence/Resurgence of Vector–borne Diseases

Vector-borne diseases—those that can infect humans through transmission by insects or ticks, or by direct contact with host animals or their body excretions (see Box 57)—were once common in the southern United States. By 1972, public health policies and the effective use of pesticides had significantly reduced the threat of diseases like malaria and dengue fever. In the last 20 years, however, new vector-borne infectious diseases are emerging and some old ones resurging (Gubler 1998a; Gubler 1998b).

Natural or human-caused environmental change affecting any segment of the vector's natural cycle might lead to the emergence or re-emergence of infectious diseases. This can take place when other species introduce existing pathogens into human populations or pathogens are further disseminated from smaller to larger populations. Ecological or environmental changes; population movements; changes in industrial processes, economic development, land use, and international travel and commerce as well as breakdowns in public health measures can create conditions that encourage the process. Natural environmental events, such as weather changes, can especially affect vector-borne diseases. Local

Box 57: Vector-borne Disease

A vector is an organism that spreads an infectious disease by amplifying and transmitting it, often without becoming ill itself. Anthropods such as mosquitoes and ticks, or mollusks such as freshwater snails typically harbor the infectious agents at some part of their life cycle. Hundreds of viruses, bacteria, protozoa, and helminths (parasitic worms) rely on such blood-sucking arthropods to be transmitted between vertebrate hosts. Yellow Fever, Dengue Fever, Lyme disease, Arboviral encephalitis, and West Nile Virus are examples of vector-borne infectious diseases (Gubler 1998a; NIAID 2001).

bio-geographical conditions, immunological history, and genetic change in pathogens are yet other factors that can influence infectious disease emergence (Epstein 1998; CDC 2000a; NIAID 2001).

Some scientists suggest that certain vectors may be expanding their geographic ranges into previously inhospitable areas in response to climate change. The International Panel on Climate Change (IPCC) reports that "changing climate conditions may lead to the northward spread of vector-borne infectious diseases and potentially enhanced transmission dynamics as a result of warmer ambient temperatures" (Watson, Zinyowara, and others 1997).

Climate change and human-induced land use change may also change predator-prey relationships, increasing the numbers of disease-carrying pests and human contact with them (EC 1998b). For example, there appears to be a relationship between the expansion of suburbs and reforestation, the abundance of deer and deer ticks, and a rise in the numbers of reported tick bites and infections of Lyme disease in the eastern United States (CDC 2001; NIAID 2001) (see Box 58).

Climate limits the distribution of vector-borne diseases, while the timing and intensity of outbreaks are affected by weather. With increases in average temperatures, studies indicate the potential for mosquito-borne encephalitis to move into northern United States and Canada (EC 1998b; Epstein, Diaz, and others 1998). Indeed, since 1990, the beginning of the hottest decade in historical record, locally transmitted malaria outbreaks—although limited to only a few cases—have occurred in numerous states during hot spells (Gubler 1998a). Warmer weather may also have influenced the occurrence of West Nile Virus in North America (see Box 59).

Climate variability is only part of a complex mix of factors that explain the emergence/resurgence of vector-borne diseases, and transmission depends on factors that are unique for each pathogen. Increased international trade can lead to the introduction of a pathogen to new populations or to an expansion in the range of vectors. The tiger mosquito *(Aedes albopictus)*, for example, was introduced to the United States in 1986 in used tires imported from Asia. It is capable of

Box 58: Lyme Disease

Lyme disease is a bacterial tick-borne infection. The *Ixodes dammini* tick, found in grassy, brushy, shrubby, and wooded areas of the northeastern United States, is responsible for most of the cases of Lyme disease. The ticks feed on a variety of warm-blooded animals including humans and domestic animals. The tick's life cycle has three stages, each of which requires separate hosts. During the nymphal stage, the tick prefers the white-footed mouse, the main reservoir of the Lyme disease bacteria, while the adult ticks prefer to feed on white-tailed deer (Miller n.d.).

Lyme disease is the leading cause of vector-borne infectious illness in the United States, with about 15,000 cases annually, although many are unreported (CDC 2001). It occurs mostly in the northeastern United States, the upper Midwest, and northern California but may also extend northward as far as Canada with climate change (EC 1998b).

Reported Cases of Lyme Disease in the United States, 1982-1997

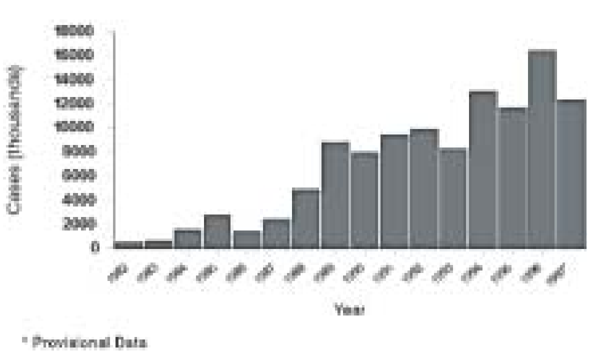

Figure 49 *Source: Gubler 1998a*

National surveillance for Lyme disease started in 1982 and since then, incidence of the disease has increased (see Figure 49) as has its geographic distribution (Gubler 1998a). Managing deer populations where Lyme disease occurs may reduce tick abundance (CDC 2001).

Box 59: West Nile Virus

The unanticipated first-time appearance of the West Nile Virus (WNV) in New York State in 1999 aroused concern over the potential for new viruses to penetrate prosperous northern climes. Mosquitoes that have fed on the blood of infected birds transmit the virus. Most people who contract the virus show no symptoms or only mild flu-like symptoms, but in rare cases the virus can cause meningitis or encephalitis, usually affecting vulnerable people such as young children, the elderly, and those with suppressed immune systems (Health Canada 2001). Of the 62 human cases of clinical illness resulting from infection with WNV in the United States in 1999, seven were fatal. Where extensive mosquito controls and risk-reduction campaigns were undertaken, no new cases occurred among humans (CDC 2000b). Its reappearance in four northeastern states in 2000, however, was evidence that it had over-wintered and transmission had increased (Brown 2000). In 2000, there were 21 human cases and two deaths (USDA 2000). When the temperature falls below 13˚C, the mosquitoes become dormant and the virus stops spreading. But it can spread once more in the spring when the mosquitoes are active and birds are migrating (Simao 2001).

WNV was first isolated in Uganda in 1937 and outbreaks have occurred in several countries outside North America, but it is not yet known how it was introduced into the United States (Health Canada 2001). It is thought that a sequence of warm winters, followed by hot, dry summers—a pattern predicted by climate change models for some regions—favors the transmission of this type of virus. To counteract increases in this and other vector-borne diseases, improved disease surveillance and prevention strategies at state and local levels will be needed as well as more research into climate-health relationships (Epstein 2000; USGCRP 2000).

transmitting viruses responsible for dengue fever and several forms of encephalitis (NIAID 2001).

Since the 1970s and the near elimination in North America of malaria and other diseases that infect humans through host carriers of pathogens, the public health infrastructure required to deal with them and prevention, surveillance, control, and training programs were discontinued or merged with other programs as less need for them was perceived (Gubler 1998a). Now, however, the need to reinvest in these measures has become evident. With the resurgence of Hantavirus pulmonary syndrome in 1993, for example (see Box 60, next page), control measures were stepped up, and although climate conditions were subsequently ripe for more outbreaks, episodes have been limited. Early-warning systems increased public awareness and prompted people to rodent-proof homes, set traps, and avoid the rodents' droppings (Bonn 1998; Epstein 2000).

The level of socioeconomic development is another significant factor in preventing vector-borne diseases. Between 1980 and 1996, for example, 43 cases of dengue fever, a mosquito-borne virus, were reported in Texas, versus 50,333 in the three adjacent Mexican states, providing clear evidence that where people prosper, they are able to take preventive measures—in this case, air conditioning and window screens were key factors—and to devote resources to public health and surveillance measures (Manning 2000; USGCRP 2000).

Better control and surveillance strategies alone may be able to counteract potential increases in vector-borne pathogens in North America (Gubler 1998a). Control strategies, however, often involve the use of pesticides, which have been called a 'double-edged sword' in view of human health problems attributed to pesticide contamination of water and food (see the land section). Increasing insect resistance is also compromising the efficacy of pesticides (Epstein, Diaz, and others 1998). Thus, more research on environmentally safe insecticides, vaccines, and alternative approaches to vector control are also crucial (Gubler 1998a). At the same time, given that climate change may have a significant and increasing role in this trend, it is also important to find solutions to the human causes of recent and future changes in climate.

Box 60: Hantavirus Pulmonary Syndrome

The example of the resurgence of Hantavirus pulmonary syndrome (HPS) in the United States illustrates the effect of 1990s climatic variability in the outbreak of vector-borne disease. Rodent species bearing HPS live throughout the United States and cases of the syndrome have occurred nationwide. It is carried primarily by the deer mouse and can be transmitted to humans when they inhale infected air from the rodents' saliva or excreta (CDC 1999). HPS is a viral disease characterized by severe pulmonary illness, with a case fatality rate of about 50 percent (WHO 1996). In the early 1990s, the population of white-footed mice in the southwest United States increased 10-fold. A combination of prolonged drought that killed its predators and subsequent heavy rains, which expanded the rodent's food supply, conspired to spur population increase and allowed the inactive or restricted virus to explode (WHO 1996; Epstein 2000). Only 191 cases have been reported since its first reappearance in 1993, but the virus is considered by some to be an important public health concern (Bonn 1998).

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