PAPER

2

THE EFFECT OF CLIMATE ON THE WEST NILE VIRUS IN ONTARIO

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ABSTRACT: As of 2002, the West Nile virus has spread to and throughout Ontario leading to one human death in the late summer. It is not known how the virus entered Ontario – whether it was an infected bird (imported, migratory or overwintering), mosquito, human or other vertebrate host. The West Nile virus is spread when infected birds that have high levels of West Nile virus in their blood are bitten by mosquitoes. The infected mosquitoes can then transmit the West Nile virus to humans or other animals. In North America, the West Nile virus cycles through 3 species of mosquitoes described as initiator, amplifier and bridger with the *Culex restuans, Culex pipiens* and *Culex salinararius* playing each role respectively. Wild birds are the principal hosts of the West Nile virus, especially the American crow which also plays an important role in signaling the epi-centre of the virus outbreak two weeks prior to peak exposures, and the onset of severe symptoms in humans. The West Nile virus fever in humans usually is an influenza-like illness, but occasionally, the more severe symptoms of meningitis or encephalitis occur. Studies have shown that only 20 percent of all humans (1 in 5) infected with the West Nile virus exhibit adverse effects. Temperature and other climate factors can be implicated in the spread and severity of the West Nile virus across North America, yet the range of influence that climatic factors play is not entirely known. Under future climate change scenarios, climatic conditions conducive to the spread and severity of the West Nile virus will increase. Recommended management options include monitoring the spread of infection; reducing human exposure to infected vectors; preventing initiation and magnification of the virus; screening blood supplies and other products capable of spreading the virus; and conducting public information campaigns.

Keywords: West Nile virus; climate; Ontario; vector borne diseases; *Culex*; mosquito; crow;

1. Introduction

In the summer of 1999, the West Nile virus was recognized in the western hemisphere for the first time when it caused an epidemic of encephalitis and meningitis in the metropolitan area of New York City, NY, USA. Intensive hospital-based surveillance identified 59 cases of West Nile virus, including 7 deaths in the region (Mostashari *et al.*, 2001). The virus has spread quickly across North America - south to the southern US states of Florida, Georgia and Louisiana, and north into Canada including the provinces of Ontario, Quebec, Manitoba and Saskatchewan. By the late summer of 2002, Ontario, Canada recorded its first human death attributed to the West Nile virus.

It has been proposed that temperature and other climate factors can be implicated in the spread and severity of the West Nile virus across North America

(Chiotti *et al.*, 2002). Hot, dry summers may promote outbreaks of the West Nile virus in humans. For example, the July 1999 temperatures in New York City were among the highest on record (59 cases of West Nile virus), while 2000 was comparatively cool (21 cases). This paper will examine the link between climate variables and the spread and severity of the West Nile virus across Ontario. Specifically, the paper will trace the spread of the West Nile virus across Ontario in spatial and temporal terms; provide a background to the West Nile Virus; associate climate variables to the spread and severity of the West Nile virus in Ontario under current climate conditions and future scenarios of climate change; and provide management options of the West Nile Virus risk.

2. The Spread of the West Nile Virus in Ontario 2000-2002

Figures 1 through 6 provide a series of maps detailing the spread of the West Nile virus into Ontario from the year 2000 to present. There was no West Nile virus recorded in 2000 for the dead birds that were tested that year, a number totaling 2,288. By 2001, the percentage of infected dead birds tested reached high levels in Toronto, Peel and Windsor-Essex Health Region (as defined by Health Canada). One year later in 2002, the West Nile virus appeared in infected dead birds tested across all of Ontario except the Sudbury Health Region. The highest percentages of infected dead birds tested appears to have moved to the north shore of Lake Erie and east to the Kingston Health Region. Yet, Toronto remains the so-called "epi-centre"[1] for the West Nile virus in Ontario when the other maps are examined. The percentage of infected mosquito pools tested in 2002 is highest in Toronto. The highest number of horses found infected with the West Nile virus in 2002 centres in the Toronto Health Region. And the highest number of confirmed human cases infected with the West Nile virus in 2002 is in the Toronto Health Region, with Canada's first confirmed fatality from the virus occuring in the Peel Health Region immediately west of Toronto. The pace of spread of the West Nile virus in Ontario is surprising and has been called "unprecedented" (McLean, 2002). But what are the conditions that led to such an unprecedented spread of this virus across North America including Canada and Ontario?

1 An epicentre is employed as a term to describe the place where the West Nile virus initiated. Usually, the term epicentre refers to the point on the Earth's surface directly above an earthquake or atomic explosion.

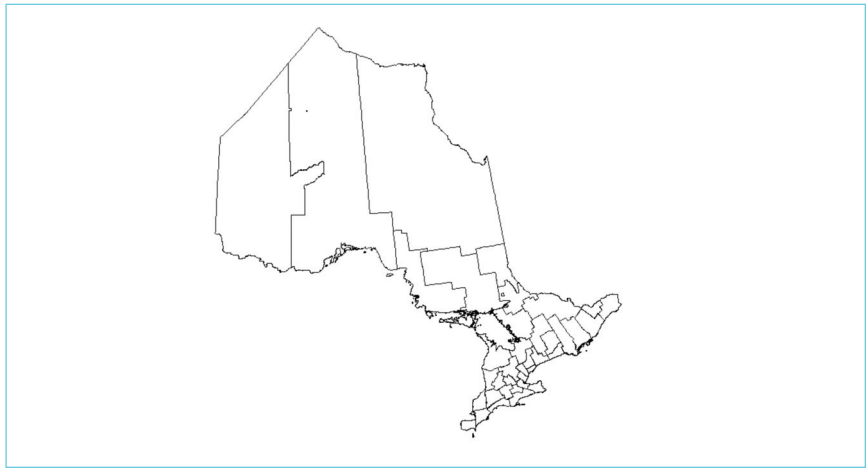

FIGURE 1
West Nile Virus in Ontario infected dead birds, 2000.
Source: Data from Health Canada, 2002.

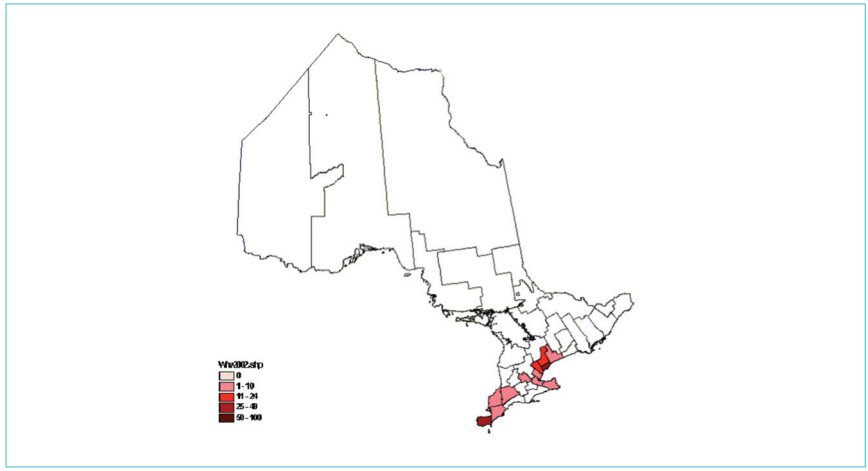

FIGURE 2
West Nile Virus in Ontario infected dead birds, 2001.
Source: Data from Health Canada, 2002.

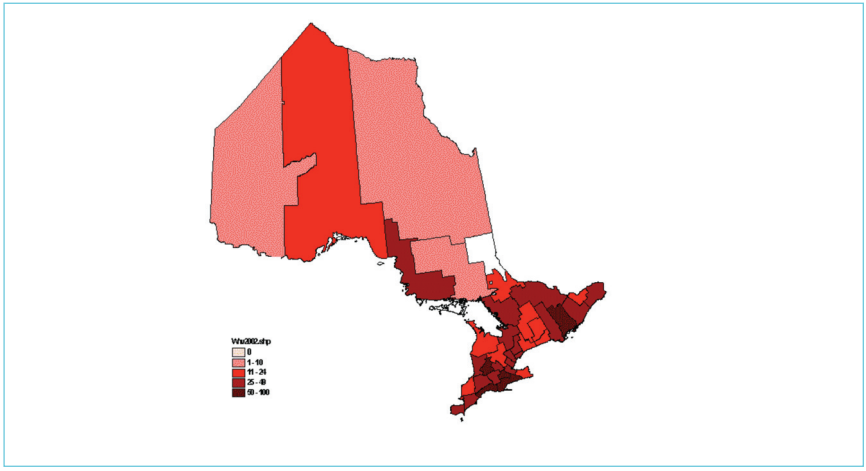

FIGURE 3
West Nile Virus in Ontario infected dead birds, 2002.
Source: Data from Health Canada, 2002.

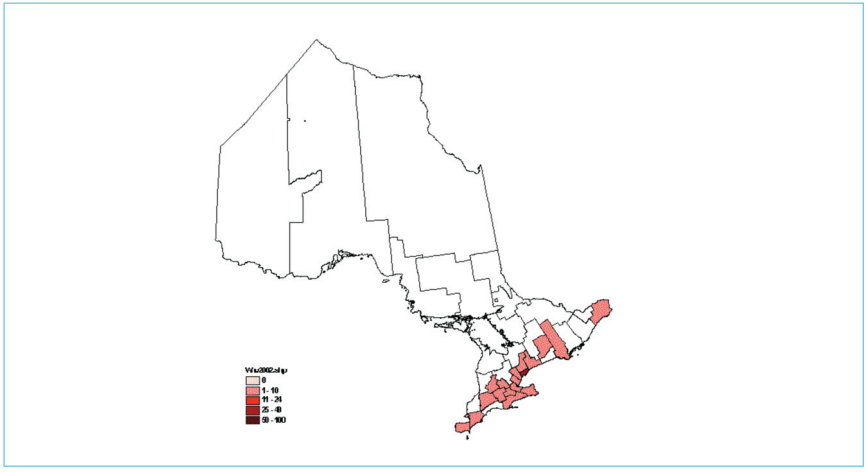

FIGURE 4
West Nile Virus in Ontario infected mosquito pools, 2002.
Source: Data from Health Canada, 2002.

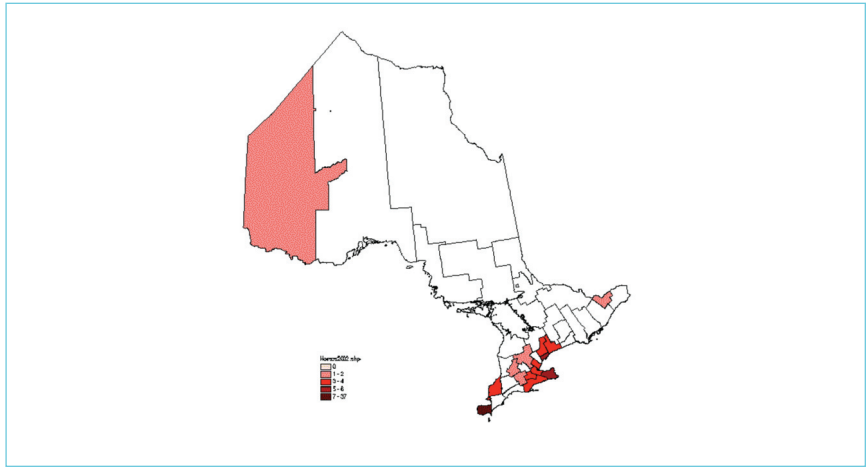

FIGURE 5
West Nile Virus in Ontario infected horses, 2002.
Source: Data from Health Canada, 2002.

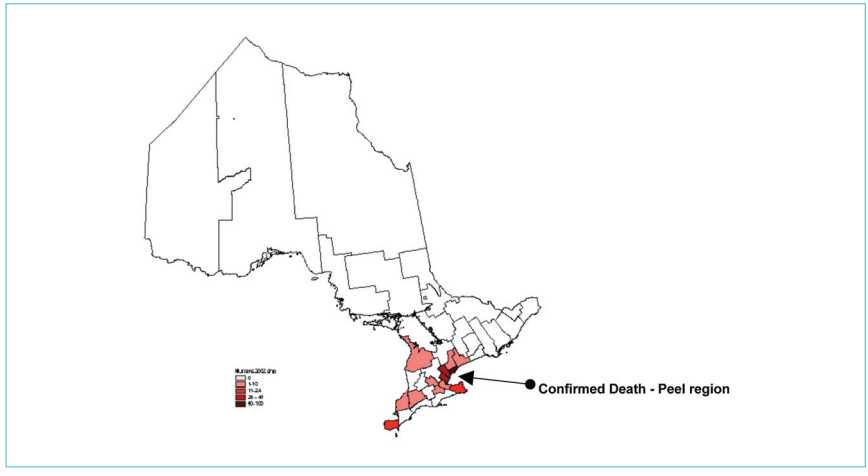

FIGURE 6
West Nile Virus in Ontario infected humans, 2002.
Source: Data from Health Canada, 2002.

3. Background

The West Nile virus, a member of the Japanese encephalitis virus serogroup in the family Falviviridae (genus *Flavivirus*) (Dohm and Turell, 2000) was first isolated in 1937 in the West Nile district of Uganda (Smithburn *et al.*, 1940). Since the original isolation of the West Nile virus, outbreaks have occurred infrequently in humans, those in Israel (1951-54 and 1957) and South Africa (1974) being the most notable (Petersen and Roehrig, 2001). Since the mid-1990s, however, there have been an increase in the frequency of outbreaks of the West Nile virus in humans and horses (Romania 1996; Morocco 1996; Tunisia 1997; Italy 1998; Russia and Israel 1999; and Israel and France 2000).

It is not known how the virus entered North America – whether it was an infected bird (imported or migratory), mosquito, human or other vertebrate host. The literature supports the possibility of all paths of entry mentioned above (Rappole, 2000). Invasive mosquito species have been recorded to arrive in countries via the water trapped in the well of recycled tires (see *Aedes japonicus* arrival in New Zealand) and spread by physcially moving along highways (Womack, 2001). It has been suggested that the West Nile virus was purposefully released in North America by the US military in order to support the sale of a West Nile virus vaccine by the pharmaceutical company, OraVax. The connection is that the military granted OraVax the license for the vacine, and that OraVax Vice President is Col. Monath, a former Ft. Detrick biowar researcher (Jannaccio, 2000). This theory is discounted by these authors. The West Nile virus responsible for the outbreak in New York City in 1999 is a close genetic relative of a virus circulating in Israel from 1997 to 2000 (Giladi *et al.*, 2001) which may help in eventually determining the path of entry.

The West Nile virus is spread when infected birds that have high levels of West Nile virus in their blood are bitten by mosquitoes. The infected mosquitoes can then transmit the West Nile virus to humans or other animals. The West Nile virus is not transmitted directly from human to human. There is no evidence that a person or other animals can be infected from handling infected birds (Toronto Public Health, 2001).

2 Note that the species *Aedes japonicus* needs to be researched further because of its characteristics of being a new species discovered in North America in 1998, the first new mosquito species in a generation. It was found in the states of New York, New Jersey and Connecticut where the West Nile virus first emerged. Yale scientists (Brigockas, 1999) predicted before the New York outbreak of 1999 the potential for this species to act as a bridge vector due to its willingness to feed on both humans and birds. The species is unique for its daytime feedings and is also "highly susceptible" to infection from the West Nile virus. Very little research has been conducted on this species in North America.

Mosquitoes, largely bird-feeding species, are the principal vectors of the West Nile virus (Hubalek and Halouzka, 1999). The virus has been isolated from 43 mosquito species, predominantly of the genus Culex and Aedes. In North America, three species of the *Culex* family – *restuans, pipiens, and salinarius* - are the assumed predominant carriers of the West Nile virus.[2] *Culex pipiens* appears to be the species most responsible for the spread of the West Nile virus in Europe (Rappole, 2000), and is now implicated in the North American outbreak (Bernard *et al.,* 2001). The West Nile virus in Europe circulates in both sylvan (forest) and urban transmission cycles involving different species and populations of mosquitoes (Savage, 1999). In North America, this cycle has been described by Andreadis *et al.* (2001) as follows: *Culex restuans* initiates the West Nile virus transmission among birds in early summer; *Culex pipiens* amplifies the virus later in the season; and *Culex salinararius* is the suspected "bridge vector" of the West Nile virus from birds to humans, horses and other mammals (Andreadis *et al.,* 2001; Bernard *et al.,* 2001). Figure 7 graphically represents the transmission dynamics of the West Nile virus from mosquitoes to mammals. Table 1 provides some results of different studies quantifying the rate of mosquito infection, rate of transmission to progeny, the transmission efficiency and the effect of ambient temperatures on infection rates.

TABLE 1

Transmission of the West Nile Virus by Mosquitoes

Rate of Mosquito infection with WNV	3.53 Minimal Infection Rate (MIR) per 000 mosquitoes in *Culex* species	Turrell *et al,* 2000.
Rate of WNV transmission to progeny	1 in 1,618 females of *Culex* species, varies by temperature	Turrell *et al,* 2000.
Mosquito WNV transmission efficiency to humans	"Nearly all" infected mosquitoes transmit WNV successfully by bite	Turrell *et al,* 2000.
Effect of ambient temperatures on mosquito infection rates	30 degrees Celsius leads to greater than 90 percent of all mosquitoes containing infection after 12 days; 18 degrees Celsius less than 30 percent contained infection after 28 days	Dohm *et al,* 2001.

Studies have shown that environmental temperature increases the ability of mosquitoes to transmit the West Nile virus (Dohm and Turell, 2000; Dohm *et al,* 2001; Nasci, 2001). Results suggest that infection rates are related directly to subsequent incubation temperatures – 30 degrees Celsius leading to greater than 90 percent of all mosquitoes containing infection after 12 days; while 18 degrees Celsius less than 30 percent contained infection after 28 days. Studies also conclude

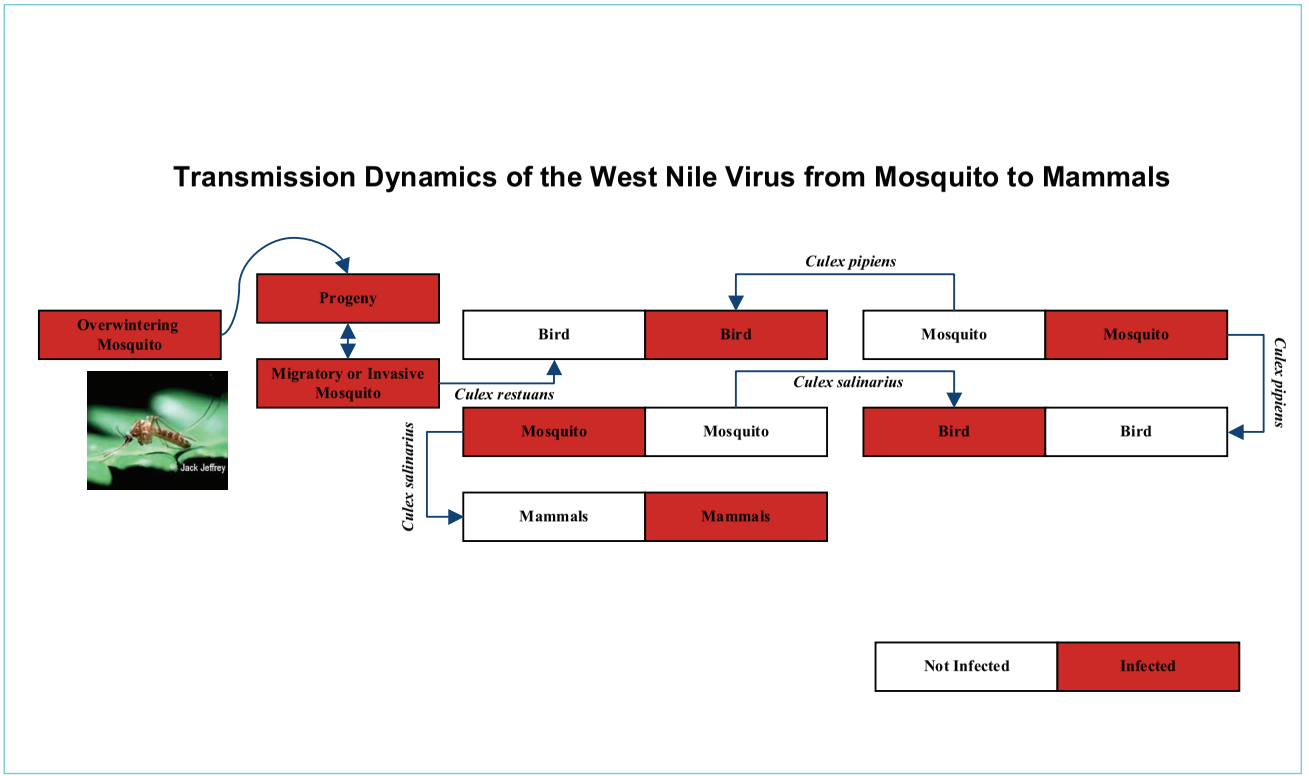

FIGURE 7
Transmission Dynamics of the West Nile Virus from Mosquito to Mammals.

that the West Nile virus can persist in vector mosquitoes at least through midwinter, suggesting that the virus would persist until spring and emerge with mosquitoes to reestablish a transmission cycle in the infected area (Nasci *et al.*, 2001).

Wild birds are the principal hosts of the West Nile virus. The virus has been isolated from a number of wetland and terrestrial bird species of North America (see Table 2). The virus persists in the organs of ducks and pigeons for 20 to 100 days (Semenov *et al.*, 1973). Migratory birds are thus instrumental in the introduction of the virus to temperate areas during migrations (Hubalek and Halouzka, 1999). Eurasian bird populations of several species in which exposure to the West Nile virus has been detected are rare migrants along the eastern seaboard of North America (Rapple *et al.*, 2000). Figure 8 graphically represents the transmission dynamics of the West Nile virus from birds to mammals.

TABLE 2

Bird Hosts of the West Nile Virus in Canada, 2001

American Crow (67%)	Fish Crow (40%)
Blue Jay (40%)	Cooper's Hawk (27%)
American Robin (9%)	House Sparrow (8%)
European Sparling (7%)	Common Grackle (7%)

Percentage of dead birds tested with positive identification for the West Nile virus in Canada

The susceptibility of crows (*Corvus brachyrhynchos*) to infection and death from the West Nile virus is a sensitive surveillance tool that is unique to North America (Komar, 2000). The percentage of crows infected by the West Nile virus was highest in the epicentre of the virus outbreak in New York City in both 1999 and 2000 supporting the importance of crows as indicators of the spread of the virus. Figure 9 shows how, in 2000, the appearance of dead crows forewarned the impending infections in humans two weeks hence. Also, data from the US Geological Survey's National Wildlife Health Center indicate that crows infected with the West Nile virus are likely to be sedentary approximately 4 days before death, suggesting that they can assist *Culex pipiens* in "amplifying" the West Nile virus in areas where crows are found (National Wildlife Health Center, 2000). The range of the American crow and its migrations can be found in Figure 10 with many overwintering in southern Ontario (Environment Canada, 2002a).

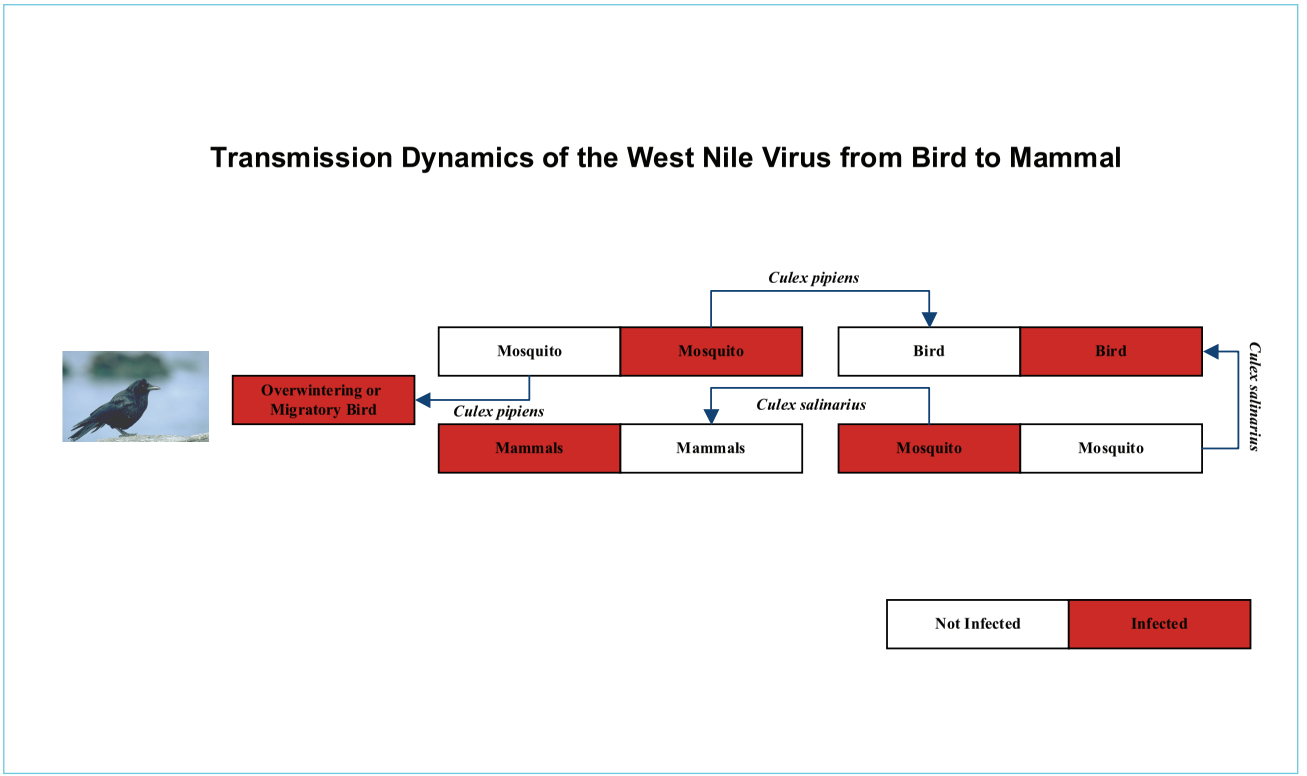

FIGURE 8
Transmission Dynamics of the West Nile Virus from Bird to Mammal.

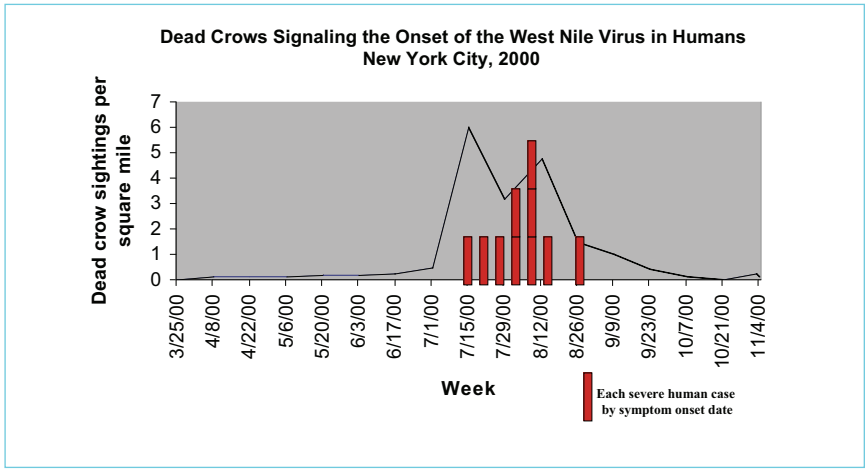

FIGURE 9
Dead Crows Signaling the Onset of the West Nile Virus in Humans, New York City 2000.
Source: Data from Eidson *et al.*, 2001.

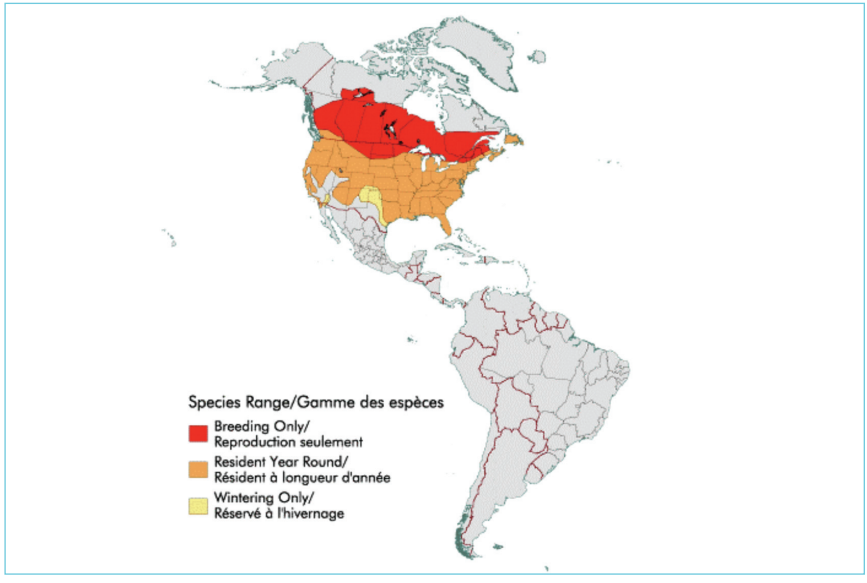

FIGURE 10
Migratory Range of the American Crow, *Corvus brachyrhynchos.*
Source: Environment Canada.

4. Human Exposure to the West Nile Virus

Humans are exposed to the West Nile virus from the bite of a mosquito infected with the virus. Studies of the outbreak of the West Nile virus in New York City in 1999 reveal some interesting results (Mostashari *et al.*, 2001) In the outbreak's epicentre, it was found that 2.6 percent of the total human population (>1200 individuals) was infected by the West Nile virus. The highest infection rates were found among individuals who spent more than 2 hours outdoors after dusk or before dawn, the peak feeding periods of *Culex* mosquitoes. Infection rates were higher for those individuals who did not use insect repellants. However, there were also high infection rates among a group of individuals who spent little or no time outdoors. Having seen a dead bird in one's neighbourhood was independently associated with higher infection rates as well. It is interesting to note that nearly all of the homes surveyed had screens on the windows (96%) and air-conditioners (92%). Other studies in temperate northern hemispheric climates showed human infection rates of up to 4 percent (Tsai, 1998; Platonov, 1999).

Studies have shown that only 20 percent of all humans (1 in 5) infected with the West Nile virus exhibit adverse effects (Mostashari *et al.*, 2001). The West Nile virus fever in humans usually is an influenza-like illness characterized by an abrupt onset (incubation period is 3 to 6 days) of moderate to high fever (3 to 5 days, sometimes with chills), headache, sore throat, backache, fatigue, diarrhea, and respiratory symptoms (Peiris and Amerasinghe, 1994). Occasionally, the more severe symptoms of meningitis or encephalitis occur causing disorientation, muscle weakness, coma and paralysis. This occurs in 3 percent of those adversely affected by the virus (1 in 30) (Mostashari *et al.*, 2001). Most fatal cases have been recorded in patients older than 50 years, and there is no mention in the literature to the susceptibility of children or immune-deficient adults to infection. Figure 11 shows the rate of illness from the West Nile virus among the human population of the New York Metropolitan Area in 1999.

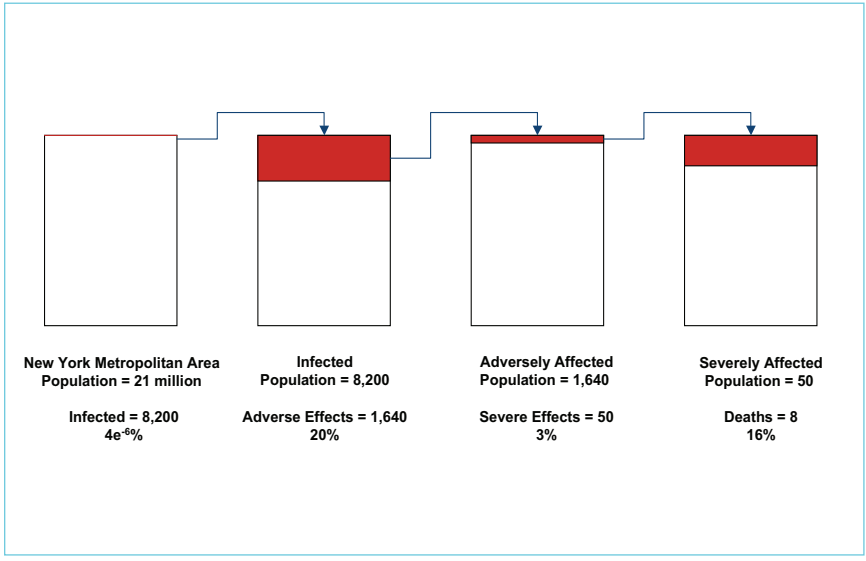

FIGURE 11
Rates of Illness in Humans, New York Metropolitan Area, 1999.
Source: data from Montashari *et al.*, 2001.

5. Linking the West Nile Virus to Climate Variables

For ten years now, scientists have understood that scenarios of global climate change hypothesize warmer, more humid weather that may produce an increase in the distribution and abundance of mosquito vectors (Reeves *et al.*, 1994) that cause the West Nile virus. Chiotti *et al.* (2002) recognized that the July 1999 temperature in New York City was among the highest on record with 59 cases of the West Nile virus recorded while 2000 was comparatively cool with only 21 cases. Recent studies (Dohm *et al.*, 2001) have also shown that mosquitoes held at high temperatures are more efficient vectors of the West Nile virus. Understanding overwintering temperatures are also important in determining the virility of the virus over seasons in the same area (Dohm and Turrell, 2000).

In addition to temperature, precipitation is an important climate variable to consider as an influence on the distribution of the West Nile virus. Epstein and Defilippo (2001) examined three large outbreaks of the West Nile virus around the world associated with drought and excessive heat - Romania, New York City and Russia. They concluded that multi-month drought, especially in spring and early summer, was associated with urban outbreaks of the West Nile virus in

Europe and the USA. Each new outbreak of the West Nile virus requires the introduction or reintroduction of the virus – primarily via birds or wildlife so there have been seasons without outbreaks despite a multi-month drought. Spread of the West Nile virus may occur, even in the absence of conditions that amplify the cycling of the virus from birds to mosquitoes to humans. Epstein and Defilippo conclude that droughts increase the "prior probability" of a significant outbreak. Once the virus becomes established in a region, other factors, such as rains that increase populations of vectors, may affect the transmission dynamics.

Epstein (2000) proposed a hypothesis linking the West Nile virus to climate variables as described in Figure 12. The hypothesis centred on four main climate connections: (1) warmer winters than normal to allow infected mosquitoes to survive the winter; (2) spring or early summer drought to concentrate vectors and hosts around pools of water, and allow for low populations of vector

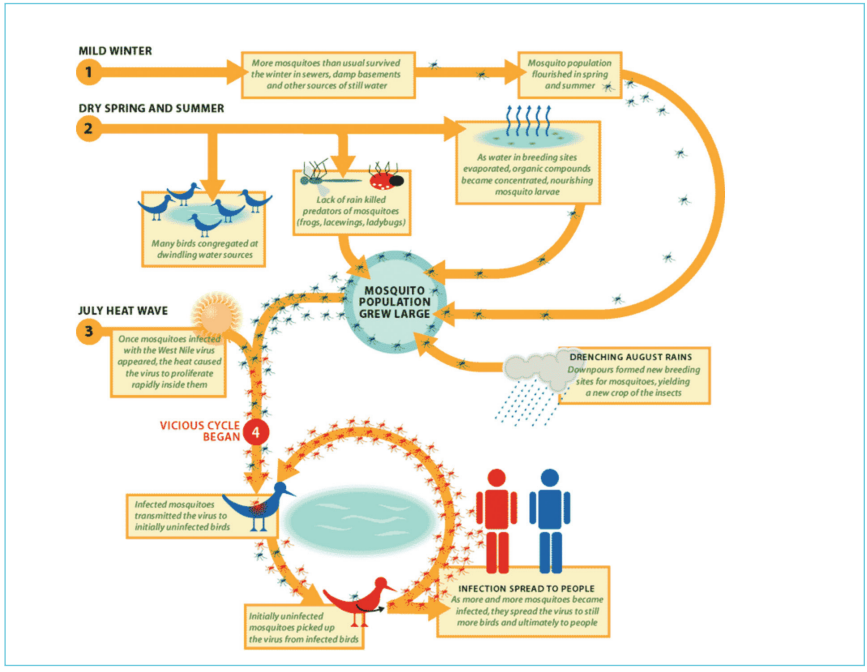

FIGURE 12
Epstein (2000) hypothesis linking West Nile Virus to climate variables.

predators such as lady beetles or amphibians; (3) summer rains to allow for expanded vector populations (mosquitoes); and (4) summer heat to allow for the incubation and transmission of the West Nile virus. Environment Canada's *Climate Trends and Variations Bulletin for Canada* (Environment Canada, 2000; 2001; 2002b) were consulted to test this hypothesis by examining the Great Lakes/St.Lawrence and the Northeastern Forest climate regions of Canada, that is, those that covered Ontario. Winter temperatures, spring precipitation[3], summer precipitation and summer temperatures were examined for the two climate zones of Canada (see Figures 13 to 16), resulting in the conclusions presented in Table 3. It appears as if not all of the conditions that would increase the "prior probability" of the West Nile virus spreading in Ontario have been met for the years of West Nile infection in southern Ontario, and thus the hypothesis needs to be re-examined. It is difficult to tease out the climate variables for the general, regionalized reports from the Climate Bulletins. Focusing on individual daily temperatures and precipitation events at specific locations such as the epi-centre in Toronto is the logical progression of research as a next step.

As a final word on climate, an examination of future climate scenarios based on a simulation output from the latest General Circulation Model – the Coupled Global Circulation Model 2 (CGCM2), developed by the Canadian Centre for Climate Modelling and Analysis of Environment Canada (Natural Resources Canada, 2002), (see Figure 17) - reveals that these climate conditions (winter temperature, summer precipitation, summer temperature) that increase the "prior probability" of the spread and severity of the West Nile virus are expected to increase in the future.

TABLE 3
Epstein Hypothesis Tested

Climate Variable	2000	2001	2002
Warmer Winter Prior	✓	×	✓
Spring Drought	×	✓	×
Summer Rains	✓	✓	✓
Summer Heat	✓	✓	✓

3 Drought is a complex term that has various definitions (Ontario Ministry of Natural Resources, 2002). Drought has been described as a prolonged period of abnormally dry weather that depletes water resources (Agriculture and Agri-Food Canada, 2002). The Ontario Ministry of Natural Resources defines drought as "weather conditions characterized by below normal precipitation that has socio-economic effects" (Ontario Ministry of Natural Resources, 2002). Thus, precipitation is examined as an indicator of drought conditions.

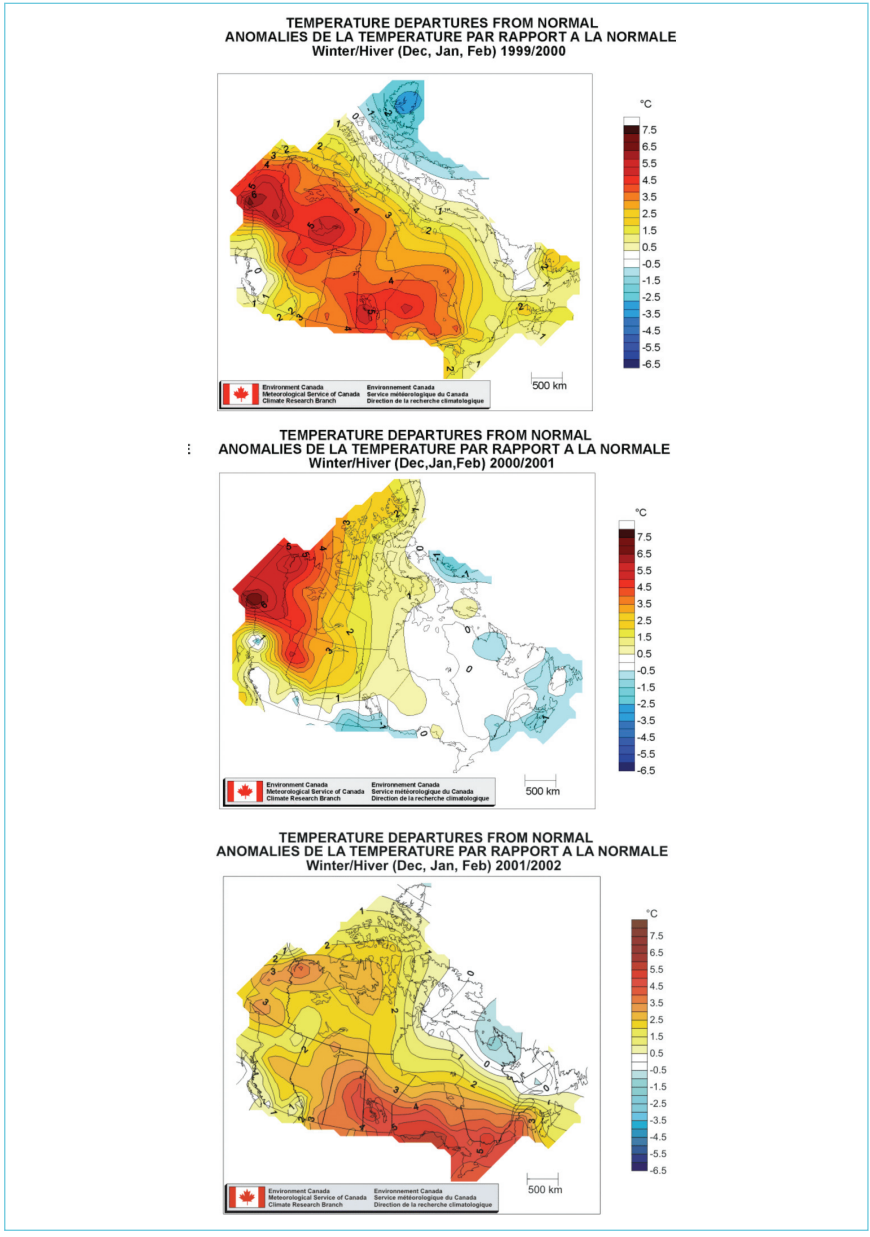

FIGURE 13
Winter Temperatures in Canada 1999-2000, 2000-2001, 2001-2002.
Source: Environment Canada.

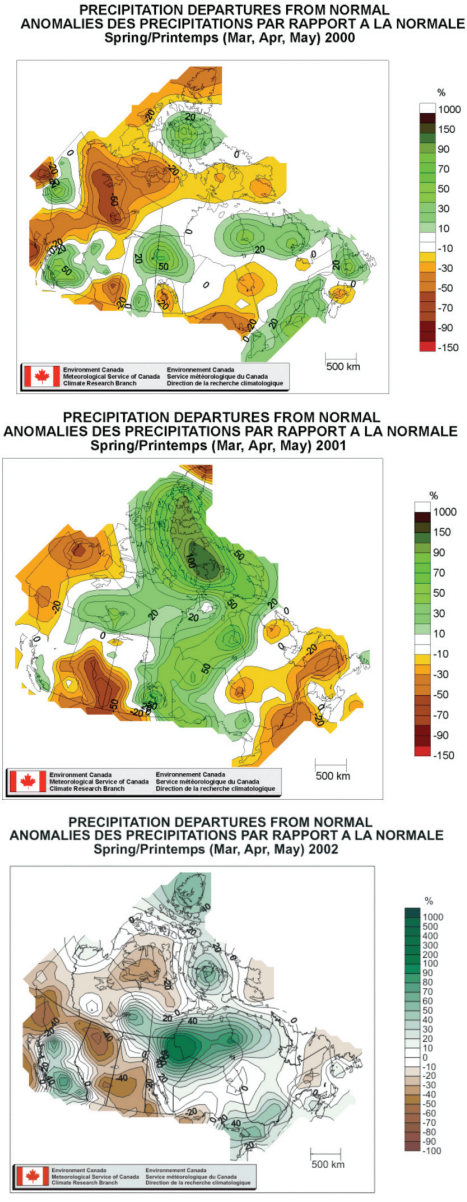

FIGURE 14
Spring Precipitation in Canada 2000, 2001, 2002.
Source: Environment Canada.

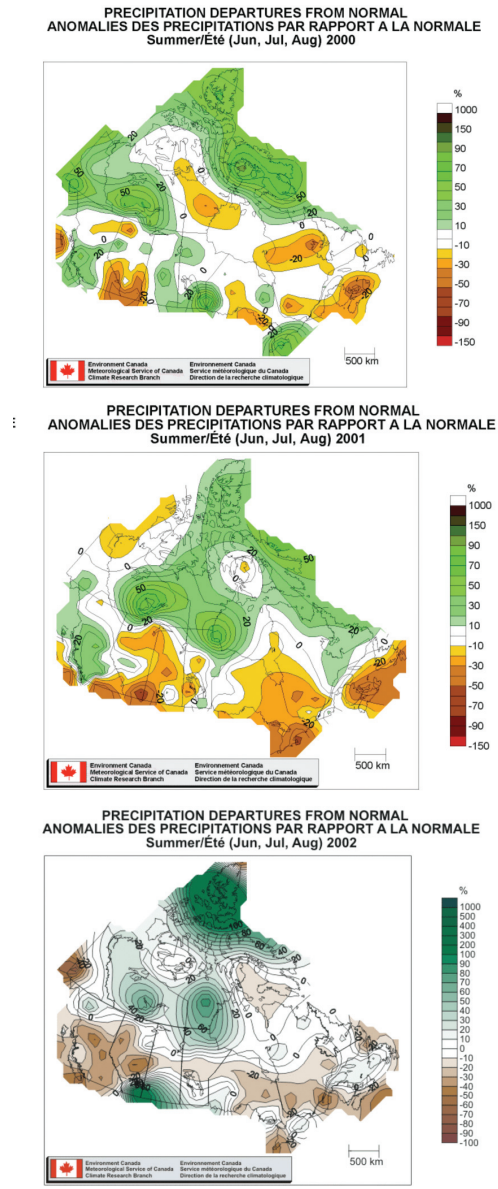

FIGURE 15
Summer Precipitation in Canada 2000, 2001, 2002.
Source: Environment Canada.

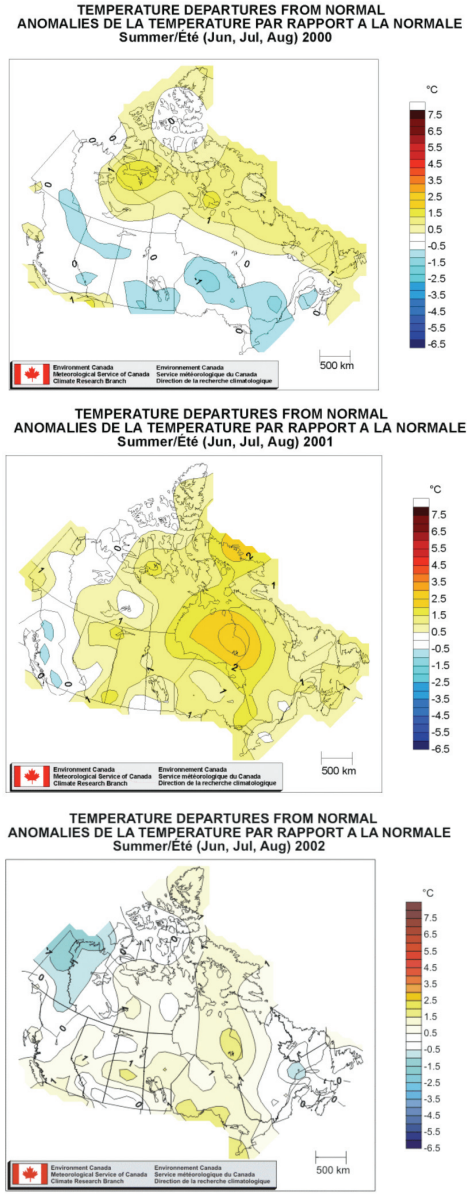

FIGURE 16
Summer Temperatures in Canada 2000, 2001, 2002.
Source: Environment Canada.

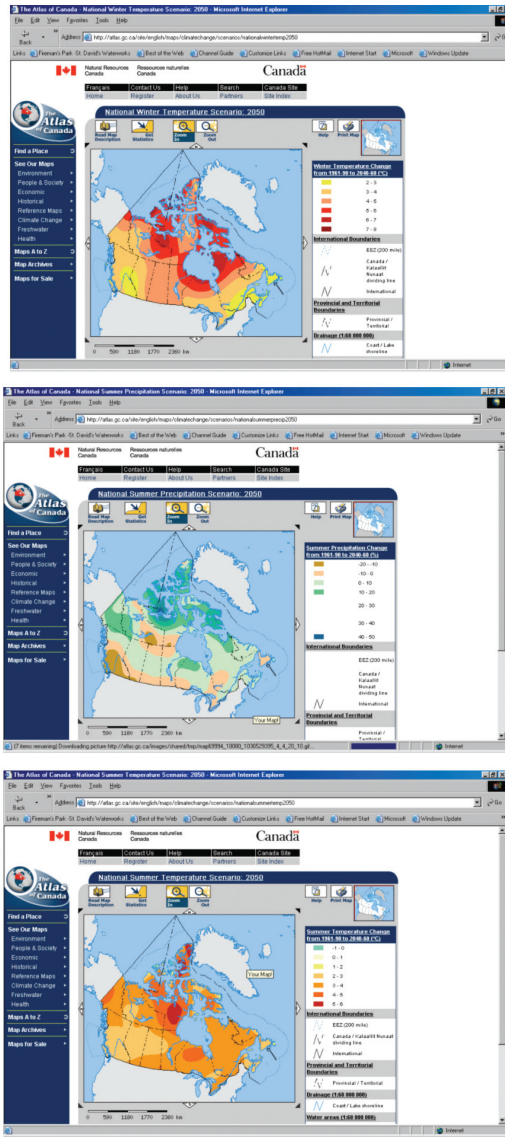

FIGURE 17
Future Scenarios of Climate Change in Canada Winter Temperature, Summer Precipitation, Summer Temperature.
Source: Natural Resources Canada.

6. Management of the Risk of the West Nile Virus

There are many opportunities to manage the risk to humans of the West Nile virus in Ontario including monitoring the spread of infection; reducing human exposure to infected vectors; preventing initiation and magnification of the virus; screening blood supplies and other products capable of spreading the virus; and conducting public information campaigns.

Monitoring infected mosquito pools, birds, mammals and humans allows for an understanding of the progression of the West Nile virus' seasonal cycle, and further analysis and research of events over time. Evidence has been collected on the probable timing of events. The numbers of dead crows play an important role in signaling the epi-centre of the virus outbreak two weeks prior to peak exposures, and the onset of severe symptoms in humans (see Figure 9). Recommendations for risk management should include monitoring crow populations to identify epi-centres of outbreaks, their severity, and to allow for early public alerts to help reduce exposure to the vector (mosquitoes).

Ways of preventing human exposure to mosquitoes include avoiding outdoor activity during peak times for mosquito feedings such as dawn and dusk; wearing insect repellant when outside; wearing long clothing made of a tight material knit to avoid mosquito stinger penetration; and ensuring gateways to indoors are secure including fixing holes in screen doors and windows. All of these personal risk management strategies are recommended, as the literature supports their success (see Mostashari *et al.,* 2001). Individuals can further protect themselves through the use of a vaccine against the West Nile virus once it has been developed. Such a vaccine would be recommended for the high risk groups such as those over 50 years of age, and the immune deficient.

New York City attempted to break the virus life cycle by spraying pesticides over infected locations in September 1999 to control the number and type of mosquitoes. This pesticide spraying has continued with success in controlling the virus over the past 3 years. The amounts of pesticides that lead to the elimination of the virus have been determined through computer modelling (Thomas and Urena, 2001). Spraying is expensive and very hazardous to other insect species important to functioning ecosystems and the environment in general. The pesticide is not always applied appropriately to its target, and pesticide residues can continue to pollute the environment over time. It is recommended that alternative means of reducing the mosquito populations be employed including reducing stagnant pools of water throughout a suspected epi-centre, and promoting the propagation of mosquitoes' natural predators such as bats, birds and lady beetles.

There is a suspected case of the West Nile virus leading to a death in Ontario from a contaminated blood sample following a blood transfusion. If inexpensive and simple tests exist, then all blood supplies and products could be screened for the virus.

Most importantly, public information programs about the preventative measures that can be taken by individuals, and the overall risk to humans are crucial. It is often quoted that influenza kills over 2,000 Canadians per year, and so far in Canada, only one confirmed death from the West Nile virus has been recorded. This comparison of risk should be presented as a means of allowing personal risk evaluation.

References

Agriculture and Agri-Food Canada. 2002. *The Health of Our Water.* **http://res2.agr.ca/research-recherche/science/Healthy_Water/e09b.html**

Andreadis, T. 1999. A survey of tire dumps across Connecticut. Connecticut Agricultural Experiment Station Report.

Andreadis, T., Anderson, J. and Vossbrinck, C. 2001. Mosquito surveillance for West Nile virus in Connecticut, 2000: Isolation from *Culx pipiens*, *Cx. restuans*, *Cx. salinarius*, and *Culiseta melanura*. *Emerg Infect Dis* 7(4) 2001.

Bernard, K., Maffei, J., Jones, S. Kauffman, E., Ebel, G., Duppuis, A. Ngo, K., Nicholas, D., Young, D., Shi, P., Kulasekera, V., Eidson, M., White, D., Stone, W., and Kramer, L. 2001. West Nile virus infection in birds and mosquitoes, New York State, 2000. *Emerg Infect Dis* 7(4) 2001.

Brigockas, M. 1999. *Press Release: New Species of Mosquito found in Connecticut by Peabody Museum Curator. July 15, 1999*. Peabody Museum of Natural History. Yale University. New Haven, Connecticut.

Chiotti, Q., Fenech, A. and Liebowitz, M. 2002. The threat of West Nile virus in Ontario: out of the blue or predictable? Presentation at the 2002 Canadian Association of Geographers Annual Meeting, Special Session on Integrated Mapping Assessment Project (IMAP). University of Toronto. May 30, 2002.

Crans, W. 2001. *Culex restauns*. Rutgers University.

Dohm, D.J. and Turell, M.J. 2000. Effect of incubation at overwintering temperatures on the replication of West Nile virus in New York *Culex pipiens* (Diptera: *Culicidae*). *Journal of Medical Entomology:* Vol. 38, No.3 pp.462-464.

Dohm, D.J., O'Guinn, M.L. and Turrell, M.J. 2001. Effect of environmental temperature on the ability of *Culex pipiens* (Diptera: *Culicidae*) to transmit West Nile virus. *Journal of Medical Entomology.* Vol. 39, No. 1, pp. 221-225.

Dooley, J.E. 1990. *Risk Analysis for Health and Environmental Management.* Environmental Management Development in Indonesia. Atlantic Nova Print. Halifax and Jakarta.

Eidson, M., Komar, N., Sorhage, F., Nelson, R. Talbot, T. Mostashari, F., and McLean, R. 2001a. Crow deaths as a sentinel surveillance system for West Nile virus in the Northeastern United States, 1999. *Emerg Infect Dis* 7(4).

Eidson, M., Miller, J., Kramer, L., Cherry, B., and Hagiwara, Y. 2001b. Dead crow densities and human cases of West Nile virus, New York State, 2000. *Emerg Infect Dis.* 7(4).

Environment Canada, 2000. Climate Trends and Variations Bulletin for Canada. **http://www.msc-smc.ec.gc.ca/ccrm/bulletin/**.

Environment Canada, 2001. Climate Trends and Variations Bulletin for Canada. **http://www.msc-smc.ec.gc.ca/ccrm/bulletin/**.

Environment Canada, 2002a. American Crow. **http://wildspace.ec.gc.ca/life.cfm?ID=AMCR&Page=Map&Lang=e**.

Environment Canada, 2002b. Climate Trends and Variations Bulletin for Canada. **http://www.msc-smc.ec.gc.ca/ccrm/bulletin/**.

Epstein, Paul R. and Defilippo, C. 2001. West Nile virus and drought. *Global Change & Human Health* 2(2):105-107

Giladi, M., Metzkor-Cotter, E., Martin, D.A., Siegman-Igra, Y., Korczyn, A.D., Rosso, R., Berger, S.A., Campbell, G.L., Lanciotti, R.S. 2001. West Nile Encephalitis in Israel, 1999: The New York Connection. *Emerg Infect Dis* Vol. 7 No. 4.

Guptill, S. 2002. Personal communication.

Health Canada. 2002. **http:\\wildlife.usask.ca/WestNileAlertHTML/**

Hubalek, Z. and Halouzka, J. 1999. West Nile fever- a reemerging mosquito-borne viral disease in Europe. *Emerging Infectious Diseases.* Vol. 5 No. 5.

Jannaccio, R. 2000. Selling the West Nile virus vaccine: If it's good for OraVax, is it also good for you? **www.nospray.org/sellingWNV.html**.

Komar, N. 2000. West Nile virus encephalitis. *Rev Sci Tech OIE.* 19:166-76.

Kosteyukov, M.A., Alekseev, A.N., Bulychev, V.P., Gordeeva, Z.E. 1986. Experimental infection of *Culex pipiens* mosquitoes with West Nile virus by feeding on infected Rana ridibunda frogs and its subsequent transmission (in Russian). *Med Parazitol (Mosk)* Vol 6. p. 76-78.

McLean, R. 2002. Personal communication.

Moore, C. and Guptill, S. 2002. Methods and data for vector-borne and zoonotic diseases. Paper presented at the June 2002 conference *Healthy People, Healthy Ecosystems* in Washington, D.C.

Mostashari, F., Bunning, M.L., Kitsutani, P.T., Singer, D.A., Nash, D., Cooper, M.J., Katz, N., Liljebjelke, K.A., Biggerstaff, B.J., Fine, A.D., Layton, M.C., Mullin, S.M., Johnson, A.J., Martin, D.A., Hayes, E.B. and Campbell, G.L. 2001. Epidemic West Nile encephalitis, New York 1999: results of a household-based seroepidemiological survey. *Lancet.* Vol. 358: 261-64

Nasci, R., Savage, H., White, D., Miller, J. Cropp, B., Godsey, M, Kerst, A. Bennett, P., Gottfried, K., and Lanciotti, R. 2001. West Nile virus in overwintering Culex mosquitoes, New York City, 2000. *Emerg Infect Dis* 7(4).

National Wildlife Health Center, 2000. Unpublished data presented at the June 2002 conference *Healthy People, Healthy Ecosystems* in Washington, D.C.

Natural Resources Canada. 2002. The Atlas of Canada. **http://head-smashed-in-a.ccrs.nrcan.gc.ca/cgi-bin/na_invent/user?name=994**.

Ontario Ministry of Natural Resources. 2002. *Definitions.* **http://www.mnr.gov.on.ca/MNR/surface_water/definitions.htm**.

Peabody Museum of Natural History. 1999. New species of mosquito fourn in Connecticut by Peabody Museum curator. *Press Release.* July 15, 1999.

Peiris, J.S.M.and Amerasinghe, F.P. 1994. West Nile fever. In Beran, G.W., Steele, J.H. editors. *Handbook of zoonoses.* Section B. Viral. 2nd edition. Boca Raton (FL). CRC press. pp. 139-48.

Petersen, L.R. and Roehrig. 2001. West Nile Virus: A reemerging global pathogen. *Emerging Infectious Diseases.* Vol. 7, No.4.

Platonov, A., Shipulin, G., Shipulina, O., Tyutyunik, E., Frolochkina, T., and Lanciotti, R. 1999. Outbreak of West Nile virus infection, Volgograd Region, Russia, 1999. *Emerg Infect Dis* 2001. 7:128-132.

Potter, N. 2000. The culprit? mosquito species that carried West Nile virus to US have been found. *ABC News.* August 24, 2000.

Rappole, J., Derrickson, S., and Hubalek, Z. 2000. Migratory birds and spread of the West Nile virus in the Western Hemisphere. *Emerging Infectious Diseases.* Vol. 6, No. 4.

Reeves, W.C., Hardy, J.L., Reisen, W.K., Milby, M.M. 1994. Potential effect of global warming on mosquito-borne arboviruses. *J Med Entomol.* Vol. 31 pp. 323-32.

Savage, H., Ceianu, C., Nicolescu, G., Karabatsos, N., Lanciotti, R., and Vladimirescu, A.. 1999. Entomologic and avian investigations of an epidemic of West Nile fever in Romania in 1996. *Am J Trop Hyg* 1999. 61:600-11.

Scott, J. 2001. *Ochlerotatus japonicus.* Rutgers University.

Semenov, B.F., Chunikhin, S.P., Karmysheva, V.Y., Yakovleva, N.I. 1973. Studies of chronic arbovirus infections in birds: Experiments with West Nile, Sindbis, Bhanja and SFS viruses (in Russian). *Vestik Akademii Medicinskikh NaukSSSR.* Vol. 2 p. 79-83.

Smithburn, K.C., Hughes, T.P., Burke, A.W., and Paul, J.H. 1940. A neurotropic virus isolated from the blood of a native of Uganda. *Am J Trop Med.* Vol 20: 471-92.

Thomas, D. and Urena, B. 2001. A model describing the evolution of the West Nile-like encephalitis in New York City. *Mathematical and Computer Modelling.* 34:771-781.

Toronto Public Health. 2001. West Nile Virus. (pamphlet)

Tsai, T., Popovici, F., Cernescu, C., Campbell, G., and Nedelcu, J. 1998. An epidemic of West Nile encephalitis in southeastern Romania. *Lancet* 1998. 352:767-71.

Turrell, M.J., O'Guinn, M.L., Dohm, D.J., and Jones, J.W. 2000. Vector competence of North American mosquitoes (Diptera: *Culicid ae*) for West Nile virus. *Journal of Medical Entomology.* Vol. 38. No. 2, pp. 130-134.

Womack, M. 2001. *Aedes japonicus japonicus.* Macon State College.