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Above All, Do No Harm -- Dealing with West Nile Virus in Canada

On September 14, 2002, a 70-year-old Mississauga man succumbed to West Nile virus, Canada's first reported WNV death (Finucane, "West" A1). Nine weeks later, the region's medical officer announced that officials were prepared to spray pesticides to control mosquitoes, the carriers of the virus, "as a last resort" (Finucane, "Peel" 14). If West Nile had come to Canada some decades ago, authorities might already have blanketed many areas with pesticides. In 1945, for instance, they might have used DDT, the miraculous new chemical that was going to win humanity's age-old war against insects and insect-borne disease. A National Geographic photograph from that year shows two boys in a New York City playground running toward a cloud of DDT, as happily unaware of its future health effects as the men in the spray truck (Colton 410). Since then, dozens of countries have outlawed DDT and many other pesticides, claiming that decades of experience showed them to be dangerous, expensive, and ineffective. However, the medical officer's reaction to last September's death suggests that the pesticide habit dies hard, and raises issues that have still not been properly debated in Canada. How serious a health threat is West Nile virus? Which strategies will most effectively mitigate this threat? And what role, if any, should pesticides have in any mitigation efforts?

West Nile virus (WNV) was first identified in 1937 in the West Nile area of Uganda, and since then has spread to South Africa, Israel, Romania and France (Petersen 611-12). **Rappole, Derrickson, and Hubalek have suggested that birds are the major means by which the virus colonizes new regions, since viral outbreaks usually coincide with the arrival of migratory birds in summer or early fall** (320-22). Once WNV is established in a region, its normal transmission cycle is from infected birds (viral "hosts") to mosquitoes (viral "vectors") and back again, with mammals such as horses and humans entering the cycle when bitten by an infected mosquito (Duebel).

The first North American outbreak started in New York City in August 1999; two months later, seven people were dead from West Nile encephalitis--inflammation of the brain--out of sixty-two diagnosed cases (Shieh 370). Weiss et al. reported that WNV tends to afflict older people with high blood pressure or damage to the blood-brain barrier (which normally blocks viral entry to the brain); for every hospitalized case in Queens, New York in 1999, there were 24 mild cases and 110 subclinical cases (those without obvious physical symptoms), with a median age for hospitalized patients of 71 years (657-58). Ontario's chief medical officer of health contrasted Canada's first WNV death with our annual toll of 2,000 influenza deaths, and claimed that "99 per cent of humans who contract West Nile virus suffer no symptoms" (Finucane, "West" A1). Furthermore, infected people develop immunity to the virus, the way childhood chicken pox protects people from future infections.

Nonetheless (as doctors now admit), because the disease is established in North America and continuing to spread, it would be a **mistake**--and a breach of medical ethics--to **downplay or underestimate it**. Medical treatment can relieve the suffering of infected people, but cannot prevent infections. Testing and approval of a human vaccine is unlikely to be completed for several years (Tesh et al. 250). Experience with malaria and other mosquito-borne illnesses shows that **breaking an established transmission cycle is almost impossible, due to the speed with which mosquitoes reproduce and fill any available ecological niches**; in **1995, forty years after the World Health Organization formally declared its intent to eradicate malaria worldwide, it was still killing more than a million children in tropical Africa alone** (Wargo 45, 61). The

only hope for real control of WNV thus appears to **lie in a reduction of the population of mosquitoes carrying the virus**. Wegbreit and Reisen found that cases of **Western equine encephalomyelitis (WEE) in California's Central Valley dropped sharply in years when low snowmelt runoff reduced the number of WEE-bearing mosquitoes** (22). Their research strongly suggests that **Canada's WNV medical case load will vary in proportion to the number of WNV-bearing mosquitoes in the country**.

**Effective mosquito control requires an understanding of the insect's four life stages**: egg, larva, pupa, and adult (Metcalf 21.8-10). Eggs of the *Culex* genus--identified as the principal WNV vector during the 1999 New York outbreak (Petersen 613)--form rafts of several hundred floating on water, looking, according to Metcalf, "like a bit of soot" (21.8). Once hatched, a *Culex* larva **floats upside down in stagnant, polluted water for two days to two weeks**, legless and wingless, feeding on organic matter, and breathing through a siphon tube located near its terminal end (Metcalf 21.9-10). In the pupal stage, the adult's eyes, legs, and wings develop inside a cocoon, tumbling around just below the water's surface for a few hours to a few weeks, until the adult emerges and flies off (Metcalf 21.10). **Culex adults reproduce several times each summer** (Metcalf 21.10).

Since mosquitoes spend three of their four life stages in water, controlling their aquatic environments is the key to controlling their numbers.

**Elimination of standing water by drainage or pumping is, according to Metcalf, "the most fundamental means of control"** (21.11). This includes **proper disposal of the 300 million tires discarded each year in North America** (Steed E4). Following a 1988 survey of discarded tires in suburban Chicago, Baumgartner concluded that they were a **significant breeding habitat for two medically important species, Culex restuans and Culex pipiens** (500). He cited research done in Minnesota and Ohio that incriminated old tires containing the *Aedes triseriatus* mosquito as the major source in cases of La Crosse encephalitis, an illness somewhat similar to West Nile encephalitis (506). Since **Culex larvae thrive in water containing human or animal**

**excrement (Metcalf 21.10), proper disinfection of fecal waste could significantly reduce mosquito breeding opportunities around farms, slaughterhouses, sewage treatment plants, and treatment lagoons.** (This would have the added advantage of controlling E. coli, the fecal bacillus that killed seven people and injured 2,300 others in Walkerton three years ago.)

Different control strategies are needed for **aquatic-stage mosquitoes in lakes, ponds, wetlands, and other bodies of water too ecologically important to drain.** An increasingly popular method uses *Bacillus thuringiensis* var. *israelensis* (Bti), a microbe that kills mosquito larvae with "minimal environmental impact," according to Brown et al. (8). **Bti and other biological larvicides have proved effective in Australia** (Brown et al.), **India (Batra, Mittal, and Adak), Arkansas (Dennett et al.), and Florida** (Hallmon et al.). Larviciding has recently been the preferred means of control in Mississauga (Rodman).

Once airborne as adults, mosquitoes can no longer be treated en masse. Rather, effective control requires that individual adults be prevented from biting and infecting potential hosts. A gel made from **neem tree oil, a natural insecticide, is 94.8 per cent effective in reducing mosquito landings on the exposed skin of treated subjects compared with untreated controls** (Caraballo 45). Cornell University's Environmental Risk Analysis Program recommends screens on homes and tents, and long-sleeved clothing in places where mosquitoes are biting, as other common sense but effective control measures ("Mosquito Hygiene"). **Adult mosquitoes returning to water to feed or breed can be controlled by predators such as dragonflies; Tillyard reported seeing a dragonfly eat more than a hundred mosquitoes within ten minutes (327-29). A Michigan pest control consultant with twenty years experience in low-toxicity pest control has listed birds, bats, frogs, fish, snails, spiders, water scorpions, ants, backswimmers, and striders as other mosquito predators** (Tvedten 855). There are thus many options available to health authorities wishing to control mosquitoes with ecologically-based methods.

**In contrast, pesticide-based mosquito control has had, at best, partial and temporary success.** Rachel Carson was documenting **pesticide resistance**--the ability of an insect population to develop immunity to pesticides--forty years ago. Carson was most urgently concerned with DDT, but **since then mosquitoes have also become resistant to malathion, an organophosphate pesticide in widespread use, as reported by Coto et al.** Their study found that organophosphates were first used in response to the high level of resistance that mosquitoes developed to DDT and other chemicals in the organochlorine class of pesticides (324). Evolution has now selected for organophosphate-resistant mosquitoes, thus making this class of pesticides increasingly useless. Coto et al. pointed out that **pesticide resistance has also been documented for a third class of pesticides, pyrethroids** (329). And

even Metcalf, though defending insecticides as necessary for complete control of some insect pests, admitted in 1993 that "[n]early every species of mosquito important to human health now shows multiple resistance to organochlorine, organophosphate, carbamate, and pyrethroid insecticides" (21.12).

A particularly striking example of pesticide resistance occurred in **Cicero Swamp**, 16 km northeast of Syracuse, New York, from 1984 to 1994 (Howard and Oliver). During each of those years, authorities sprayed naled, an organophosphate, to control **Culiseta melanura**, the vector of the eastern equine encephalitis (EEE) virus. By 1994, the population of Cs. Melanura had increased 15-fold. After analyzing several possible reasons for this increase, Howard and Oliver concluded that evolutionary pressures were responsible, and stated that this long-term case study "further discredits the rationale that preventive applications of naled reduce the risk of EEE" (324).

Of the 2.5 million tons of pesticides applied worldwide each year, Pimentel estimated that less than 0.1 per cent actually reach their intended pests. The remaining 99.9 per cent drift into and contaminate nearby soil, water, or air. The resulting adverse effects on the health of humans, and organisms beneficial to humans, were already well documented by the time Rachel Carson wrote *Silent Spring*. Subsequent research has only strengthened her case for banning pesticides. The recent debate about pesticide use on lawns and yards shows the public is more aware than ever of the link between pesticide exposure and cancer. However, the full scope and severity of pesticides' health hazards is still largely unrecognized. One example is the **potentially devastating effect on brain development in young children noted by Weiss (246)**. After summarizing **some clinical studies of young children who became "profoundly retarded" after their mothers were exposed to a common organophosphate pesticide in pregnancy, Weiss assessed the consequences of a pesticide-caused five per cent downward shift in average IQ**. He first quoted a 1996 paper on PCBs and development that showed IQ scores dropped 6.2 per cent due to PCB exposure; he then noted that PCBs are chemically similar to many of the pesticides now stored in people's bodies as a result of fifty years of worldwide use. A five-point drop, Weiss calculated, would reduce the number of people attaining IQ scores above 130 by more than half; it would also increase by 60 per cent the number of people with IQ scores below 70. The financial impact, for a population the size of the U.S., would be an accumulated loss of seven trillion dollars.

One effect of pesticides is especially troubling in light of the recent WNV outbreaks. The virus causes encephalitis only when it crosses the blood-brain barrier. **Gupta et al. found that several common pesticides increase the permeability of this barrier**. Since the response of many communities to WNV has been to spray pesticides, many of them known to damage the blood-brain barrier, the disturbing possibility exists that, far from preventing WNV outbreaks, pesticides may have worsened some of them.

Those who promoted the use of pesticides in the 1940s did not mean to cause harm. DDT at first seemed truly a "Powerful Insecticide, Harmless to Humans," as the placard on the side of the New York City spray truck said (Colton 410). But later, as evidence of harm accumulated, the same people made the mistake of believing that the answer to complex problems in biology and ecology lay just one more high-tech fix away. This attitude, naive but understandable in 1945, is now at odds with the well-being, and possibly the survival, of our species (Colborn). We know enough about WNV to deal with it without chemicals that, at best, cannot control it effectively, and, at worst, will amplify the harm the virus causes.

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