WEST NILE VIRUS, VERMONT, AND PESTICIDES

Understanding risks—Preventing harm

A Guide for the Public, Policymakers and News Media

March 2001

Vermont Public Interest Research Group
141 Main St. Ste. 6 Montpelier, VT 05602
802-223-5221 vpirg@vpirg.org www.vpirg.org

Table of Contents

Executive Summary 1
Viruses, mosquitoes and birds 2
West Nile virus background information 2
Mosquito background information 3
West Nile virus comes to America and Vermont 5
West Nile Virus—only one of many minor human diseases 6
Symptoms of West Nile virus infection in humans 7
Antibody response to West Nile virus 8
Vermont Draft West Nile Virus Surveillance and Response Plan 9
Premise for action 9
Three tiered course of action 9
Phase I: Surveillance—No Detection 9
Phase II—Documentation of West Nile virus establishment in Vermont 9
Phase III—Implementation of Vector Management Program 10
Use of larvicides to control mosquito larvae 10
Three larvicides are proposed for use in Vermont 10
Potential non-target consequences of larviciding in wetlands 11
Use of synthetic pyrethroids to kill adult mosquitoes 12
Resmethrin (Scourge) 12
Sumithrin (Avid) 13
Permethrin (Aqua-reslin) 13
Inert ingredients in pyrethroid formulations 14
Vermont Pyrethroid application specifics 15
Problems associated with use of synthetic pyrethroids 16
Human health effects from pyrethroid exposure 18
Non-target effects of spraying synthetic pyrethroids 20
Public over-reaction to the West Nile outbreak 21
Poisonous arsenal at our fingertips 21
Insect repellents 22
DEET—the official choice 22
Other ongoing arbovirus vector control programs 24
VPIRG statements for responsible West Nile virus public response 25
VPIRG proposals for responsible mosquito control 26
CONCLUSION 29
Glossary 30
References 33

Executive Summary

West Nile virus, known as WNV, is transmitted by mosquitoes. WNV first appeared in the U.S. in 1999, when at least 62 people in the New York City area developed WNV encephalitis and seven people died.

After months of testing birds and mosquitoes, WNV presence was confirmed in Vermont on October 2000. A Hermit Thrush found in Putney on September 29, 2000 was the first bird in Vermont to test positive for WNV[1].

WNV presence in neighboring states and adjacent counties in New York indicate WNV has likely been present, although not detected in Vermont in both bird and mosquito populations.[2] Public health officials now agree West Nile virus is probably widespread in the bird population of the eastern U.S., and that it is only a matter of time before the West Nile virus is regularly detected in Vermont.

Because mosquitoes are the vectors of WNV, massive pesticide spraying programs have been carried out in New England in the last two years. Emphasizing responsible use of larvicides to keep mosquito populations low, the State of Vermont is also planning to advise municipalities on aerial and ground spraying with synthetic pyrethroids if needed.

Although under continuous review and discussion regionally, Vermont's current proposed triggers for initiation of pyrethroid spraying are one of the following: one confirmed human WNV clinical infection, one confirmed horse WNV clinical infection, or multiple confirmed active bird infection with corresponding local infected mosquitoes.[3] The spray zone originally proposed by the Arboviral Task Force was a two-mile radius circle, an area of roughly 8,700 acres, around established WNV diagnosis.[4] This spray zone is no longer considered necessary or effective by the Center for Disease Control, and will not be used by Vermont officials in determining spray zones.

Implementation of a spray program would be the responsibility of local municipal officials, under advisement from the Vermont Department of Agriculture. Spraying would be conducted either by truck or by aerial application.

VPIRG believes spraying thousands of acres of Vermont with synthetic pyrethroids is difficult to justify, when:

- For most humans, WNV is a low risk disease. Most people are not aware of being infected. Death results in approximately 0.001% of infected individuals.
- Widespread pesticide use is documented to harm sensitive ecosystems and vulnerable individuals.
- There is already much people can do to protect themselves from mosquitoes, including using lower risk bacterial larvicides, disrupting larval habitat, and wearing protective clothing.

To ensure any pesticides used in the West Nile virus response plan are applied in a manner which will have minimal environmental and human health implications, VPIRG has proposed strict parameters be set prior to implementing a statewide mosquito control strategy.

The following information overviews WNV, Vermont's proposed Response Plan, and VPIRG's concerns and recommendations for an appropriate response to the presence of WNV in Vermont.

Year 2000 Update

In the U.S in 2000, there were 59 equine cases of clinical WNV infection, of which 36 survived and 23 (39%) died. The total number of human cases for the year 2000 was 21, including two deaths. A total of 4,323 birds have been documented as infected with WNV in 12 States plus the District of Columbia since May 2000. Since May 2000, a total of 143 counties in 12 States and the District of Columbia had con-firmed findings of WNV in a mosquito, bird, or mammal.[5]

Viruses, mosquitoes and birds

West Nile virus background information

West Nile virus is one of many global arbo-viruses

WNV is one of many arthropod borne viruses (referred to as arboviruses), including many viruses transmitted by mosquitoes with similar patterns of viral outbreaks. Other biting flies, and both hard and soft ticks, often using birds as main host, also transmit arboviruses.[6]

WNV is related to yellow fever and dengue fever to which serious mosquito control efforts are directed. WNV is closely related to St. Louis encephalitis virus, already found throughout the United States, and the target of mosquito control programs in other states.

WNV is a bird virus, transmitted by mosquitoes from bird to bird

WNV belongs to a family of viruses (Flaviviridae) adapted over millions of years to live sub-lethally in the world's bird population. West Nile virus is established throughout Africa, the Mediterranean, the Middle East and southern temperate and tropical Asia.

Birds are the primary WNV hosts. An infected bird has the active virus in its bloodstream for one to four days. During that time it may become ill, then develop antibodies and immunity, or less commonly, be killed by the virus.[7]

In areas where WNV is newly arrived, the natural pattern is a die-off of birds that have not developed immunity, followed by development of immunity in the general bird population.[8]

Mosquitoes become infected when they feed on infected birds, which may circulate the virus in their blood for seven to ten days. After an incubation period of 10 days to 2 weeks, infected mosquitoes can then transmit West Nile virus to another bird or other vertebrates, including humans, while biting to take blood.

An infected mosquito is able to transmit the virus throughout its life.[9] Infected mosquitoes may overwinter and emerge in the spring capable of transmitting WNV.

The virus is located in the mosquito's salivary glands. During blood feeding, the virus is injected into the animal or human, triggering immune system response. Where WNV finds weakened immune response, it may multiply, causing illness.[10]

Migratory birds contribute to successful WNV spread

The pattern of outbreaks in southern Europe suggests that migratory birds infected with WNV contribute to movement of the virus. American bird migration patterns are well documented and are already used by the CDC in its surveillance and tracking programs. [11]

WNV antibodies were found in 41% of domestic birds in Romania studied after the 1996 Romanian outbreak.[12] Sporadic, minor outbreaks of WNV in birds in southwestern Romania after 1996 are consistent with locally maintained bird-to-bird viral transmission.[13]

Bird to bird transmission documented

Scientists from the U.S. Geological Survey National Wildlife Health Center in Madison, Wisconsin have confirmed in October 2000 that the West Nile Virus can be transmitted from bird-to-bird in a confined laboratory setting.[14] It had previously been thought that the virus was only transmitted through mosquito bites.

Alternative hosts for West Nile virus include mammals and amphibians

In addition to birds, WNV infects a variety of vertebrate hosts, including cattle, horses, dogs, and humans. Mammals are less important than birds in maintaining transmission cycles of the virus. [15]

Mammals are considered "dead-end" hosts for WNV. Levels of WNV in mammalian blood rarely reach high enough levels for successful transmission by mosquitoes.[16]

Dogs can become infected, but do not play an important role in the transmission of WNV.[17] Other mammals with documented infections include cats, raccoons, chipmunks, skunks, squirrels, and domestic rabbits. That list also includes more than a dozen infected bats collected from five counties representing eastern, central, and western New York.[18] The role of bats in re-transmittal is unknown.[19]

Frogs can harbor the virus, and their blood can transmit WNV to *Culex pipiens*, according to lab studies.[20]

Humans and horses are poor WNV hosts

Humans are generally considered to be poor hosts for WNV and are insignificant in re-transmittal of the virus. Horses appear to have moderate ability to support high enough levels of WNV to support WNV locally.[21]

Bird mortality not a good indicator of human risk

Review of data generated by states affected by WNV in 2000 concludes that WNV mortality in native birds shows little correlation with human WNV incidence. Birds are so sensitive they begin to die off well before WNV poses a risk to humans. Birds are thus a useful indicator of WNV, but are not a reliable indicator of human disease risk.[22]

A combination of factors must be present at the same time for human infection

Environmental factors, including human activities, enhance population densities of vector mosquitoes. These include heavy rains followed by floods, irrigation, higher than average temperatures, or the creation of ecological niches that enable the mass breeding of mosquitoes.[23]

When these conditions are present with sufficient active WNV infection in resident and migratory bird populations, in proximity to human populations, local WNV outbreaks in the human population can occur.

Human living conditions play a role in human WNV outbreaks

As with many human diseases living condition plays a role. In Romania in 1996, mosquitoes in the home were associated with WNV infection, and among apartment dwellers, flooded basements were a risk factor.[24]

Mosquito background information

Common mosquitoes are the main vectors of WNV

Internationally, WNV is transmitted by at least 43 species of mosquitoes[25] (known as vectors), including the resident Vermont northern house mosquito, *Culex pipiens*. Other mosquitoes found in New England have been identified as WNV vectors, including members of the genus *Aedes*, which are also found in Vermont. Information on mosquito distribution in Vermont is still being compiled.[26]

Culex pipiens, Vermont's most common mosquito, can carry WNV if it is present in the bird population. In the 1996 Romanian WNV outbreak, *Culex pipiens* was the likely vector.[27]

WNV transmitted by bite of infected female mosquito

When adult mosquitoes emerge from the aquatic larval stages, they mate, and the female seeks a blood meal to obtain the protein needed for the development of her eggs. The females of a few species may produce a first batch of eggs without this initial blood meal. After a blood meal is digested and the eggs are laid, the female mosquito again seeks a blood meal to produce a second batch of eggs.

Young adult female mosquitoes taking their first blood meal do not transmit diseases. When an older female picks up a disease organism in her first blood meal, she can then transmit the disease during her next blood meal. Depending on her stamina and the weather, she may repeat this process many times without mating again.

Life cycle and daily habits differ for each mosquito species

Culex pipiens—the common Vermont house mosquito

Culex mosquitoes lay their eggs on the surface of fresh or stagnant water. The water may be in tires, tin cans, birdbaths, ornamental ponds, swimming pools, basements, puddles, creeks, ditches, or marshy areas. Mosquitoes prefer water sheltered from the wind by grass and weeds.

Culex mosquitoes usually lay their eggs at night, one at a time, sticking them together to form a raft of from 200 to 300 eggs. A raft of eggs looks like a speck of soot floating on the water and is about 1/4 inch long and 1/8 inch wide. A female mosquito may lay a raft of eggs every third night during its life span. Tiny mosquito larvae emerge from the eggs within 24 hours. *Culex* mosquitoes prefer to seek a blood meal at dusk and after dark, and readily enter dwellings. Domestic and wild birds are preferred over humans, cows, and horses.

Aedes—daytime biters found in Vermont

Aedes mosquitoes are painful and persistent biters. They search for a blood meal early in the morning, at dusk and into the evening. Some are daytime biters especially on cloudy days and in shaded areas. They generally do not enter dwellings, and they prefer to bite mammals like humans.

Aedes mosquitoes lay their eggs singly on damp soil. *Aedes* eggs hatch only when flooded with water, such as in treeholes and flooded stream bottoms.

Mosquito lifespan

The length of life of the adult mosquito depends on several factors: temperature, humidity, sex of the mosquito and time of year and predation.

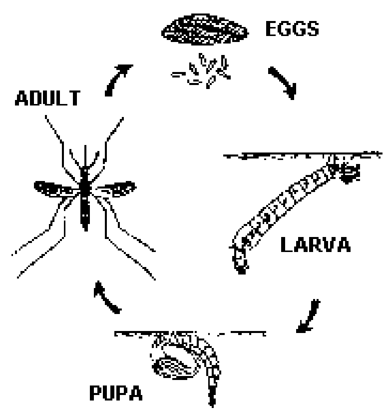
Being relatively fragile insects, the life span of female mosquitoes is several days to three months, while the male's life span is 10 to 20 days only. *Culex* mosquitoes typically live only a few weeks during the warm summer months.

The vast majority of mosquitoes die by serving as food for bird, dragonflies and spiders or are killed by the effects of wind, rain or drought.

Dispersal from breeding site

The flight habits of mosquitoes depend on the species. *Culex* mosquitoes are generally weak fliers and do not move far from home, although they have been known to fly up to two miles. The flight range for females is usually longer than that of males. Wind is a frequent factor in the dispersal of mosquitoes. Most mosquitoes stay within a mile or two of their source. *Aedes* mosquitoes are strong fliers and can fly many miles from their breeding sites.

Mosquito Life Cycle



Overwintering mosquitoes

Many mosquito species overwinter in the egg stage, awaiting the spring thaw, when waters warm and the eggs hatch. *Culex* and other species spend the winter as adult, mated females, resting in protected, cool locations, such as cellars, sewers, crawl spaces, and well pits. With warm spring days, these females seek a blood meal and begin the cycle again. The male mosquito does not take a blood meal, but feeds on plant nectar and lives for only a short time after mating.

West Nile virus comes to America and Vermont

1999—West Nile virus comes to America from Eurasia

WNV is thought to have come to America from the Middle East,[28] most likely carried in infected birds. Using DNA analysis, scientists identified the virus as a WNV virtually identical to a West Nile virus that was found in an outbreak in Israel in 1998. It is also very similar to a virus found in Egypt.[29]

In 1999 WNV was detected for the first time in the Western Hemisphere as a widespread bird die-off and human encephalitis outbreak in New York City and Long Island, NY. Over 60 clinical cases of WNV occurred in the New York area during late summer. Subsequent blood surveys revealed that there might have actually been thousands of sub-clinical human infections (producing few or no noticeable symptoms) with resultant antibody production.[30]

Unexpected WNV features heighten alarm

Several new or unusual features of the North American 1999 WNV outbreak alarmed epidemiologists. Simultaneously occurring human epidemics and avian outbreaks had not previously been reported. In addition, a finding of severe, diffuse muscle weakness in afflicted humans had not been described in previous WNV outbreaks overseas.[31]

Picking up on the potential of the unknown, national news media led the charge in spreading the word on the "potentially deadly" West Nile invasion. As if on cue municipal and state officials disbursed millions of dollars on ill-advised pesticide spraying.

The American Response—unprepared and over-reactive

Despite daily movement of hundreds of global bio-invaders, American health authorities were not prepared for the arrival of WNV. Overreaction by local, state and federal authorities resulted in widespread spraying of organophosphate and pyrethroid insecticides to kill adult mosquitoes. Multiple incidences of pesticide exposure to humans included spraying participants in a New York City street fair with synthetic pyrethroids.[32]

West Nile virus spreads into New England and Vermont

Over the summer and fall of 2000, reports of positive virus identification in dead birds and in mosquito populations were recorded in New York, New Jersey, Connecticut, Massachusetts, Rhode Island, New Hampshire, Pennsylvania, Virginia, Washington D.C. and Vermont. It is likely that the virus is more widespread than reports from these states suggest.[33]

Effectiveness of WNV spray programs uncertain

According to Charles H. Calisher, Ph.D., an epidemiologist specializing in the evolution of arboviruses, "It is likely that the control programs that have been employed have been less a factor this year [2000] than have the natural conditions in the northeastern U.S."[34]

Unexpected lobster and crab die-off after WNV spraying

Environmental ramifications of widespread spraying in the New York City area have yet to be fully tallied, but may include the unanticipated widespread die-off of crabs and lobsters in Long Island Sound. Initial testing of lobsters indicated high levels of pyrethroids in body tissues.[35]

Absence of WNV immunity heralds establishment

High bird mortality in 1999 along the East coast reflects the absence of WNV immunity in the Americas. A foray into mammals, such as the 62 New Yorkers with diagnosed WNV encephalitis, typically results in a dead-end for the virus, as re-transmission from mammals rarely occurs. Since migrating birds carry WNV, the New York City outbreak heralds eventual establishment in the Americas.

The Vermont Response—more cautious

The fervor of the 1999 New York City spray programs, reinforced by year 2000 detection and knee-jerk spray responses in New York, Connecticut, Massachusetts, Rhode Island, and New Hampshire, and the subsequent public outcry against spraying, convinced Vermont authorities to take a more cautious approach in setting up a Vermont response plan.

Development of State WNV Response Plan underway

Using a variety of existing mosquito control, pesticide regulation, and health emergency statutes, the Vermont Department of Health and the Vermont Department of Agriculture have jointly prepared a draft emergency response plan. The draft plan identified a range of surveillance and control options involving personnel from the Department of Health, Department of Agriculture and Agency of Natural Resources.

Establishment of a statewide surveillance program is VT goal

To avoid duplication of New York's controversial spraying programs, Vermont is directing considerable resources into WNV surveillance programs, including mosquito monitoring, avian monitoring and human health monitoring. A supplemental legislative appropriation is being sought in 2001 for expanding the Agriculture Department's existing arthropod vector surveillance program.

Synthetic pyrethroid spray programs in Vermont are planned

To keep mosquito populations low, Vermont's response focuses first on killing mosquito larvae using bacterial insecticides. To kill adult mosquitoes, aerial and ground spraying of synthetic pyrethroids is planned.

See page 9: State of Vermont Draft West Nile Virus Surveillance and Response Plan.

West Nile Virus—only one of many minor human diseases

West Nile virus historical record

Where WNV is well established and diagnosed it is recognized as a minor disease in humans. Years, even decades, may pass between outbreaks.[36] Short-lived and geographically limited human and horse WNV outbreaks in Europe occurred in the western Mediterranean and southern Russia in 1962-64, Belarus and Ukraine in the 1970's and 1980's, Romania in 1996-97, Czech Republic in 1997, and Italy in 1998.[37]

WNV epidemic in Romania resulted in low loss of life

Even where WNV has occurred in epidemics, the health impacts on human populations have been relatively small. Europe's largest documented WNV epidemic occurred in Romania in 1996. An estimated 94,000 were infected, of whom less than 400 had acute central nervous system infection serologically linked to WNV. 17 people, mostly elderly, died.[38]

WNV endemic in Israel

WNV is endemic to Israel and outbreaks have been noted sporadically. An outbreak is currently underway. According to the Jerusalem Post "...Israeli health ministry officials note that it [WNV] has been around locally for years. In the early '90's, a check of army reservists indicated that as much as 15 percent of the adult population may have been exposed to it—often without even knowing... Most WNV victims have relatively mild, flu-like symptoms.... Only rarely does it develop into encephalitis or meningitis, which can ...be fatal."[39]

One in 300,000 people bitten are at risk of WNV disease

According to the New York City Health Department, individuals bitten by a West Nile virus-carrying mosquito have a one in 300,000 chance of getting sick.[40] Evidence from other WNV epidemics suggests less than one-tenth of one percent of people bitten by infected mosquitoes develop clinical encephalitis symptoms. Less than 10% of those who develop encephalitis are at risk of severe or fatal infection.[41]

While the death of any person is a serious concern, the chance of a human becoming seriously ill from WNV is very small.

Elderly and immune-compromised most at risk

Individuals most at risk from serious encephalitis manifestations tend to be elderly, immune-compromised, or both. Examples of susceptible individuals are people receiving immune suppression therapy for cancer treatment or individuals with suppressed immunity, such as HIV infected individuals. Median age for confirmed WNV infection during New York outbreak was 68 years.[42] In Israel's current outbreak, of those who died, 7 were close to 80 years old. The latest victim, a 54-year-old woman, suffered from a disease that suppressed her immune system.[43]

The flu is far deadlier than WNV

Further evidence of the low risk associated with WNV is apparent in the New York metropolitan area. Out of ten million people, an estimated 533 to 1903 individuals were infected (based on blood surveys).[44] Seven died of WNV infection in 1999, and these were either elderly or had immune compromised systems. In contrast, more than 2,600 people in New York City die every year of the various strains of the flu.[45]

Symptoms of West Nile virus infection in humans

Human infection may produce a wide spectrum of disease from sub clinical infections to severe encephalitis resulting in death.

WNV usually produces asymptomatic infection in humans.

Most people are not aware of being infected. A mild fever, sometimes accompanied by rash, head and muscle ache and a sore throat occurs in a small percent of infected individuals.

Febrile systemic illness

After inoculation there is an incubation period of five to fifteen days. Where human immune response is weak, an abrupt onset of disease corresponds with increasing levels of viruses in the blood. Disease progression includes a sudden onset of fever with chills; headache; muscle, bone and joint pains; nausea and vomiting, accompanied by rash and swollen lymph nodes. These clinical features may be mild or very severe and prostrating. The disease is typically self-limiting and short lived (few days to a week).[46]

West Nile Encephalitis

Almost all arboviruses may infect the central nervous system, but some characteristically produce encephalitis in humans. Symptoms start with fever, progressively severe headache, nausea and vomiting, neck stiffness, paralysis, decreased consciousness, convulsions and death in severe cases (approximately 0.001% of infected individuals).

Misdiagnosis of WNV encephalitis has likely been common

Mis-diagnosis of acute as unknown encephalitis has likely been common, especially prior to widespread WNV serological tests.[47] Until recently in Israel, absence of local WNV diagnostic capability has likely meant mis-diagnosis in past encephalitis cases, and speculation is that WNV has been historically active at levels seen in the current outbreak.[48]

Antibody response to West Nile virus

Because naturally acquired immunity to WNV is not yet established in the Americas, human and animal health implications of the recent introduction of WNV into the Western Hemisphere are not yet known.[49]

Avian antibody response allows co-existence

As with other arboviruses, host immunity co-evolves with WNV, allowing both host populations and viral populations to co-exist. Presence and detection of WNV antibodies in blood serum of birds does not indicate potential for transmission of active virus by mosquitoes to humans,[50] only that individual birds have been exposed to the virus. WNV antibodies are also present in birds that have developed immunity.

Lack of immunity in the New World reflected in bird die-offs

Historically, previously unexposed birds have suffered severe initial population losses due to introduced diseases in many parts of the world.[51] When WNV enters new territory, such as the Americas, susceptible birds perish, the number of immune birds rises, and WNV eventually establishes itself at a sub-lethal level. Birds become less susceptible to mortality, and increasingly carry less virulent viruses. A decline in transmission of the virus from birds to birds and from birds to dead-end hosts, such as humans, naturally follows. Over cycles lasting decades,[52] the virus exploits unexposed hosts, and WNV flares up in new rounds of local infection.

Human antibody response to WNV is generally strong

Much like a vaccine, presence of WNV stimulates antibody production to varying degrees in humans. A person once infected with WNV acquires immunity against re-infection.[53] Most infected individuals will experience minimal symptoms and thereafter be immune.

Where WNV is endemic, human antibody production is widespread

In Madagascar, virological, serological and entomological research conducted since 1975 indicates WNV is widespread on the island.[54] Serological investigations in Madagascar in 1990 found WNV antibodies in 29.9% out of human sera tested.[55] Presence of WNV antibodies in human blood serum is considered a marker of past exposure and is not a reliable indicator of the potential for near future encephalitis outbreaks.

Following New York City's 1999 WNV outbreak, city health officials identified the epicenter as a section of northern Queens. Based on random testing among area residents, they estimated that between 533 and 1,903 individuals in the epicenter had been infected with the West Nile virus, a finding that suggested both a pervasive presence and a high percentage of asymptomatic infections.[56]

WNV immunity is shared with related viruses

WNV immunity appears to also offer a limited immunity for related mosquito borne bird viruses. Partial immunity to Japanese encephalitis appears to be imparted to monkeys[57] and humans[58] upon experimental exposure to WNV.

WNV vaccine proposed

Development of a vaccine is underway.[59] Proposed use would be by immune compromised or elderly individuals. The WNV vaccine would consist of attenuated genetically altered Japanese Encephalitis viral vaccine (WNV is a Japanese Encephalitis variation.) For individuals with healthy immune response, WNV virus provides superior immunization to itself, better than a synthesized vaccine.

Vermont Draft West Nile Virus Surveillance and Response Plan

Anticipating the arrival of WNV in Vermont in 2000, Agriculture and Health Department officials initiated a WNV response planning process, and released a draft WNV Response Plan in June 2000.[60] The final plan will be released in Spring 2001.

Premise for action

The Vermont Draft Plan states as a premise for action: “It is imperative that the State of Vermont takes a proactive approach in dealing with this potential disease threat. WNV may have serious implications for human health, as well as for both avian and equine populations. Both our domestic and wild avian populations are at risk because WNV is lethal to many species of birds.”

The Vermont Plan quotes from the New York State plan: “With a coordinated response, we can achieve the overall goal of this plan—to minimize the risk of West Nile Virus infection to humans and domestic animals by using a graded response that causes the least adverse impact on the environment.”

Three tiered course of action

The State of Vermont has set in place a three-tiered course of action (Phase I, Phase II, Phase III) to be implemented when WNV is detected in Vermont. Each phase deals with an increasing level of detection and management.

Phase I: Surveillance—No Detection

Arboviral Task Force formed

Phase I of Vermont’s WNV Response Plan was launched in 2000. An Arboviral Task Force was set up to prepare for the presence of WNV. The Task Force consists of members of the following Vermont agencies: VT Dept. of Agriculture, Food and Markets, VT Dept. of Health, VT Dept. of Forests, Parks and Recreation, VT Dept. of Fish and Wildlife, and the UVM College of Medicine.

Public education campaign

Phase I has seen the beginning of a public education campaign about WNV. Fact sheets,[61] multiple web sites, and much media air time and print space has been devoted to discussions WNV and the steps individuals can take to reduce mosquito breeding areas and protect themselves from mosquitoes.

Human health surveillance

Conducting human surveillance involves outreach by the Vermont Department of Health and the CDC to Vermont physicians.[62]

Phase I calls for recording possible health effects from pesticide exposure as part of the campaign. Information and protocols for assessing health effects will be disseminated and coordinated by the Vermont Department of Health.

Mosquito surveillance strategy is the key to appropriate response

An objective of Phase I is the development of a statewide mosquito surveillance strategy to determine larval habitat distribution and species distribution data for Vermont. Collection of this data is seen as imperative for responding to any future insect borne diseases. Insect surveillance data were not compiled by New York City authorities prior to ill-advised 1999 spraying programs. New Jersey, with a surveillance program in place, largely avoided spraying.

Phase II—Documentation of West Nile virus establishment in Vermont

Phase II is implemented when WNV is detected in or near Vermont (in any county in a neighboring state that borders on Vermont).[63]

Phase II objectives

Phase II objectives involve documentation of WNV establishment; alerting health officials, health care providers, and residents about WNV presence; and conducting health surveillance for encephalitis and viral meningitis. These processes are already underway. A role of the Arbovirus Task Force is to determine whether conditions warrant moving to Phase III.

Phase III—Implementation of Vector Management Program

According to the proposed VT Response Plan, Phase III will be initiated when the Task Force determines the potential threat of WNV in Vermont exceeds any risk associated with a "no action" level.

Phase III involves using ground and aerially applied larvicides and adulticides.

Established Vermont mosquito control program is the model

Mosquito control in the Lake Dunmore area for nuisance mosquito control has been ongoing since the Brandon, Leister, Salisbury (BLS) Insect Control District was established in 1990, and now includes the town of Goshen. The BLS District serves as the Vermont model for implementation of a mosquito control program.[64]

Note. The BLS Insect Control District has also applied the synthetic larvicide Methoprene and aerially applied Malathion and resmethrin. Breakdown metabolites of methoprene have been implicated in frog deformities in the laboratory.[65]

Use of larvicides to control mosquito larvae

Mosquito larvae are not infected with WNV, so larviciding efforts are intended to reduce local mosquito populations when infected mosquitoes are discovered. Otherwise, larviciding is largely presumptive based on potential for WNV establishment in future adult mosquito populations.

When WNV is detected in local mosquito populations, killing mosquito larvae in water habitats reduces the likelihood that synthetic adulticides will be needed. Larvicide applications typically begin in April, and must be continued until WNV is no longer detected. The American Bird Conservancy recommends larviciding only in disturbed or man-made bodies of water, which are of lesser ecological importance than naturally occurring water bodies.[66]

Three larvicides are proposed for use in Vermont

Three larvicides are proposed by the State to control mosquito larvae in waters where they breed: *Bti*, Bacillus sphaericus, and monomolecular oil.[67]

Bti—*the larvicide of choice*

The principal larvicide proposed for use is known as *Bti*. The active ingredient is a bacteria called Bacillus thuringiensis Var.israelensis.

Bti exert a toxic effect on aquatic larvae of members of the fly family.[68] *Bti* grows in the gut of fly larvae, releasing toxins and breaking down the mosquito gut wall. *Bti* must be ingested to be effective.[69]

Bti has a history of application at label rates to control mosquitoes. Despite claims that effects on applicators, livestock, or wildlife, including beneficial insects, annelid worms, flatworms, crustaceans, mollusks, fish, amphibians, reptiles, birds, and mammals are minimal,[70] effects have not been widely documented and ecosystem effects are largely unknown.

Bti application timing critical to effectiveness

To work effectively, timing of *Bti* application is extremely important. Optimal benefits are obtained when treating 2nd or 3rd instar mosquito larvae. Treatments at other development stages may provide less than desired results. Evidence that mosquito larvae reduce feeding prior to instar molting suggests that *Bti* application during these stages may result in *Bti* crystals settling out before larvae resume feeding, resulting in control failure.[71] Therefore, a disadvantage of using *Bti* is the limited treatment window available.

Bti is not problem free

Bti application problems include high cost, variable product performance, poor water penetration and subsequent dispersal problems due to wind blowing *Bti* off target, and variable lapse time before onset of mosquito larval mortality. Often treatments must be repeated due to inadequate performance.[72] For these reasons, ground applied *Bti* is preferable to aerial applications. Resistance to *Bti* is recognized as a growing potential problem.[73]

Bacillus sphaericus for use in polluted waters

A secondary larvicide for use in highly polluted waters is Bacillus sphaericus. Sewage treatment plants, storm drain traps, and manure pits and runoff ponds are examples where this larvicide would be employed. Reapplication is generally required every one to four weeks.

The sphaericus endotoxin destroys the insect's gut similarly to *Bti* and works on larvae of many mosquito genera such as Aedes, Culex, Culiseta, and Psorophora. The toxin works on the feeding larval stages and must be partially digested before it becomes activated.[74]

Monomolecular oil to coat water surfaces

A third larvicide proposed for use by Vermont insect control officials is the monomolecular oil known as Agnique. It spreads quickly over the surface of the water in a thin film. The effect is to lower the surface tension of the water, subsequently inhibiting proper orientation by larvae at the “on-water” surface and by wetting tracheal structures and causing anoxia. Larvae normally use surface tension to suspend for long periods when breathing and resting. Emerging and egg-laying adults cannot be supported on the water surface when oil is present and drown.[75]

A disadvantage of surface oil is the effect on non-target surface dwelling insects, such as mayfly nymphs and water striders. The use of surface oils is generally not recommended where non-target insects are considered to be an important resource, such as in natural areas.

Potential non-target consequences of larviciding in wetlands

Vermont wetlands will bear the brunt of larviciding

Because Vermont wetlands are mosquito breeding areas, many will be the targets of selective larviciding programs. Although the use of larvicides to control mosquito populations is preferable to use of synthetic pesticides, widespread use of fly specific larvicides and surface oils can have potential negative ecological effects.

Bti affects many aquatic fly larvae

Bti, the larvicide of choice affects the aquatic larval stage of flies (Order Diptera), such as mosquitoes, crane flies, midges, and black flies. Non-target activity on larvae of insect species normally present alongside mosquito larvae in aquatic habitats has been observed in larvae in the Order Diptera, Suborder Nematocera, Families Chironomidae (Midges), Ceratopogonidae (Biting Midges), and Dixidae (Dixid Midges). These non-target insect species, taxonomically closely related to mosquitoes and black flies, contain the necessary gut pH and enzymes to activate the *Bti* toxins.[76]

Fly larvae are integral in Vermont’s wild food webs

Fly larvae, although inconspicuous to the casual observer, exist in phenomenally large numbers in wetlands, ponds, and lakes. They are a critical component of Vermont’s aquatic food webs. Adult flies with aquatic larval stages are important food for insects, like dragonflies, as well as food for amphibians, reptiles, birds and mammals, such as bats and shrews. Broad removal of fly larval biomass inevitably ripples up the food web and results in changes to other dependent species.

Use of synthetic pyrethroids to kill adult mosquitoes

When infected birds with active West Nile virus in their blood are sufficiently present in a geographic area, re-transmission by adult mosquitoes increases, and the likelihood of human exposure also increases.

To reduce populations of infected adult mosquitoes, Vermont's WNV Response Plan calls for the targeted spraying of synthetic pyrethroids, by truck in urban settings and by air in rural areas.

Synthetic pyrethroids are more potent and stable than natural pyrethrum

Pyrethroids are based on the chemical structure of pyrethrum, derived from an extract of plants in the genus *Chrysanthemum*. Development of synthetic pyrethroids has involved extensive modifications to increase toxicity and reduce degradation by light.[77]

Mode of action: pyrethroids are neurotoxins

The principal mode of action of pyrethroids is believed to be disruption of the permeability of the nerve membranes to sodium atoms. The nervous system is strongly excited and becomes hypersensitive to stimulation. The mode of action is similar to that of DDT.[78] Site of action is the peripheral nervous system.[79]

Proposed triggers for pesticide spraying—one infected human or one horse

Although under continuous review and discussion regionally, Vermont's current proposed triggers for initiation of spraying are one of the following: one confirmed human WNV clinical infection, one confirmed horse WNV clinical infection, or multiple confirmed active bird infection with corresponding local infected mosquitoes at a level suggesting a high risk of human infection.[80] The scientific community no longer recognizes bird deaths as a trigger for the use of adulticides. CDC recommends adulticides be considered for use only where large populations of infected human biting mosquitoes are found, or other indicators of human risk are identified, and only after other control options are exhausted.[81]

The spray zone originally proposed by the Arboviral Task Force was a two-mile radius circle, an area of roughly 8,700 acres, around established WNV diagnosis.[82] This spray zone is no longer considered necessary or effective by the CDC, and will not be used by Vermont officials in determining spray zones.

Three synthetic pyrethroids have been proposed for use in Vermont's WNV program

- Resmethrin
- Sumithrin
- Permethrin

Resmethrin (Scourge)

The pesticide ingredient of Scourge is resmethrin. One formulation of Scourge consists of 4.14% resmethrin, 12.42% piperonyl butoxide and more than 80% polyethylbenzene. Manufactured by AgrEvo, owned by Hoechst and Schering. Resmethrin is a restricted use pesticide due to its acute toxicity to fish. Resmethrin was registered by EPA in 1971.

Manufacturer's warning label

Precautionary Statements: Hazards to Humans and Domestic Animals. CAUTION. Harmful if swallowed or absorbed through skin. Avoid contact with skin, eyes, or clothing.

Health indications issued by a United States government authority for Scourge (resmethrin)

- Recognized human developmental toxicant – California Proposition 65 list.
- Suspected gastrointestinal or liver toxicant – Environmental Protection Agency.
- Suspected neurotoxicant – US EPA
- "May be hazardous to the environment; special attention should be given to fish and honeybees" Center for Disease Control.

According to the International Chemical Safety Card for resmethrin, application of mists should be avoided due to risk of inhalation.[83]

Persistence

When released to the air as a fine mist, resmethrin will degrade with an estimated half-life of 25 minutes in the presence of sunlight. The photo-degradation half-life in sunlight (at water's surface or as a thin film on dry surfaces) can be as fast as several hours or less. Because spraying is planned for dusk, when flying mosquitoes are present, photo-degradation will be delayed, increasing likelihood of resmethrin settling onto the ground.

Sumithrin (Avid)

Sumithrin was registered by US EPA in1975.

Manufacturer's warning label

The following information is included on the label: "This product is toxic to fish… For terrestrial uses, do not apply directly to water, or to areas where surface water is present…"

Health indications issued by United States government authorities for Sumithrin

- Suspected gastrointestinal or liver toxicant – U.S. Environmental Protection Agency
- Suspected kidney toxicant – National Institute for Occupational Safety and Health (NIOSH) Registry of Toxic Effects of Chemical Substances.
- Suspected neurotoxicant – NIOSH Registry of Toxic Effects of Chemical Substances.
- Listed on the Toxic Release Inventory list under the Emergency Planning and Community Right-to-Know Act.
- No tolerable levels have been established – International Programme on Chemical Safety/Commission of the European Communities.

Sumithrin formulation ingredients

Avid products are typically formulated with four ingredients: sumithrin; piperonyl butoxide; polyethylbenzene, also known as heavy aromatic solvent naphtha (petroleum); and "white mineral oil," also known as hydrotreated light paraffinic petroleum distillate.

Permethrin (Aqua-reslin)

Permethrin has been registered by EPA since 1977. Permethrin is widely used in the U.S. for control of insects in agriculture. Homeowner use in yard and garden is estimated at over 18 million applications, and use in homes is estimated at over 100 million applications.[84] Permethrin is an active ingredient in flea and tick collars and shampoos, and in backyard foggers and aerosol sprays.

Manufacturer's warning label

Information on the manufacturer's product label includes: "Drift and runoff from treated areas may be hazardous to aquatic organisms in neighboring areas… Do not apply when winds exceed 10 MPH. Do not apply over agricultural crops that are used for food, forage or pasture... Avoid prolonged inhalation of spray droplets."

Adverse health effects of permethrin are well documented

Like other synthetic pyrethroids, permethrin is neuro-toxic. It inhibits a variety of nervous system enzymes, and inhibits a nervous system receptor.[85] Exposure to permethrin causes eye and skin irritation. Permethrin appears to adversely affect the immune system at exposure levels that do not cause overt toxicity.[86] Permethrin affects both male and female reproductive systems.[87] [88] Permethrin is mutagenic to mammals.[89] Permethrin is listed by EPA as a possible human carcinogen. Chronic effects have also been noted on the liver, adrenal glands, and kidneys in mice. [90]

Long permethrin half-life increases non-target risk

Vapor phase permethrin (ultra-low volume spray) degrades in the ambient environment upon exposure to sunlight. Half-life for photo-degradation is estimated at less than 10 days. Photo-degradation half-lives of 5-17 days were determined for permethrin on thin films suggesting that permethrin may be removed from the atmosphere via direct photolysis.[91]

Degradation of pyrethroids in the soil is mainly by chemical and microbial action. The rate of breakdown is highly variable depending on soil and microbial characteristics. Permethrin binds strongly to soil. A degradation half-life of 30 days was estimated for permethrin in soil,[92] a factor that results in track-in of residues to home interiors following exterior applications.

Inert ingredients in pyrethroid formulations

"Inert" ingredients also pose health risks

In mosquito sprays, pyrethroids are combined with other hazardous ingredients. Technical grade pyrethroids are formulated with carriers and solvents prior to use in residential and commercial applications. The toxicity of these other ingredients, the so-called "inerts", must be taken into consideration when assessing the toxicity of the final pesticide product.

Piperonyl butoxide—hazardous droplets

One "inert" ingredient, a synergist called piperonyl butoxide, is suspected of causing a range of human health effects including cancer, dermal irritation, renal and hepatic damage, prenatal damage, vomiting and weight loss. Piperonyl butoxide enhances the toxicity of pyrethroids by inhibiting naturally occurring enzymes that would otherwise degrade the insecticide, effectively slowing resistance to pyrethroids. Piperonyl butoxide can compose up to 90% of a pyrethroid formulation.[93]

Health indications issued by a U.S. government authority for pyrethroid "inerts"

Piperonyl butoxide

- Suspected carcinogen – Environmental Protection Agency Office of Pesticide Programs.
- Suspected gastrointestinal or liver toxicant – National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances.
- Suspected neurotoxicant – National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances.

Polyethylbenzene (heavy aromatic solvent naphtha (petroleum))

- Listed on Environmental Protection Agency's Office of Pesticide Programs' Inert Pesticide Ingredients List No 2, a list of 64 substances the EPA "believes are potentially toxic and should be assessed for effects of concern."
- Closely related to ethyl benzene, which is listed as a suspected reproductive toxicant and a suspected respiratory toxicant by the EPA.

White mineral oil (hydrotreated light paraffinic petroleum distillate)

- Listed on Environmental Protection Agency's Office of Pesticide Programs' Inert Pesticide Ingredients List No 2.

Vermont Pyrethroid application specifics

Certified application only

Application of insecticides in the WNV Response Plan will be by or under the immediate supervision of certified pesticide applicators only.

All pesticide applications for West Nile virus mosquito adulticiding are subject to 6 V.S.A Chapter 87 Section IV—Restrictions on the use and application of pesticides.

Larvicide applications require state of Vermont Aquatic Nuisance Control permits.

Local officials to make final spray determinations

The State of Vermont does not currently plan to undertake a state-run mosquito abatement program. Municipal authorities, under guidance and advisement of the Dept. of Agriculture, will be responsible for implementing mosquito control operations, much as Vermont's BLS Insect Control District does for the towns of Brandon, Leicester, Salisbury and Goshen.

Larvicide applications by air and ground

Along large wetland boundaries, larviciding will be done mainly by aerial application. For smaller targeted wetlands and manure storage pits, larvicide application will be done by hand.

Adulticide applications by air and ground over thousands of acres

Application of adulticides will be done by aerial distribution in rural areas, and by ground application from truck in urban and suburban settings. Surveillance and determination of WNV infection in adult mosquito populations will determine extent of adulticide applications.

Aerial application in rural areas

Aerial application will be done in rural areas in response to WNV confirmed presence surrounding the focal point of an outbreak. Light wind is necessary for proper dispersal of mists. Under ideal conditions, pesticide sprays are distributed for several miles downwind of release.[94]

Truck applications in residential areas

Trucks will be used only where enough roads are present to allow fairly complete spraying coverage. Areas receiving greatest spray coverage will be residential in nature. Rutland City is cited by the Task Force as a locale suited for truck spraying.[95]

Ultra-low volume insecticides kill on contact

Application of synthetic pyrethroids is by what is known as ultra-low volume (ULV). This refers to the application of concentrated pesticide active ingredient by spraying fine aerosol droplets over a large area. The droplets are very fine, and stay suspended in the zone most likely to contact adult flying mosquitoes. Mortality is achieved when flying insects contact suspended droplets.[96]

Highly specialized ULV equipment is used to disperse insecticides. Pesticides are applied in a concentrated form (referred to as technical grade) and at very low volumes. A typical application would be one to two ounces per acre.

Problems associated with use of synthetic pyrethroids

Effectiveness of spraying limited under actual circumstances

The established mosquito control program in Harris County, Texas is able to achieve a 30% efficacy rate, with rigorous testing to affirm results. To achieve a 30% rate, treatment occurs 3 nights per week in areas where mosquitoes are found to be positive for St Louis encephalitis virus. Trap experiments in residential areas in Florida, the state with the most extensive mosquito-control experience, generally show a reduction of about 30 percent after a spraying.[97]

Studies consistently show that, on average, less than one-tenth of 1 percent of the millions of droplets released during a typical spraying run actually hits a mosquito, according to David Pimentel, a professor of entomology at Cornell University and a longtime pesticide researcher. In addition, he said, if there are numerous trees, buildings or other obstructions in the area, the spray cloud will almost certainly miss large areas of the target zone, including mosquitoes inside buildings.[98]

Local atmospheric conditions determine effectiveness of spray operations.

Conditions must be ideal for aerial spraying. These conditions include spraying when mosquitoes are most active, when meteorological conditions are conducive to keeping the spray cloud close to the ground, and when inversion of temperatures and wind is not excessive (wind does not exceed 10 MPH). Application should be done at an altitude of less than 50 meters.

Spraying effectiveness limited by differing mosquito flying habits

Because each species has its own daily habits—flying times, resting patterns, and habitat requirements—the correct mosquito must be targeted for control efforts. WNV can be transmitted by seven or more mosquito species, each with different breeding, flying and biting habits.

Repeat spraying is necessary to overcome rapid mosquito rebound

Because pesticide efficacy never approaches 100% and mosquitoes from adjacent unsprayed areas emigrate into sprayed zones, repeated spraying to control adult mosquito populations is required. Spraying an area just one time is not effective.[99]

Truck spraying misses all areas away from roads, such as backyards, many probable mosquito habitats are bypassed, allowing rapid re-population, necessitating repeated spraying and undermining the initial justification for spraying.

The New York State WNV Response Plan recognizes "the likelihood that vectors in nearby areas not subject to control measures will migrate from the area if not subject to control."[100]

Resistance to Pyrethroids well documented

Insecticide use breeds insecticide resistance, and insecticide resistance has become a problem in all insect groups serving as disease vectors. Development of resistance to pyrethroids is documented in a wide variety of target pests.[101] Each resistance development is unique and involves complex patterns of exposure, survival and reproduction.[102]

Because natural selection in mosquitoes occurs over days, weeks and months due to rapid regeneration cycles, repeated pesticide spraying directly results in new generations of pesticide-resistant mosquitoes. Under ideal conditions, single applications to previously unexposed mosquito populations can achieve kill rates of 95%. Even under this extreme circumstance, selection pressure is strong for development of resistance. Reproduction by even one pesticide-resistant mosquito can result in hundreds of resistant offspring who then mate and create an entire population with genetic pesticide resistance—often in a matter of weeks.[103]

Spraying pesticides may enhance WNV infection in mosquitoes, birds and humans

Scientific evidence suggests mosquito spraying may actually enhance infection rates in the birds harboring the virus by directly compromising the avian immune response thus making them more susceptible to infection. Additionally, mosquitoes that survive insecticide exposure may be affected in ways that make them more efficient transmitters of the infectious virus.[104]

Pesticide use may result in increases in infected mosquito numbers

Studies done in New York state for mosquitoes carrying Eastern Equine Encephalitis found a 15 fold increase in mosquitoes after repeated spraying, virtually all of who were now pesticide-resistant.[105] Experts believe the pesticide induced immune suppression in all living things makes mosquitoes, birds and humans far more vulnerable to viral infections. In addition, mosquitoes exposed to pesticides can more readily get and transmit the infection due to damage to their stomach lining.[106]

Pesticide spraying may ultimately lead to increased rates of human infection

Evidence suggests the following sequence may increase the rate of human infection:[107]

1) Pesticides are sprayed over an area to knock down adult mosquitoes, resulting in
2) More subtle genetic flaws in mosquitoes, resulting in
3) More mosquitoes now vulnerable to viral infection, resulting in
4) More mosquitoes actually infected with WNV, resulting in
5) More bites on humans by WNV carrying mosquitoes, resulting in
6) More humans becoming infected with encephalitis.

Off-target drifting of pyrethroids

One consequence of using ultra-low volume mists is off-site drifting of pesticides. Because droplets stay suspended for hours, they move readily on light breezes and convection currents. This increases the likelihood that unplanned human pesticide exposures will occur. On warm evenings, ULV droplets rise and are eventually deposited elsewhere, often as dew.

Pyrethroid breakdown limited in absence of sunlight

Applications of adulticides are most effective when done during peak mosquito flying time. For *Culex spp*. this is a few hours after sunset.[108] In the absence of sunlight, little pyrethroid breakdown occurs overnight. Unexpected rain events, such as occurred after spraying in New England in 1999 and 2000, result in residual pesticides being washed into nearby surface water.

Persistence prolonged where pyrethroids are drawn into homes

With any aerial and truck spraying, much of the chemical is deposited in shaded areas, on leaves, in cracks and crevices, as well as being drawn into in air-conditioning filters and heating vents and in other places where direct sunlight does not penetrate. Pesticides settling on the ground can be tracked or can drift into people's homes, where they can persist for weeks, months or years waiting to be inhaled, eaten or absorbed through the skin.

Requests for exemption to spray programs reduce spray effectiveness

According to the Vermont Dept. of Agriculture, anyone may request exemption from WNV spray programs. Due to the large number of certified organic farms in Vermont, the Department of Agriculture is committed to working with organic farmers to ensure their farms will not be sprayed with synthetic pyrethroids.[109] Others wishing to be exempted from spray programs will need to proactively contact the Dept. of Agriculture to request exemption. The presence of exempt areas adjacent to sprayed areas will result in a patchwork of sprayed and unsprayed areas. As a result, spray efficacy may be diminished due to rapid re-infestation from unsprayed areas.

Human health effects from pyrethroid exposure

Human exposure to synthetic pyrethroids from WNV spraying is inevitable

Due to difficulty of staying indoors on spray days, and the active, highly scheduled life of Vermont's citizens, human exposure to aerial and ground application of pyrethroids is inevitable when spraying occurs.

In New York City, multiple cases of human exposure were documented, and physicians reported seeing hundreds of people with neurological complaints attributed to spraying.[110]

As reported in Audubon magazine, individuals reported that they and their children has been sprayed while playing in a park, and baseball fans watching a game were sprayed while at the ball bark.[111]

Typical of inadvertent exposures, a Manhattan woman was accidentally doused with a synthetic pyrethroid when spray crews had difficulty maintaining advertised schedules and sprayed two hours earlier than announced.[112] In another incident, a group of Manhattan residents ventured out on an evening when no spraying had been announced, only to be blanketed by mist when passing spray trucks caught them in the open.[113]

Even where individuals have requested exemptions from spray programs, inadvertent exposure occurs. In the Brandon Leicester Salisbury Insect Control District, spraying stops and starts at the property corner of individuals requesting an exemption. According to one individual who requested a spraying exemption, drift from adjacent spraying renders her request meaningless. The same individual reported she was sprayed by passing trucks while walking her dogs along the road near her home.[114]

Pyrethroids were registered by EPA prior to human health studies

Long-term human health studies on pyrethroids have not been done, and permethrin was "approved" by the EPA for use in killing mosquitoes in the 1970's years before EPA upgraded testing standards. Permethrin won't be re-evaluated for several more years by the EPA, but has been determined to be a potential carcinogen.[115]

Harmful acute effects of pyrethroid exposure

Inhalation of sumithrin can cause coughing, wheezing, shortness of breath, nausea, vomiting, runny or stuffy nose, chest pain or difficulty breathing, as well as delayed long-term neurotoxic effects, including damage to optic and peripheral nerves. Skin contact can cause rashes, itching or blisters. Young children, seniors and people with asthma are most at risk from sumithrin exposure. The Pesticide Management Education Program at Cornell University reports, "Asthmatic wheezing may be precipitated by exposure of predisposed individuals."[116]

Pyrethroids are nerve poisons

Pyrethroids are nerve poisons, referred to as neurotoxins. They affect both peripheral nerve cells and the central nervous system.[117] Although not directly toxic to nerve cells, their effect is to prolong neural excitation with potent activation of sympathetic nerves.[118]

Low dose studies show evidence of harm

A study on mice found that "low-dose exposure" to pyrethroids resulted in "irreversible changes in adult brain function in the mouse" when exposed during the growth period. This occurred at levels of exposure less than what was found to affect adult mice. The study also found "neonatal exposure to a low dose of a neurotoxic agent can lead to an increased susceptibility in adults to an agent having a similar neurotoxic action, resulting in additional behavioral disturbances and learning disabilities."[119]

Pyrethroids are endocrine disruptors

Pyrethroids have been shown to interfere with human and wildlife endocrine systems, as well as effects on reproduction and thyroid function. Sumithrin has been demonstrated to affect endocrine function and induce growth in cancer cells.[120] Chronic exposure of humans and animals to pyrethroids results in endocrine disruption relating to action of androgen.[121]

Pyrethroids and childhood brain cancer

A study of pesticides and childhood brain cancers reveals a strong relationship between brain cancers and pyrethroids used to kill fleas and ticks, according to a report published in Environmental Health Perspectives. The study concludes, “The specific chemicals associated with children's brain cancers were pyrethrins and pyrethroids (which are synthetic pyrethrins, such as permethrin, tetramethrin, allethrin, resmethrin and fenvalerate) and chlorpyrifos.”[122]

Permethrin and human immune system damage

Researchers at the National Center of Sanidid Ambiental in Madrid Spain found that the pesticide permethrin was able to induce “structural chromosome aberrations” in human immune system cells as well as in the reproductive cells in laboratory animals. The chromosome damage became apparent after 2 hours of exposure at levels of 150-200 ug/ml (micrograms per milliliter). Chromosome damage was also detected at lower levels.[123]

Pyrethroids and breast cancer

Researchers at Mt. Sinai Hospital in NY have found sumithrin induces cell proliferation of MCF-7 human breast carcinoma cells and that “each pyrethroid compound is unique in its ability to influence several cellular pathways. Pyrethroids should be considered to be hormone disruptors, and their potential to affect endocrine function in humans and wildlife should be investigated.”[124]

Other researchers in a different program at Mt. Sinai found that “[fenvalerate and] sumithrin demonstrated significant estrogenicity” and that “exposure to certain pyrethroids may contribute to reproductive dysfunction, developmental impairment, and cancer.”[125]

Pyrethroids act synergistically with insect repellents and organophosphates

Permethrin acts synergistically with the common insect repellent DEET.[126] Pyrethroids act synergistically with widely available organophosphates, resulting in increased nervous system effects.[127]

Health effects on aerial and ground applicators

Pesticide health effects on pesticide applicators have been extensively recognized, documented, and treated by health professionals.[128] Aerial applicators suffer risks from chronic exposure and from the likelihood that neurotoxic pesticides “impair coordination, balance, and other neuromotor functions essential for safe flying.”[129] Ground applicators are at risk from prolonged breathing of mists.[130]

Non-target effects of spraying synthetic pyrethroids

While many of the most serious concerns about the use of synthetic pyrethroids have to do with their impact on human health (neuro-toxic effects, immune suppression, chromosomal aberrations), environmental impacts occur as well. Sustained use of insecticides puts the environment at considerable risk.

Beneficial insects at risk from pyrethroid spraying

Vermont has hundreds of species of beneficial insects. Synthetic pyrethroids are toxic to many, including all bees and honeybees and other essential pollinators. Predatory wasps, such as those that now provide our first line of defense against defoliating caterpillars are susceptible to pyrethroids.

Butterflies, an important piece of Vermont's visual and ecological landscape, are susceptible to pyrethroid exposure.[131]

Natural mosquito predators populations knocked down and recover slowly

Aerial and ground spraying of synthetic pyrethroids to kill adult mosquitoes risks also killing off natural mosquito predators, setting the stage for larger future problems.[132]. Some mosquito predators, such as damselflies, have longer population recovery times, setting the stage for larger future mosquito outbreaks.

Effects strongest on aquatic organisms

Non-target creatures killed by pyrethroids include anything aquatic, such as crayfish, fish, and salamanders. Permethrin causes deformities and other developmental problems in tadpoles, a factor that may be related to frog deformities.[133]

LC50's (concentration of chemical in water which will kill 50% of population of aquatic test animals) is at a value less than 1ppb for many small aquatic animals. Surface dwelling insects, such as mayfly nymphs are extremely sensitive, as are zooplankton and bottom dwelling organisms, and crustaceans, such as crayfish.[134]

Pyrethroids are highly toxic to most fish; about 40% if the LC50 values for fish are less than 1ppb. Presence of synergists in pyrethroid formulations enhances toxicity to aquatic organisms.[135] Rainbow trout appear to be extremely sensitive to the neurotoxic activity of pyrethroids.[136]

Unplanned lobster and crab die-off following 2000 WNV spraying

Evidence suggests the lobster decline in Long Island Sound was a non-target effect related to pyrethroid exposure following aerial application to combat WNV in the New York City area. Heavy rains following aerial application washed pyrethroids into the sound via storm sewage drains. Lobster deaths resulted from direct mortality or by lowering lobster immune function. Preliminary tests on lobsters confirm traces of pyrethroids near fatal thresholds.[137]

Public over-reaction to the West Nile outbreak

Over-reaction by public health officials and the media guarantees public over-reaction

Media hyperbole about the "potential deadly" nature of WNV, combined with the readiness of public health officials to combat adult mosquitoes with insecticide spray programs, virtually guarantees "an irrational response to a potential threat"—in effect, the opposite of one of the stated goals of Vermont's West Nile Response Plan.

In the current WNV outbreak in Israel (2000), Yossi Inbar, deputy director general of the Environment Ministry is quoted by the Jerusalem Post: "Most local authorities who have sought permission to fog want to do it for the psychological effect. They want to appease their residents. Some of them don't even have mosquitoes, but think fogging has a preventative effect."[138]

Poisonous arsenal at our fingertips

Use of highly toxic insecticides by homeowners is likely

There is a real danger that people will be overexposed to pesticides because they apply repellents, spray insecticides at home, and then may be additionally exposed through broadcast spraying.

As soon as WNV is detected in an area, worried homeowners and parents will head for the local hardware, garden supply or grocery store to purchase insecticides.

Products now on shelves are acutely toxic

Products now on shelves for general homeowner use include synthetic pyrethroids, as wells as organophosphates such as Dursban and diazinon, and malathion. In addition, pre-formulated insecticidal aerosols and backyard foggers are sold specifically for backyard mosquito control. These compounds are both acutely toxic and pose chronic health dangers to children and adults. Organophosphates also act synergistically with pyrethroids to increase cholinesterase inhibition.[139]

Use of backyard pesticide foggers

Backyard foggers are available off-the-shelf for purchase by homeowners. Their use poses a variety of dangers to residents, neighbors and pets. Harmful effects result from inhalation of both concentrated and dilute pesticide fogs and contact with droplets that have settled on the ground. Fogging is generally ineffective—it does not kill the adult mosquito that flies in 20 minutes later. Handling and disposal of depleted containers presents additional exposure risks.

Businesses and public building managers can also spray

In addition to homeowners, businesses, such as restaurants, will be likely to spray to avoid possible risks of mosquito contact with customers. Schools, day care centers, nursing homes, hospitals, correctional facilities, and public buildings managers all have unrestricted access to a wide variety of organophosphate insecticides.

If the past and present are a guide to the future, misapplication of these compounds will be commonplace.

WNV Pesticide education is essential

An essential component of a responsible insect control program is a broad public campaign about the dangers of home insecticide use. Focus should be on non-chemical ways of avoiding mosquitoes, and information about the potential health dangers posed by excessive pesticide or repellent use should be strengthened.

Insect repellents

A wide variety of insect repellents are available to individuals. Chemical repellents provide varying degrees of protection against ticks and insects that can transmit diseases. When used improperly, however, chemical repellents may pose a risk of adverse health effects. To reduce these risks, one must carefully read and follow all label directions before each use, and not allow children to apply repellents.

DEET—the official choice

The most widespread active ingredient found in repellents is DEET (N,N-diethyl-m-toluamide). Recommendations for the use of the personal insecticide DEET have been both widespread and controversial. Virtually all public health information sources tout the use of DEET as the repellent of choice.

EPA re-registration of DEET highlights dangers to children

EPA recently re-registered DEET due to occasional adverse reactions, especially to children. EPA did not restrict use of DEET, citing the ability of consumers to read and follow label precautions. EPA advises the following precautions for use of DEET:[140]

- Read and follow all directions and precautions on this product label.
- Do not apply over cuts, wounds, or irritated skin.
- Do not apply to hands or near eyes and mouths of young children.
- Do not allow young children to apply this product.
- Use just enough repellent to cover exposed skin and/or clothing.
- Do not use under clothing.
- Avoid over-application of this product.
- After returning indoors, wash treated skin with soap and water.
- Wash treated clothing before wearing it again.
- Use of this product may cause skin reactions in rare cases.

Efficacy of DEET

Efficacy tests on mosquitoes indicate that products with higher DEET concentrations provide marginally improved repellency. Overall, data indicate that the use of repellents containing concentrations of DEET greater than 30 percent increases exposure and the risk of adverse reaction, with little or no increased benefit in protection.[141]

Toxic reactions to children from DEET exposure

Although DEET is considered the most effective mosquito repellent, toxic reactions can occur from the use of DEET.[142] Some children have experienced adverse health reactions after applications of DEET. These health problems can be severe. Generalized seizures have been temporally associated with the use of DEET-containing insect repellent on skin. The toxic effects on the brain have been characterized by agitation, weakness, disorientation, ataxia, seizures, and coma and, in three cases resulted in death.[143]

Effective non-DEET mosquito protection alternatives widely available

Other non-DEET insect repellents have been evaluated for efficacy and shown to be effective.[144] [145]

Examples of non-DEET repellents include commercial repellents made from natural root, flower, herb and essential oils, and homemade repellents. Many are available as retail products. The Internet is a good source for non-DEET insect repellents. Consumers need to be cautious about synthetic chemical ingredients in non-DEET repellents, which can include non-traditional repellents such as skin care lotions.

Mosquito proof clothing

Polyester insect mesh clothing and headnets are already widely used by individuals sensitive to black fly and mosquito bites. Their use can be recommended for immune compromised individuals when WNV is detected in an area.

Use of Mosquito Magnets

Increasingly used by municipalities, Mosquito Magnet, manufactured by American Biophysics Corp., is a non-toxic method for removing mosquitoes from a one acre area. The machine emits a plume of carbon dioxide, heat, and moisture to naturally attract mosquitoes and other blood-sucking insects, and then draws them into a trap where they perish. Due to the high cost (around $1000), widespread use may be restricted to public facilities and private businesses looking to protect customers.[146]

Bug zappers

Electric high voltage insect traps using “black” or ultraviolet light provide little in the way of mosquito control. The primary effect is to kill insects indiscriminately, including mosquito predators.

Electronic mosquito repellers

Electronic mosquito repellers use ultrasound technology that matches the wing speed vibration (noise) of a male mosquito. Female mosquitoes instinctively steer clear of the aggressive male mosquito. The sound causes the female mosquito to stay away from the area. Electronic mosquito repellers also emit the frequency of the wing beat of a dragonfly, a predator the female naturally avoids.

Citronella candles

The effectiveness of citronella candles depends on the amount of citronella in the candle, the number of candles being used, the number of mosquitoes in the area and the wind direction. Citronella candles should not be burned indoors. Citronella plants are not considered to be effective against adult mosquitoes.

Other ongoing arbovirus vector control programs

Mosquito control has a long history in the United States. A sample of current programs follows:

Harris County, Texas (Houston)—St. Louis encephalitis is the focus of an active monitoring program, enabling identification of infected areas up to a month before any human cases are detected. As a result, Harris County has not resorted to use of aerial pesticides for years, saving the county millions of dollars. Effective monitoring allows pathogen detection weeks before humans are affected.[147]

Leon County, Louisiana— Due to local presence of Eastern Equine Encephalitis and St. Louis encephalitis, the county undertakes ongoing mosquito control efforts. The primary approach is on education, surveillance, larviciding and source reduction. Adulticiding is done by truck and hand held sprayer only (no aerial applications), using two pyrethroids.

New Jersey West Nile approach— The New Jersey State Mosquito Control Commission was established in 1956. A mosquito control network and arbovirus surveillance programs have been maintained throughout the years, principally to keep mosquito populations low and identify and control mosquitoes carrying Eastern Equine Encephalitis. As a result of WNV detection, NJ's programs are undergoing enhancement throughout the state. Surveillance includes the use of sentinel chicken flocks placed strategically in each county and monitored for presence of WNV antibody; collection, antibody testing and release of sparrows and starlings; and the use of crow morbidity/mortality and testing. NJ considers source reduction to be the most efficient method of mosquito control. A natural predator, the mosquitofish (*Gambusia iffinis*) is raised and selectively used to control mosquito populations and reduce the need for pesticides

Hedera, Israel—Once seriously plagued by malaria infested mosquitoes, Hedera is now largely mosquito free, without the use of chemical pesticides. Targeted larviciding using *Bti*, combined with other ecological pest control methods, such as mosquito habitat reduction, fish which eat mosquito larva, and the use of an organic based oil spray, known as MLO, have reduced mosquito populations and brought financial savings to the town.[148]

State of Florida—Both insect control authority and protection of environmentally sensitive habitats are legislatively mandated. Chronic effects of insect control pesticides on humans, wildlife and Florida ecosystems are officially recognized, as are the effects on beneficial species. Florida law mandates that mosquito control methods on biologically productive state lands shall impose the least hazard to fish, wildlife, and other natural resources protected or managed in such areas, and requires environmental management agencies to take a conservative approach to insect vector management on state lands.[149]

Connecticut— State and local governments have implemented an aggressive West Nile virus surveillance and response plan. Focusing on source reduction, the program also uses aerial and truck applications of resmethrin to knock down adult mosquitoes. The Department of Environmental Protection has established a registry for people who are chemically sensitive to be alerted prior to pesticide spray operations.

New York City—In 1999, following several deaths from WNV encephalitis, New York City launched widespread ground and aerial pesticide spraying programs using Malathion and pyrethroids. In the absence of a mosquito surveillance program, initial spraying was done in the fall when mosquito flying was minimal. Municipal authorities assured the public that the spaying was safe. Numerous reports of inadvertent pesticide exposure were recorded, and a number of lawsuits against the city were filed.

VPIRG statements for responsible West Nile virus public response

Personal mosquito protection is the goal for immune compromised individuals

Individuals with compromised immune systems and the elderly can take maximum personal precautions, such as using mosquito-proof bed nets to assure protection from adult mosquitoes. Others, especially the elderly, can wear protective clothing and judiciously apply insect repellents.

Steps individuals can take to minimize local mosquito populations[150]

- Identify and remove items that can hold stagnant water, such as old tires.
- Empty water from buckets, toys, and containers, and store them in places where they will not collect rain.
- Drill holes in the bottoms of recycling bins and any other containers that must be kept outdoors.
- Drain the water from birdbaths, fountains, wading pools, plant saucers, plant pots and drip trays twice a week.
- Check for other ways water may be collecting around your house, such as puddles beneath air conditioners.
- Clean out gutters and fix gutters that sag or do not drain completely. Check for areas of standing water on flat roofs.
- If you have a swimming pool, outdoor sauna, or hot tub, make sure rainwater does not collect on the cover.
- Clear vegetation and trash from any drains, culverts, ponds or streams near your house so that water drains properly.
- Mow grass and trim shrubs to minimize hiding places for adult mosquitoes.
- Eliminate standing water in basements and crawlspaces.
- To minimize the likelihood of being bitten inside your house, make sure window and door screens fit properly and replace outdoor lights with yellow "bug lights."
- To avoid being bitten outdoors, wear hats, long sleeves and long pants in the evenings, when mosquitoes are most active.
- People should be especially careful when in “mass gatherings” where the CO_2 given off by the crowd attracts more mosquitoes from greater distance.

Implementing the precautionary principle in the face of unknown pesticide risks

▲ WNV is not a highly contagious killer disease, though it is a public health concern for the elderly and immune compromised individuals.

▲ Insect borne diseases are best dealt with by using non-chemical means. Toxic chemical controls are often less effective than mechanical and least toxic control methods.

We do not adequately understand the human health and environmental impacts of pesticide use.

▲ The more we know about the health effects of pesticides the more dangerous we understand them to be.

▲ Because of the unknowns surrounding the health impacts of pesticides and other toxics, we must take a cautious approach—dispassionately evaluating the real level of danger presented by a pest problem (WNV), and using all available preventive measures and non-chemical interventions before considering chemical controls.

▲ Synthetic pyrethroids, proposed by the State of Vermont for WNV mosquito spraying, are neuro-toxic—that is they disrupt the function of the nervous system. They have also been shown to interfere with the immune and endocrine systems, affecting reproduction and thyroid function. An epidemiological study found that use of pet flea products containing pyrethroids and plant-derived pyrethrins was associated with an elevated incidence of childhood brain cancer.[151] A recent lab study found breast cancer cells to proliferate more rapidly when exposed to pyrethroids.[152]

▲ Over the past century we have repeatedly found that we have underestimated the damaging effects of toxic chemicals released to the environment. The proposal to spray square miles of areas where WNV infected mosquitoes, birds, and humans are found will potentially expose non-target insects, wildlife, people and domestic animals to significant risks associated with the pesticides proposed. Where substances have been shown to have the potential for health and environmental damage, they should not be used.

VPIRG proposals for responsible mosquito control

To ensure a uniform Statewide approach to managing mosquito populations associated with West Nile virus and protect the environment and human health from the unintended effects of synthetic mosquito control agents, VPIRG believes all public health and mosquito management authorities must agree to a binding set of conditions prior to initiation of any WNV spray programs. The conditions include the following components:

1. Full public participation in the development of the State of Vermont Surveillance and Response Plan

Prior to approval and adoption of the Vermont Plan, regional public hearings should be held to solicit formal public comment on the development of the WNV Response Plan.

2. Public hearings prior to implementation of local mosquito control programs

Prior to adoption of municipal mosquito control programs, public hearings should be held by municipal officials to solicit public comment on proposed actions. Public officials should make formal response to all public comments.

3. Coordination of local mosquito control programs by the Department of Agriculture

To ensure optimal regional mosquito management and preclude ineffective mosquito management practices, to ensure local control programs are in full compliance with guidelines established by the Vermont Task Force and to respect no-spray requests, the Department of Agriculture should advise and coordinate all local mosquito control programs.

4. Raise the proposed pesticide spray triggers

The current proposed triggers for initiating spraying of pesticides to kill adult mosquitoes may be too low and do not indicate the potential for a WNV "epidemic". These triggers need to be balanced with comprehensive information on the presence of WNV in area mosquito populations, and an indication that human health is actually at risk, prior to initiation of any local spray program.

5. Complete public disclosure of all pesticide formulation ingredients and breakdown metabolites

Vermont citizens have the fundamental right to know about any pesticides used in their towns, their communities, and on neighboring properties. Full disclosure to the public about all chemical components of each and every insecticide used is basic information citizens must have to protect themselves from potentially harmful exposure. The WNV Response Plan should honor the public's right to know.

Inert ingredients

Prior to use of mosquito control agents, the complete public disclosure of all ingredients, including inert ingredients, should be made for all approved pesticides.

Breakdown metabolites

Because metabolites can be both persistent and toxic, knowledge of their presence is essential for reducing public exposure. Wherever known, full disclosure of active half-lives and resultant breakdown metabolites should be available to the public.

6. Development of binding mosquito control parameters

All municipal mosquito control programs should fall within binding parameters established by the Task Force. These guidelines should include, but not be limited to:

Implementation of tiered approach to local mosquito control prior to use of any synthetic pesticides, including pyrethroids

VPIRG advocates adherence to an ecologically sound tiered approach to mosquito management, such as promoted by Beyond Pesticides/ National Coalition Against the Misuse of Pesticides.[153] Prior to implementation of any adulticide program, all least toxic approaches should be exhausted locally. These should include proper preventive action aimed at public education and programs to reduce backyard-breeding areas, accompanied by biological controls to disrupt mosquito larvae.

Ensure that mosquitoes are infected with WNV prior to initiating adulticide control operations

Large local or regional mosquito populations are not a sufficient reason to initiate spraying of pyrethroids. Given the low risk to the general population of serious West Nile infection, broadcast spraying of pyrethroids to knock down adult mosquitoes may have limited ability to achieve desired results. Because pyrethroids are toxic to a wide variety of non-target organisms, large-scale spraying of pyrethroids can cause unpredictable alterations in Vermont's ecological balance. Therefore, pyrethroids should be applied only when infected mosquito populations are verified. Statistically valid sampling of mosquito populations for presence of WNV should be done prior to initiating any spray programs.

Determine species of infected mosquitoes

Because several species of mosquito can transmit WNV, and each has unique daily flying habits, determination of infected species is critical to ensuring effective spray timing and location.

Adulticide use only with pre-approval from the Department of Agriculture and Department of Health

Use of adulticides by municipal heath authorities should be preceded by written approval of the Department of Agriculture and the Department of Health.

Use least toxic insecticides from pre-approved list only

If spraying for mosquitoes must occur, botanical-based chemicals such as synthetic pyrethroids must be used. Although synthetic pyrethroids are neurotoxic and pose a special threat to those with allergies they are the least toxic widely available adulticide. All pesticide products and formulations should be pre-approved by the Vermont Arboviral Taskforce.

Use of insect control agents not specified in the Vermont Plan should be prohibited.

Use of insect control agents (larvicides and adulticides) by municipalities other than those specific agents approved for use by the Task Force, should be prohibited.

Meeting pesticide label requirements

The Department of Agriculture should monitor pesticide application procedures to verify that there is strict compliance with all label instructions, including prohibitions on spraying and drifting of certain pesticides over bodies of water, as well as requirements for storage and disposal, and equipment cleaning.

Pesticide residue monitoring

Wherever requested, the Dept. of Agriculture should monitor pesticide levels in the environment through wipe tests of outdoor and indoor surfaces, checking air conditioner filters, evaluating water samples from bodies of water, and conducting soil and food residue tests from gardens and farms.

7. Pesticide injury preparedness

The Department of Health should alert hospitals about the health effects of the pesticides used as well as the health effects of commonly available off-the-shelf pesticides and provide information on appropriate treatment measures for those who are injured by pesticide use.

Pesticide injury monitoring

The Department of Health should monitor the public for adverse health effects resulting from WNV mosquito control pesticides by setting up a hotline for receiving reports, collecting hospital records and requiring physician reporting of pesticide exposure incidents.

8. Establishment of a spray hot-line and web site

A telephone hot line and web site should be established for the public to find out where and when pesticides are being used in WNV control efforts and to record public concerns and requests for spray exemptions.

9. Public notification prior to implementation of mosquito control measures

The general public should be notified one week in advance of any spraying so that they may take precautionary measures to avoid exposure.

Prior special notification

Individuals requesting personal advance notification should be contacted individually by the Department of Agriculture one week in advance of spraying. The Department of Health should advise hospitals, schools and others with especially vulnerable populations to take extra precautionary measures to prevent pesticides from making their way inside buildings.

10. Honor requests for exemptions from insect control programs

All requests to be exempt from pesticide spray programs received in writing, over the telephone, via e-mail or personal communication to the Department of Agriculture and or the local municipal authority should be considered legal notification. Protection from pesticide drift should be the ultimate responsibility of the certified pesticide applicator following notification by the Department of Agriculture.

11. Restrict spraying around Vermont schools

Because many Vermont schools stand near or on waterlogged ground, spraying around school buildings is highly likely. Spraying should include an allowance to spray only with case-by-case authorization by the Department of Health and should not be left to the discretion of individual principals or superintendents.

12. Non-target monitoring before and after mosquito control implementation

The Agency of Natural Resources should monitor aquatic organisms and non-target beneficial organisms both before and after aquatic larviciding and chemical adulticiding to measure effectiveness and non-target effects. Sampling of target and non-target populations before and after spraying should be statistically valid.

13. Public education campaign about minimizing pesticide exposure

A broad education campaign should proceed all adulticide operations, including education of the public about precautionary measures such as leaving the area, closing windows, turning off air intake on cooling or air handling equipment, bringing lawn furniture, toys and other outdoor items inside and covering swimming pools.

Hospitals, schools and other buildings with especially vulnerable populations should be advised to take extra precautionary measures to try to prevent pesticides from making their way inside buildings.

14. WNV pesticide education about home insecticide and repellent use

A broad public campaign about the dangers of home insecticide use should be an essential component of a responsible insect control program. Focus should be on non-chemical ways of avoiding mosquitoes, and information about the potential health dangers posed by excessive pesticide or repellent use should be strengthened. Because DEET-containing repellents can be hazardous to children, special emphasis should be given to alternatives to DEET for children.

CONCLUSION

Unlike the flu and many other common viruses, West Nile virus (WNV) poses a low risk to general public health. Mosquito management programs using pesticides should therefore be reflective of the low risk inherent in WNV.

When WNV enters new territory, such as the Americas, susceptible birds perish, the number of immune birds rises, and WNV eventually establishes itself at a sub-lethal level. Birds become less susceptible to mortality, and increasingly carry less virulent viruses. A decline in transmission of the virus from bird to bird and from birds to dead-end hosts, such as humans, naturally follows. Over cycles lasting decades, the virus exploits unexposed hosts, and WNV flares up in new rounds of local infection.

Evidence from WNV outbreaks suggests less than one-tenth of one percent of humans bitten by infected mosquitoes develop clinical encephalitis symptoms. Less than 10% of those who develop encephalitis are at risk of severe or fatal infection.

Individuals most at risk from serious encephalitis manifestations tend to be elderly, immune-compromised, or both.

Much like a vaccine, presence of WNV stimulates antibody production to varying degrees in humans. A person once infected with WNV acquires immunity against re-infection.

Because surveillance is the key to the development of any appropriate response, the comprehensive mosquito and vector surveillance program currently underway by the Vermont Department of Agriculture should be strengthened.

A public education campaign aimed at removal of backyard mosquito breeding habitats and steps individuals can take to protect themselves should precede all mosquito control programs.

Where WNV has been historically present, targeted use of bacterial larvicides can be implemented to reduce adult populations.

When WNV is detected in adult mosquito populations, area residents who are most susceptible to WNV infection should be advised to take maximum personal precautions.

When infected adult mosquito populations are detected in proximity to inhabited areas, larvicides should be used to reduce area mosquito populations. If public health officials opt to use pesticides, ground application of least toxic pesticides should be undertaken in a highly targeted manner, following strict application protocols, and respecting public requests not to be sprayed.

Because the use of synthetic pyrethroids has inherent human health risks, every effort should be made to minimize human exposure and track reported health effects of exposure.

Surveillance efforts should include statistically valid sampling to determine presence of WNV, spray efficacy and to track non-target ecological effects.

Ultimately, it remains to be seen how much of a threat the West Nile Virus poses to public health. We already know the many dangers from sustained use of insecticides. The State of Vermont and its citizens must now recognize these risks before they embark on a costly war on mosquitoes.

Glossary

Adulticide: pesticides used to control insects at the adult stage of their development.

Antibody: (*plural* antibodies) a protein produced in the body in response to the presence of an antigen, for example, a bacterium or virus. Antibodies are a primary form of immune response in resistance to disease and act by attaching themselves to a foreign antigen and weakening or destroying it.

Antigen: a substance, usually a protein, on the surface of a cell or bacterium that stimulates the production of an antibody.

Arbovirus: any of various RNA viruses transmitted by arthropods. "*A*rboviruses" are not a taxonomic group. (From arthropod borne virus)

Arthropod: invertebrate animals in the phylum Arthropoda, a group that have a segmented body, jointed appendages, a usually chitinous exoskeleton molted at intervals, and a dorsal anterior brain connected to a ventral chain of ganglia. Includes insects, mites, ticks, spiders, and crustaceans.

Beneficial insects: Insects serving a known useful function, such as wasps that prey on defoliating caterpillars, bees that pollinate agricultural crops, or dragonflies that eat mosquitoes.

Diptera: a large group (Order) of insects abundant almost everywhere and include mosquitoes, flies, gnats and midges. Diptera (from di = two and ptera = wing) are distinguished from other Orders of insects by having only one pair of wings, with the second pair instead reduced to small knobbed structures that aid in maintaining equilibrium. Diptera include vectors of diseases such as malaria, yellow fever, filariasis, and the arboviruses, while others are important pollinators and pest predators and parasites. Diptera undergo complete metamorphosis, which means that they change form during development (see *larvae* and *pupae.*)

Drift: the airborne movement of a pesticide during or immediately after its use or application to a site not intended for its use or application.

Efficacy: effectiveness or ability to produce the desired results. In the case of mosquito adulticides, expressed as the percentage of adult mosquitoes killed by a single application.

Encephalitis: an inflammation of the brain that can be caused by viruses and bacteria, including viruses transmitted by mosquitoes.

Endemic: belonging or native to a particular region and thus continuously present at varying frequencies of occurrence; (*endemic* diseases; an *endemic* species).

Endocrine disruptors: Chemicals that mimic or block hormones or interfere with normal hormone activity, often at extremely small doses.

EPA: The United States Environmental Protection Agency.

Epidemic: affecting or tending to affect a disproportionately large number of individuals within a population, community or region at the same time, *i.e.*, at a higher than expected frequency. Used to refer to diseases that are not consistently present in an area, and which are brought in from the outside or a temporary increase in the number of cases of an *endemic* disease.

Epizootic: relating to an epidemic of animal disease. Used to describe an outbreak of disease that rapidly affects many animals in a given area at the same time.

Half-life: For pesticides, the length of time required for one-half of the active ingredient to degrade into a secondary chemical metabolite.

Host: a living animal or plant affording subsistence or lodgment to a virus or parasite. Parasites are organisms that are metabolically dependent upon the host.

Immune system: the interacting combination of all the body's ways of recognizing cells, tissues, objects, and organisms that are not part of itself, and initiating the immune response to fight them.

"Inert" ingredient: Any ingredient in a pesticide formulation other than the active ingredient, including carriers, emulsifiers, and synergists. Inert ingredients are often hazardous.

Insecticide: a chemical substance used to kill insects.

Larva, (plural **larvae**): the immature stages between the egg and the pupa, of an insect with complete metamorphosis. The form of the insect during the larval stage differs radically from the adult.

Larvicide: pesticides used to control insects at the larval stage of their development.

Metabolite: a chemical resulting from breakdown of another chemical. Also referred to as a breakdown product.

Neurotoxin: a substance that damages, destroys, or impairs the functioning of nerve tissue.

Non-target organism: An organism negatively affected by a pesticide application other than the intended target.

Organophosphates: A family of insecticides containing phosphorus and having a common mode of action: they inhibit a nerve chemical called cholinesterase. Examples are malathion, diazinon, and Dursban.

Parasite: generally a plant or animal that lives on or in another, usually larger, host organism in a way that either harms or is of no advantage to the host.

Persistence: In regard to pesticides, the length of time pesticides remain active. For many pesticides, persistence results in unintended non-target effects. Persistence is enhanced when pesticides are carried indoors.

Pesticide: a chemical substance used to kill pests, especially insects.

Piperonyl butoxide: A synergist added to pyrethroids to enhance toxicity and inhibit naturally occurring insect enzymes that would otherwise degrade the pyrethroids.

Precautionary principle: the concept of risk avoidance—when there is suspicion of potential harm, taking action to prevent harm before there is full scientific understanding of the consequences.

Predation: a way of life in which food is primarily obtained by the killing and consuming of animals. *E.g.*, mosquito fish are "predaceous" on mosquitoes.

Pupa, (plural **pupae**): the immature stage between the larva and adult, of an insect with complete metamorphosis.

Pyrethrin: either of two oily liquid complex organic compounds obtained from pyrethrum flowers and used as a contact insecticide. Despite their quick lethal action on insects, pyrethrins are valued for their relatively low toxicity to humans and other animals, but they are highly allergenic.

Pyrethroid: a synthetic complex organic compound with insecticidal properties similar to those of pyrethrin. Synthetic pyrethroids are extensively modified to enhance toxicity and reduce degradation by light.

Reservoir: a population or group of populations of vertebrate or invertebrate hosts in which the pathogen is endemic (*i.e.*, permanently maintained at varying levels). Although human populations can form reservoirs of this kind, the concept is usually applied to non-human populations, from which the pathogen periodically escapes, causing individual infections or epidemics in humans or epizootics in other animals.

Residue: A portion of a pesticide or its breakdown metabolite remaining on surfaces after treatment.

Resistance: the ability to remain unaltered by the damaging effect of a pesticide. Genes for pesticide resistance may be passed to subsequent generations.

Seroconversion: the production of antibodies in response to an antigen.

Seropositive: having or being a positive serum reaction, especially in a test for the presence of an antibody.

Seroprevalence survey: a standard tool used by public health officials. “Sero” (from the Latin *serum)* refers to testing of blood for antibodies to an infectious organism; “prevalence” refers to the percentage of people with a particular characteristic at a given point in time. A "West Nile Virus Seroprevalence Survey" is a survey to determine the percentage of persons with antibodies to West Nile Virus, at a given point in time, within the geographic area sampled.

Surface water: any river, stream, creek, brook, reservoir, marsh, swamp, pond, lake, spring and any other body of water, whether natural or artificial.

Synergism: the phenomenon in which the combined action of two chemicals, for example, pesticides and/or inert ingredients, is greater than the sum of their effects individually.

Synergist: A chemical added to a pesticide to enhance its effect.

Ultra-low-volume insecticides: High potency insecticides applied at very small droplet size over a large area. In the case of pyrethroids, contact by mosquitoes with individual droplets results in death.

Vector: carrier of a pathogen from one host to another. In describing any species as a vector, what is meant is that some individuals of a species are capable of carrying a given pathogen. Because of genetic differences within species, it is generally not the case that all individuals in a species are competent vectors.

Virus: a minute particle that lives as a parasite in plants, animals, and bacteria and consists of a nucleic acid core within a protein sheath. Viruses can only replicate within living cells and are not considered to be independent living organisms.

References

[1] Vermont Department of Health. October 13, 2000. *Hermit Thrush Tests Positive for West Nile Virus in Putney; No Spraying Planned*. www.state.vt.us/health/record/westnile5.htm.

[2] Vermont Department of Health. Disease Control Bulletin. *West Nile Virus*. Volume 2, Issue 2, 2000.

[3] Peter Galbraith Vermont State Epidemiologist, in presentation to the Vermont Pesticide Advisory Council. August 14, 2000. Montpelier, VT.

[4] Phil Benedict, Vermont Department of Agriculture, Food and Markets in presentation to the Vermont Pesticide Advisory Council. August 14, 2000. Montpelier, VT.

[5] USDA Animal and Plant Health Inspection Service http://www.aphis.usda.gov/oa/wnv/wnvstats.html

[6] Hubalek Z, Halouzka J. *West Nile Fever—a Reemerging Mosquito-Borne Viral Disease in Europe*. Emerging Infectious Diseases. Center for Disease Control. 2000. Internet: www.cdc.gov/ncidod/EID/vol5no5/hubalek.htm.

[7] Center for Disease Control, Division of Vector Borne Infectious Diseases. Vertebrate Ecology. http://www.cdc.gov/ncidod/dvbid/westnile/birds&mammals.htm.

[8] Work TH, Hurlbut HS, Taylor RM. Indigenous wild birds of the Nile Delta as potential West Nile circulating reservoirs. Am J Trop Med Hyg. 1955;4:872-8.

[9] Pitigoi D, Popa MI, Streinu-Cercel A. *The epidemiological process of West Nile viral infection*. Bacteriol Virusol Parazitol Epidemiol. 1998 Oct-1998 Dec.31; 43(4):281-8.

[10]Center for Disease Control and Prevention. 2000.Information posted on internet at www.cdc.gov/ncidod/dvbid/westnile/q&a.htm#map.

[11] Rappole, John H., Scott R. Derrickson, and Zdenek Hubalek. 2000. Migratory Birds and Spread of West Nile Virus in the Western Hemisphere. Emerging Infectious Diseases. 6(4). www.cdc.gov/ncidod/eid/vol6no4/rappole.htm. 2000.

[12] Tsai, T.F., Popovici, F., Cernescu, C., Campbell, G.L., and Nedelcu, N.I. 1998. West Nile encephalitis epidemic in southeastern Romania. Lancet 352(9130 Sept 5):767-71.

[13] Cernescu, Costin, Nicolae-Ion Nedelcu, Gratiela Tardei, Simona Ruta, and Theodore F. Tsai. 2000. Continued Transmission of West Nile Virus to Humans in Southeastern Romania, 1997-1998. The Journal of Infectious Diseases 181 (Feb 8):710-712.

[14] Press Release. October 25, 2000. U.S. Department of the Interior, U.S. Geological Survey. Reston, VA.

[15] Karabatsos N, editor. International catalogue of arboviruses, including certain other viruses of vertebrates. 3rd ed., and Supplements 1986-98. San Antonio: American Society of Tropical Medicine and Hygiene. 1985.

[16] Gochfeld M. *Public Panic over West Nile Virus*. American Butterflies. Summer 2000. Available on the Internet at http://naba.org/wnvirus.html.

[17] Blackburn NK, Reyers F, Berry WL, and Shepard AJ. *Susceptibility of dogs to West Nile virus: a survey and pathogenicity trial*. J Comp Pathol. 1989 Jan; 100(1):59-66. Rec#: 480.

[18] Chomsky MS. The Enduring West Nile Virus. Newsday. 10/03/2000. http://email.rutgers.edu/cgi-bin/wa?A2=ind0010&L=arbo_bytes&F=P&S=&P=500.

[19] West Nile Virus Confirmed for the First Time in Bats. State of New York Department of Health August 30 Press Release. http://www.cfe.cornell.edu/risk/WNV-LArchive/8-31-00.html.

[20] Kostyukov MA, Alekseev AN, Bulychev VP, Gordeeva ZE. Experimental infection of *Culex pipiens* with West Nile virus by feeding on infected *Rana ridibunda* frogs and its subsequent transmission. Med Parazitol. 1986;6:76-8.

[21] Rodhain, F, Petter J, Albignac R, Coulanges P, Hannoun C. Arboviruses and lemurs in Madagascar: experimental infection of *Lemur fulvus* with yellow fever and West Nile viruses. Am J Trop Med Hyg. 1985; 34:816.

[22] State of Rhode Island Response Protocol for Mosquito-Borne Diseases. Amended March 2001. http://www.health.state.ri.us/wnv/protocol.htm.

[23] Hubalek Z, Halouzka J. *West Nile Fever—a Reemerging Mosquito-Borne Viral Disease in Europe*.[23] Emerging Infectious Diseases. Center for Disease Control. 2000. Internet: www.cdc.gov/ncidod/EID/vol5no5/hubalek.htm.

[24] Han Linda L., Florin Popovici, James P. Alexander Jr., Velea Laurentia, Leslie A. Tengelsen, Costin Cernescu, Howard E. Gary, Jr., Nicolae Ion-Nedelcu, Grant L. Campbell, and Theodore F. Tsai. 1999. Risk factors for West Nile virus infection and meningoencephalitis, Romania, 1996. Journal of Infectious Diseases 179:230-233.

[25] Ibid.

[26] Graham, AC, Turmel JF, and Darsie RF. New State Mosquito Records for Vermont including a Checklist of the Mosquito Fauna. Jounal of the American Mosquito Control Association. Vol.7, No.3. Sept. 1991.

[27] Tsai, T.F., Popovici, F., Cernescu, C., Campbell, G.L., and Nedelcu, N.I. 1998. West Nile encephalitis epidemic in southeastern Romania. Lancet 352(9130 Sept 5):767-71.

[28] Lanciotti RS et al. 1999. Origin of the West Nile Virus Responsible for an Outbreak of Encephalitis in the Northeastern United States. Science 286 (17 December): 2333-2337.

[29] Lipkin I, Jie X, Brieze T, Jordan I, Rambadt A, Hen Chang Chi, Mackenzie J, Hall R, and Scharret J. Dec. 1999. Lancet. unisci.com/stories/19994/1203994.htm.

[30] University of Florida, Institute of Food and Agricultural Sciences, Gainesville, FL 32611 Public Health Pest Control Manual – Pests and Public Health. www.ifas.ufl.edu/~pest/vector/chapter_02.htm.

[31] Vermont Department of Health. Disease Control Bulletin. *West Nile Virus*. Volume 2, Issue 2, 2000.
[32] Callahan E. UNTENABLE CHOICES. Westchester Weekly. www.westchesterweekly.com/articles/mosquitos.html
[33] Health Canada, Population and Public Health Branch. West Nile Virus Activity: States of New York, New Jersey, Connecticut, Massachusetts, Rhode Island, and New Hampshire. www.hc-sc.gc.ca/hpb/lcdc/osh/wnv2000_e.html.
[34] FWD from ProMED mail posting from Charles H. Calisher, Ph.D., Sept 21, 2000. www.cfe.cornell.edu/risk/WNV-LArchive/9-27-00b.html.
[35] Martindale, Diane. Dead in the water: Lobsters are the first victims of New York's pesticide frenzy. New Scientist. 12 August 2000. www.newscientist.com/news/news.jsp?id=ns225127.
[36] Gochfeld M. *Public Panic over West Nile Virus*. American Butterflies. Summer 2000. http://naba.org/wnvirus.html.
[37] Hubalek Z, Halouzka J. *West Nile Fever—a Reemerging Mosquito-Borne Viral Disease in Europe*.[37] Emerging Infectious Diseases. Center for Disease Control. 2000. www.cdc.gov/ncidod/EID/vol5no5/hubalek.htm.
[38] Tsai, T.F., Popovici, F., Cernescu, C., Campbell, G.L., and Nedelcu, N.I. 1998. West Nile encephalitis epidemic in southeastern Romania. Lancet 352(9130 Sept 5):767-71.
[39] *Israel unfazed by virus that panicked New York*, Jerusalem Report; August 28, 2000; page 5.
[40] No Spray Coalition http://www.nospray.org/WNVodds.html.
[41] Gochfeld M. *Public Panic over West Nile Virus*. American Butterflies. Summer 2000. http://naba.org/wnvirus.html.
[42] Vermont Department of Health. Disease Control Bulletin. *West Nile Virus*. Volume 2, Issue 2, 2000.
[43] WEST NILE VIRUS, HUMAN CASES – ISRAEL. Source: Associated Press, Fri 15 Sep 2000
[44] New York City Department of Health Bureau of Communicable Disease. West Nile Virus Questions and Answers on Serosurvey. March 21, 2000. www.ci.nyc.ny.us/html/doh/html/wnv/wnvqa.html.
[45] Gonzalez, Juan. *City' Cure's More Deadly than the Disease*. New York Daily News. July 25, 2000.
[46] Vermont Department of Health. Disease Control Bulletin. *West Nile Virus*. Volume 2, Issue 2, 2000.
[47] Lev, Boaz, M.D. Deputy General, Israeli Health Ministry. Quoted in Jerusalem Post. September 22, 2000.
[48] Frucht, LE. *Anatomy of an Epidemic*. Jerusalem Post. September 22, 2000.
[49] Center for Disease Control and Prevention. *Epidemic/Epizootic West Nile Virus in the United States: Guidelines for Surveillance, Prevention, and Control*. From a workshop cosponsored by the Department of Health and Human Services, CDC and the U.S. Department of Agriculture. Fort Collins, CO. November 8-9, 1999.
[50] Rappole, John H., Scott R. Derrickson, and Zdenek Hubalek. 2000. Migratory Birds and Spread of West Nile Virus in the Western Hemisphere. Emerging Infectious Diseases. 6(4). www.cdc.gov/ncidod/eid/vol6no4/rappole.htm. 2000.
[51] Pesticide Use and West Nile Virus. Position statement from the American Bird Conservancy. www.abcbirds.org.
[52] Gochfeld M. *Public Panic over West Nile Virus*. American Butterflies. Summer 2000. http://naba.org/wnvirus.html.
[53] New York State Department of Health. Communicable Disease Fact Sheet. Arboviral Infections www.health.state.ny.us/nysdoh/consumer/arbo.htm
[54] Fountenille D, Rodhasin F, Digoutte JP, Mathiot C, Morvan J, Coulanges P. Transmission cycles of the West Nile virus in Madagascar, Indian Ocean. Ann Soc Belg Med Trop. 1989 Sep; 69(3):233-43.
[55] Morvan J, Chin LH, Fontenille D, Rakotoarivony I, Coulanges P. Prevalence of antibodies to West Nile virus in youngsters from 5 go 20 years old in Madagascar. Bull Soc Pathol Exot. 1991; 84(3):225-34.
[56] Chomsky MS. The Enduring West Nile Virus. Newsday. 10/03/2000 http://email.rutgers.edu/cgi-bin/wa?A2=ind0010&L=arbo_bytes&F=P&S=&P=500.
[57] Goverdhan MK, Kulkarni AB, Gupta AK, Tupe CD, Rodrigues JJ. Two way cross-protection between West Nile and Japanese encephalitis virus in bonnet macaques. Acta Virol. 1992 May; 36(3):277-83.
[58] Rodrigues FM, Mohan Rao CV, Mandke VB, Pinto BV, Pavri K. Neutralizing antibody response to Japanese encephalitis inactivated mouse brain vaccine among laboratory personnel. Trans R Soc Trop Med Hyg. 1986. 80(2):301-4 Rec#580.
[59] Oravax Developing West Nile Virus Vaccine http://biotech.about.com/industry/biotech/library/weekly/aa081800a.htm.
[60] State of Vermont, Department of Agriculture and Department of Health. Draft West Nile Virus Surveillance and Response Plan. June 2000.
[61] Vermont Department of Health. FACT SHEET: Preventing West Nile Virus. August 7, 2000.
[62] Vermont Department of Health. Disease Control Bulletin. *West Nile Virus*. Volume 2, Issue 2, 2000
[63] State of Vermont, Department of Agriculture and Department of Health. Draft West Nile Virus Surveillance and Response Plan. June 2000.
[64] Vermont Department of Agriculture, Food and Markets. *Mosquito Management*. www.state.vt.us/agric/pidmosquito.htm.
[65] La Clair JJ, Bantle JA, Dumont J. 1998. Photoproducts and Metabolites of a Common Insect Growth Regulator Produce Developmental Deformities in Xenopus. *Environmental Science & Technology*. Vol. 32, No. 10 Pp. 1453-1461.

[66] Pesticide Use and West Nile Virus. Position statement from the American Bird Conservancy. www.abcbirds.org.
[67] Jon Turmel, Vermont State Entomologist, in presentation to the Vermont Pesticide Advisory Council. August 14, 2000. Montpelier, VT.
[68] Florida Coordinating Council on Mosquito Control. 1998. Florida Mosquito Control: The state of the mission as defined by mosquito controllers, regulators, and environmental managers. University of Florida. Chapter 5. Mosquito control benefits and risks. www.ifas.ufl.edu/~veroweb/whitep/ch5.htm
[69] Ibid.
[70] Ibid.
[71] Ibid.
[72] Vermont Department of Agriculture, Food and Markets. *Mosquito Management*. On the Internet at www.state.vt.us/agric/pidmosquito.htm.
[73] Goldman, I.F., J. Arnold and B.C. Carlton. 1986. Selection for Resistance to *Bacillus thuringiensis* subsp. *israelensis* in Field and Laboratory Populations of the Mosquito *Aedes aegypti.* J. Invert. Path. 47:317-324.
[74] Florida Coordinating Council on Mosquito Control. Ibid.
[75] Ibid.
[76] Ibid.
[77] Meuller-Beilschmidt, Doria. *Toxicology and Environmental Fate of Synthetic Pyrethroids*. Journal of Pesticide Reform. 1990 Vol.10 No. 3.
[78] Vijverberg, HPM and J van den Bercken. 1990. Neurotoxicological effects and the mode of action of synthetic pyrethroids. *Crit. Rev. Toxicol.* 21:105-126.
[79] Meuller-Beilschmidt, Doria. *Toxicology and Environmental Fate of Synthetic Pyrethroids*. Journal of Pesticide Reform. 1990 Vol.10 No. 3.
[80] Peter Galbraith Vermont State Epidemiologist, in presentation to the Vermont Pesticide Advisory Council. August 14, 2000. Montpelier, VT.
[81] State of Rhode Island Response Protocol for Mosquito-Borne Diseases. Amended March 2001. http://www.health.state.ri.us/wnv/protocol.htm.
[82] Phil Benedict, Vermont Department of Agriculture, Food and Markets in presentation to the Vermont Pesticide Advisory Council. August 14, 2000. Montpelier, VT.
[83] Center for Disease Control. International Chemical Safety Card. Resmethrin. ICSC:0324. www.cdc.gov/niosh/ipcs0324.html
[84] Whitmore RW, Kelly JE, Reading PL. 1992. National home and garden pesticide use survey. Final report, Volume 1: Executive summary, results, and recommendations. Research Triangle Park, NC: Research Triangle Institute.
[85] Ramadan AL, et al. 1988. Action of pyrethroids on $GABA_A$ receptor function. *Pest. Biochem. Physiol.* 32:97-105.
[86] Blaylick AL, et al. 1995. Suppression of cellular immune responses in BALB/c mice following oral exposure to permethrin. *Bull. Environ. Contam. Toxicol.* 54:768-774.
[87] Eil C, Nisula BC. 1990. The binding properties of pyrethroids to human sin fibroblast androgen receptors and sex hormone binding globulin. *J. Steroid Biochem.* 35:409-414.
[88] Spencer F, Berhane Z. 1982. Uterine and fetal characteristics in rats following a post-implantational exposure to permethrin. *Bull. Environ. Contam. Toxicol* .29:84-88.
[89] Barrueco C, et al. 1994. Induction of structural chromosomal aberrations in human lymphocyte cultures and CHO cells by permethrin. *Terat. Carcin. Mutag.* 14:31-38.
[90] U.S. EPA Office of Pesticide Programs. Health Effects Division. June 24, 1997. Tox one liners: Permethrin. Washington, DC.
[91] Spectrum Hydrolysis Chemical Abstract: Permethrin. www.speclab.com/compound/c5264553.htm
[92] Ibid.
[93] Piperonyl Butoxide Technical. www.bugpage.com/pbo.html
[94] Erstfeld KM. Environmental fate of synthetic pyrethroids during spray drift and field runoff treatments in aquatic microcosms. Chemosphere. Vol. 39 No. 10 pp1737-1769. Oct. 1999.
[95] Jon Turmel, Vermont State Entomologist, in presentation to the Vermont Pesticide Advisory Council. August 14, 2000. Montpelier, VT.
[96] Jon Turmel, Ibid.
[97] Parsons, RE. Effectiveness of Spraying - Putting the Record Straight. Nov. 10, 2000. Harris County Mosquito Control. Houston, TX. Communication to West Nile listserv archive. www.cfe.cornell.edu/risk/WNV-LArchive/11-14-00.html
[98] Fagin, Dan. Doubts about spraying—some experts call it ineffective against West Nile virus. Newsday. November 7, 2000.
[99] Gillett, James. Cornell Center for the Environment. Reported by James, R. *Cornell: Pesticides wrong reply to West Nile threat—Ecologists say spraying is risky to humans. More studies are needed, they add*. Syracuse Post-Standard. Sept. 21, 2000.
[100] New York State West Nile Virus Surveillance Agency Response Triggering Thresholds. www.health.state.ny.us/nysdoh/westnile/final/appendixa.htm
[101] Cox, Caroline. *Permethrin—Insecticide Factsheet*. Journal of Pesticide Reform. 1998. Vol. 18, No.2.
[102] Brogdon, William G. and Janet C. McAllister. Insecticide Resistance and Vector Control Emerging Infectious Diseases 1998. 4 (4 October-December):605-613.
[103] NCAMP. 2000. The effectiveness of mosquito adulticiding.
[104] Haas, George. West Nile virus, spraying pesticides the wrong response. American Bird Conservancy, October 23, 2000. http://email.rutgers.edu/cgi-

bin/wa?A2=ind0010&L=arbo_bytes&F=PP&S=&P=158 8.
[105] Journal of the Am Mosquito Control Assoc, Dec; 13(4):315-25, 1997 Howard JJ, Oliver New York State Department of Health, SUNY-College ESF, Syracuse 13210, USA. www.chem-tox.com/brevard/index.htm.
[106] Ibid.
[107] Pressinger R (M.Ed.) Evidence Suggests a Possible Link. 2000. www.chem-tox.com/brevard/index.htm.
[108] Florida Coordinating Council on Mosquito Control. 1998. Florida Mosquito Control: The state of the mission as defined by mosquito controllers, regulators, and environmental managers. University of Florida. Chapter 6. Mosquito control benefits and risks. www.ifas.ufl.edu/~veroweb/whitep/ch6.htm.
[109] Jon Turmel, Vermont State Entomologist, in presentation to West Nile Virus Panel Discussion, November 1, 2000, Carpenter Auditorium, Given Building, UVM, Burlington, VT.
[110] "Officials Defend Spraying to Curb West Nile Virus". New York Times. April 1, 2000. B3.
[111] Boyle, Robert. Flying Fever. Audubon. July-August 2000.
[112] James, R. *Cornell: Pesticides wrong reply to West Nile threat—Ecologists say spraying is risky to humans. More studies are needed, they add*. Syracuse Post-Standard. Sept. 21, 2000.
[113] Personal communication with the author. 02/17/01.
[114] Personal communication with the author. 02/17/01.
[115] Office of Pesticide Programs, U.S. Environmental Protection Agency. 1979. Tolerances and exemptions from tolerances for pesticide chemicals in or on raw agricultural commodities; permethrin. *Federal Register* 44(81): 24287-24288.
[116] No-spray Coalition. Pyrethroid pesticides are health hazards. http://nospray.freeyellow.com/technical.html.
[117] Bloomquist, Jeffery R. Insecticides: Chemistries and Characteristics. In E.B. Radcliffe and W.D. Hutchinson [eds.], Radcliffe's IPM World Textbook, http://ipmworld.umn.edu University of Minnesota, St. Paul. 1996.
[118] Ray D.E., Forshaw P.J. Pyrethroid insecticides: poisoning syndromes, synergies, and therapy. J Toxicol Clin Toxicol. 2000. 38(2):95-101.
[119] Department of Environmental Toxicology. Uppsala University, Sweden. Neurotoxicology 1997;18(3):719-26
[120] Go, Vera, Joan Garey, Mary S. Wolff, and Beatriz G.T. Pogo. 1999. Estrogenic Potential of Certain Pyrethroid Compounds in the MCF-7 Human Breast Carcinoma Cell Line. Environmental Health Perspectives 107(3): 173-177.
[121] Eil C., Nisula B.C. 1990. The binding properties of pyrethroids to human skin fibroblast androgen receptors and to sex hormone binding globulin. Journal of Steroid Biochemistry 35(3-4):409-14.
[122] Pogoda JM and Preston-Martin S. "Household Pesticide sand Risk of Pediatric Brain Tumors," Environmental Health Perspectives, Vol. 105, No. 11 (November 1997), pages. 1214-1220.
[123] Teratogenesis, Carcinogenesis, and Mutagenesis, 14:31-38, 1994. www.chem-tox.com/brevard/index.htm
[124] Go, Vera, Joan Garey, Mary S. Wolff, and Beatriz G.T. Pogo. 1999. Estrogenic Potential of Certain Pyrethroid Compounds in the MCF-7 Human Breast Carcinoma Cell Line. Environmental Health Perspectives 107(3): 173-177.
[125] Garey, J and MS Wolff, Biochemical and Biophysical Research Communications 1998; 2551: 3: 855-9.
[126] Abou-Donia, MB et al. 1996. Neurotoxicity resulting from coexposure to pyridostigmine bromide, DEET, and permethrin: Implications of Gulf War chemical exposures. *J Toxcol. Environ. Health* 48:35-56.
[127] Abiola FA et al. 1988. Cholinesterase depression among Senegalese crop protection workers exposed to organophosphorus pesticides. *Bull. Environ. Contam. Toxicol.* 41:483-488.
[128] Reigart JR, Roberts, JR. Recognition and Management of Pesticide Poisonings. USEPA. 1999.
[129] Cantor, Kenneth P. and Warren Silberman. Mortality Among Aerial Pesticide Applicators and Flight Instructors: Follow-up from 1965-1988. American Journal of Industrial Medicine. 1999, 36(2): 239-247.
[130] Gonzalez J. Skeeter Spray Nightmare. NY Daily News. 1/24/2001.
[131] Gochfeld M. *Public Panic over West Nile Virus*. American Butterflies. Summer 2000. Available on the Internet at http://naba.org/wnvirus.html.
[132] Florida Coordinating Council on Mosquito Control. 1998. Florida Mosquito Control: The state of the mission as defined by mosquito controllers, regulators, and environmental managers. University of Florida. Chapter 9. Mosquito control benefits and risks. www.ifas.ufl.edu/~veroweb/whitep/ch9.htm.
[133] Cox, Caroline. *Permethrin—Insecticide Factsheet*. Journal of Pesticide Reform. 1998. Vol. 18, No.2.
[134] Smith, Tara M., and Glenn W. Stratton. Effects of synthetic pyrethroid insecticides on non-target organisms. *Residue Reviews* 1986. 97:93-120.
[135] Kallaji, M. Mosquito/black fly adulticide (Brand Name Scourge ® proposed for aerial spray applications in the Adirondack Park. Memorandum, New York Department of Law and Environmental Protection Bureau, March 1990, #676.
[136] Eshleman AJ, Murray TF. Pyrethroid insecticides indirectly inhibit GABA-dependent super(36)CI super(-) influx in synaptoneurosomes from the trout brain. Neuropharmacology. 1991. Vol.30 No. 12A pp1333-1341.
[137] Martindale, Diane. Dead in the water: Lobsters are the first victims of New York's pesticide frenzy. New Scientist. 12 August 2000. www.newscientist.com/news/news.jsp?id=ns225127.
[138] Inbar, Yossi. Deputy Director General of Israeli Environment Ministry. Quoted in the Jerusalem Post. September 22, 2000.
[139] Meuller-Beilschmidt, Doria. *Toxicology and Environmental Fate of Synthetic Pyrethroids*. Journal of Pesticide Reform. 1990 Vol.10 No. 3.

[140] Reregistration of the Insect Repellent DEET. www.epa.gov/pesticides/citizens/deet.htm.
[141]The Pesticide Management Education Program at Cornell University. N,N-diethyl-m-toluamide (Deet) Background Information http://pmep.cce.cornell.edu/profiles/insect-mite/ddt-famphur/deet/deet-background-info.html.
[142] Fradin, Mark S. Mosquitoes and Mosquito Repellents: A Clinician's Guide 1998. Annals of Internal Medicine June 1, 1998. 128(June 1): 931-940.
[143] The Pesticide Management Education Program at Cornell University. N,N-diethyl-m-toluamide (Deet) Background Information http://pmep.cce.cornell.edu/profiles/insect-mite/ddt-famphur/deet/deet-background-info.html.
[144] Lindsay, L.R., G.A. Surgeoner and J.D. Heal. Field Evaluation of the Efficacy of Three Druide Reg. Citronella-Based Repellents to Protect Against Aedes Species Mosquitoes in Ontario: Final Report. 8 pp. 1996. Unpublished.
[145] Rutledge, L.C . and R.K. Gupta. Variation in the protection periods of repellents on individual human subjects: an analytical review. Journal of the American Mosquito Control Association 1999. 5 (3 Sept.): 348-55.
[146] Mosquito Magnet™—Taking Back Neighborhoods One Yard at a Time. www.mosquitomagnet.com/main.html.
[147] Environmental Advocates, New York Public Interest Research Group, New York Coalition for Alternatives to Pesticides. *Towards Safer Mosquito Control in New York*. January, 2000.
[148] Frucht, LE. *Anatomy of an Epidemic*. Jerusalem Post. September 22, 2000.
[149] Florida Coordinating Council on Mosquito Control. 1998. Florida Mosquito Control: The state of the mission as defined by mosquito controllers, regulators, and environmental managers. University of Florida. Chapter 9. Mosquito control benefits and risks. www.ifas.ufl.edu/~veroweb/whitep/ch9.htm.
[150] U.S. Environmental Protection Agency. “Mosquitoes: How to control them.” www.epa.gov/pesticides.citizens/mosquitoes.htm.
[151] Pogoda JM and Preston-Martin S, “Household Pesticides and Risk of Pediatric Brain Tumors,” Environmental Health Perspectives, Vol. 105, No. 11 (November 1997), pages. 1214-1220.
[152] Go, Vera, Joan Garey, Mary S. Wolff, and Beatriz G.T. Pogo. 1999. Estrogenic Potential of Certain Pyrethroid Compounds in the MCF-7 Human Breast Carcinoma Cell Line. Environmental Health Perspectives 107(3): 173-177.
[153] Beyond Pesticides/National Coalition Against the Misuse of Pesticides. *Approaches to Dealing with Mosquito Borne Diseases*. Pesticides and You. Vol. 19, No. 4, 1999-2000.