Overkill

Why Pesticide Spraying for West Nile Virus in California May Cause More Harm Than Good

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Acknowledgments

"We should no longer accept the counsel of those who tell us that we must fill our world with poisonous chemicals; we should look about and see what other course is open to us."

Rachel Carson, *Silent Spring*, 1962

The most important person to acknowledge in this paper is Rachel Carson. Her book *Silent Spring* is the wellspring from which this continuing work to protect the public from toxic pesticides flows.

This report is heavily indebted to earlier editions that described problems with pesticide spraying for West Nile Virus control in Maine and Massachusetts. The Maine Environmental Policy Institute board of directors (Kevin Mattson, Tom Federle, Matt Scease, and Susie O'Keefe) supported the first edition, which benefited from the careful review of Heather Spalding, Rob Baldwin, Mitchel Cohen, Sharon Tisher, RussellI Libby, Kathleen McGee, George and Laura Appell, Paul Donahue, Mitch Lansky, Will Everitt, Kim DeFeo, and Elizabeth Spalding. Without their advice and generosity this report would not have been possible.

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In addition, we would like to acknowledge numerous scientists and public education staff at California mosquito and vector control agencies who provided timely, localized information about preparations for West Nile Virus already ongoing in the state. In researching this report, we found that these men and women, on the front lines of protecting public health from West Nile Virus in California, are highly sensitized to the risks entailed by indiscriminate pesticide spraying, and are working hard to educate and activate the public to help take preventative measures before the virus arrives in the Golden State.

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Table of Contents

Executive Summary

Since its emergence in New York City in 1999, West Nile Virus (WNV) has spread rapidly across the United States. The disease, borne by wild birds and transferred to humans by bird-biting mosquitoes, is likely to reach California shortly. If and when WNV does arrive, California communities must be prepared to respond in a manner that prevents harm to human health and the environment. In doing so, California can and should avoid the massive pesticide spraying programs that have been triggered in other states at the first sign of West Nile Virus.

Broadcast pesticide spraying, by truck or aerial application, has not been proven effective in curbing WNV:

- The Centers for Disease Control and Prevention have stated that ground and aerial spraying targeted at adult mosquitoes is one of the least effective mosquito control techniques.
- Northeastern communities (Boston, NYC) that first responded to WNV with massive spraying subsequently scaled back their use of adulticides, prioritizing preventative measures and establishing stricter criteria to limit adulticide spraying.
- Despite three years of widespread spraying to control WNV, no scientific studies have demonstrated that such spraying has effectively reduced the human risk of infection.

Spraying may cause more harm than good:

- Pesticide spraying may actually increase the number of mosquitoes by killing off insect predators such as dragonflies that feed on mosquitoes and their larvae.
- Pesticide spraying may increase infection rates by leading mosquitoes to develop resistance, live longer, exhibit more aggressive biting behavior, and become more susceptible to infection by WNV.
- Pesticide spraying may create a false sense of security, diminishing public participation in preventative public health measures that are necessary to effectively reduce the risk of

contracting WNV. Such measures include wearing protective clothing and helping reduce mosquito habitat by eliminating stagnant water that serves as a breeding ground for mosquitoes.

Pesticide spraying will expose human beings and nontarget organisms to chemicals known to affect human health and the environment:

- For spraying to be effective at all, it must be timed during the hours when the mosquitoes are most active (for most species, the early evening). However, these same times entail the greatest risk of exposure to the general population.
- The chance of any one individual becoming seriously ill from exposure to West Nile may be significantly lower than an individual's chance of becoming ill from pesticide exposure. For example, in 1999 there were 59 known cases of meningitis due to WNV infection in New York City, and 187 individuals who reported experiencing illness after malathion exposure.

California's current West Nile Virus Response Plan is overly permissive of dangerous and ineffective pesticide spraying:

- Current pesticides approved for mosquito control in the state include organophosphates (malathion) and pyrethroids (Pyrethrin, Sumethrin, Resmethrin) known to have serious human health impacts.
- Human health risk assessment studies, conducted to show these pesticides are theoretically "safe" if applied correctly, routinely fail to account for errors in application rates and vulnerability of certain populations, such as infants and the elderly.

To ensure minimal environmental and human health impact, and maximum effectiveness in mosquito control, the state plan should be revised to:

- Include strict parameters limiting the use of health-threatening pesticides.
- Include specific benchmarks to help promote public outreach, communication, and education activities essential for a preventative public health strategy.

In addition, local mosquito and vector control agencies, which will have significant decision-making power to choose among mosquito control options, should immediately initiate a public process in which concerned community members can be involved in outreach and education about mosquito prevention activities as well as the establishment of strict local thresholds to reduce or eliminate the use of pesticide sprays in mosquito control.

Introduction

If and when West Nile virus hits the West Coast, officials are prepared to pull out the big guns. California mosquito control, now quelling larvae with environmentally compatible hormones and bacteria, would expand to include air and ground spraying with insecticides to kill adult mosquitoes, a state health official said.

Wall Street Journal Aug 13, 2002[1]

The mosquito-borne West Nile Virus has traveled across the nation as far as the Rockies and Washington State, and is expected to eventually reach California. When it does, local leaders and mosquito vector control districts may be sorely tempted to "pull out the big guns," supplementing normal mosquito control programs with widespread aerial and ground spraying of toxic pesticides. Considering the toxicity of such pesticides to human beings, the ecological damage they may cause, and their lack of proven effectiveness in curbing West Nile, mounting such an offensive may pose a greater threat to public health than the West Nile Virus itself.

As the disease has spread rapidly throughout the nation, states and municipalities have been forced to scramble to develop emergency control plans for mosquitoes. Too often, this crisis management has relied on spraying entire neighborhoods, fields, and water bodies in an attempt to wipe out adult mosquitoes.

Only after the initial crisis has subsided have health officials and local leaders

taken time and resources to develop effective control plans emphasizing mosquito surveillance, prevention, and public education—and ensuring a response appropriate to the level of risk that West Nile Virus actually poses to most people.

Fortunately, California communities are in a unique position to avoid the overkill that has characterized the response to West Nile Virus in so many parts of the country. California has had time to absorb the lessons from WNV control in the northeastern and Gulf states. Furthermore, California has a robust infrastructure in place to prevent mosquito-borne diseases, several of which are endemic to the Golden State.

More than 50 mosquito control districts have been established throughout the state, with budgets ranging from several hundred thousand to several million dollars. These districts rely on guidelines from the California Department of Health, which has designed a West Nile Virus response plan focused on preventative measures that limit the need for pesticide spraying—prioritizing elements of an ideal strategy to effectively control mosquito populations while minimizing the spraying of harmful (and largely ineffective) pesticides.

While the state plan includes guidance about how to determine when pesticide spraying is appropriate, local agencies are left to decide when and where to do so. Ultimately, community leaders, health experts, and concerned citizens will need to work on the local level to ensure that the agencies, and the public, are not forced to make a false choice between "doing something" to stop WNV by spraying pesticides, or allowing West Nile to spread by not using pesticides. Rather, the true choice is between addressing West Nile Virus with rational control measures that have been proven effective or spraying pesticides that may do more harm than good.

Background

The 2002 WNV epidemic in the U.S. was the largest arboviral meningoencephalitis epidemic documented in the Western Hemisphere and the largest reported WNME [West Nile Meningoencephalitis] epidemic. Epizootic and epidemic activity was most intense in the central U.S., especially in the Great Lakes region, and extended to the West Coast [indicating] complete transcontinental movement of WNV within 3 years.

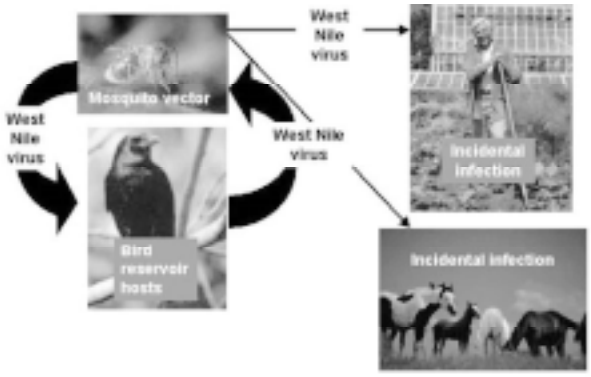
Centers for Disease Control, *Morbidity and Mortality Weekly*, December 20, 2002

Transmission of WNV

Mosquitoes transmit WNV to humans after biting infected birds, the primary hosts of WNV. In addition to humans, horses, bats, and other small mammals can all serve as alternate hosts. There is some evidence that amphibians such as frogs can host WNV as well.[2] The WNV transmission cycle is depicted in Figure 1.

WNV is in a family of arboviruses (arthropod-borne viruses). It is closely related to Western Equine encephalitis and St. Louis encephalitis, mosquito-borne diseases for which many states have already developed mosquito control programs.

Figure 1. West Nile Virus Transmission Cycle



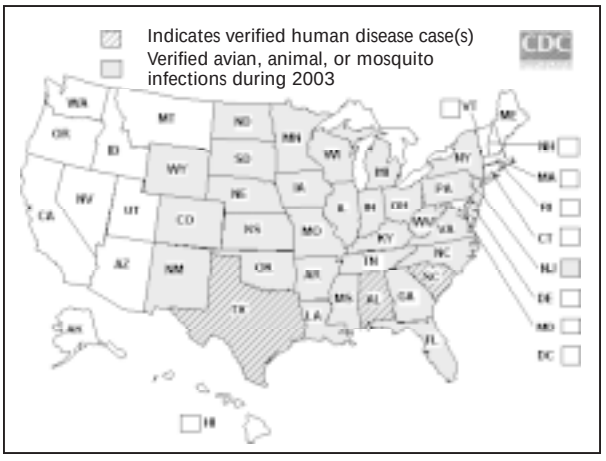
The Westward Spread of West Nile Virus

WNV originated in Africa, from which it spread to the Mediterranean, the Middle East, and parts of Asia. In 1999, it emerged in the Western Hemisphere for the first time in New York City. Infected wild birds carried the disease up and down the Eastern Seaboard, then westward through the Gulf States and up to the Rockies.[4] By the end of 2002, WNV had been detected in 2,289 counties in 44 states across the US, an increase from 359 counties in 27 states and Washington, D.C. in 2001.[5]

Experts now believe WNV will never be eradicated from the United States but rather will become endemic throughout the country in areas where related illnesses such as Western Equine encephalitis and St. Louis encephalitis are found. According to the Centers for Disease Control and Prevention (CDC), from 1964 to 1998, there were 122 confirmed human St. Louis encephalitis cases in California.

Over the past three years, WNV has demonstrated its ability to adapt to different types of mosquitoes — the vectors that transfer the virus from one host to another — allowing WNV to thrive in Illinois' long summer days as well as the hot, humid weather of Louisiana, Mississippi and Texas.[6] The map in Figure 2 depicts states where infected birds, mosquitoes, or animals have been discovered as of July 2003.

Figure 2. West Nile Virus in the United States as of July, 2003



The Public Health Impact of West Nile Virus

Rates of Human Infection by WNV

As the disease has spread across the country, the number of people infected by West Nile has also steadily increased. From 1999 to 2001, the CDC confirmed 149 cases of human illness and 18 deaths attributed to WNV. Last year, as the disease traveled to the Midwest and South, the number of laboratory-confirmed human infections grew to 4,156, including 284 deaths.[7]

In 2002, the Midwest was especially hard hit by WNV. A provisional analysis by the CDC estimated that 5 states experienced 64% of the nation's known WNV illness in 2002: Illinois, Michigan, Ohio, Louisiana, and Indiana. The first four of these states, together with Texas, accounted for 67% of reported meningitis resulting from West Nile infection.[8]

The data show that WNV poses a small but real risk to the general population. In New York State, where the disease first emerged, studies showed that less than one-tenth of one percent of people bitten by infected mosquitoes evinced any symptoms of the disease, and even fewer exhibited serious symptoms such encephalitis or meningitis, in which the brain or its casing becomes inflamed.[9] A Louisiana study found that in St. Tammany Parish, eight people in 100,000 showed any WNV symptoms (also less than one-tenth of one percent), but of

those, a significant number developed encephalitis, and the risk of death was 4 in 1 million.[10]

In general, elderly and immuno-compromised individuals face the greatest risk of serious illness associated with a West Nile infection. According to a CDC analysis of WNV cases from January through November 2002, the median age of WNV-infected people was 55 years, and the median age of people who experienced meningitis was 59 years. Of the 2,354 people with meningitis, 199, or 9%, died; in addition, 2 elderly people (more than 80 years old) died of the normally less-serious West Nile Fever. The median age of those who died from West Nile-associated illness was 78 years.[11]

Many believe that these infection rates will subside as West Nile Virus becomes "endemic" to the United States, and will be characterized by low baseline infection rates interrupted by sporadic outbreaks. In Africa, where West Nile Virus has been recognized for more than sixty years and where it is widespread, very few human epidemics have been identified. The same has been observed in the United States with related infections, such as St. Louis encephalitis and Eastern equine encephalitis, where 30 or more years may pass between human outbreaks.

New York City's Experience with WNV and Mosquito Control

In the summer of 1999, a physician noted a cluster of patients in Queens, New York City, who were thought to be infected with St. Louis encephalitis. This was later determined to be WNV, the first known emergence of the disease in the United States.[12]

City officials, lacking a robust mosquito control plan, followed CDC recommendations to embark on an aerial insecticide spraying program. A $5 million program of repeated aerial applications of malathion, a pesticide related to chemicals developed for military use in World War II, ensued. Some neighboring counties sprayed heavily as well, some even in the absence of confirmed human infection. Other counties did not spray.

This emergency management measure occurred with minimal assessment of the relative risks to human health associated with exposures to the sprayed insecticides versus those of contracting WNV. Indeed, little was known about WNV or whether the outbreak could be limited geographically by intensive aerial spraying.

During the first spray season, 187 people reported health symptoms associated with malathion exposure to New York City's Poison Control Center.[13]

In 2000, after significant public opposition to aerial spraying and hundreds of complaints from people reporting pesticide exposure, the NYC Health Department switched from its aerial campaign to largely ground-based spraying of Anvil, a pyrethroid.[14] A private contractor hired by the city sprayed this pesticide (which consists of the active ingredients sumithrin and piperonyl butoxide) in a two-mile radius around places where WNV infections or infected dead birds were reported.

By 2001, new data indicated that spraying should be further restricted to a one-mile radius, and even then only as a "last resort." Furthermore, city officials switched to an emphasis on prevention. "While last year we had a formulaic and somewhat reflexive approach . . . this year we're going to look very carefully to determine where the greatest risk to people is," City Health Commissioner Dr. Neal Cohen told a *New York Post* reporter.[15] In a separate interview with the *New York Times*, city officials stated: "To reduce the reliance on pesticides in the battle against West Nile virus, the city will use a more conservative, concentrated approach to spraying this summer."[16]

Infection Rates Case Study: A Closer Look at NYC

Surveys of blood samples taken from New Yorkers have revealed that many people infected with the virus never evinced any symptoms. A New York City Health Department survey of blood samples taken from people who lived in northern Queens, the epicenter of the 1999 outbreak, showed that 19 out of 677 tested positive for the virus, but none had become seriously ill, and all either reported no symptoms or mild illness, such as a low-grade fever.

The survey's statistical analysis concluded that between 1.2 percent and 4.1 percent of the 46,000 residents (533 and 1,903 people) in that three-square-mile area had probably been infected. Of the infected group, four people in the sample had non-specific aches, pains or fever. The others presented no symptoms.[17]

However, some people did become ill from WNV, and some deaths were recorded. Out of New York City's population of more than 7 million, 62 people — or less than .0009% — became ill with the virus, and 7 died (one in one million).

While this is a real and quantifiable public health impact that should not be dismissed, one must question whether the pesticide spraying campaign that New York City embarked upon was an appropriate response to the West Nile threat.

A comparison of WNV infection rates to rates of influenza in New York City in 1999 can provide some context, revealing that 2,474 individuals in New York City died from influenza or pneumonia in 1999, representing 400 times the number of WNV mortalities.[18]

Preparing for WNV in California

We fully expect that, over time, the virus will make it to the West Coast. What the timing of it will be is unknown at this time. It's unknown whether the virus will make it to California or the West Coast this year or next year or the year after that. It's completely a matter of conjecture.

Dr. Peterson, Medical Epidemiologist,
CDC Center for Infectious Diseases[19]

WNV in California?

Given the rapid spread of WNV in the three years since its introduction on the East Coast, it is likely that WNV will arrive here this summer.

To date, no wild birds, sentinel chickens, or mosquito pools have tested positive for WNV in California. One known instance of human infection has been documented. However, since the person lived near Los Angeles International Airport and no other tests have revealed the presence of WNV, this infection is generally attributed to a bite from an infected mosquito that arrived on board an airplane.

California is home to more than 40 mosquito species. The state's urban areas, coastal bays and wetlands, and the drainage ditches and irrigation canals of the Central Valley provide a range of potential habitats. Laboratory experiments have indicated that several California species are likely to transmit West Nile Virus.

While mosquitoes are found in all parts of the state, officials have noted that southern areas, such as the Imperial Valley and Riverside County, have been most

vulnerable to other forms of mosquito-born encephalitis, and are therefore likely to be most vulnerable to WNV as well.[20]

Culex tarsalis, the "encephalitis mosquito"

In the western United States, *Culex tarsalis* is the primary carrier of Western Equine encephalitis and St. Louis encephalitis, and is therefore expected to be a significant vector of WNV should it arrive in California. This mosquito, which bites mostly between sunset and midnight, has been shown in laboratory experiments to readily become infected by and transmit WNV.

C. tarsalis is especially abundant in the Central Valley and coastal regions. It can live in all but the most polluted waters, ranging from wetlands and salt marshes to puddles and containers. In most places *C. tarsalis* is most active in the spring and fall, but in Southern California it is active all winter long.[21] After years of intense efforts to keep this endemic species under control, vast populations in the Central Valley have become resistant to nearly all the common chemical insecticides.[22]

In populated areas, other species such as *Culex quinquefasciatus* and *Culex pipiens* are also expected to play a significant role in the transmission of WNV.[23]

Photo courtesy of NASA

Figure 3: A NASA-funded study mapped satellite imagery of temperature and vegetation to help predict where West Nile virus will spread.

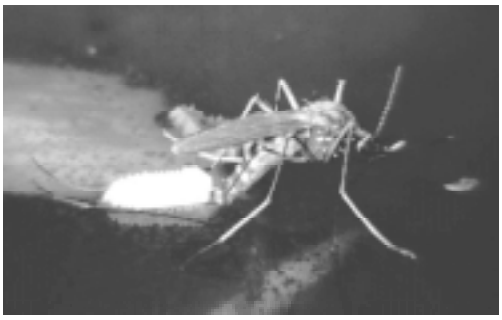


Photo: Courtesy of CDC

Figure 4: *Culex pipiens,* the house mosquito

Culex pipiens, the "house mosquito"

In residential and urban areas, the common house mosquito, *C. pipiens*, is expected to play a significant role as well. *C. pipiens* has been the primary WNV vector in much of the nation, and breeds in stagnant, standing fresh water. It can be found in high concentrations at sewage treatment plants and often lives underneath buildings, in storm drains, and in catch basins. It bites primarily in the evening and after dark, and is not active in daylight. Because *C. pipiens* rarely travels distances greater than a half mile, local efforts to eliminate breeding sites can play a major role in controlling human health impacts.

Ochlerotatus squamiger

The salt marsh mosquito, *Ochlerotatus squamiger*, inhabits coastal regions from Sonoma County down to the Baja peninsula. Unlike *Culex* species, this mosquito breeds in brackish tidal waters. A strong flyer, it easily reaches nearby cities during its early morning and late afternoon flights, and is considered a significant nuisance in cities like San Francisco.[24] Preliminary studies show it is not as readily infected with WNV as *C. pipiens.* However, it is of concern due to its abundance and more aggressive biting behavior.

"'Carpet bombing, like what some states have done, would be our last resort,' and only 'if the powers that be agree that it's necessary to protect human life.'"

Ted Toppin, Spokesperson, Mosquito and Vector Control Association of California, *Boston Globe* April 3, 2003[25]

California's West Nile Virus Surveillance and Response Plan

Following the West Nile outbreak in New York in 1999, California leaders recognized the need to update the state's mosquito control strategy to ensure detection and prevention of the spread of WNV.

The state plan was developed in a joint effort of the California Department of Health Services (DHS), the Mosquito and Vector Control Association of California (MVCAC), and the University of California at Davis and Berkeley. It provides guidelines for local agencies to use in responding to the WNV threat.

This plan, available on the Web at westnile.ca.gov/CA_WNV, emphasizes public education, prevention, and monitoring as critical strategies to effectively reduce the risk of WNV while minimizing the use of harmful pesticides. However, the plan does not rule out or set strict thresholds to limit the systematic broadcast of pesticides to kill adult mosquitoes.

Mosquito Monitoring and Surveillance

Monitoring and surveillance are the first line of defense against mosquito-borne illnesses. California's mosquito control plan includes an extensive monitoring and surveillance network to ensure prompt detection and identification of WNV. Such surveillance can play a critical role in helping towns and counties avoid unnecessary spraying. California's surveillance plan includes the following:

Mosquito Surveillance: Because mosquitoes are the vectors of viruses like West Nile Virus, monitoring mosquitoes provides a somewhat accurate estimate of the immediacy of risks to humans. Mosquitoes are tested using fixed trap sites. These sites provide information regarding mosquito numbers, virus prevalence and estimation of WNV risk. More intensive mosquito trapping will be employed in response to increased virus activity in specific areas.

Sentinel Chickens and Wild Bird Surveillance: WNV is fatal to birds, with a particularly high mortality rate in American crows. Therefore, dead birds are potential indicators of virus activity in an area, and bird reporting and testing will be an important component of California's efforts. Approximately 200 chicken flocks, known as "sentinel chickens," are strategically placed throughout the state and are tested routinely during the mosquito season to detect evidence of infection from West Nile and other related viruses.

In addition, the California Animal Health and Food Safety Laboratory screens dead wild birds and sends tissue samples to UC-Davis and the Department of Health Services for testing.[25]

Equine Surveillance: Because many horses are vaccinated against viruses borne by mosquitoes, they are not the ideal species to study to keep track of the spread of these viruses. Veterinarians are contacted annually by DHS and the California Department of Agriculture (CDFA) to ensure that horses are vaccinated and to describe diagnostic services

that are available in the event of a suspected case of WNV or related diseases, such as western equine encephalitis.

Human Surveillance: Specimens from clinical human cases of encephalitis will be screened in order to determine the possible cause of infection. In addition, hospitals will be contacted in the geographic areas of increased virus activity.

Education

The Mosquito-Borne Virus Surveillance & Response Plan notes the importance of public education in teaching people how to protect themselves, and others, from WNV. It refers to the important role of residents, farmers, and duck club owners in eliminating standing water, and the need for education of the medical community. The plan does not make specific prescriptions of how such education should be conducted, however.

Mosquito Control Measures

There are three general kinds of mosquito control articulated in California's plan: environmental management, biological control, and chemical control.

Environmental Management

Physical control measures discussed in the California plan include water management and vegetation management. These include measures that increase the water disposal rate through evaporation, recirculation, or drainage, as well as restricting growth of vegetation to decrease habitat availability for immature mosquitoes. These measures can be considered "source reduction," since they decrease the number of breeding sites for mosquitoes. According to the CDC, such measures are the most effective and economical methods of providing long-term mosquito control in many habitats.[26]

Biological Controls

Biological control entails the intentional use of natural predators or parasites to control mosquito populations. According to the state Surveillance and Response Plan, the most widely used biological control agent in California is the Mosquitofish, which can be released annually in rice fields, small ponds, and canals.

In addition, Bti and Bacillus sphaericus, two microbial control agents, are recommended for use in larval control. Since these biological agents are applied to treat water bodies in a manner similar to chemical pesticides, they are discussed further in the following section on chemical control.

Photo courtesy of CDC

Figure 5: Mosquitofish feed on mosquito larvae

Chemical Control

In addition to physical and biological control measures, the State Mosquito Control Plan explicitly lists a range of pesticides "approved for use" in California. These pesticides include adulticides, usually pesticide sprays, which target adult mosquitoes, and larvicides, generally liquids that are applied to the pools of water where mosquitoes breed. Many of these insecticides are chemicals known to have significant impacts on human health and other organisms in the environment. A partial list of pesticides "approved" for use in California can be found in Table 1.

The plan acknowledges that some pesticides, such as organophosphates, should be used infrequently because of their impact on non-target organisms and the environment, but does not expressly limit the use of these pesticides, beyond stating that adulticides in particular are used "when larval control is not possible or has been used to the fullest extent possible."

Table 1: Pesticides Approved For Use In California Mosquito Control Larvicides

Larvicides	
Bacillus thuringiensis israelensis (BTI: e.g. Vectobac, Teknar)	Use: Approved for most permanent and temporary bodies of water.
Bacillus sphaericus (e.g. Vectolex)	Use: Approved for most permanent and temporary bodies of water.
Methoprene (e.g. Altosid)	Use: Approved for most permanent and temporary bodies of water.
Diflurobenzamide (e.g. Dimilin)	Use: Impounded tailwater, sewage effluent, urban drains and catch basins.
Larviciding oils (e.g. Golden Bear 1111, BVA Chrysalin)	Use: Ditches, dairy lagoons, floodwater. Effective against all stages, including pupae.
Monomolecular Films (e.g. Agnique MMF)	Use: Most standing water including certain crops.
Adulticides	
Organophosphates:	
a. Malathion (e.g. Fyfanon)	Use: May be applied by air or ground equipment over urban areas, some crops including rice, wetlands.
b. Naled (e.g. Dibrom, Trumpet EC)	Use: Air or ground application on fodder crops, swamps, floodwater, residential areas.
Note: Many Cx. tarsalis populations in the Central Valley are resistant to label organophosphate application rates.	
Pyrethrins (natural pyrethrin products: e.g. Pyrenone Mosquito Spray, Pyrocide)	Use: Wetlands, floodwater, residential areas, some crops.
Pyrethroids (synthetic pyrethrin products containing resmethrin or permethrin: e.g. Scourge)	Use: All non-crop areas including wetlands and floodwater.

California's Pesticide Spray Policy

The California Surveillance and Response Plan identifies three levels of mosquito control in response to three levels of threat: normal season, emergency planning, and epidemic conditions.

The plan states that adulticide spraying "may be recommended" as an appropriate response in the "emergency planning" stage, as determined by the following indicators:

- Snow pack and rainfall above average;
- Significant increase in adult mosquito populations;
- One or more WNV isolations from mosquitoes;
- One to three chickens carrying the virus antibodies per flock of 10 birds;
- One or two equine cases;
- One human case statewide;
- Viral activity in small towns or suburban area; and
- Evidence of recent infection in wild birds.

However, the plan does not set a strict threshold that must be reached in a community before a mosquito control district can decide to spray. In the absence of such thresholds, California mosquito control districts may be vulnerable to politically-motivated calls for spraying, potentially resulting in unwarranted application of hazardous pesticides. They do not have to prove that there is a public health threat to conduct widespread spraying.

Like the federal guidelines, the state plan leaves this decision in the hands of local mosquito and vector control districts.

Federal Guidelines

On the federal level, the Centers for Disease Control and Prevention, Division of Vector-Borne Infectious Diseases, has developed West Nile Virus response guidelines for individual states to use.[27]

These federal guidelines for surveillance, prevention, and control of WNV have changed with time, raising the threshold at which pesticide spraying should be considered and placing more emphasis on preventative measures.

In 2000, for example, the guidelines recommended chemical control (pesticide spraying) of adult mosquitoes within approximately a 2-mile radius around the area where a WNV-positive dead bird or infected mosquitoes were found.[28] By the following year, however, the CDC had removed this direct recommendation of broadcast spraying of adulticides. The guidelines now state, "Control activity should be initiated in response to evidence of virus transmission [to humans], as deemed necessary by local health departments."[29]

These revisions acknowledged the fact that there is no truly objective evidence to determine when and if spraying should occur. "There is no simple formula for determining how large an area to treat around a positive surveillance indicator or a suspected or confirmed human case of WNV. Nor is there adequate information to determine the degree of vector population suppression that must be attained, or for how long this suppression must be maintained to reduce risk of disease."

In the absence of scientific evidence to support a specific spray policy, the revised federal guidelines give state and local officials significant flexibility in determining how large an area to treat around a positive surveillance indicator or a suspected or confirmed human case of WNV, or even whether to spray at all.

The Role of Local Agencies in Mosquito Control

In California, mosquito control is conducted by more than 70 local agencies. This includes 53 mosquito and vector control districts, servicing areas inhabited by 80% of the state's population, and dozens of environmental health and county health departments. In areas without defined vector-borne disease control programs, the California Department of Health Services provides oversight.

These local mosquito control districts have significant leeway to decide to defer pesticide spraying even if "Emergency Conditions" specified by the state plan are met. Conversely, these agencies have significant leeway to increase use of chemical pesticides even before a known threat has emerged. Many of these districts routinely spray pesticides to control mosquito populations, even in the absence of an "emergency" situation.

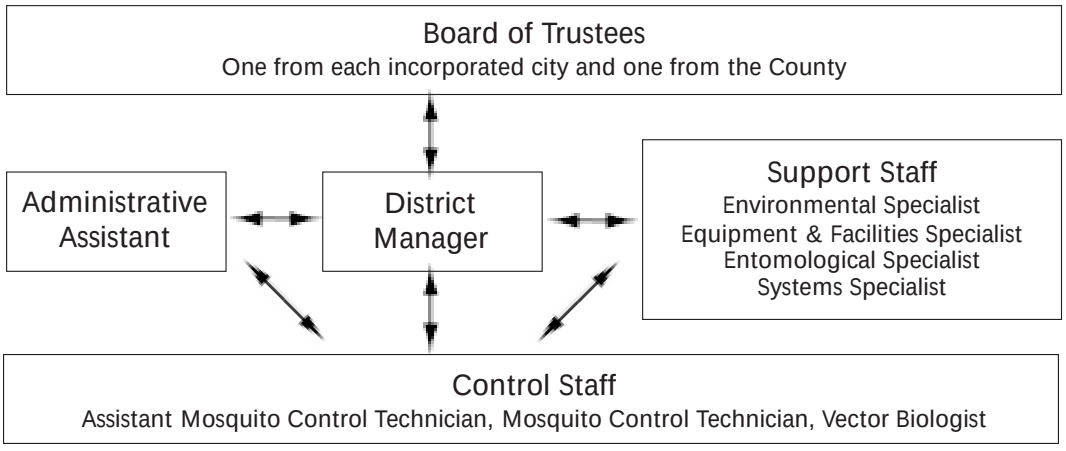
Mosquito Control Districts operate under Sections 2200-2398 of the Health and Safety Code of California. They develop mosquito control plans based on guidelines developed by the CDC and the California Department of Health Services, which outline acceptable mosquito control practices and pesticide usages. They also report pesticide use to the County Agricultural Commissioner each month.

Mosquito control districts typically cover half a dozen or more municipalities, and many have a county-wide jurisdiction. They are governed by boards of trustees comprised of representatives appointed from each member city and the county at large. In most districts, the Board of Trustees oversees the fiscal administration of the organization but leaves day-to-day operation and decision-making in the hands of the District Manager. (See Figure 6.) Funding is provided by a combination of property taxes and other special taxes authorized by local voters. For a complete listing of California Mosquito Control Districts and contact information, see Appendix A.

East Bay vector control districts already are doing their part. Wednesday, a team of all-terrain vehicles sprayed a flooded pasture in Bethel Island, while a helicopter dropped insecticide pellets into blackberry thickets and other hard-to-reach areas.

News report on March 14, 2003 in *Contra Costa Times*[30]

Figure 6. Organizational Flow Chart of a Mosquito Control District (www.mosquitoes.org/PDF/Prog99.pdf:)



Local districts have considerable decision-making power in choosing to use, or refrain from using, pesticides. The Alameda County Mosquito and Vector Control District (CMVCD) is particularly open about its decision-making process, which is outlined in the Control Plan posted on its Web site.

In 2002, the Alameda CMVCD reported "regularly using" methoprene and larvicides, and "occasionally using" the adulticides pyrethrins, resmethrin, and permethrin. In fact, adulticides, totaling less than 1 ounce, were only applied on two occasions in that year.

The following is an excerpt from their control plan, available on the web at www.mosquitoes.org/PDF/Prog99.pdf:

> *The District uses a phased approach to pesticide treatments. In the choice of material to use District personnel will use the material with the least impact to control larvae and as a last resort, localized adulticiding may be chosen. In general this progression of choices would be:*
>
> *2Bti, Duplex (Bti + methoprene), Methoprene, Oil or Agnique, Pyrethroids*
>
> *Decisions on where and when to treat are based on thresholds....These thresholds are meant to be guidelines since each site is different and other factors play a role in the levels of mosquitoes that can be tolerated. Some of these factors are as listed:*
>
> - *The proximity of homes or heavy human use areas to the source.*
> - *The age and distribution of the immature mosquitoes in a source.*
> - *The number of mosquito service calls attributed to the source from previous seasons.*
> - *The expected weather conditions and the season of the year.*
> - *The accessibility to the source (including special restrictions).*
> - *The pest or disease significance of the mosquito to be controlled in the source.*
> - *The size of the source (staff and equipment needs increase with size).*
> - *The sampling method used to check the source.*
> - *The number of active sources and available personnel and equipment.*

We surveyed Web sites of 20 mosquito control districts, finding that control plans are generally not posted on their Web sites. However, these plans are public information and should be available upon request.

Pesticide Spraying May Do More Harm Than Good

The average person thinks the way you control mosquitoes is you spray for them. That's absolutely not true. Spraying is a last, last resort.

Dr. Wayne J. Crans, Director of Mosquito Research and Control at Rutgers University, *New York Times*, Sept 8, 1999[31]

Pesticide Spraying Not Proven Effective In Curbing Human Infection Rates

Does spraying pesticides to kill mosquitoes have a significant impact on the transmission rates of the West Nile Virus? Does spraying prevent more illness than unintended pesticide exposures cause?

Despite three years of potential data, in which pesticide spraying was used in an attempt to stop the spread of WNV, these are critical questions that have not been answered by scientific study. At the moment, given the relative lack of knowledge about WNV, the Centers for Disease Control and Prevention are not conducting the statistical analysis necessary to identify the effect of insecticide spraying on infection rates in communities throughout the country. In fact, few studies have even been conducted to answer a simpler question: How effective is spraying at killing targeted mosquitoes?

There's not enough evidence that all this spraying has changed the dynamic of the outbreak, and that's in part because the studies really haven't been done to find out.

Michael Hansen, the chief pesticide researcher at Consumers Union, which publishes *Consumer Reports* magazine, *Newsday*, November 7, 2000[32]

Effectiveness of Spraying in Controlling Mosquito Populations is Limited

One study conducted by the Connecticut Agricultural Experiment Station in 2002 found that mosquito populations did not drop notably after trucks sprayed pesticides in the cities of Greenwich and Stamford.[33] However, very few studies have been conducted to document the effectiveness—or lack of effectiveness—of pesticide spraying in curbing mosquito populations under real-word conditions.

Most studies on the impact of pesticide spraying are performed under outdoor "lab" type conditions. In such studies, caged mosquitoes are placed at measured distances from spraying, at differing pesticide potencies. Some cage-trap experiments in residential areas have shown a reduction in mosquito populations of about 30 percent after a spraying.[34]

Such studies, however, may overestimate the effectiveness of spraying since they do not take into account the many variables that are involved in ground spraying. Real-world mosquitoes are not trapped in one place. Rather, they can hide under leaves and in vegetation. As a result, extrapolating the efficacy numbers from cage or trap studies to actual spraying programs is questionable.

"In order to work, the insecticide must hit the mosquito directly," Cornell University researcher Dr. David Pimentel reported in a November 2000 interview with *Newsday*. "But since spray trucks are only fogging the street side of buildings, I doubt that more than one-tenth of 1 percent of the poison is actually hitting its target. And you have to put out a lot of material to get that one-tenth of a percent onto the mosquito."[35]

In fact, scientists have estimated that less than 0.0001% of ULV (Ultra Low Volume) pesticide sprays actually reach the target insects.[36] So for every droplet that reaches a mosquito, hundreds of thousands more droplets circulate pointlessly in the environment.[37]

The CDC has also noted that, "ground applications are prone to skips and patchy coverage in areas where road coverage is not adequate or in which the habitat contains significant barriers to spray dispersal and penetration."[38]

In a 1998 study, it took two to three times more insecticide to kill 90% of the mosquitoes in residential settings than it took to kill 90% of the mosquitoes in open areas. Spraying high enough levels of insecticide to kill most of the mosquitoes in residential areas would require violating current labeling safety guidelines.[39]

Many factors decrease the effectiveness of pesticide spraying in urban areas. For example, the West Nile Virus Advisory Group to Cambridge, MA has pointed out the following factors:

- Most mosquitoes prefer to bite birds, particularly birds at rest, of which there are few in the street and building-front areas at the time the spray is applied.
- Mosquitoes may be located in roosting areas that are higher than the reach of the spray.

- Buildings and trees close to the street may block the spray from spreading to reach mosquito habitat.
- Backyard roosting areas are not effectively reached because close spacing of buildings limits penetration beyond the buildings.
- The period that the spray is effective and airborne is of relatively short duration.[40]

Furthermore, in many places mosquitoes may already be resistant to pesticides applied at health-protective label rates. California's Mosquito Control Plan notes that mosquitoes in the Central Valley have now developed sufficient resistance to organophosphates like malathion that spraying at the levels permissible under current labeling requirements is ineffective.

Too often, agencies will assume a high rate of effectiveness from spraying that is never backed up with experimental verification. For example, Deputy Commissioner Carl Johnson of the New York State Department of Environmental Conservation, which regulates pesticide use around the state, told reporters that local governments had anecdotally reported "60 to 80 percent reductions" after spraying.

Yet although New York City Health Department researchers told reporters in November, 2000, that they were conducting studies to determine the effectiveness of pesticide spraying in WNV control, such studies have not been released to the public. As of June 6, 2003, a health department official told Pesticide Watch that the data were still being analyzed, and may be available to the public in four to six weeks. Three weeks later, the same official reported that the principal investigator had resigned, delaying the study's release indefinitely.[41]

Pesticide Spraying Could Even Make WNV Worse

Spraying pesticides for mosquito control may be worse than ineffective—it may even make the West Nile virus situation worse, contributing to higher infection rates.

Pesticide spraying may build resistance, leading to resurgence of mosquito-borne disease

Ecologist Garret Hardin has stated that "every biocide selects for its own failure." This means that mosquitoes can and will become resistant to chemical efforts to destroy them. Overuse of pesticides may create resistant super-mosquitoes that require ever increasingly toxic chemicals to kill them.[43]

Few studies have been conducted to document the actual impact of aerial and ground adulticide spraying for West Nile Virus control on mosquito resistance. However, there is documentation that spraying may have contributed to a global resurgence in mosquito-borne disease over the last twenty years. The year before West Nile emerged in the United

You're going to see a resistant strain of these insects. It's like every time you get the sniffles, you don't use an antibiotic. We're running out of those. You want to use the worst treatment for the worst cases—you don't want to use the extreme approach if the risk is not that high.

Sheldon Krimsky, Professor of Urban and Environmental Policy, Tufts University[42]

States, a CDC researcher, Dr. Duane Gubler, wrote that while the factors contributing to this resurgence are complex, "the technical problems of insecticide and drug resistance, as well as too much emphasis on insecticide sprays to kill adult mosquitoes, contributed greatly to the resurgence of diseases such as malaria and dengue." Furthermore, according to Dr. Gubler, the lack of emphasis on preventative measures "and emphasis on high-tech solutions to disease control have led most physicians, health officials, and the public to rely on 'magic bullets' to cure an illness or control an epidemic."[44]

Such "magic bullet" spraying targeted at *Aedes aegypti*, the mosquito species responsible for spreading dengue fever, has been ineffective at both controlling the mosquito population and influencing the course of dengue epidemics. Though this may be due to features of *Aedes aegypti* natural ecology not shared by all other mosquito species, the fact that spraying programs of long standing were ultimately found futile indicates that pesticide efficacy (not the simple of efficacy of killing exposed mosquitoes but the broader efficacy of controlling populations and curtailing disease) is an open question with each new climate/mosquito species/disease combination that arises.

Pesticides May Kill Off Natural Mosquito Predators

Spraying can increase mosquito populations by killing off natural predators (fish, other arthropods, birds, etc.) of the mosquitoes and their larvae, thereby removing natural checks on population levels.

A 1997 study looked at trends in populations of a mosquito primarily responsible for transmitting eastern equine encephalitis (EEE) among birds. Over a period of eleven years, Cicero Swamp in central New York State was sprayed fifteen times with the insecticide Dibrom (naled). Instead of declining, the mosquito population grew fifteen-fold during

Photo: Courtesy of CDC

Figure 7. Pesticide spraying can increase populations of harmful pests.

this period. The study suggests that the pesticides may have altered the ecological balance of the swamp, killing organisms whose presence would ordinarily help limit the mosquito population.[45]

Other studies have shown that spraying malathion, another pesticide approved for mosquito control in California, may have similar results. For example, in California in the early 80s, widespread aerial applications of malathion were used in attempts to eradicate the Mediterranean Fruit Fly, an agricultural pest. An observed increase in the population of another pest, Old Black Scale, was attributed to the effect of the pesticide spray on beneficial insects. In Florida, where malathion was also used in an attempt to control Medfly infestations, the spraying did not kill mosquito larvae but did kill the larvae of an important mosquito predator, the dragonfly.[46]

Populations of scale insects on citrus trees exploded following medfly eradication sprays in California. This occurred because parasitoids that normally keep scale populations under control were killed by the malathion spray.

Pesticides Can Make Animals More Susceptible to WNV Infection

Low level chemical exposures to pesticides can decrease the quality of animals' immune system function, leading some to speculate that wildlife with compromised immune systems may be more susceptible to becoming infected by encephalitis when bitten by an infected mosquito.

This in turn would increase the numbers of formerly healthy mosquitoes developing encephalitis (since their chance of biting encephalitis infected wildlife has also increased), contributing to the spread of the illness.

Studies have shown that impurities and by-products present in malathion can further disrupt immune system function.[47] Immunosuppression may enhance susceptibility of mammalian systems to bacterial, viral, or parasitic infection or possible increased tumor formation.[48]

Use of these pesticides for WNV mosquito control could actually end up suppressing human and avian immune systems in the areas sprayed, putting each species at greater risk than before of spreading, contracting, and becoming seriously ill from WNV.

Another theory, still untested, is that mosquitoes that are sprayed but not killed may themselves experience genetic damage that would increase their infection rates—by weakening a stomach barrier known to play a role in preventing ready infection by the virus, for example.

Pesticide researcher Richard Pressinger advanced this theory to help explain observed increases in rates of encephalitis infection among sentinel chickens in Florida counties over the past decade. "Every time a mosquito spray plane or truck sprays these proven genetically damaging pesticides over the area, they are very likely increasing the amount of subtle genetic damage in the mosquito population, and hence, increasing the number of mosquitoes with genetic flaws which could in theory, allow the encephalitis virus to take hold and grow more rapidly," he surmised.[49]

Some scientists have disputed Pressinger's theory.[50] Clearly, more research is needed to explain the increasing infection rates despite widespread pesticide spraying.

Another way spraying can contribute to increased infection rates is simply by aggravating biting behavior. In an interview with the New York Public Interest Research Group, Dr. Ray Parsons, who heads the Harris County Mosquito Control Division in Houston, observed that

malathion may actually aggravate *Culex*, causing an increase in aggressive biting behavior for an hour or two after spraying.[51]

Pesticide Spraying May Reduce Participation in Other Important Public Health Measures

When cases of WNV show up in California, citizens may understandably want government officials to "do something" to prevent them from being bitten by WNV-carrying mosquitoes. A massive spraying campaign runs the risk of giving residents a false sense of security, encouraging them to think they are less likely to be bitten after the spraying, and less likely to implement non-toxic preventative measures.

Pesticide Spraying Entails Significant Risk of Public Exposure

Aerial and ground spraying of pesticides in urban and residential areas is of particular concern due to the heightened risks of exposure to the general population. Whenever these pesticides are broadcast, there are unintended impacts on human health and the environment. Even pesticides with relatively low acute toxicity to adults may pose a significant threat to young children with immature nervous systems, asthmatics, the elderly, and other individuals with unusual sensitivities to pesticides or other chemicals. Furthermore, many pesticides continue to be widely used, despite large volumes of clinical and laboratory evidence that exposure to these pesticides can have severe, sometimes fatal, human health impacts.

The Centers for Disease Control have noted, "For adult mosquito control, insecticide must drift through the habitat in which mosquitoes are flying in order to provide optimal control benefits." This kind of drift inevitably entails exposure to human populations if spraying is conducted in urban areas. Furthermore, since most mosquitoes are active nocturnally, spraying must occur during the evening hours, times of maximum exposure to those living in residential areas, for the spraying to have the desired impact on the targeted mosquitoes.

Although public notification efforts such as television broadcasts or reverse 911 calls may caution listeners to remain indoors during spraying, the time of spraying at any one location can be difficult to predict and many people cannot or choose not to remain indoors all day on the announced day of spraying. Furthermore, it is not easy to keep these pesticides from entering people's homes. Indeed, outdoor air pollutants tend to accumulate at higher levels indoors than out.

Widely used adulticides such as malathion (an organophosphate) and the synthetic pyrethroids sumithrin (Anvil) and resmithrin (Scourge) have significant, well-documented, human health impacts that are discussed in the following pages.

Known Health and Environmental Impacts of Pesticides Approved for Use in California

Pyrethroids

"Pyrethroids" are a class of chemicals modeled on natural insecticides derived from chrysanthemum flowers, called "pyrethrins."[52] Synthetic pyrethroid compounds vary in their toxicity, as do the natural pyrethrins.

Many health effects of pyrethroid exposure have been well documented. The Cornell University Program on Breast Cancer and Environmental Risk Factors in New York State lists over 125 journal studies on the health effects of pyrethroids on its Web site.[53]

Acute pyrethroid insecticide poisoning can result in tremors, salivation, hyperexcitability, choreoathetosis (involuntary movements), and seizures, as well as numbness and tingling in exposed body parts, and gastrointestinal irritation when ingested.[54]

Despite these known human health impacts, synthetic pyrethroids such as resmethrin (sold under the trade name Scourge), sumithrin (sold under the trade name Anvil), and permethrin (sold under the trade names Ambush or Pounce) have been widely used in mosquito control.

New Yorkers exposed to sumithrin when the compound was sprayed to control for WNV reported symptoms typical of pyrethrum inhalation, including asthmatic breathing, sneezing, nasal stuffiness, headache, nausea, poor coordination, tremors, convulsions, facial flushing and swelling, and burning and itching sensations. The most severe poisonings have been reported in infants.[55]

A report in the *New York Daily News* told the story of a woman who was sprayed directly on the street in Manhattan with sumithrin who ended up in the emergency room after experiencing blurry vision, nausea, itching, coughing, choking and a swollen tongue. "I threw up three days in a row, I really thought I was going to die," said the unidentified woman. In the story, a New York City Health Department spokesperson stated that this incident was one of 200 complaints from people who called the city's pesticide hotline in 2000 reporting illness due to pesticide spraying.[56]

Inert ingredients are often added to delay the enzyme action in pyrethroids

so a lethal dose is assured. These inerts may include toxic organophosphates, carbamates, or other synergists. The inerts in resmetherin (sold under the trade name Scourge) include piperonyl butoxid and petroleum distillates. Piperonyl butoxide has been shown to cause liver tumors in rats and mice.[16]

Natural Pyrethrins

Natural pyrethrins are contact poisons that quickly penetrate the nerve system of the insect. A few minutes after application, the insect cannot move or fly away. Natural pyrethrins can be swiftly detoxified by enzymes in the insect. Thus, some pests will recover.

Links between pyrethroids and hormonal disruption

Numerous studies have indicated that pyrethroids disrupt the endocrine system by mimicking the effects of the hormone estrogen, which can cause breast cancer in women and lowered sperm counts in men.[57]

A Mount Sinai School of Medicine study examined four pyrethroid pesticides, including sumithrin. It concluded that pyrethroids "should be considered to be hormone disruptors, and their potential to affect endocrine function in humans and wildlife should be investigated."[58]

A study at the Roger Williams General Hospital of Brown University on pyrethroids concluded, "chronic exposure of humans or animals to pesticides containing these compounds may result in disturbances in endocrine effects."[59]

A Cambridge University report issued in June 2000 by the Royal Society in England called for international cooperation to deal with the dangers posed by endocrine-disrupting chemicals, including pyrethroids, and recommended reducing human exposure to these chemicals.[60]

Links between pyrethroids and childhood brain cancers

Studies have found nervous-system damage from pyrethroids to be comparable to damage from DDT.[61]

A report of pesticides and childhood brain cancers published in Environmental Health Perspectives revealed a strong relationship between brain cancers and pyrethroids used to kill fleas and ticks. 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 (trade name: Dursban)."[62]

Links between pyrethroids and neurological damage

A study conducted by the Physiological Institute at Ludwig Maximilians University in Munich, Germany, found that neurological effects of pyrethroid poisoning were still seen in patients after more than two years.

Among these long-term symptoms were:

1) reduced intellectual performance with 20%-30% reduction of endurance during mental work;
2) personality disorders;
3) visual disturbances and tinnitus (ringing in the ears);
4) sensomotor-polyneuropathy, most frequently in the lower legs;
5) increased heat-sensitivity and reduced exercise tolerance due to circulatory disorders.[63]

This has been corroborated by Swedish lab studies showing 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."[64]

Links between pyrethroids and thyroid damage

A pesticide study conducted on rats concludes, "[E]xposure to organochlorine, organophosphorus, and pyrethroid insecticides for a relatively short time can suppress thyroid secretory activity in young adult rats." The study also said a decrease in body weight seen "suggests that pyrethroid insecticides can inhibit growth rate."[65] "We tested four frequently encountered pyrethroids, fenvalerate, sumithrin, d-trans allethrin, and permethrin, for estrogen and progesterone agonist/antagonist activities. Through these hormonal pathways, exposure to certain pyrethroids may contribute to reproductive dysfunction, developmental impairment, and cancer."[66]

Wildlife impacts

All pyrethroids are extremely toxic to beneficial insects, including bees. They are also extremely toxic to aquatic life, such as bluegill and lake trout, while slightly toxic to bird species, such as mallards. Toxicity increases with higher water temperatures and acidity.[67] EPA warnings on the pesticide labels include restrictions that prohibit the direct application of products to open water or within 100 feet of lakes, streams, rivers or bays. Because most pyrethroids were registered with the EPA before 1984, when comprehensive health assessment reviews were first required, EPA has scheduled such a review of pyrethroids for 2004.[68]

Organophosphates

Malathion

In response to WNV, New York City embarked on a control program relying on malathion, one of the most widely used organophosphate insecticides in the United States and throughout the world. Eradication programs for pests such as mosquitoes and fruit flies have exposed thousands of people to malathion applied in aerial applications, in many cases provoking citizen complaints of allergic reactions and flu-like symptoms.[69]

Proponents of malathion use often refer to the chemical's relatively low acute mammalian toxicity. But like DDT and other pesticides that have been found to cause irreparable damage to human and environmental health, malathion may pose a greater risk than the product label would lead one to believe.

Shown to be mutagenic; a possible carcinogen; implicated in vision loss, reproductive and learning problems, immune system disruption and other negative health effects in human and animal studies; damaging to non-target organisms; and containing highly toxic impurities, malathion has a legacy of serious problems.[70]

Acute Malathion Poisoning

Numerous incidents of acute poisoning have been documented for this widely-used pesticide. For example, in June 2001, the *Glens Falls Post-Start* reported that 37 fourteen and fifteen year-old girls became ill at a softball game after being exposed to malathion, which was being applied to an area adjacent to the field.

Organophosphates such as malathion are in the same chemical class as the nerve gas Sarin. These chemicals act as neurotoxins, disrupting the nervous system by inhibiting the enzyme cholinesterase. High exposures can produce fatal poisoning.[71]

Table 2: Symptoms of Organophosphate Insecticide Poisoning[73]

Common early or mild signs/symptoms	Symptoms of moderate or severe poisoning	Symptoms of life-threatening poisoning
Headache Nausea/Vomiting Dizziness Muscle weakness Drowsiness/lethargy Agitated/anxiety	Tightness in chest Difficult breathing Bradycardia Tachycardia Hypertension Hypotension Pallor/cyanosis Abdominal pain Diarrhea Anorexia Tremor/Ataxia Fasciculations Lacrimation Heavy salivation Profuse sweating Bronchorrhea Blurred vision Pinpoint pupils Poor concentration Confusion/delusions Memory loss	Coma Seizures Incontinence Respiratory arrest Pulmonary edema Loss of reflexes Flaccid paralysis

In laboratory animals, malathion exposure has caused stomach ulcers, testicular atrophy, chronic kidney disease, increased liver and kidney weights, adverse gastrointestinal tract effects, and changes in the adrenal glands, liver, and blood sugar levels.[72]

Table 2 lists symptoms of organophosphate exposure compiled by the United States Environmental Protection Agency.

Link between malathion and blood disorders

During a malaria mosquito eradication spray program in Pakistan in 1976, 2,800 people became poisoned from malathion and five died.[79] Physicians at Travis Air Force Base Medical Center in California have observed seven children with bone marrow disorders over the past eight years. The physicians believe organophosphate pesticides caused the blood disorders in all cases. All blood disorders occurred shortly after exposure to the pesticides DDVP/propoxur and malathion.[80]

Malathion and reproductive disorders

Juvenile male rats exposed to daily doses of malathion had decreased numbers of sperm forming cells.[81] In sheep, malathion exposure of pregnant ewes resulted in an increase in aborted fetuses, stillbirths, and low birth weight babies. Longer duration and earlier initiation of malathion exposure resulted in more severe problems.[82]

Case Study: Malathion Spraying to Control Medflies in California

Despite strong public opposition, malathion spraying was repeatedly (and ultimately unsuccessfully) used in California from 1980 through the early 1990s in an attempt to eradicate an agricultural pest called the Mediterranean fruit fly, commonly known as the Medfly.

An infestation of this pest, which can feed on and damage more than 200 species of fruit and vegetables, first appeared in California in 1975, but quickly subsided. However, a bigger infestation in 1980 led the United States Department of Agriculture to order an unprecedented campaign of widespread aerial malathion spraying. Citizen and local government opposition, including city council resolutions, helped stave off the spraying for nearly a year, when the governor authorized an all-out aerial assault. From July 10, 1981, through September 1982, more than 1,300 square miles of land were subjected to aerial spraying each week, resulting in several deaths due to accidental poisoning.[74]

Similar waves of infestation and aerial malathion spraying occurred despite public opposition throughout the 80s and early 90s. Often, the aerial spraying occurred in populated areas, exposing thousands of residents in Santa Clara County in 1983 and 1984, and 1.6 million people in the Greater Los Angeles area over a six-month period in 1989 and 1990.[75]

Dr. Jorge Mancillas, a neurobiologist at UCLA and professor at the UCLA School of Medicine, calculated that during the Los Angeles spraying, a 50-pound child exposed to malathion on a surface equivalent to that of a dollar bill would have been subjected to an exposure exceeding the EPA's "acceptable daily intake level." Although in theory the spray levels were set low enough to be "safe" (amounting to 1.4 milligrams per square foot) the actual rate of deposition of the chemical exceeded the predicted rates by 40% or more, resulting in clearly unsafe exposure levels.

"... a 50-pound child exposed to malathion on a surface equivalent to that of a dollar bill would have been subjected to an exposure exceeding the EPA's *acceptable daily intake level*."

In 1990, Ventura County successfully filed an injunction to prevent malathion spraying, helping end the aerial campaign. In 1992, one study of aerially applied malathion for Medfly control in California found an association between malathion exposure during the second trimester of pregnancy and the occurrence of gastrointestinal abnormalities in infants.[76] By that time, 10% of residents in affected areas refused to allow access to their backyards for spraying.[77]

After nearly a decade of repeated aerial bombardments, communities finally won an end to the aerial spraying program, which was replaced by release of sterile Medflies to prevent any introduced Medflies from mating successfully. According to a senior economic entomologist with the Medfly Prevention Release Program based in Orange County, since the shift to a preventative sterile release program in 1996, "there has been only one minor infestation of Medflies within the boundaries of the program."[78] After years of harmful spraying, the preventative approach turned out not only to be the least threatening to public health, but also the most effective in controlling the pest.

Malathion and vision disorders

Between 1957 and 1971, Japanese school children experienced a tremendous increase in cases of myopia (nearsightedness), which correlated with the increased use of organophosphate insecticides, including malathion.[83] Reduced visual keenness was discovered in 98 percent of the children examined from Saku, an agricultural area where malathion was regularly applied. Other examples of what is now called "Saku disease" in both children and adults were reported throughout Japan where organophosphate pesticides were applied.

In California, one incident involved a 15-year-old boy who was declared legally blind after being outside while helicopters were spraying malathion. An ophthalmologist and a pesticide expert both agreed that the boy may have Saku disease.[84]

Malathion and immunosuppression

Impurities and by-products present in malathion can further disrupt immune system function.[85] Immunosuppression may enhance susceptibility of mammalian systems to bacterial, viral, or parasitic infection or possible increased tumor formation.[86]

Ironically, use of these pesticides for WNV mosquito control could actually end up suppressing human and avian immune systems in the areas sprayed, putting each species at greater risk than before of spreading, contracting, and becoming seriously ill from WNV.

Link between malathion and cancer

In April 2000, a U.S. Environmental Protection Agency (EPA) committee reviewed a series of studies on mice and rats exposed to malathion. Based on this review, the committee concluded that there was "suggestive evidence of carcinogenicity."[87] For the moment, malathion remains listed by EPA as "not classifiable" with regard to carcinogenicity.[88] However, recent evidence suggests that organophosphates such as malathion can cause Non-Hodgkin's Lymphoma (NHL).[89] Use of malathion by farmers in Iowa and Minnesota has recently been linked to an increased risk of one type of NHL.[90]

Wildlife impacts

Malathion is lethal to beneficial insects, snails, microcrustaceans, fish, birds, amphibians, and soil microorganisms. Sublethal exposure of these species can cause a variety of behavioral and physiological abnormalities.[91]

Naled and Related Pesticides

Naled (trade name Dibrom) is an organophosphate with many of the same characteristics and concerns as malathion.

Naled can cause cholinesterase inhibition in humans; that is, it can overstimulate the nervous system causing nausea, dizziness, confusion, and at high exposures, can cause respiratory paralysis and death.

Dichlorvos: toxic byproduct of naled

One of the byproducts of degradation of naled is dichlorvos, another registered organophosphate.[92] Researchers at the Cornell University Program on Breast Cancer and Environmental Risk Factors in New York State prepared a fact sheet reviewing several studies on dichlorvos. They found the following:

- Female mice that were fed high doses of dichlorvos over a long period of time had a higher frequency of stomach cancers than untreated mice.
- High doses of dichlorvos fed over two years caused an increase in the number of male rats that had pancreatic tumors and leukemia.

- A higher number of leukemia cases were reported in one study among male farmers who used dichlorvos for more than ten days per year, compared to those who had not used dichlorvos.
- A higher number of childhood brain cancer cases were reported among families that used dichlorvos than among families that did not.[93]

In addition, Russian researchers found fish exposed to dichlorvos demonstrated slower growth rates. Researchers believe it may be due to the subtle neurotoxin actions of the pesticide and its effects upon the areas of the brain involved in feeding or food search mechanisms.[94]

Trichlorfon: ingredient in naled

The pesticide trichlorfon is a common ingredient in the mosquito pesticide Dibrom (naled). In one study, trichlorfon was found to cause a "severe reduction" in brain weight (and shape) in test animals exposed. The timing of exposure to the developing offspring appeared to be the key factor in determining neurological damage (known as the "critical brain growth period"). It occurred when the chemical was administered between 40-50 days gestation for the guinea pig, which scientists say, correlates with the brain growth spurt period for the animal.[95]

Wildlife impacts

Naled is characterized as very highly toxic to bees and aquatic invertebrates. It is moderately to highly toxic to fish and slightly toxic to upland game birds and waterfowl.[96] There is potential for chronic risk from naled to estuarine invertebrates.[97]

Temephos

Temephos (Abate) is an organophosphate insecticide used to control mosquito, midge, and black fly larvae. It is used in lakes, ponds, and wetlands. It also may be used to control fleas on dogs and cats and to control lice on humans. The compound is sometimes found in mixed formulations with other insecticides including trichlorfon. As an organophosphate, it has many of the same concerns and characteristics as malathion and naled.

Symptoms of acute exposure to Temephos are similar to other organophosphates and may include nausea, salivation, headache, loss of muscle coordination, and breathing difficulties.[98] Some studies show that Temephos may greatly increase the observed toxicity of malathion when used in combination with it at very high doses.[99]

Wildlife impacts

Tests with various wildlife species indicate that the compound is highly toxic to some bird species. The compound is also highly toxic to bees.[100] Temephos shows a wide range of toxicity to aquatic organisms, including salmon.[101] Freshwater aquatic invertebrates such as amphipods are very highly susceptible to temephos, as are some marine invertebrates.[102] Temephos is very highly toxic to saltwater

"Naled can cause cholinesterase inhibition in humans; that is, it can overstimulate the nervous system causing nausea, dizziness, confusion, and at high exposures, can cause respiratory paralysis and death."

species such as the pink shrimp, and presumably to lobsters as well.

Temephos has the potential to accumulate in aquatic organisms. In one study, the bluegill sunfish accumulated 2,300 times the concentration present in the water.[103]

Larvicides

Larvicides applied to mosquito breeding pools are generally considered to have lesser impacts on public health than adulticide sprays. For this reason, many mosquito and vector control programs rely on larviciding as the primary intervention strategy to limit mosquito populations. Indiscriminate use of larvicides, however, may have harmful impacts on wildlife and contaminate drinking water supplies.

Methoprene

Methoprene is applied to water bodies such as sewers, wetlands, ditches, and ponds for the purpose of killing mosquito larvae. According to EPA human toxicity ratings, the larvicide methoprene (Altosid) is considered to be practically nontoxic to humans.[104]

Interrupting the normal life cycle of an insect, methoprene prevents larvae from maturing to the adult stages, and thus prevents them from reproducing. To be effective, it is essential that this growth inhibitor be administered at the proper stage of the target pest's life cycle. Methoprene is not toxic to the pupal or adult stages. Treated larvae will pupate but adults do not hatch from the pupal stage.[105]

Methoprene may be the larvicide of choice in many mosquito control districts, due to the fact that one application remains effective for significantly longer than a single application of biological agents such as Bti, discussed in the following section. This can reduce the labor costs of ongoing larviciding by more than fifty percent. Methoprene mimics the action of an insect growth regulation hormone. However, application of methoprene may have significant ecological impacts.[106]

Wildlife impacts

Studies have documented methoprene to be slightly toxic to birds and slightly to moderately toxic to fish.[107] Methoprene residues may have a slight potential for bioconcentration in bluegill sunfish and crayfish.[108] Methoprene is very highly toxic to some species of freshwater, estuarine, and marine invertebrates.[109] Methoprene harms shrimp development.[110] Studies at the laboratory of researcher Charles McKenney have shown that methoprene inhibits the metamorphic success of larval estuarine shrimp and crabs with exposure to concentrations used in killing salt marsh mosquitoes.[111]

Methoprene and birth defects in vertebrates

Some researchers have hypothesized that methoprene may cause birth defects and deformities that have been observed in frogs throughout the United States.[112]

The larvicide methoprene has been linked to frog deformities, particularly extra limbs growing from various parts of a frog's body or head.[113] These deformities are thought to result from exposure to methoprene acid, a chemical that is formed when methoprene breaks down. This byproduct may function as a retinoid, a compound that stimulates gene transcription in vertebrates.[114] Changes in exposure to retinoids during certain critical stages can cause birth defects in all vertebrates, including humans, and may be contributing to the global epidemic of skeletal deformities in frogs.[115]

A recent controlled study published in *Aquatic Toxicology* demonstrated that

Spraying to Kill Mosquitoes and Killing Lobsters Instead

In 1999, immediately after Hurricane Floyd hit the eastern seaboard, lobstermen who fish in the Long Island Sound noticed a sharp decline in the local lobster population.

By the following year, experts estimated that more than 10 million lobsters, or 90% of the stock, had died off in the western part of the Long Island Sound.[116] President Clinton declared the Sound a natural resource disaster area, and Congress appropriated $13.9 million for research and financial assistance to licensed lobstermen.[117]

The lobstermen, arguing that the lobster had survived polluted runoff in the past, believe that WNV spraying in the summer of 1999 was the cause for the decimation of their fishery, and filed a $125 million putative class action lawsuit against insecticide manufacturers.[118]

This hypothesis is potentially bolstered by studies that have documented that exposure to pyrethroids used in mosquito control can kill lobsters and shrimp.[119] Although shellfish appear very different from mosquitoes, they share many life characteristics and a common evolutionary history with insects. Insects, for example, an external skeleton and development from a larval stage through a series of molts.

"This hypothesis is potentially bolstered by studies that have documented that exposure to pyrethroids used in mosquito control can kill lobsters and shrimp."

Some scientists, such as those studying the lobsters at the University of Connecticut, have hypothesized that insecticides may have been indirectly responsible for the lobster die-off. For example, pesticide exposure may have lowered their immune system, allowing a parasitic infection to overwhelm the population.

The EPA has launched an investigation into the cause of the lobster crash. Scientists estimate it will take at least 10 years for the population to recover. Research is still being conducted to determine the effects that mosquito control pesticides might have on lobsters, particularly sub-lethal effects at low levels. If lobstermen are right and widespread use of mosquito control pesticides was responsible for the crash in Long Island's lobster population, California's lobster industry could be similarly imperiled.[120]

California's Lobster Industry

Recently, the California lobster harvest has rebounded to the highest levels in over fifty years, totaling over 950,000 pounds. The economic value of California's fishing industry to the state is estimated at more than $800 million annually. The industry ranks among the top 5 seafood-producing states in the U.S. (472 million pounds in 1999).[121]

It is not clear how the California lobster and shellfish industry could be damaged if pesticides are used more widely to control mosquito populations. However, it would be irresponsible and shortsighted to introduce these chemicals onto land or water bodies without knowing the effects they might have on lobsters.

frog embryos exposed to high concentrations of methoprene did not show any developmental defects. However, the study found that methoprene can degrade into other compounds that do cause developmental toxicity at concentrations significantly higher than those expected to result from proper application of the larvicide.[122]

The U.S. Environmental Protection Agency risk assessment for methoprene did not include an evaluation of its chemical breakdown products, retinoids. This illustrates a common failure of the agency's risk assessments, which do not evaluate the breakdown by-products of the chemical pesticides under consideration.

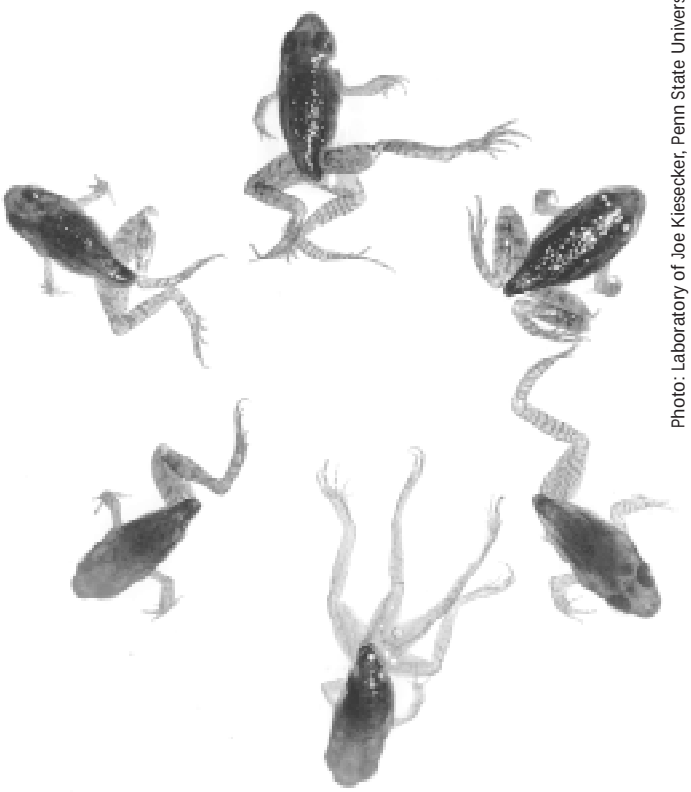


Photo: Laboratory of Joe Kiesecker, Penn State University

Figure 8. Some researchers have hypothecized that methoprene may breakdown into compounds that cause deformities in vertebrates.

Biopesticides

Biopesticides have emerged as important alternatives to traditional chemical pesticides with fewer known human health impacts. However, they, too, should be used sparingly. Like traditional chemical pesticides, significant research needs to be done on the ecological effects of biopesticides. What non-target invertebrates that are important in the food chain are also affected by their use? How will a potential decrease in this part of the food chain affect fish and amphibians, and the birds and animals that feed on them? Many unanswered questions make it difficult to estimate the potential risk of biopesticide use.

Bacillus thurengiensis israelensis (Bti) and *Bacillus sphaericus*

Bti is a biological pesticide that contains naturally occurring soil bacteria in different strains that target specific insects. It is not known to be toxic to animals, birds, humans, fish or beneficial insects. Bti is required to have EPA warning and caution labels, as is the requirement by law for any registered pesticide.[123]

Based on extensive testing, no harmful effects to the public are expected to occur when biopesticide products are applied according to label directions. Because there is the potential for skin and eye irritation, applicators are warned to avoid direct contact with the granules or a concentrated spray mix. Various tests revealed no expected harm to non-target organisms.[124]

Biopesticides closely related to Bti are widely used in organic farming. Some trade names are Aquabac, Teknar, and LarvX. *Bacillus sphaericus* (VectoLex) is another naturally occurring biopesticide. It was registered in 1991 for use against mosquito larvae, which ingest the bacteria and die after the toxin in the bacteria disrupts their gut function.

If Bti and variants are too widely used, insects may develop immunity to these pesticides, thereby limiting their effectiveness for mosquito control and for use by organic farmers. The University of California Working Group on Organic Farming has estimated that this industry has a value exceeding $225 million in the state of California alone.[125]

The Threat to Agriculture

All of the aforementioned chemicals are designed to kill insects, many of which are responsible for pollinating wild and cultivated plants in California. The future of agriculture depends on pollinators. Insect pollination is a necessary step in the production of most fruits and vegetables we eat and in the regeneration of many forage crops utilized by livestock.

California growers of almonds, apples, and many other crops depend on insect pollinators — both managed and wild — to produce fertile seeds and full-bodied fruit. Recent surveys document that more than thirty genera of animals — consisting of hundreds of species of floral visitors — are required to pollinate the 100 or so crops that feed the world. Domestic honey bees service only 15% of these crops, while at least 80% are pollinated by wild bees and other wildlife.[126]

Researchers have estimated severe revenue losses to both almond growers and honey producers in California resulting from a pesticide-induced decline in the numbers of pollinators where pollination by honeybees alone is valued at over $14.6 billion.[127]

Organic crops are also at risk, should the state choose the method of aerial or ground spraying of pesticides. It is unlikely that sprayed farms will lose their certified status, but sprayed crops and plant material may not be able to be marketed as 'organically produced.'[128]

Unknown Health Impacts of Mosquito Control Pesticides

"Inert" Ingredients Escape Public Disclosure

The true nature and health threat of a pesticide is difficult to analyze, since many of its ingredients may never be made public.

The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the nation's primary pesticide control law, classifies pesticide ingredients into two categories—active and inert. The active ingredients are those designed to kill pests while the inerts are added to make the active ingredient more potent and easier to use. Inert ingredients can make up a significant percentage of the material that is actually sprayed. Yet these inerts, which are often highly toxic, are often classified as "trade secrets" under law and are not listed on the label. In September 1997, U.S. EPA issued a memo encouraging pesticide manufacturers to voluntarily substitute the term "inert ingredients," with the term "other ingredients," noting that:

Many of these compounds are potentially harmful, even more so than the active ingredient in the pesticide. Many consumers are misled by the term "inert ingredient", believing it to mean "harmless." Since neither the federal law nor the regulations define the term "inert" on the basis of toxicity, hazard or risk to humans, non-target species, or the environment, it should not be assumed that all inert ingredients are non-toxic.[129]

Since the technical (chemically pure) grade of a pyrethroid is usually formulated (mixed with carriers, solvents, synergists, etc.) for use in commercial pest control, the toxicity of these other ingredients must be taken into consideration when assessing the toxicity of a formulated product. Researchers found a ten-fold difference in toxicity between formulations with the same active ingredient, but with different carriers, solvents, etc.[130]

Some mixtures of Anvil are made up not only of 10% artificially manufactured Sumithrin but 10% piperonyl butoxide (PBO), a suspected carcinogen, and 80% "inert" ingredients such as polyethylbenzene, which is listed by the EPA as being "potentially toxic."[131]

PBO is added to make the pyrethroids more effective. It acts by inhibiting naturally occurring enzymes that would otherwise degrade the insecticide. PBO breaks

through the insect's defense, making the insecticide more powerful. The EPA's Office of Pesticide Programs suspects PBO of being a carcinogen. The National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances lists it as a suspected gastrointestinal or liver toxicant, and a suspected neurotoxicant. It has also been reported as a suspected reproductive toxicant.[132] In addition, there is some evidence that PBO-pyrethroid mixes can affect the human immune system.[133]

Polyethylbenzene (PEB), a heavy aromatic solvant also known as naphtha, is widely used in pesticides. PEB is listed on the EPA Office of Pesticide Programs' Inert Pesticide Ingredients List No 2, which is a list of 64 substances the EPA "believes are potentially toxic and should be assessed for effects of concern. Many of these inert ingredients are structurally similar to chemicals known to be toxic; some have data suggesting a basis for concern about the toxicity of the chemical."

PEB, for example, is related to ethylbenzene, which is listed as a suspected reproductive toxicant and a suspected respiratory toxicant by the EPA. White mineral oil, also known as hydro-treated light paraffinic petroleum distillate, is also listed on the EPA's Inert Pesticide Ingredients List No. 2 of potentially toxic chemicals.[134]

Although they may be off the public radar screen, inert chemicals may have significant impacts on public health and the environment.

Pesticides Are Not Proven Safe

Many people assume that a pesticide is safe to use if it has been approved for use and is available on the store shelves at their local hardware store. However, thousands of chemicals on the market lack adequate testing to demonstrate that they will not harm human health or the environment.

Pesticides are routinely approved before their health consequences have been accurately determined, as evinced by the fact that nearly 100 pesticides have been banned or severely restricted by the EPA since their introduction.[136] As recently as June 8, 2000 the EPA announced a ban on virtually all uses of Dursban (chlorpyrifos) in residential and commercial buildings. Diazinon, one of the most widely used pesticides in the United States, will be phased out of home and garden use by 2004 because of health concerns.

Yet it can be years or even generations before a dangerous compound is banned or its use restricted. Consider the examples of lead in paint and gasoline,

Current policies such as risk assessment and cost-benefit analysis give the benefit of the doubt to new products and technologies, which may later prove harmful. And when damage occurs, victims and their advocates have the nearly-impossible task of proving that a particular product or activity was responsible.[135]

Peter Montague, Environmental Research Foundation

DDT in pesticides, and DES and thalidomide for pregnant women.

This demonstrates a faulty system in which pesticides, not public health, are given the benefit of the doubt in regulatory decision-making. The risk assessment models used by the state to evaluate the chemicals, although they enjoy widespread use in the regulatory community, are often inadequate in determining whether the introduction of these compounds into the environment will adversely affect humans, wildlife, and entire ecosystems.

In order to protect public health and the environment, pesticides should be subjected to standards like those used by the Food and Drug Administration, in which a product is considered harmful until it is proven safe.

Risk assessments are used to demonstrate the relative safety of using a given toxic chemical when exposure is limited to a certain level. The officials and applicators will assure the public, based on their risk assessments, that the levels of chemicals they will be exposed to will be so low, and so infrequently applied, that there will be no effect on the environment and human health, or that the compound's toxicities quickly degrade.

These assessments may be unreliable for a number of reasons. First, many of these chemicals may have significant to subtle negative health and environmental effects at extremely low levels. Secondly, pesticides are never applied under ideal conditions as planned. There will always be mistakes, spills, and oversprays. The compounds, although analyzed for safety and degradation characteristics under ideal laboratory conditions, will be applied by real people in the real world.

Risk assessments may be designed and conducted to prove that a pesticide spray program is safe. Yet, significant uncertainties often underlie these assessments. A working group of the U.S. Environmental Protection Agency identified the following uncertainties in current knowledge which limit regulators' "ability to make decisive assessment conclusions and take fully informed actions to prevent or mitigate pesticide problems:"

- What is the efficacy of spraying, especially ground spraying without aerial?
- What should agencies do when public health comes head-to-head with environmental risks?
- Communication of risk of disease vs. risk of control.
- What are effects of multiple spraying (risks)?
- More measurements of (outdoor and indoor) pesticides via spraying.
- Is turning air-conditioning off effective in reducing exposure; what about restarting?
- When is it safe to allow children and pets out after spraying?
- What are the results of environmental spraying and implications to the lower end of food web/chain?[137]

Only rarely will risk assessments performed to justify proposed spraying programs clearly delineate the above uncertainties.

Balancing the Risks

If we're just spraying all over and not doing a damn bit of good, then this is a waste of time and money, and it's also a hazard.

Dr. David Pimentel, Professor of Entomology, Cornell University, *Newsday*, November 7, 2000

As Michael Gochfeld, Professor of Environmental and Community Medicine at the Robert Wood Johnson Medical School and School of Public Health, Rutgers University has written:

In weighing the risks and benefits of mosquito control, we should consider the disease itself and the risk to the human population. The media always paired the words "lethal" or "deadly" with "West Nile" or "encephalitis," reinforcing in the public's mind the danger from the disease. But it would be equally propriate to characterize West Nile virus infection as "unapparent," "usually asymptomatic," or "occasionally serious." Seven deaths in a population of over 10 million people over a one month period is certainly tragic, but pales beside the number of deaths from many other diseases that are addressed less aggressively.[138]

Dr. Gochfeld and other experts have argued further that we have insufficient evidence to know how to control WNV-type diseases or how our control measures may affect them. Filling in these data gaps will be crucial in assessing the risk tradeoffs essential to public health decisions in this area.[139]

Why the Push for Pesticide Spraying?

Considering the lack of evidence demonstrating the effectiveness of pesticide spraying, it may seem surprising at first that the vast majority of government officials have responded to the emergence of West Nile Virus with broadcast spraying programs.

The push to spray comes from several very different sources: the urge to do something highly visible to show that action is being taken to address the health threat; momentum resulting from the fact that pesticide spraying has been the dominant approach to such problems for the past thirty or forty years; and the influence of pesticide manufacturers on local, state, and national decision-making processes.

In states lacking mosquito control plans when WNV first appeared, pesticide spraying provided a quick and easy solution to a very complicated and multifaceted problem. Thinking they had to "do something," most government officials put their finger firmly on the pesticide trigger, picking the easiest and quickest, but not the safest, least costly, or most effective response to address the WNV. These officials could say that they had done "something," highly visible to the affected communities, even though their solution may have caused more harm than good.

In states with more prevalent and long-standing mosquito control problems, the decision to spray may be based in years of precedent in which spraying has been the main approach to mosquito control.

There is also big money to be made by spraying pesticides. Pesticide manufacturers and applicators stand to profit from manufacturing and applying sprays for WNV mosquito control. In New York City, for example, Clarke Environmental Mosquito Management, Inc. was paid $650/hour per truck in a $4.6 million New York City contract.[140] (The company's bid for a three year contract to spray was in excess of $50 million.[141] This bid was rejected by the state, and Clarke was recently fined $1 million for violating New York State's pesticide application laws.)

"... the line between publicly-funded mosquito control and for-profit chemical companies is consistently blurred by "public/private partnerships."

The corporations who manufacture the pesticides are often the same entities funding research to document the effectiveness of those same pesticides. Furthermore, the line between publicly-funded mosquito control and for-profit chemical companies is consistently blurred by "public/private partnerships." For example, many pesticide companies have direct links to the California Mosquito and Vector Control Association's (MVCAC) website, and are corporate sponsors or "members" of this governmental agency. "Sustaining members" who made significant financial contributions to MVCAC in 2002 include large chemical pesticide manufacturers and distributors, such as Aventis Chemical Corporation, Clarke Mosquito Control Products, Inc., Electramist, Inc., Fen-nimore Chemicals, Pigott & Associates, Inc., Valent Biosciences Corporation, Vopak USA, and Zoecon Professional Products.[142]

In this sense, the state of knowledge of WNV control is analogous to the state of understanding of Medfly control in California in the mid 1980s (described on page 31) when there was no clear-cut technical or scientific evidence to support a program of ground spraying, aerial spraying, or the mass application of pesticides in any form. In the case of the Medfly infestations, pesticides were given the benefit of the doubt until exposed communities organized against the onslaught. It took more than a decade of ineffective pesticide spraying before preventative, nonchemical control programs were implemented in their place.

This time, communities can be prepared. In many parts of the country, the West Nile Virus outbreak has been accompanied by intensive media coverage, including daily or near-daily reports highlighting each additional case discovered in humans or birds. The impacts of pesticides on nontarget organisms rarely have been given comparable attention. An important first step may be actively working with journalists in California to ensure that media representations of WNV include clear discussion of the risks and known impacts of pesticides, rather than simply inflaming public fears of a new health threat.

"An important first step may be to actively work with journalists in California to ensure that media representations of WNV include clear discussion of the risks and known impacts of pesticides, rather than simply inflaming public fears of a new health threat."

Principles for Safe, Effective Mosquito Control Measures on the State and Local Level [143]

I. Give Public Health, Not Pesticides, the Benefit of the Doubt

In order to safeguard public health in the state, a balanced approach to West Nile Virus must weigh the threats posed by pesticide use to the general population against the threat posed by West Nile Virus. In the absence of evidence demonstrating that spraying helps limit transmission of the disease to humans, pesticide spraying should not be part of the WNV control plan.

1. Before any decision to use pesticides, community-specific assessments of health and environmental hazards of proposed products that take into consideration all pesticide ingredients (including inerts) should be conducted, with full public input.
2. Reevaluate and eliminate spraying conducted for nuisance reasons. Such spraying generally relies on the same potentially hazardous pesticides used in WNV control. Furthermore, indiscriminate use of these pesticides builds up resistance in mosquito populations, making targeted use for disease control even less effective.

II. To Protect Public Health, Prioritize Alternatives To Pesticide Spraying

Public education and outreach, behavioral changes and preventative measures that reduce mosquito breeding habitat can effectively minimize risk of WNV while reducing momentum for dangerous pesticide spraying programs.

While public outreach and education of this nature may be a significant part of a mosquito control district's strategies for

controlling mosquito populations, the state plan does not lay out specific parameters for it. Community leaders and activists can work with district managers to ensure that mosquito control resources are focused on prevention and education, rather than chemical spraying response.

Focus on source reduction

Source reduction encompasses a broad range of activities. It can be as simple as steps taken by individuals where they live, such as turning over empty containers, removing used tires and cleaning rain gutters and bird baths. Source reduction also encompasses extensive regional water management projects conducted by mosquito control agencies or fish and wildlife officers. Comprehensive source reduction activities can eliminate or substantially reduce mosquito breeding and the need for repeated applications of insecticides in the affected habitat.

- Stock manmade ponds and other appropriate bodies of water with mosquito-eating fish. In some cases, it may be appropriate to use bacterial larvicides or mechanical controls such as vegetable-based oils that smother mosquito eggs floating on the surface of the water (see larvicide section).[144]
- Keep waterways clean so that fish and other mosquito predators can survive. Ensure vegetation is cleaned out of natural sloughs in marshy areas to keep water flowing, preventing mosquito habitat from forming.

Healthy Wetlands Help Control Mosquito Populations

Laboratory studies have shown that salt marsh mosquitoes are unlikely to be major vectors of West Nile Virus. However, they are among the mosquitoes most commonly sprayed for nuisance reasons. Restoring degraded wetlands can help limit both the public health threat and nuisance of mosquito populations. For example, the Ora Loma Marsh in the San Francisco area was recently restored for a number of sensitive species, including the endangered salt marsh harvest mouse. According to a paper by Wes Maffei, the manager of the Napa County Mosquito Abatement District, the marsh design "was altered to improve tidal flow, thereby reducing the amount of stagnant water in which mosquitoes thrive. Although it has only been a couple of years since the restoration occurred, mosquito breeding has been markedly reduced and it is quite apparent that the health of a degraded marsh is now returning."[145]

"Restoring degraded wetlands can help limit both the public health threat and nuisance of mosquito populations."

On the flip-side, wetland restoration projects that lack adequate design controls and funding for ongoing maintenance may actually undermine attempts at effective mosquito control.

Open Marsh Water Management, or OMWM, was developed to control mosquitoes by facilitating access of their natural fish predators to areas on salt marsh where mosquitoes breed.[146] Through a system of pools and pannes connected by radial ditches, small fish that eat mosquito larvae can reach the larvae during high tide, then retreat to sumps or reservoirs at low tide. Robert Scheirer, a coordinator with the US Fish and Wildlife Service, has written that "This has been found to be an effective, long-term method of controlling mosquito populations without using sprays."[147]

Involve and Engage the Community

- On a municipal or county level, set up a system for citizens to report standing water near their homes.[148]
- Educate the public about what people can do at home to minimize mosquito exposure and eliminate breeding sites through press releases, Web sites, school presentations, mailings, and distribution of brochures in public offices. Public health education is a good investment of resources and will pay off better than quick-fix expenditures on chemical sprays.
- Respect public requests not to spray, both on the individual and municipal level.
- Hold public hearings that offer community members the opportunity for meaningful input into local mosquito control decisions involving the use of pesticides.
- Continuously evaluate the effectiveness of all mosquito control measures.

Steps Individuals Can Take

1. Learn about local mosquito control policies.

Contact your mosquito control district to obtain a copy of their mosquito control plan, including information about their policies regarding pesticide use (what indicators will "trigger" spraying, how will they notify the public of their intent to spray?)

Find out when there may be opportunities for public input (e.g. schedule of mosquito control board meetings.)

2. Reduce standing water and other mosquito habitat.

- Get rid of any unnecessary items on your property that can hold stagnant water, such as old tires. If you use old tires for farming or gardening, drill holes in them and empty them regularly.
- 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 bird baths, fountains, wading pools, plant pots and drip trays twice a week. Call your local mosquito control district to learn whether stocking fountains or ponds with mosquito fish might be appropriate.
- Check for other ways water may be collecting around your house, such as puddles beneath air conditioners.
- Clean out your 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 on your property so that water drains properly.
- Keep grass cut short and trim shrubs to minimize hiding places for adult mosquitoes.
- Eliminate standing water in your basement.

3. Report dead birds.

Reports of dead birds can be made to the California Department of Health Services Surveillance program by calling (877) WNV-BIRD or by clicking the

"Report Dead Bird" link: westnile.ca.gov/Dead_Birds. Cal DHS will initiate the pick up of bird specimens.

4. Mosquito-proof your house and body.

- 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.

Table 3. Checklist of Possible Mosquito Sources Around the Home[149]

Mosquito Sources	What to Do to Reduce Mosquitoes
Ponds	Stock pond with Mosquitofish. Each fish can eat 100 to 500 larvae per day. They play an important role in mosquito control in ponds, canals, irrigated fields and some other freshwater sources. The fish live two to three years; they are live-bearing and produce 3 to 4 broods each year. Remove excess vegetation.
Swimming pools	Keep water off cover. Maintain water quality at all times.
Tree holes	Fill hole with sand or mortar.
Plastic pools	Drain water when not in use, or cover so mosquitoes cannot lay eggs.
Containers	Empty water. Store in an inverted position. Dispose. Cover so mosquitoes cannot lay eggs.
Bird baths	Change water at least once a week.
Standing water	Eliminate by draining. Fill in low areas.
Watering troughs	Stock with fish, or change water weekly.
Cooler drains	Prevent water from standing.
Street gutter or catch basins	Keep litter and garden debris out of gutter. Do not over-water yard.
Cesspool or septic tanks	Seal and cover opening so mosquitoes can't lay eggs.
Roof gutters	Clean once a year to remove debris.
Irrigated lawns or fields	Avoid over-irrigation. Drain standing water.

What You Should Know About Personal Protection and Insect Repellents

The most effective method of personal protection from mosquito bites is to avoid places where mosquito densities are high and to avoid being out-of-doors at times of the day when mosquito activity is at its highest. Wearing protective clothing such as hats, long sleeves and pants can help limit exposure.

If you choose to use insect repellents, treat clothing, rather than skin, whenever possible, and wash off repellents with soap and water after returning indoors.[150]

DEET

DEET has been demonstrated to be an effective mosquito repellent. However, use of DEET may entail the risk of serious side effects.

A recent study published in the New England Journal of Medicine found a formulation containing 23.8 percent DEET offered complete protection from mosquito bites for 5 hours, on average, compared to a soybean-oil-based repellent (see Bite Blocker/Buzz-Off section below), which protected against mosquito bites for an average of 94.6 minutes.[151]

More than 50 cases of serious toxic side effects experienced by people using the insecticide DEET have been documented in the medical literature. The U.S. Environmental Protection Agency (EPA) acknowledges fourteen cases in which individuals reported seizures associated with exposure to DEET.[152] Twelve were children, three of whom died.

A press release from Duke University Medical Center research pharmacologist Mohamed Abou-Donia, Ph.D., whose animal studies have shown that DEET has potential interactions in humans, argues that "safe is better than sorry."[153]

Dr. Abou-Donia recommended:

1. Never use insect repellents on infants, and be wary of using them on children in general.
2. Never combine insecticides with each other or use them with other medications. Even so simple a drug as an antihistamine could interact with DEET to cause toxic side effects.

Some state Bureaus of Health in the USA and Health Canada do not recommend using DEET at all on infants and/or children under 2, and only 10% (or less) DEET preparations on kids 2-12. Health Canada also recommends that adults not use preparations with over 30% DEET, and will not register products with a higher concentration than 30% after 2004.

Plant-Based Insect Repellents

University of California Pest Management Guidelines note that plant oils such as those from birch, bluestem grass, geranium, pine, rosemary, spearmint, yarrow, lantana, and neem have been shown to be somewhat repellent to mosquitoes, but most are not available in commercial mosquito repellents.[154]

Two commercially available plant oil-based repellents are Bite Blocker and Buzz-Off.[155] Studies published in the *New England Journal of Medicine* have shown that repellents containing oil of eucalyptus provided protection for an average of two hours, and a product containing soybean oil (Bite Blocker for Kids, HOMS) was effective for an average of 90 minutes.[156]

Citronella repellents and candles are non-toxic and somewhat effective

Studies show that citronella can be an effective repellent, but it provides shorter complete protection time than most DEET-based products. Frequent reapplication of the repellent can partially compensate for this.[157]

Canadian researchers studied, under field conditions, the efficacy of three citronella-based products (lotion, milk and sun block formulations (active ingredients: 10% oil of citronella and 5% terpene of citronella) to protect against biting mosquitoes. All of the repellents "reduced the number of mosquitoes biting by 95% over the 1st and 2nd 30 minutes after application."[158]

The same group of researchers assessed the efficacy of 3% citronella candles and 5% citronella incense in protecting against mosquito bites under field conditions. "Although significantly fewer bites were received by subjects at positions with citronella candles and incense than at nontreated locations, the overall reduction in bites provided by the citronella candles and incense was only 42.3% and 24.2%, respectively."[159]

Avon Skin-So-Soft ™

When tested under laboratory conditions against Aedes aegypti mosquitoes, this product was shown to be mildly effective. However, with a half-life of 30 minutes, frequent reapplication is necessary to maintain a protective layer of the oil on the skin, which works by forming a barrier that insect mouthparts have difficulty penetrating.[160]

Mosquito traps

A range of devices are being marketed that have been shown to trap and kill measurable numbers of mosquitoes over a geographic range. Such traps may be an adjunct to other precautionary measures, but homeowners should be aware that depending upon their placement, such traps may attract more mosquitoes into an area than they can catch.

If pesticide spraying occurs in your community, take precautions to limit exposure:

(adapted from "Fight the Bite," New York State Department of Health, April 2003, downloaded on June 6, 2003)

1. Keep windows closed during and immediately after spraying. If possible, also turn off window air conditioners.
2. Stay inside and keep children and pets inside during spraying and until the next morning after spraying. Pregnant women should take special precautions to avoid exposure.
3. Bring in or cover portable outdoor furniture, toys, laundry, pet dishes and tools.
4. Cover larger outdoor items such as barbecue grills or sand boxes. Swing sets and items that cannot be covered should be rinsed thoroughly after the spraying.
5. Cover ornamental fish ponds because pesticides are highly toxic to fish.
6. Cover vegetable gardens if you can with plastic sheeting; wash any exposed vegetables before storing, cooking or eating.
7. Remove shoes when entering the home after spraying because pesticides can be tracked indoors and remain toxic for months in synthetic carpet fibers. Pesticides used for mosquitoes are most easily degraded in direct sunlight and are sheltered when inside where they do not degrade quickly.
8. Hose off window screens, door handles and hand railings after spraying occurs to avoid direct contact.
9. If you suffer symptoms such as dizziness, headache, nausea, vomiting, weakness, blurred vision, breathing difficulties, or irritation of the eyes, nose, lips, mouth or throat, see your doctor immediately.

APPENDIX: California Mosquito Control Contacts

Who do I contact if I have more questions about mosquito control or pesticide use in California?

Public Interest Advocacy Organizations

- Pesticide Watch at (213) 251-3690 ext. 308
- Environment California, at (415) 206-9185
- The Pesticide Action Network of North America at (415) 981-1771
- Californians for Pesticide Reform at (888) CPR-4880 or (888) 277-4880
- Your local Mosquito and Vector Control District or Environmental Health Department. (See following table.)

Mosquito and Vector Control Association of California: www.mvcac.org

California Department of Health Services/ Vector-Borne Disease Section
arbovirus@dhs.ca.gov or
www.dhs.cahwnet.gov/ps/dcdc/disb/disbindex.htm

The U.S. Environmental Protection Agency (EPA) Web site: www.epa.gov/pesticides/factsheets/skeeters.htm

California Mosquito Control Districts
www.mvcac.org/Download/map.pdf

California Mosquito Control Districts, available on the web at:
www.mvcac.org/Download/map.pdf

Agency	Contact Info
Coastal Region	
Alameda County MAD	23187 Connecticut St., Hayward,-94545 John R. Rusmisel 510/783-7744 (510/783-3903) acmad@mosquitoes.org
Alameda County VCSD	1131 Harbor Bay Parkway, Alameda, CA 94502 William Pitcher 510/567-6800 (510/337-9137) bpitcher@co.alameda.ca.us
Contra Costa MVCD	155 Mason Circle Concord, CA 94520 Craig Downs 925/685-9301 (925/685-0266) cdowns@ccmvcd.net
Marin-Sonoma MVCD	595 Helman Lane, Cotati, CA 94931 Jim Wanderscheid 707/285-2200 (707/285-2210) jimw@msmosquito.com
Napa County MAD	Post Office Box 10053, American Canyon, CA 94503 Wesley A. Maffei 707/553-9610 (707/553-9611)
No. Salinas Valley MAD	342 Airport Blvd., Salinas, CA 93905 Peter B. Ghormley 831/422-6438 (831/422-3337) pbg217@aol.com
San Mateo County MAD	1351 Rollins Road, Burlingame, CA 94010 Robert Gay 650/344-8592 (650/344-3843) rgay@smcmad.org
Santa Clara County VCD	976 Lenzen Drive, San Jose, CA 95126 Tim D. Mulligan 408/792-5010 (408/298-6356) timothy.mulligan@deh.santa-clara.ca.us
Santa Cruz County MVCD	640 Capitola Road, Santa Cruz, CA 95062 Paul Binding 831/454-2590 (831/464-9161) agc020@agdept.com
Solano County MAD	2950 Industrial Court, Fairfield, CA 94533 Jon A. Blegen 707/437-1116 (707/437-1187) solmad@aol.com
Sacramento Valley Region	
Burney Basin MAD	Post Office Box 1049, Burney, CA 96013 Michael S. Churney 530/335-2133 (530/335-2663) bbmad@frontiernet.net

Butte County MVCD	5117 Larkin Road, Oroville, CA 95965 James A. Camy 530/533-6038 (530/534-9916) bcmvcd@global411.net
Colusa MAD	Post Office Box 208, Colusa, CA 95932 David B. Whitesell 530/458-4966 (530/458-0818) colmad@mako.com
Durham MAD	Post Office Box 386, Durham, CA 95938 Aaron A. Amator 530/345-2875 (530/345-1792)
El Dorado Co. V.C.-CSA3	1170 Rufus Allen Road, S. Lake Tahoe, CA 96150 Virginia Huber 530/573-3450 (530/542-3364) vhuber@co.el-dorado.ca.us
Glenn County MVCD	165 Co. Rd. G, Willows, CA 95988 Richard T. Ramsey 530/934-4025 (530/934-5971)
Lake County VCD	Post Office Box 310, Lakeport, CA 95453 Arthur Colwell, Ph.D. 707/263-4770 (707/263-3653) lcvcd@mchsi.com
Oroville MAD	Post Office Box, CA 940, Oroville, CA 95965 Jeff Cahn 530/534-8383 jajens1@cwnet.com
Pine Grove MAD	Post Office Box 328, McArthur, CA 96056 William Clark 530/336-5740 (530/336-6866)
Placer MAD	Post Office Box 216, Lincoln, CA 95648 Charlie Dill 916/435-2140 (916/435-8171) charlied@placermosquito.org
Sacramento-Yolo MVCD	8631 Bond Road, Elk Grove, CA 95624 David Brown 916/685-1022 (916/685-5464) dabrown@sac-yolomvcd.com
Shasta MVCD	Post Office Box 99033, Redding, CA 96099 William C. Hazeleur 530/365-3768 (530/365-0305) mosquito@snowcrest.net
Sutter-Yuba MVCD	Post Office Box 726, Yuba City, CA 95992 Ronald L. McBride 530/674-5456 (530/674-5534) rmsymvcd@pacbell.net
Tehama County MVCD	Post Office Box 1005, Red Bluff, CA 96080 D. Andrew Cox 530/527-1676 (530/527-3353) dacox@cwnet.com

North San Joaquin Valley Region

East Side MAD	2000 Santa Fe Avenue, Modesto, CA 95357 Claude L. Watson 209/522-4098 (209/522-7841) esmad@thevision.net

Merced County MAD	Post Office Box 909, Merced 95341 Allan D. Inman 209/722-1527 (209/722-3051) mcmadmanager@mercednet.com
San Joaquin County MVCD	7759 S. Airport Way, Stockton 95206 John R. Stroh 209/982-4675 (209/982-0120) sjcmvcd@worldnet.att.net
Turlock MAD	4412 North Washington Road, Turlock 95380 Jerry M. Davis 209/634-8331 (209/634-4103) mosquito@cwnet.com

South San Joaquin Valley Region

Coalinga-Huron MAD	Post Office Box 447, Coalinga 93210 Ralph Baiza 559/935-3198
Consolidated MAD	Post Office Box 278, Selma 93662 Steve Mulligan 559/896-1085 (559/896-6425) conmad@pacbell.net
Delano MAD	Post Office Box 220, Delano 93216 Ralph T. Alls, Ph.D. 661/725-3114 (661/725-3179) dmad@lightspeed.net
Delta VCD	Post Office Box 310, Visalia 93279 Michael W. Alburn 559/732-8606 (559/732-7441) deltavcd@aol.com
Fresno MVCD	2338 McKinley Ave, Fresno 93703 David G. Farley 559/268-6565 (559/268-8918) fmvcd@pacbell.net
Fresno Westside MAD	Post Office Box 125, Firebaugh 93622 Elizabeth A. Cline 559/659-2437 (559/659-2193) lizcline@inreach.com
Kern MVCD	4705 Allen Road, Bakersfield 93312 Robert A. Quiring 661/589-2744 (661/589-4913) kmvcd@lightspeed.net
Kings MAD	Post Office Box 907, Hanford 93232 Lue Casey 559/584-3326 (559/584-3310) kingsmad@attglobal.net
Madera County MVCD	900 North Gateway Dr., Madera 93637 Kevin Pinion 559/674-6729 (559/674-6004) madrmosq@inreach.com
Tulare MAD	Post Office Box 1476, Tulare 93275 Marshall Norgaard 559/686-6628 (559/686-2013) tmad@lightspeed.net
West Side MVCD	Post Office Box 205, Taft 93268 Don W. Black 661/763-3510 (661/763-5793) wsm.mosq@verizon.net

Southern California Region

Antelope Valley MVCD	Post Office Box 1192, Lancaster, CA 93584 Cei Kratz 661/942-2917 (661/940-6367) avmos2@earthlink.net
Coachella Valley MVCD	43-420 Trader Place, Indio, CA 92201 Donald E. Gomsi 760/342-8287 (760/342-8110) cvmosquito@cvmvcd.org
Compton Creek MAD .	1224 So. Santa Fe Avenue, Compton, CA 90221 Mitchel R. Weinbaum 310/639-7375 (310/639-4768)
Greater L. A. County VCD	12545 Florence Avenue, Santa Fe Springs, CA 90670 Jack Hazelrigg, Ph.D. 562/944-9656 (562/944-7976) glacvector@mgci.com
Long Beach -Vector Control Prog	2525 Grand Ave, Rm 220, Long Beach 90815 Donald D. Cillay 562/570-4132 (562/570-4038) docilla@ci.long-beach.ca.us
Los Angeles Co. W. VCD	6750 Centinela Ave, Culver City, CA 90230 Robert Saviskas 310/915-7370 (310/915-9148) rsaviskas@lawestvector.org
City of Moorpark/VC .	799 N. Moorpark Ave, Moorpark, CA 93020 John Brand 805/517-6267 (805/529-0267) jbrand@ci.moorpark.ca.us
Northwest MVCD	1966 Compton Avenue, Corona, CA 92881 Major S. Dhillon, Ph.D. 909/340-9792 (909/340-2515) mdhillon@nwmvcd.com
Orange County VCD	Post Office Box 87, Santa Ana, CA 92702 Robert Sjogren, Ph.D. 714/971-2421 (714/971-3940) ocvcd@ocvcd.org
Owens Valley MAP	207 W. South Street, Bishop, CA 93514 Ernest Poncet 760/873-7853 (760/873-3236) ovmap@qnet.com
San Bernardino Co. VCP	2355 E. 5th Street, San Bernardino, CA 92410 Joan Mulcare 909/388-4600 (909/386-5148) jmulcare@dph.sbcounty.gov
San Gabriel Valley MVCD	1145 N. Azusa Canyon Rd, West Covina, CA 91790 Steve A. West 626/814-9466 (626/337-5686) swest@sgvmosquito.org
Santa Barbara Coastal VCD	P.O. Box 1389, Summerland, CA 93067 Mitchell J. Bernstein 805/969-5050 (805/969-5643) vector@silcom.com
West Valley MVCD	13355 Elliot Avenue, Chino, CA 91710 Min-Lee Cheng, Ph.D. 909/627-0931 (909/627-0553) wvmvcd@wvmvcd.org

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