• ALTERNATIVES

MEETING THE CHALLENGE OF WEST NILE VIRUS WITHOUT POISONS

By Deanna McKinney

West Nile virus, a mosquitotransmitted disease, was first documented in North America in the summer of 1999 when cases of human illness occurred in New York City. Since that time, West Nile virus has progressively moved across the country.¹ People have been infected in most states in the eastern and midwestern regions of the United States and recently in southern California.²

The virus's rapid expansion across the U.S. has led to increased concern for mosquito control in northwestern states. State and county agencies are currently enhancing mosquito control programs as the virus arrives in the region.³

Mosquito control strategies that rely on pesticides to kill adult mosquitoes are inefficient and use toxic chemicals that pose hazards for our health and the health of the environment. Successful management of West Nile virus requires that communities learn how to control mosquito populations by reducing breeding habitats and learn how to protect themselves from mosquito bites. Strategies aimed at these two measures will ensure that the use of pesticides does not add to the problems caused by this disease.

What is West Nile Virus?

West Nile virus (WNV) is a mosquito-transmitted virus first identified in the West Nile District of Uganda in 1937.¹ Until 1999 when the first documented cases occurred in New York City, the virus was found only in the Eastern Hemisphere in parts of Africa,

Deanna McKinney is an environmental studies student at the University of Oregon.



Asia, the Middle East, and Europe.¹ In areas where the virus is endemic, it is a childhood disease with an immune adult population.⁴

WNV is closely related to St. Louis encephalitis, a virus historically found in the U.S.⁵ Most people infected with WNV show no symptoms of illness or only mild ones. On rare occasions, however, infection can result in a potentially fatal illness known as encephalitis or inflammation of the brain.^{1,2}

Only about 1 in 5 individuals infected with the virus develops mild flu-like symptoms such as fever, head and body aches, swollen lymph glands, and a body rash. About 1 in 150 infected persons develops encephalitis.

Age is the most significant risk factor for severe disease. People age 50 and older have an increased risk of becoming seriously ill if infected, and "patients older than 70 years of age are at particularly high risk."¹

As of November 15, 2002, there were 212 reported deaths from West Nile virus in the United States during the year 2002.² While these deaths are clearly a significant public health issue, in comparison approximately 20,000 deaths per year are attributed

to influenza in the U.S.⁶

, How Does West Nile Virus Affect Animals?

Birds, horses, and other animals are at risk of infection from West Nile virus. It has been detected in dead birds of at least 110 species. In fact, wild bird mortality is one method that is used to monitor the geographic spread of WNV.^{7,8}

Birds belonging to the corvid family (crows, ravens, jays and magpies) appear to be the most susceptible species to date. Raptors also appear susceptible. Many other bird species, such as chickens, usually survive infection.⁷

WNV does not often cause severe illness in dogs and cats. A study of dogs in New York City following the 1999 cases indicated that dogs are frequently infected, but rarely become ill. There has been one documented case of illness in cats.⁹



Horses are more susceptible to West Nile virus than are other livestock and pets.

Of the mammals documented with infection, horses appear to be the most vulnerable. While many (75 percent) horses infected with WNV do not develop any illness, of those that do, between 20-40 percent die or need to

be euthanized.7,10,11

Clinical signs of disease in horses consist of central nervous system problems such as ataxia (stumbling, staggering, wobbly gait, or incoordination), circling, head tilt, hind limb weakness, muscle tremors, and sometimes inability to stand. A WNV vaccine has recently become available for horses.¹¹

According to the U.S. Department of Agriculture, other livestock "do not commonly show any illness if infected with WNV."¹⁰

How is West Nile Virus Transmitted?

West Nile virus is an arbovirus (arthropod borne virus) spread by mosquitoes. Birds serve as the host for the virus, and mosquitoes spread it through their bites. A mosquito first acquires the infection by feeding on a bird infected with the virus. The virus is then transmitted when the mosquito bites a person or animal.¹

Current evidence shows that only mosquitoes can spread the disease. Except for four cases linked to organ transplants, there is no evidence that WNV can be spread from one person to another or from birds and mammals to a person.^{5,9}

The virus is prevalent when adult mosquitoes are abundant. "Among humans, the incidence of disease peaks in late summer and early fall."⁵

Migrating birds are an efficient means of geographic spread of the virus.⁵

Mosquito Biology

Mosquitoes are classified as Diptera (true flies). They play an important role in many ecosystems as a major food source for wildlife, including fish, birds, bats, frogs and other insects.¹²

Mosquitoes pass through four life stages (egg, larva, pupa, and adult). Female mosquitoes require a blood meal to obtain the nutrients required for development of their eggs, so only female mosquitoes bite and are capable of transmitting disease.¹³

Most female mosquitoes lay their eggs directly on the surface of still or slow moving water. After passing through the aquatic stages of egg, larva, and pupa, adult mosquitoes emerge from their water nurseries. Mosquitoes can breed in any still water and some can complete their life cycle in a few days.¹³

The type of standing water in which the female lays her eggs depends on the species. Breeding sites are usually still pools of water that offer shelter



Mosquito larvae (much larger than actual size!).

from wind and water flow. Predators such as fish and other insects in permanent ponds, lakes, and streams usually keep larvae populations under control. Semipermanent ponds, marshes, clogged ditches, puddles, and artificial containers that do not contain predators or have adequate water flow are potential breeding sites. Fastmoving waters such as streams and seas are generally unsuitable habitats for mosquito breeding.¹³

Which Mosquito Species Transmit West Nile Virus?

Most species of mosquitoes do not transmit the disease. Out of the 200 species of mosquitoes found in the U.S., about 30 have tested positive for WNV. However, virus infection does not necessarily mean a species is involved in active transmission.^{1,14}

In the U.S., WNV is primarily associated with *Culex* mosquitoes. *Culex pipiens* and *Culex tarsalis* are the species believed most likely to transmit the virus in the Northwest.^{3,15} Knowing the breeding and feeding habits of these species can help communities reduce mosquito populations and reduce the chances of being bitten.

Culex pipiens (northern house mosquitoes) are widespread in the Northwest, reaching their greatest numbers in urban and suburban areas.

They prefer to lay eggs in polluted water high in organic content such as catch basins, stormwater ponds, and areas where raw sewage drains. They also often deposit eggs in tin cans, tires, birdbaths, backyard ponds, and other containers. Egg-laying occurs from May through September. These mosquitoes usually enter homes and bite humans at night.¹⁶

Culex tarsalis are the most important transmitters of St. Louis and western equine encephalitis in western North America. Larvae of this species tolerate a wide range of water conditions, but are often found in wetlands, salt marshes, and short-lived pools.¹⁷ Larvae are also found in containers, like *Culex pipiens.*³ In most areas adults are active in spring and fall, but in southern California they are active all winter. Adults are most active just after sunset.¹⁷

According to studies conducted by California entomologists, most *Culex* females travel less than 1 kilometer (0.6 miles); the maximum distance traveled was 13 kilometers (7 miles).¹⁸

Mosquito Control

The most important action you can take to control mosquito populations is to reduce mosquito breeding sites around your home and neighborhood, often referred to as "source reduction." The U.S. Centers for Disease Control and Prevention's (CDC's) evaluation of source reduction is that it "remains the most effective and economical method of providing long-term mosquito control in many habitats."¹⁹



Backyard ponds are excellent mosquito habitat.

It is important to remember that even the smallest pool of water can be the source of a large number of mosquitoes. "A coffee can with an inch of water can produce over a thousand mosquitoes every 7 days!"²⁰

In the Northwest, mosquito breeding occurs between early spring and fall, so diligent efforts are particularly important during this time of year.³

To reduce mosquito breeding sites around the home, remove, turn over, or drill holes in the bottom of any container that can hold water, such as tin cans, buckets, ceramic pots, plastic covers, and toys. Old tires should be recycled or stored inside a garage or shed. Change water often in birdbaths, fountains, wading pools, and potted plant trays. Empty water from wheelbarrows and boats. Turn these items over if possible. Cover large boats to prevent accumulation of water and keep drains open.^{21,22}

Make sure roof gutters drain properly and clean them in the spring and fall. Fix leaky outdoor faucets and sprinklers. Drain (or fill with dirt) unneeded or unwanted pools or puddles. Do not dispose of garden debris into catch basins, storm drains or creeks. Clean obstructions to promote flow. Clean vegetation and debris from edges of ponds.^{21,22}



An educational flier prepared by the Washington State Department of Health.

Clean swimming pools. If not in use, tightly cover and keep rain water from accumulating on the cover.^{21,22}

Mosquito fish (*Gambusia affinis*) are commonly used in ponds to control mosquito larvae. While this nonnative fish can potentially cause ecological problems if released into open ecosystems, correct use of the fish can provide effective control of mosquito larvae in enclosed bodies of water, such as garden ponds.²³ Contact your local vector control agency to obtain more information on the use of mosquito fish in your area.

Protection from Mosquito Bites

You can further reduce your risk of infection by protecting yourself from mosquito bites.

Make sure window and door screens are "bug tight." Repair or replace if needed. Gaps can be filled with caulk or weather-stripping. Replace any incandescent outdoor lighting with yellow "bug" lights, which are less likely to attract mosquitoes.²¹

Restrict outside activities from dusk to dawn during mosquito season (early spring to fall) if West Nile virus is a problem in your community. Many mosquito species are active during these times. This is particularly important for the elderly.²¹

When you go outside, wear a long sleeved shirt and long pants. Loose fitting and/or thicker clothes can make it more difficult for mosquitoes to bite. Net shirts and head nets can be important when going into areas with a large number of mosquitoes, such as wetlands or woods.²¹

See "Protection from Mosquito Bites," www.pesticide.org, for more information.

Protection for Domestic Animals

There are several effective steps you can take to protect pets from WNV. These steps are particularly important for protection of horses.

Reduce mosquito breeding sites by scheduling pasture irrigation to minimize standing water. Thoroughly clean water bowls or troughs regularly. Consider stocking troughs with fish that consume mosquito larvae.^{10,11} (See



Emptying buckets, cleaning gutters, and disposing of old tires are simple ways to reduce mosquito habitat.

paragraph on mosquito fish above.)

Reduce exposure to mosquitoes by housing animals in structures with wellmaintained screening. Be careful that mosquitoes are not trapped inside the screened area.^{10,11}

Stable horses during active mosquito feeding time (dusk to dawn). Use of fans may reduce the ability of mosquitoes to feed on horses.^{10,11}

In addition, there is now a WNV vaccination that may help prevent

illness in horses. Contact your veterinarian to find out more about its use in your area. Because complete information on the effectiveness of the vaccine is not yet available, it is important that mosquito control measures still be taken.^{10,11}

Community-wide Efforts

West Nile virus makes headlines in late summer or early fall when mosquito populations and the number of reported virus cases is high. The best time to prevent mosquito problems, however, is much earlier in the year when source reduction efforts can be effective. If your community is facing West Nile virus, don't procrastinate. Find out if there is an existing mosquito control program. If not, encourage your local government to start a prevention program. Take an active role in shaping the management strategies. If your community has a control

GUIDELINES FOR MOSQUITO ABATEMENT PROGRAMS THAT INCLUDE PESTICIDES

NCAP is committed to promoting alternatives and does not recommend the use of pesticides. However, we recognize that many communities faced with West Nile virus outbreaks will begin or expand mosquito spray programs. NCAP recommends the following guidelines for pesticide programs developed in response to West Nile virus:

Prevention

- Communities should act carefully and thoughtfully. Planning should begin early so that crisis actions are not necessary. Educate the local community about West Nile virus.
- No pesticides should be used unless there are also "source reduction" efforts to reduce mosquito breeding habitat. In addition to management of public areas, source reduction should include cooperative efforts with residents and businesses. Comprehensive public education about source reduction is essential.

• Introduce mosquito-eating fish where appropriate.

Control

- Focus on programs to kill mosquito larvae. Using pesticides to kill larvae is, according to the Centers for Disease Control and Prevention, "typically more effective and target-specific"¹ than trying to kill adult mosquitoes, "the least efficient mosquito control technique."¹
- Pesticide applications should target areas where mosquitoes are abundant. Before using a pesticide, vector control districts should conduct systematic monitoring to identify where mosquito population levels are high.
- Pesticide applications should be conducted when they are most effective. For example, applications of the larvicide *Bacillus thuringiensis israelensis* should be scheduled when mosquito larvae are at a susceptible stage.

Guidelines for Pesticides, continued on page 6

program, find out what techniques they use. Encourage mosquito managers to act before a crisis develops and focus on long-term nonchemical source reduction efforts instead of chemical treatments. Source reduction efforts are most effective when government, individuals, and businesses cooperate; be sure your community educates all stakeholders.

Conclusion

Reduction of mosquito problems around homes and neighborhoods can be successfully achieved with just a few simple steps. Focus on the reduction or elimination of mosquito breeding habitats, any place or container that collects standing water. Individuals and communities can have a large impact on reducing the risk of West Nile infection without using pesticides.

References

 Petersen, L.R., and A.A. Marfin. 2002. West Nile virus: A primer for the clinician. Annals of Internal Medicine. 137(3):173-179.

- Centers for Disease Control and Prevention. 2002. West Nile virus basics. www.cdc.gov/ ncidod/dvbid/westnile/index.htm.
- Washington State Dept. of Health. 2002. Mosquito-borne disease response plan.
- Kettle, D.S. 1995. Medical and veterinary entomology. 2nd ed. Chap. 24. Arboviruses. Wallingford, U.K.: CAB International. Pp. 489-516.
- Petersen, L.R., J.T. Roehrig, and J.M. Hughes. 2002. West Nile virus encephalitis. *New England Journal of Medicine*. 347(16):1225-1226.
- Bridges, C.B. et al. 2002. Prevention and Control of Influenza. Morbidity and Mortality Weekly Report 51:1-31. www.cdc.gov/mmwr/preview/ mmwrhtml/rr5103a1.htm, Apr. 12.
- Centers for Disease Control and Prevention. 2002. West Nile virus: Vertebrate ecology. www.cdc.gov/ncidod/dvbid/westnile/ birds&mammals.htm.
- U.S. Geological Survey. National Wildlife Health Center. 2002. West Nile virus continues to move west. Wildlife Health Alert #02-01. www.nwhc.usgs.gov/whats_new/wha/ wha0201.html.
- Komar, N., N.A. Panella, and E. Boyce. 2001. Exposure of domestic animals to West Nile virus during an outbreak of human encephalitis, New York City, 1999. *Emerg. Infec. Dis.* 7:736-738.
- U.S. Dept. of Agriculture. Animal and Plant Health Inspection Service. 2002. Prevention and control of West Nile virus infection in equine and other livestock or poultry. www.aphis.usda.gov/oa/wnv/prv.html.
- California Dept. of Food and Agriculture. Animal Health Branch. Undated. West Nile virus: Information for horse owners. www.cdfa.ca.gov/ahfss/ ah/pdfs/WNV%20fact%20sheet%20fc5.pdf.

- 12. National Park Service. 1998. National Park Service integrated pest management manual: Mosquitoes. www.mature.nps.gov/wv/ipm/mosquito.html.
- Service, Mike. 2000. Medical entomology for students. 2nd ed. Cambridge, U.K.: Cambridge University Press. Pp. 6-12.
- Turell, M.J. et al. 2001. Vector competence of North American mosquitoes (Diptera: Culicidae) for West Nile virus. *J. Med. Entomol.* 38: 130-134.
- Oregon Dept. of Human Services. West Nile virus-moving wester. 2002. *CDSummary* 51(18).
 Crans, Wayne. Undated. *Culex pipiens* Linnaeus.
- www-rci.rutgers.edu/~insects/pip2.htm.
 17. Reisen, W. 1993. The western encephalitis mos-
- Reisen, w. 1993. The western encephalitis mosquito, *Culex tarsalis. Wing Beats* 4(2):16. wwwrci.rutgers.edu/~insects/sp6.htm.
- Reisen, W.K., M.L. Milby, and R.E. Meyer. 1992. Population dynamics of adult Culex mosquitoes (Diptera: Culicidae) along the Kern River, Kern County, California, in 1990. J. Med. Entomol. 29:531-543.
- Centers for Disease Control and Prevention. 2001. Epidemic/epizootic West Nile virus in the United States: Revised guidelines for surveillance, prevention, and control. www.cdc.gov/ ncidod/dvbid/westnile/resources/wnv-guidelinesapr-2001.pdf.
- Multnomah Co. Health Dept. 2001. About mosquitoes. www.mchealthinspect.org/vector/mosq.htm.
- 21. U.S. EPA. Office of Pesticide Programs. 2002. Mosquitoes: How to control them. www.epa.gov/ pesticides/citizens/mosquito.htm.
- Alameda Co. Mosquito Abatement District. Undated. Are you raising mosquitoes in your backyard? www.mosquitoes.org/Publicinfo/ControlGuide.html.
- Alameda Co. Mosquito Abatement District. 1999. The Alameda Co. mosquito abatement district control program. Hayward, CA.

Guidelines for Pesticides, continued from page 5

- Choose application techniques to minimize exposure of people and the environment. Ground applications can be more precisely targeted than aerial applications.
- Choose pesticides that selectively target mosquitoes.
- Choose pesticides to minimize hazards to people and



Community spray programs are a common response to West Nile virus outbreaks.

the environment. See "Hazards of Pesticides Used to Kill Adult Mosquitoes," page 7 and "Hazards of Pesticides Used to Kill Mosquito Larvae," page 8.

- As part of a decision to use pesticides, a communityspecific assessment of the health and environmental hazards of proposed products should be conducted. National or state-level assessments are not adequate to protect local communities. Assessments must include all pesticide ingredients.
- Community residents and businesses should be notified at least 72 hours prior to any pesticide application.
- Community residents should be informed about what they can do to minimize pesticide exposure.

Monitoring

- Monitor mosquito populations before and after pesticide applications in order to target applications and document their efficacy.
- Monitor pesticide contamination of the environment to identify potential problems.
- Establish a "hotline" to collect reports of any suspected pesticide-related illnesses from residents and physicians. —*Caroline Cox*
- Centers for Disease Control and Prevention. 2001. Epidemic/epizootic West Nile virus in the United States: Revised guidelines for surveillance, prevention, and control. www.cdc.gov/ncidod/dvbid/westnile/resources/ wnv-guidelines-apr-2001.pdf.

Name	Selectivity	Health Hazards	Environmental Hazards
Malathion	Kills all insects	Malathion is in the organophosphate chemical family, and like all members of this family, disrupts nervous system function by inhibiting the activity of an important enzyme.(1) It has similar effects on the human nervous system,(2) causing headaches, nausea, and diarrhea.(3) Other health hazards identified in laboratory studies include genetic damage (chromosome aberrations) ,(4) damaged sperm,(4) altered immune system function,(5) and increased incidence of breast tumors.(6) Use of malathion is also associated with an increased risk of the cancer non-Hodgkin's lymphoma.(7)	Malathion frequently contaminates water. In birds (quail) malathion exposure caused ovaries to stop functioning and reduced the number of eggs that successfully hatched. The U.S. Environmental Protection Agency (EPA) classifies malathion as highly or very highly toxic to most species of fish. Fish kills of up to thousands of fish have been reported to EPA following mosquito spraying.(8)
Naled	Kills all insects	Like malathion, naled is in the organophosphate chemical family and disrupts nervous system function, causing headaches, nausea, and diarrhea in exposed people.(3) Naled is more toxic when exposure occurs by breathing contaminated air than through other kinds of exposure.(9,10) In laboratory tests, exposure to naled's breakdown product dichlorvos caused increased aggressiveness and a deterioration of memory and learning.(11,12) Dichlorvos also interferes with prenatal brain development (13) and is classified as a cancer causing chemical by the International Agency for Research on Cancer.(14)	According to EPA, naled is moderately to highly toxic to birds and fish.(15) In laboratory tests, exposure to naled reduced egg production and hatching success in birds and reduced the growth of juvenile fish.(15) Naled mosquito spraying has been "directly correlated" with precipitous declines in populations of an endangered butterfly.(16)
Resmethrin	Kills all insects	Resmethrin acts by disrupting nervous system function; the World Health Organization refers to resmethrin as a "neuropoison."(17) Its effects on the human nervous system are similar to its effects in insects.(17) In laboratory studies resmethrin's most important health hazards relate to its ability to disrupt reproduction. For example, in two studies in which rats were exposed to resmethrin during pregnancy, the insecticide caused an increase in the number of stillborn offspring. The increase was significant even at the lowest exposure tested in these experiments.(18)	Low concentrations of resmethrin are toxic to fish; for example, 3 parts per billion (ppb) kill rainbow trout. Even lower concentrations (less than one ppb) kill juvenile fish and reduce their growth. Scourge, a common resmethrin-containing mosquito insecticide product, is over five times more toxic to rainbow trout than resmethrin itself. Resmethrin also bioconcentrates in fish tissue.(19)

- U.S. EPA. 2000. Malathion: Human health risk assessment for the reregistration eligibility decision (RED) document. Memo from P.A. Deschamp, Health Effects Div., to P. Moe, Special Review and Reregistration Div. p. 1,9. www.epa.gov/pesticides.
- Ware, G.W 2000. The pesticide book. Fresno, CA: Thomson Publications. Pp. 180-183.
- Reigart, J.R. and J.R. Roberts. 1999. Recognition and management of pesticide poisonings. Fifth edition. U.S. EPA. Office of Pesticide Programs. p. 34. www.epa.gov/pesticides/safety/healthcare.
- Giri, S. et al. 2002. Genotoxic effects of malathion, an organophosphorus insecticide, using three mammalian bioassays in vivo. *Mutat. Res.* 514: 223-231.
- Johnson, V.J. et al. 2002. Increased T-lymphocyte dependent antibody production in female SJL/J mice following exposure to commercial grade malathion. *Toxicol.* 170:119-129.
- Cabello, G. et al. 2001. A rat mammary tumor model induced by the organophophorous pesticides parathion and malathion, possibly through acetylcholinesterase inhibition. *Environ. Health Persp.* 109: 471-479.
- McDuffie, H.H. et al. 2001. Non-Hodgkin's lymphoma and specific pesticide exposures in men: Cross-Canada study of pesticides and health. *Canc. Epidemiol., Biomark. and Prev.* 10: 1155-1163.
- U.S. EPA. 2000. Malathion reregistration eligibility document. Environmental fate and effects chapter. Pp. 28-48, 51-52, 60-62, 74. www.epa.gov/pesticides.
- Berteau, P.A. and W.A. Dean. 1978. A comparison of oral and inhalation toxicities of four insecticides to mice and rats. *Bull. Environ. Contam. Toxicol.* 19: 113-120.
- 10. U.S. EPA. Office of Pesticide Programs. Health Effects Division. 1999.

Human health risk assessment: Naled. www.epa.gov/pesticides/op/ status.htm. Pp. 26-28.

- Sarin, S. and K.D. Gill. 1998. Biochemical and behavioral deficits in adult rat following chronic dichlorvos exposure. *Pharmacol. Biochem. Behavior* 59: 1081-1086.
- Sarin, S. and K.D. Gill. 1999. Dichlorvos induced alterations in glucose homeostasis: Possible implications on the state of neuronal function in rats. *Mol. Cell. Biochem.* 199:97-92.
- Mehl, A. et al. 1994. The effect of trichlorfon and other organophosphates on prenatal brain development in the guinea pig. *Neurochem. Res.* 19:569-574.
- International Agency for Research on Carcinogens. 1991. Occupational exposures in insecticide applications, and some pesticides. *IARC Monographs* 53: 267.
- U.S. EPA. 2002. Interim reregistration eligibility decision for naled. Case number 0092. (Unpublished document.) p. 32,34-35.
- Emmel, T.C. 1991. Overview: Mosquito control, pesticides, and the ecosystem. In *Mosquito control pesticides: Ecological impacts and management alternatives*, ed. T.C. Emmel and J.C. Tucker. Gainesville FL: Scientific Publishers, Inc.
- World Health Organization. 1989. Resmethrins: Resmethrin, bioresmethrin, cismethrin. Environmental Health Criteria 92. Geneva: United Nations Environment Programme, International Labour Organization, and World Health Organization. Pp.11-12.
- U.S. EPA. 1988. Integrated Risk Information System (IRIS): Resmethrin (CASRN 10453-86-8). www.epa.gov/iris.
- 19. Rand, G.M. 2002. Hazard assessment of resmethrin I. Effects and fate in aquatic systems. *Ecotoxicol.* 11: 101-111.

Name	Selectivity	Health Hazards	Environmental Hazards
Temephos	Kills all insects	Temephos is in the organophosphate chemical family. It disrupts nervous system function similarly to malathion and naled. Temephos was first registered for use as a pesticide in 1965, and its toxicology testing is out of date. According to EPA there are "several data gaps"(1) and "most of the available studies were conducted in the 1960s and 1970s and do not meet the current requirements"(1) The symptoms occurring at the lowest exposures in these tests are caused by temephos's neurotoxicity.(1)	Temephos is highly toxic to a variety of aquatic animals including stoneflies, water fleas, oysters, and shrimp. It bioconcentrates in fish. The temephos emulsifiable concentrate product is very highly toxic to rainbow trout. Temephos is highly toxic to quail.(2) Pheasants, pigeons, and sparrows are also particularly susceptible to temephos.(3)
Golden Bear Oil	Kills aquatic insects that breathe at the water surface by forming a suffocating barrier.(4)	Golden Bear Oil is a petroleum distillate.(5) In a laboratory test, this petroleum distillate caused skin cancer, although the frequency of cancers was less than the frequency of cancers caused by other oils with higher concentrations of aromatic hydrocarbons.(6) It also causes genetic damage in assays with bacteria.(7)	Applications of Golden Bear OII "had a strong negative impact" on an abundant insect in California marshes (water boatmen). Golden Bear Oil causes reduced hatching success and malformations in duck eggs exposed to the oil at concentrations about three times the recommended application rates. These are concentrations that might occur because of drift or misapplication.(4) It is toxic to fish and resistant to biodegradation.(8)
Methoprene	Insect growth regulator that prevents larval mosquitoes becoming adults; similar effects occur in many insect species.(9)	EPA's 1991 evaluation of methoprene concluded that it causes "no significant adverse toxicological effects" in vertebrates.(10) However, more recent research shows methoprene stimulates gene activity in vertebrates and acts like retinoids, compounds that "play essential roles in many aspects of development, metabolism, and reproduction."(11) It also has a "weak mutagenic effect" in laboratory tests.(12)	Methoprene is moderately toxic to fish(10) and reduces reproduction in crustaceans.(13) It can delay the development and population growth of aquatic animals that are a food resource for birds (14) and "drastically reduced" diversity when applied repeatedly to wetlands.(15) Exposure of tadpoles to low concentrations (1 ppb) of methoprene's breakdown products causes deformities.(16)
Bacillus thuringiensis israelensis (Bti)	"Highly specific"(17); Bti is toxic to many species of flies but to only a handful of other insects.(18)	Bti is a naturally occurring soil bacteria. Its few human toxicity concerns include the following: it has infected a person when combined with a common skin bacteria; commercial Bti can contain a toxin that causes diarrhea; and laboratory animals with compromised immune systems develop skin lesions when exposed to Bti.(18) Based on laboratory studies, EPA concluded that "toxicity and infectivity risks" to nontarget animals are "minimal to nonexistent."(19)	A University of Minnesota field study showed that repeated applications of Bti drastically reduced the diversity of nontarget insects and other invertebrates.(15)

- 1. U.S. EPA. 1999. Temephos RED. Pp. 4,5,7-9. www. epa.gov/pesticides, Oct. 6.
- U.S. EPA. 1999. EFED RED chapter for temephos. www.epa.gov/pesticides, Oct. 6.
- World Health Organization. 1978. Data sheets on pesticides No. 8 Rev. 1: Temephos. Food and Agriculture Organization. www.inchem.org.
- Miles, A.K. et al. 1999. Experimental assessment of the toxicity of the mosquito larvicide Golden Bear Oil (GB-1111): (1) Field evaluations on duckling, target, and non-target prey survival; (2) Laboratory evaluations on reared mallard and bobwhite eggs, and wild redwing blackbird eggs. U.S. Geological Survey; University of California, Davis. http:// pacific.fws.gov/ecoservices/envicon/pim/reports/.
- Clarke Mosquito Control Products, Inc. Undated. Golden Bear Oil sample label. Roselle, IL.
- 6. Broddle, W.D. et al. 1996. Chronic dermal studies of petroleum streams in mice. *Fund. Appl. Toxicol.* 30: 47-54.
- Brooks, T.M. et al. 1995. Evaluation of modified bacterial mutagenicity assays for the genotoxicity testing of mineral oils. *Mutag.* 10: 409-415.
- Gulf Oil International. Undated. Material safety data sheet: Gulf Transcrest H, Gulf Transcrest HD Type 1 and 2, Transformer Oil N. www.gulfoilmanila.com.
- Ware, G.W 2000. The pesticide book. Fresno, CA: Thomson Publications. Pp. 274-275.
- U.S. EPA. 1991. Reregistration eligibility document: Isopropyl (2E,4E)-11-methoxy-3,7,11-trimethyl-2,4-dodecadienoate. www.epa.gov/pesti-

cides. Pp. 3,5,14.

- Harmon, M.A. et al. 1995. Activation of mammalian retinoid X receptors by the insect growth regulator methoprene. *Proc. Natl. Acad. Sci.* USA 92:6157-6160.
- Marec, F., R. Socha, and I. Gelbic. 1987. Mutagenicity testing of the juvenoid methoprene (ZR-515) by means of the *Drosophila* wing spot test. *Mutat. Res.* 188:209-214.
- Olmstead, A.W. and G.L. LeBlanc. 2001. Low exposure concentration effects of methoprene on endocrine-regulated processes in the crustacean *Daphnia magna. Toxicol. Sci.* 62:268-273.
- Lawrenz, R.W. 1984/1985. The response of invertebrates in temporary vernal wetlands to Altosid® SR-10 as used in mosquito abatement programs. J. Minn. Acad. Sci. 50:31-34.
- Hershey, A.E. et al. 1998. Effects of *Bacillus thuringiensis israelensis* (Bti) and methoprene on nontarget macroinvertebrates in Minnesota wetlands. *Ecol. Appl.* 8: 41-60.
- La Clair, J.J., J.A. Bantle, and J. Dumont. 1998. Photoproducts and metabolites of a common insect growth regulator produce developmental deformities in *Xenopus. Environ. Sci. Technol.* 32:1453-1461.
- World Health Organization. 1999. *Bacillus thuringiensis*. Environmental Health Criteria 217. International Programme on Chemical Safety. p.3.
 Glare, T.R. and M. O'Callaghan. *Bacillus thuringiensis: Biology, ecology,*
- and safety. Chichester U.K.: John Wiley & Sons. Pp. 33, 66-67.
 U.S. EPA. Prevention, Pesticides and Toxic Substances. 1998.
- U.S. EPA. Prevention, Pesticides and Toxic Substances. 1998. Reregistration Eligibility Decision: *Bacillus thuringiensis*. p. 16.