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STATEMENT OF
SHAWNEE HOOVER, SPECIAL PROJECTS DIRECTOR
BEYOND PESTICIDES
ON
H.R. 1749
PEST MANAGEMENT AND FIRE SUPPRESSION ACT
TO AMEND THE
FEDERAL WATER POLLUTION CONTROL ACT (33 U.S.C. 1342(1))
aka CLEAN WATER ACT
BEFORE THE
SUBCOMMITTEE ON WATER RESOURCES AND ENVIRONMENT
U.S. HOUSE OF REPRESENTATIVES

SEPTEMBER 29, 2005

APPENDICES A - D

APPENDIX A

- In 2003 the city of **Boulder, CO** did not use adulticides to combat the presence of West Nile virus (WNV) and showed an 80% reduction in mosquito populations reporting as many as 94 million mosquitoes killed prior to becoming biting adults. The city also reported lower attack rates (or rates of serious illness) per population than surrounding cities where adulticiding took place.[1]

- Despite high mosquito counts and large percentages of infected birds, **Shaker Heights, Ohio** refused to adulticide, like its neighboring cities in Cuyahoga County, due to concerns of health hazards and efficacy. 2002 results showed that Shaker Heights had only 2 human WNV cases out of the county's 219 cases.[2]

- The counties of **Goshen and Plate, WY** rely heavily on adulticides and in 2003 counted 80 WNv cases, 8 fatalities and 77 cases, 3 fatalities, respectively. The neighboring county of **Cheyenne**, with 2 times the population and 3 times the landmass, used only larvicides and had 20 cases of WNv and 1 fatality.[3]

- **Lyndhurst, Ohio**, passed a landmark ordinance in 2003 prohibiting the spraying of pesticides for WNv. During a Task Force sponsored forum, a panel of experts discussed the hazards and low efficacy of adulticides. The Council stated, "[T]here is substantial belief that the more effective way of controlling the mosquito populations is by larvacide treatment and thorough education..." Concluding that, "[T]he dangers of WNV are minimal and affect a very small segment of the population and that the long-term health and environmental risks of spraying with synthetic pesticides poses a much greater risk."[4]

- **Washington, DC** health officials continue their no-spray policy stating that pesticide spraying is inappropriate in a heavily populated area with asthmatics. Instead, officials focus on larval control and pubic education, with education materials distributed in four languages. The Department of Health is also implementing a Tire Round-Up program for residents to discard old tires, a major breeding site for mosquitoes.[27]

- In **York County, Virginia**, officials distribute the mosquito eating fish, *Gambusia holbrooki*, to residents in order to decrease pesticide use for mosquito control. Several thousand of the fish have been bred by the county's fishery as part of its mosquito prevention program.[5]

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[1] City of Boulder WNV Surveillance and Control Plan, 2003 Season.
[2] Lynch, Joe. Cuyahoga County Board of Health, and Ryan Sullivan, Shaker Heights WNV Task Force. Personal Communications, June/July 2004, Beyond Pesticides.
[3] Lee, Robert A. Director Environmental Management, City of Cheyenne and Larimer County. Personal Communication, April 2004, Beyond Pesticides.
[4] Beyond Pesticides. 2003. "Ohio City Adopts Landmark Law to Stop Pesticide Spraying for West Nile Virus." *Daily News*. Washington, DC. July 14.
[5] York County Environment and Development Services. Div. Drainage and Mosquito Control. Hhttp://www.yorkcounty.gov/eds/fishhatchery.htmH (July 2, 2004)

- In **Dallas, Texas**, the City Council's Health, Environment and Human Services Committee adopted a mosquito control plan in 2003 that calls for more public education and allows the use of pesticide sprays only as a last resort and upon approval of the pertinent council member.[6]

- **Ft. Worth, Texas** has not sprayed for mosquitoes since 1991. In 2003, Ft. Worth had 3 WNV cases and no deaths. Brian Boerner, Director of Environmental Management, states, "the spraying of chemicals also has the potential of contaminating our waterways, killing the beneficial fish and organisms that feed on mosquito larva, adding harmful volatile organic chemicals to the atmosphere-a precursor chemical to ozone formation-and providing a potential inhalation or ingestion hazard to residents." [7]

- **Nassau County, New York** joins others in using predacious fish in hard to reach salt-water marshes.[8]

- **Marblehead, MA** has a WNV Response Plan that requires a town hall meeting before the use of adulticides (and only after a locally-acquired human death).[9]

- In 2003, **Boulder, Colorado** focused on larviciding, surveillance and public education without the use of adulticides and offered free WNv information workshops for neighborhood groups and distributes free samples of Mosquito Dunks, a least-toxic larvacide product, for use in stagnant water.[14]

- In preparation for WNv, **Lane county, OR** have an easy to read public educational flyer that is put in local newspapers and distributed with utility bills early in the season.[10]

- In 2003, **Seattle, Washington** adopted an Integrated Pest Management Plan for Mosquito Control, which identifies public education, personal protection, and breeding source reduction on public property as, "…the most effective and appropriate techniques for the City to use."[11]

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[6] Beyond Pesticides. 2003. "Virginia and Texas Towns Find Alternatives for West Nile Virus Control." *Daily News*. June 12, 2003.
[7] Ft. Worth Public Health Department, Mosquito Prevention and Control. Hhttp://www.fortworth.gov/health/HP/mosqinees.aspH (viewed July 6, 2004)
[8] Turrillion, G. 2002. Director of Mosquito Control Program in Nassau County, NY. Personal Communication. March.
[9] Town of Marblehead, MA West Nile Virus Protocol and Response Plan. 2002. Hhttp://www.beyondpesticides.org/mosquitoH (July 6, 2004)
[10] Northwest Coalition for Alternatives to Pesticides. July 2004. Personal Communication.
[11] City of Seattle. Office of Sustainability and Environment. February 20, 2002. Integrated Pest Management Plan for Mosquito Control. Hhttp://www.cityofseattle.net/environment/Documents/WNV%20IPM.pdfH (July 2, 2004)

APPENDIX B

West Nile Virus and Mosquito Control

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INTRODUCTION

The West Nile virus, which causes serious encephalitis in Americans, was introduced from Africa into northeastern United States in 1999. No one knows exactly how the virus was transported here, but with rapid air travel and large numbers of people and goods being moved throughout the world, the West Nile virus could have been carried to the United States by an infected bird, person, or even by a mosquito.

By the year 2003, the Centers for Disease Control (CDC) reported there were 8900 reported human infections of the West Nile disease with 218 deaths, with many of the infections and deaths occurring in Ohio. The rate of infections and deaths is running significantly ahead of last year, with most of the infections and deaths occurring in Colorado where the incidence has increased from only 14 infections in 2002 to 635 West Nile infections by August 2003.

BIRD RESERVOIRS

The prime reservoir of West Nile is the bird population. At least 125 species of birds have been reported infected with West Nile,[1] with crows, blue jays, sparrows, hawks, eagles, and others identified as reservoirs. Birds appear to be especially susceptible to the virus and are more likely to die of an infection than are humans. In some localities crows and blue jays have all but disappeared. Estimates are that 20,000 birds were killed last year from West Nile in the United States. Because birds travel long distances in their seasonal migrations, infected birds spread the disease to humans, horses, and other animals. Mosquitoes obtain the virus mostly from infected birds and in turn infect humans by biting them.

MOSQUITO VECTORS

In the Northeast, the prime mosquito vector between birds and humans is *Culex pipens*, the house mosquito. In New York and New Jersey, when 32,000 mosquitoes were examined by the CDC,[2] the great majority associated with West Nile were *Culex pipens*.[3] In Colorado, the prime mosquito vectors are *Culex tarsalis* and *Culex pipens*. Other mosquito species capable of transmitting the West Nile virus include other *Culex* species, *Anopheles* sp, *Coquilletidia* sp., *Ochlerotatus* spp., and *Psorophora* sp.

Male mosquitoes feed primarily on nectar and do not bite humans. The female mosquito requires a blood meal and when she bites an infected bird she then transmits the West Nile virus to humans by biting them.

The life cycle of *Culex* mosquito is about 14 days at temperatures of about 21°C (70°F). The female obtains her blood meal from birds, humans, and other animals. She mates either before or after her blood meal. Then she lays about 250 eggs in pools of water, including bird baths, flower pots, tin cans, old tires, as well as other pools of collected water. The egg stage lasts 1 to 2 days and the emerging larvae feed on algae, bacteria, and other organic matter in the water. The larval stage lasts 7 days followed by the pupal stage that lasts 2 to 3 days. Adult mosquitoes emerge from the pupae and the life cycle begins again. The adult mosquitoes normally live a week or two, but also hibernate in protected locations during the winter (Fig. 1).

Adult mosquitoes are not strong fliers and usually travel only a few hundred feet from the place of emergence. They may be carried by the wind several miles. In general, when the wind is blowing above 5 mph they will not fly. Female mosquitoes feed most often during the evening and morning.

MOSQUITO LARVAL CONTROL

The CDC advises that mosquito control should focus primarily on mosquito larval control and secondarily on the less efficient adulticiding.[4] Effective larval control curtails the supply of adult mosquitoes.

In aquatic habitats, mosquito larvae have many predators, but few parasites. The predators include damselfly larvae, back swimmers, dragonfly larvae, water boatman, dytiscid beetles, frogs, fishes, and salamanders. However, none of these predators is effective because they usually inhabit permanent water bodies, whereas most mosquito larvae live in temporary pools of water.

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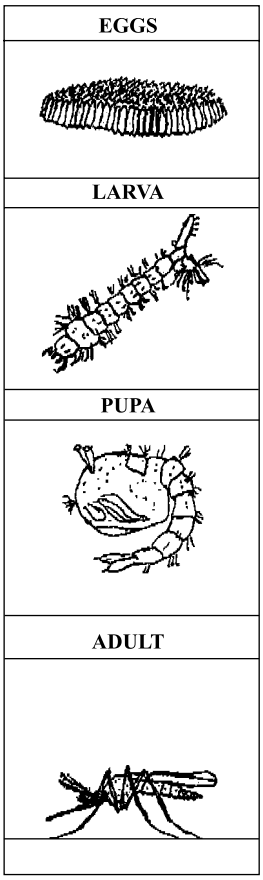

Fig. 1 *Culex* mosquito eggs, larva, pupa, and adult female.

Although mosquito larvae can be killed by bacteria, protozoans, nematodes, and fungi, none of these provides control for large mosquito populations. One exception, a strain of *Baccilus thringiensis isrraeliensis* or BT has proven effective. Various commercial formulations of this bacterium are available for application to ponds and pools where larvae are found.

In addition to eliminating all mosquito breeding sites, such as bird baths, flower pots, tires, ponds, and pools of water, such breeding habitats may also be treated, provided some water remains in them. BT is an effective larvacide that is safe for humans and pests, but it may kill some beneficial insects in water bodies.

In some small bodies of water, a thin layer of light oil can be spread over the surface. This will kill both mosquito larvae and pupae in the water. However, the oil also may have negative impacts on small fish and arthropods in the water.

Most insecticides are banned from water bodies because they are highly toxic to most aquatic organisms, such as fish, frogs, salamanders, and arthropods.

ADULT MOSQUITO CONTROL

Instead of focusing control efforts on larval mosquitoes as suggested by CDC, most homeowners and municipalities focus on adult mosquito control.

Adult Mosquito Control with Predators

Adult mosquitoes have relatively few predators because they are so small and not a large meal for a predator. Dragonflies, bats, and small birds such as purple martins feed on a few adult mosquitoes, but none of these animals can be counted on to control large populations of adult mosquitoes.

ULTRALOW VOLUME SPRAYING

Before municipalities spray for mosquitoes, the mosquito population should be measured for 5 days before spraying and 5 days after spraying using various mosquito traps. Such data will assist the government officials to determine whether the several thousand or millions of dollars spent in spraying was effective.

Homeowners should require warning 72 hr in advance of community spraying. During spraying, the windows and doors should be closed and the people should stay inside away from the insecticide spray.

When many West Nile infected birds are found and the mosquito population is relatively abundant, municipalities are often pressured into spraying pyrethroid insecticides for mosquito control. This spraying is carried out using trucks mounted with ultralow volume (ULV) sprayers. The insecticide spray produced from these units is like a smoke or fine mist and is carried downwind. Even assuming that the spraying is carried out in the evening when wind is minimal, the spray is carried downwind in an open area, for instance, on a golf course. Downwind, from 150 to 300 ft and at 3 ft height, the mosquito kill will range from 25% to 75%.[5] However, ZERO mosquitoes will be killed upwind by the insecticide spray. Thus the average upwind and downwind kill is only 21% to 45%. Note, the insecticide spray does not penetrate buildings, and mosquitoes behind buildings are not killed. Further, dense vegetation hinders spray treatment and desired mosquito control. For example, downwind in a dense stand of trees, mosquito kill is reported to be only 34% to 58%.[5]

For effective mosquito control, at least 90% of the adults must be killed. Only a few scientific studies of the effectiveness of spraying for mosquito control have been reported. These results are relatively discouraging. For

example, in Greenwich, CT, only a 34% mosquito population reduction was reported after ground spraying, and in Houston, TX, only a 30% reduction occurred after spraying.[6] Then in Cicero Swamp, FL, populations of disease-carrying mosquito populations increased 15-fold after spraying,[6] when the mosquito population was measured 11 days after spraying. However, it is doubtful that the insecticide spray caused the increase in the mosquito population, but clearly the insecticide provided insufficient adult mosquito control.

Aerial ULV Spraying

The aerial application of insecticides for adult mosquito control has some advantages over ground applications. Reports on the effectiveness of aerial ULV spraying range from 42% to 93%.[7,8] However, using ULV aerial equipment results in only 10% to 25% of the insecticide reaching the target area, whereas up to 90% drifts away from the target into the environment at large.[9,10] Aerial application covers a larger area faster than the ground application equipment, but it is more expensive than ground application, costing from $250 to $1000 per hour (truck spraying costs from $150 to $250 per hour). Also to be considered are the serious public health and environmental problems associated with the application of insecticides from aircraft.[11]

Insecticide Effectiveness in Reaching Target Mosquitoes

With ULV spraying, the spray particles are minute and measure from 7 to 22 μm. The lethal dose of a pyrethroid insecticide is one particle 18 to 20 μm. Based on the fact that many billions of spray droplets are produced per kilogram of insecticide for both ground and aerial spraying, less than 0.0001% of the insecticide applied is reaching the target mosquitoes.[12] Thus by both ground and aerial application 99.999% of the insecticide spreads into the environment, when it can cause public health and other environmental problems.

Because many adult mosquitoes remain after spraying and more adult mosquitoes will emerge, if the mosquito larvae are not controlled, then insecticide spraying is required every 7 days. Costs of spraying every 7 days are prohibitive.

PERSONAL PROTECTION

Homeowners should drain standing water in pools, gutters, and flower pots in the yard. Water in bird baths and wading pools should be changed every 3 days. If outdoors during dawn or dusk when mosquitoes are most abundant and the wind is not blowing, then long pants and a long-sleeve shirt made of heavy material, such as denim, should be worn. Adult mosquitoes easily bite through a light T-shirt.

Various adult mosquito traps and zappers are sold to homeowners for control, but rarely do these units provide continuous satisfactory control of mosquitoes.[13] While outside, homeowners may use an insecticide fogger or can of insecticide spray for temporary control of mosquitoes. However, if the wind is blowing sufficiently strong (5 mph or stronger), the mosquitoes will not be a problem because the mosquitoes will not fly in the wind.

Of the numerous chemical repellants, the most popular is the pesticide, DEET. DEET should be applied only to the outer layer of heavy clothes. The chemical should only be used, if there is a serious West Nile threat. DEET has been known to cause rashes, restlessness, lethargy, confusion, slurred speech, clumsiness, seizures, and in a few cases death.[14] For some individuals, the DEET pesticide is reported to cause allergic reactions and may interfere with the immune and endocrine systems for some people.

Located on a patio or other small area, a large fan blowing air about 5 mph or higher will discourage the presence of mosquitoes.

CONCLUSION

West Nile virus is a health hazard to humans, birds, horses, and other animals. *Culex* mosquitoes are important vectors in the United States. The prime method of control is the elimination of the breeding habitats for larval mosquitoes, such as water accumulating in bird baths, flower pots, old tires, and other containers.

Widespread ULV spraying from ground equipment or aircraft for control of mosquitoes and West Nile virus is relatively ineffective, costly, and has been associated with environmental and public health risks.

During the evening and early morning, repellants can protect humans from mosquito bites. However, the pesticide DEET and related chemicals should not be applied directly to the skin of children or adults, because they pose serious public health risks.

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APPENDIX C

There is mounting scientific evidence that synthetic pyrethroids, increasingly the most popular mosquito pesticide, are capable of disrupting the endocrine (hormonal) system in wildlife and humans at extremely low doses (ppb).

The following are a sample of studies to illustrate this point.

Aziz MH, Agrawal AK, Adhami VM, Shukla V, Seth PK. 2001. Neurodevelopmental consequences of gestational exposure (GD14-GD20) to low dose **deltamethrin** in rats. Neurosci Lett 300(3):161-165.

Abstract: Effect of low level in utero exposure to deltamethrin (DT) (1 mg/kg wt.) during gestation day 14-20 was studied on selected neurobehavioral, neurochemical, immunohistochemical parameters in rats at 6 and 12 weeks postnatal period. The significant increase in acetylcholinesterase activity and decrease in H-3-quinuclidinyl benzilate binding in the hippocampal region of DT exposed animals, suggesting impairment in cholinergic (muscarinic) receptors. A significant decrease in the learning and memory performances was also observed both at 6 and 12 weeks, which is directly correlated with decrease in muscarinic receptor binding. Immunohistochemistry and image analysis of growth associated protein-43, a neuron specific protein present in axonal growth cone and a marker for neuronal differentiation and synaptogenesis, exhibit aberrant increase in its expression in the hippocampus in DT exposed rats at both time periods. The data suggests that low level exposure to DT in utero during brain growth spurt period adversely affects the developing brain and the changes persist even upto 12 weeks postnatal period in rats. Although there is no significant recovery at 12 weeks assessment but still significant impairment persist on biochemical and behavioural parameters.

[Deltamethrin = Pyrethroid insecticide]

Berrill M, Bertram S, Wilson A, Louis S, Brigham D, Stromberg C. 1993. Lethal and sublethal impacts of **pyrethroid insecticides** on amphibian embryos and tadpoles. Environmental Toxicology & Chemistry 12:525-539.

Abstract: Amphibian populations are potentially sensitive to aquatic contaminants such as pesticides. We exposed embryos and larvae of five amphibians (the frogs *Rana sylvatica, Rana pipiens, Rana clamitans*; the toal *Bufo americanus*; the salamander *Ambystoma maculatum*) to one or both of the pyrethroid pesticides permethrin and fenvalerate. Concentrations ranged from 0.01 ppm to 2 ppm, and exposures lasted 22 or 96 h. No significant mortality of embryos, anuran tadpoles, or salamander larvae occurred during or following exposure to pyrethroids. However, tadpole growth was delayed following exposure, and tadpoles and salamander larvae responded to prodding not by darting away but by twisting abnormally. Both effects may result in greater vulnerability to predation. Recovery of normal avoidance behavior occurred more rapidly at 20 than at 15şC and following exposure to lower concentrations of the pesticides, indicating both temperature and dose effects. Tadpoles exposed later in development did not feed for a period of days following exposure but were still capable of metamorphosis.

Of the five tested species, *Ambystoma maculatum*, a tadpole predator, was particularly sensitive. An amphibian community is therefore likely to be sensitive to low-level contamination events.

Chen AW, Fink JM, Letinski DJ. 1996. Analytical methods to determine residual **cypermethrin** and its major acid metabolites in bovine milk and tissues. Journal of Agricultural & Food Chemistry 44(11):3534-3539.
Abstract: Several analytical methods were developed to determine cypermethrin and its acid metabolite residues in bovine milk, cream, kidney, liver, muscle, and fat samples. These methods used solvent extraction or acid reflux, liquid-liquid and/or solid phase extraction, with or without chemical derivatization, and quantitation by gas chromatograph with electron capture or mass selective detector. The LOQ and LOD for milk were set at 10 and 2 ppb, respectively. The average method recoveries for cypermethrin, cis-DCVA, trans-DCVA, and m-PBA in cow milk were 81% (n=39), 96% (n=22), 99% (n=22), and 106% (n=22), respectively. For bovine tissues and cream samples, the LOQ and LOD were 50 and 10 ppb, respectively. The overall average method recoveries for cypermethrin, cis-DCVA, trans-DCVA, and m-PBA in cream and tissue samples were 92% (n=27), 97% (n=25), 103% (n=25), and 98% (n=25), respectively. Satisfactory recoveries were also obtained with higher fortification levels for milk and fat samples.
[Cypermethrin = Pyrethroid insecticide]

Eriksson P. 1997. Developmental neurotoxicity of environmental agents in the neonate. Neurotoxicology 18(3):719-726.
Abstract: The development of an organism includes periods that can be critical for its normal maturation. One such appears to occur during perinatal development of the brain, the so-called 'brain growth spurt'. This period in the development of the mammalian brain is associated with numerous biochemical changes that transform the feto-neonatal brain into that of the mature adult. We have observed that **low-dose exposure** to environmental agents such as DDT; **pyrethroids, organophosphates**, nicotine paraquat and polychlorinated biphenyls (PCBs) during the 'brain growth spurt' can lead to irreversible changes in adult brain function in the mouse. The induction of behavioural and cholinergic disturbances in the adult animal appears to be limited to a short period during neonatal development, around postnatal day 10, and following doses that apparently have no permanent effects when administered to the adult animal. Furthermore, 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 behavioural disturbances and learning disabilities.

Eriksson P, Fredriksson A. 1991. Neurotoxic effects of two different pyrethroids, bioallethrin and deltamethrin, on immature and adult mice: changes in behavioral and muscarinic receptor variables. Toxicol Appl Pharmacol 108(1):78-85.
Abstract: We have recently shown that two **pyrethroids**, bioallethrin and deltamethrin, affect muscarinic cholinergic receptors (MAChR) in the neonatal

mouse brain when given to suckling mice during the period of rapid brain growth. Such early exposure to these pyrethroids can also lead to permanent changes in the MAChR and behavior in the mice as adults. In the present study, male NMRI mice were given bioallethrin (0.7 mg), deltamethrin (0.7 mg), or a 20% fat emulsion vehicle (10 ml) per kilogram of body weight per os once daily between the 10th and 16th postnatal day. The mice were subjected to behavioral tests upon reaching the age of 17 days and at 4 months. Within 1-2 weeks after the behavioral tests the mice were killed by decapitation and crude synaptosomal fractions (P2) were prepared from the cerebral cortex, hippocampus, and striatum. The densities of MAChR were assayed by measuring the amounts of quinuclidinyl benzilate ([3H]QNB) specifically bound in the P2 fraction. The proportions of high-affinity (HA) and low- affinity (LA) binding sites of MAChR were assayed in a displacement study using [3H]QNB/carbachol. The behavioral tests at an adult age of 4 months indicated a significant increase in spontaneous motor behavior in both bioallethrin- and deltamethrin-treated mice. There was also a significant decrease and a tendency toward a decrease in the density of MAChR in the cerebral cortex in mice receiving bioallethrin and deltamethrin, respectively. The proportions of HA- and LA-binding sites of MAChR were not changed. This study further supports that disturbances of the cholinergic system during rapid development in the neonatal mouse can lead to permanent changes in cholinergic and behavioral variables in the animals as adults.

Eriksson P, Talts U. 2000. Neonatal exposure to neurotoxic pesticides increases adult susceptibility: a review of current findings. Neurotoxicology 21(1-2):37-47.
Abstract: An environmental mischance commonly occuring in nature is the combination of neonatal exposure and later adult exposure to various toxic substances. During neonatal life, offspring can be affected by toxic agents either by transfer via mother's milk or by direct exposure. In many mammalian species the perinatal period is characterized by a rapid development of the brain--'the brain growth spurt' (BGS). We have observed that exposure to pesticides, such as DDT and **bioallethrin**, during the BGS in mice can potentiate susceptibility to bioallethrin or paraoxon in adult life. This combined neonatal and adult exposure caused spontaneous behavioural aberrations and changes in muscarinic cholinergic receptors and led to impairment of the faculties of learning and memory. Our studies indicate that neonatal exposure to pesticides--even in low doses--can potentiate and/or modify the reaction to adult exposure to xenobiotics, and thereby accelerate dysfunctional processes.
[Bioallethrin = Pyrethroid insecticide]

Go, V, Garey J, Wolff MS, Pogo BGT. 1999. Estrogenic Potential of Certain **Pyrethroid Compounds** in the MCF-7 Human Breast Carcinoma Cell Line. Environ Health Perspect 107(3):173-177.
Abstract: Estrogens, whether natural or synthetic, clearly influence reproductive development, senescence, and carcinogenesis. Pyrethroid insecticides are now the most widely used agents for indoor pest control, providing potential for human exposure. Using the MCF-7 human breast carcinoma cell line, we studied the estrogenic potential of several synthetic pyrethroid compounds *in vitro* using pS2

mRNA levels as the end point. We tested sumithrin, fenvalerate, *d-trans* allethrin, and permethrin. Nanomolar concentrations of either sumithrin or fenvalerate were sufficient to increase pS2 expression slightly above basal levels. At micromolar concentrations, these two pyrethroid compounds induced pS2 expression to levels comparable to those elicited by 10 nM 17ß-estradiol (fivefold). The estrogenic activity of sumithrin was abolished with co-treatment with an antiestrogen (ICI 164,384), whereas estrogenic activity of fenvalerate was not significantly diminished with antiestrogen co-treatment. In addition, both sumithrin and fenvalerate were able to induce cell proliferation of MCF-7 cells in a dose-response fashion. Neither permethrin nor *d-trans* allethrin affected pS2 expression. Permethrin had a noticeable effect on cell proliferation at 100 µM, whereas *d-trans* allethrin slightly induced MCF-7 cell proliferation at 10 µM, but was toxic at higher concentrations. Overall, our studies imply that each pyrethroid compound is unique in its ability to influence several cellular pathways. These findings suggest that pyrethroids should be considered to be hormone disruptors, and their potential to affect endocrine function in humans and wildlife should be investigated.

Greenlee AR, Ellis TM, Berg RL. 2004. **Low-dose agrochemicals and lawn-care pesticides** induce developmental toxicity in murine preimplantation embryos. Environ Health Perspect 112(6):703-709.

Abstract: Occupational exposures to pesticides may increase parental risk of infertility and adverse pregnancy outcomes such as spontaneous abortion, preterm delivery, and congenital anomalies. Less is known about residential use of pesticides and the risks they pose to reproduction and development. In the present study we evaluate environmentally relevant, low-dose exposures to agrochemicals and lawn-care pesticides for their direct effects on mouse preimplantation embryo development, a period corresponding to the first 5-7 days after human conception. Agents tested were those commonly used in the upper midwestern United States, including six herbicides [atrazine, dicamba, metolachlor, 2,4-dichlorophenoxyacetic acid (2,4-D)], pendimethalin, and mecoprop), three insecticides (chlorpyrifos, terbufos, and permethrin), two fungicides (chlorothalonil and mancozeb), a desiccant (diquat), and a fertilizer (ammonium nitrate). Groups of 20-25 embryos were incubated 96 hr in vitro with either individual chemicals or mixtures of chemicals simulating exposures encountered by handling pesticides, inhaling drift, or ingesting contaminated groundwater. Incubating embryos with individual pesticides increased the percentage of apoptosis (cell death) for 11 of 13 chemicals (p less than or equal to 0.05) and reduced development to blastocyst and mean cell number per embryo for 3 of 13 agents (p less than or equal to 0.05). Mixtures simulating preemergent herbicides, postemergent herbicides, and fungicides increased the percentage of apoptosis in exposed embryos (p less than or equal to 0.05). Mixtures simulating groundwater contaminants, insecticide formulation, and lawn-care herbicides reduced development to blastocyst and mean cell number per embryo (p less than or equal to 0.05). Our data demonstrate that pesticide-induced injury can occur very early in development, with a variety of agents, and at concentrations assumed to be

without adverse health consequences for humans.

Lazarini CA, Florio JC, Lemonica IP, Bernardi MM. 2001. Effects of prenatal exposure to **deltamethrin** on forced swimming behavior, motor activity, and striatal dopamine levels in male and female rats. Neurotoxicol Teratol 23(6):665-673.
Abstract: The effects of prenatal exposure of rat pups to 0.08 mg/kg deltamethrin (DTM) on physical, reflex and behavioral developmental parameters, on forced swimming and open-field behaviors, and on striatal monoamine levels at 60 days of age were observed. Maternal and offspring body weight, physical and reflex development were unaffected by the exposure to the pesticide. At 21 days of age, open-field locomotion frequency and immobility duration of male and female offspring were not different between control and exposed animals. However, male rearing frequency was increased in experimental animals. A decreased immobility latency to float and in general activity after the swimming test in male offspring was observed at adult age; no interference was detected in the float duration during the swimming test. In addition, these animals presented higher striatal 3,4-dihydroxyphenylacetic acid (DOPAC) levels without modification in dopamine (DA) levels and an increased DOPAC/DA ratio. These data indicate a higher activity of the dopaminergic system in these animals. Noradrenaline (NA) levels were increased, while MHPG levels were not detectable in the system studied. Serotonin (5-HT) and 5-hydroxyindolacetic acid (5-HIAA) levels, as well as the homovanillic acid (HVA)/DA ratio, were not modified by the exposure to the pesticide. No changes were observed in swimming and open-field behaviors nor were there any changes in striatal monoamines or their metabolites in the female experimental group. In relation to the pesticide formula, the present data showing that prenatal exposure to DTM alters latency to float and the activity of striatal dopaminergic system might reflect a persistent effect of the pesticide on animal motor activity, mainly in males. On the other hand, the decrease in general activity observed in experimental male rats suggests higher levels of emotionality induced by previous exposure to the swimming behavior test in relation to control animals. Data gathered in the present study may be important for the assessment of the safety of pyrethroid insecticides.
[Deltamethrin = Pyrethroid insecticide]

Leng G, Gries W. 2005. Simultaneous determination of **pyrethroid** and pyrethrin metabolites in human urine by gas chromatography-high resolution mass spectrometry. Journal of Chromatography B-Analytical Technologies in the Biomedical & Life Sciences 814(2):285-294.
Abstract: A new developed gas chromatographic-high resolution mass spectrometric method for the sensitive simultaneous determination of trans-chrysanthemumdicarboxylic acid, cis- and trans-3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid, cis-3-(2,2-dibromovinyl)2,2-dimethylcyclopropane carboxylic acid, 3-phenoxybenzoic acid and 4-fluoro-3-phenoxybenzoic acid in human urine is presented. These metabolites are biomarkers for an exposure to pyrethrum, allethrin, resmethrin, phenothrin, tetramethrin, cyfluthrin, cypermethrin, deltamethrin or permethrin. Therefore, with the help of this method for the first time a complete assessment of exposure

to pyrethroid and pyrethrin insecticides is possible. After acid hydrolysis and extraction with tert-butyl-methyl-ether the residue is derivatized with 1,1,1,3,3,3-hexafluoroisopropanol and analyzed by GC/HRMS in electron impact mode (detection limits <0.1 mug/l) as well as in negative chemical ionization mode (detection limit <0.05 mug/l urine).

Monteiro-Riviere NA, Baynes RE, Riviere JE. 2003 Feb 1. Pyridostigmine bromide modulates topical irritant-induced cytokine release from human epidermal keratinocytes and isolated perfused porcine skin. Toxicology 183(1-3):15-28. Abstract: Gulf War personnel were given pyridostigmine bromide (PB) as a prophylactic treatment against organophosphate nerve agent exposure, and were exposed to the insecticide **permethrin** and the insect repellent N,N-diethyl-m-toluamide (DEET). The purpose of this study was to assess the effects of PB to modulate release of inflammatory biomarkers after topical chemical exposure to chemical mixtures containing permethrin and DEET applied in ethanol or water vehicles. Treatments were topically applied to isolated perfused porcine skin flaps (IPPSFs). Concentrations of interleukin-8 (IL-8), tumor necrosis factor-alpha (TNF-alpha) and prostaglandin E-2 (PGE(2)) were assayed in perfusate to probe for potential inflammatory effects after complex mixture application. IPPSFs (n = 4/treatment) were topically dosed with mixtures of permethrin, DEET, and permethrin/DEET, in ethanol. Each treatment was repeated with perfusate spiked with 50 ng/ ml of PB. Perfusate was also spiked with 30 ng/ml diisopropylfluorophosphate to simulate low level organophosphate nerve agent exposure. Timed IPPSF venous effluent samples (0.5,1,2,4, and 8 h) were assayed by ELISA for IL-8 and TNF-alpha and by EIA for PGE(2). Overall, PB infusion caused a decrease or IL-8 and PGE(2) release. Effects on TNF-alpha were vehicle dependent. To probe the potential mechanism of this PB effect, human epidermal keratinocyte HEK cell cultures were exposed to permethrin DEET permethrin/DEET, with and without PB in DMSO. IL-8 was assayed at 1, 2, 4, 8, 12 and 24 h. PB suppressed IL-8 in permethrin and ethanol treatment from 4 to 24 h confirming the IPPSF results. In conclusion, these studies suggest that systemic exposure to PB suppressed IL-8 release at multiple time points in two skin model systems. This interaction merits further study.

Nassif M, Brooke JP, Hutchinson DBA, Kamel OM, Savage EA. 1980. Studies with **permethrin** against bodylice in Egypt. Pesticide Science 11:679-684. Abstract: Approximately 350 people, the inhabitants of two villages in the Fayum district of Egypt, were individually dusted with 50 g of powder containing 2.5 or 5.0 g permethrin kg(-1). The inhabitants of a third village were left untreated as a control. Before treatment, approximately two-thirds of the population of all three villages were infested with bodylice. Fourteen days after treatment, the permethrin dust at the lower strength reduced the infestation by 98.8% and at the highest strength of residual control for at least 91 days. The other gave a lower level of control at this time. Urine samples, taken from subjects in each of the treated villages before and after dusting, were analyses for permethrin metabolites. Results indicated that the maximum amount of permethrin absorbed, orally, through the skin, or by inhalation, was 39 ug/kg(-1) body weight, 24 h

after treatment. No residue was found 30 days and 60 days after treatment. It was concluded that there was a very substantial safety margin when permethrin dusts were used on man for bodylice control.

Sheets LP, Doherty JD, Law MW, Reiter LW, Crofton KM. 1994. Age-dependent differences in the susceptibility of rats to **deltamethrin**. Toxicol Appl Pharmacol 126:186-190.

Abstract: Separate groups of weanling and adult rats were exposed to both behaviorally active and lethal doses of deltamethrin to examine age-dependent toxicity of a pyrethroid over a wide dose range. The acoustic startle response (ASR) was selected for comparison at low doses since it is a sensitive, quantifiable biological indicator of pyrethroid effects in rats. Acute mortality was included for comparison at the upper limit of the dose-response. Deltamethrin was administered by gavage as a single dose in corn oil for all tests. Effects on the ASR were comparable in 21- and 72-day-old rats, with a 4-mg/kg dose decreasing ASR amplitude by approximately 50% (ED50) at both ages. By comparison, LD50 values in 11-, 21-, and 72- day old rats were 5.1, 11 and 81 mg/kg, respectively. Thus, 11- and 21- day-old male rats were 16 and 7 times, respectively, more sensitive than adults to acute lethality. The concentration of deltamethrin was measured in whole-brain tissue from weanling and adult males treated with ED50 and LD50 doses. The brain concentration of deltamethrin at the ED50 dose of 4 mg/kg was higher in weanling rats than adults. This suggests a possible functional difference, with weanling rats being less susceptible than adults to a low dose. By comparison, there was an equivalent concentration of deltamethrin in brain tissue following an LD50 dose of 12 mg/kg in weanling rats and 80 mg/kg in adults. These results support age-related differences in pharmacokinetics as the basis for the markedly greater sensitivity of young rats to a lethal dose of deltamethrin.

van Haaren F, Haworth SC, Bennett SM, Cody BA, Hoy JB, Karlix JL, Tebbett IR. 2001 May-2001 Jun 30. The effects of pyridostigmine bromide, **permethrin** and deet alone, or in combination, on fixed-ratio and fixed-interval behavior in male and female rats. Pharmacol Biochem Behav 69(1-2):23-33.

Abstract: Concurrent exposure to pyridostigmine bromide (PB), permethrin (PERM) and/or N,N-diethyl-m-toluamide (DEET) may have contributed to the development of a syndrome that appears to have afflicted military personnel who served during the Gulf War. The present experiment sought to evaluate the behavioral effects of these compounds alone, or in various combinations, in male and female rats. Subjects were exposed to a multiple fixed-ratio (FR) 50, fixed-interval (FI) 2-min schedule of reinforcement. PB dose-dependently decreased FR and FI response rates. FR responding was disrupted by lower doses and there were no differences between the sexes. PERM vehicle administration decreased response rates maintained by both schedules of reinforcement; this was offset by an increase in response rate after the administration of the intermediate dose of PERM. The highest dose of PERM decreased both FR and FI response rates. FR rates in male rats were more disrupted than those in female rats. Only the highest dose of DEET decreased FR and FI response rates in male and female rats. FR

rates were more disrupted in female rats than in male rats. Synergistic effects were only observed when FI response rates decreased in male rats upon exposure to half the low dose of PB with half the low dose of PERM or half the low dose of PB with half the low dose of DEET. The results of this experiment thus show that small doses of PB, PERM and DEET disrupt well-established, schedule-controlled behavior in male and female rats in a schedule- and gender-dependent manner; schedule-dependent and gender-dependent synergistic effects were also observed. The mechanism by which the compounds exert these behavioral effects remains to be determined.

Walker AN, Bush P, Puritz J, Wilson T, Chang ES, Miller T, Holloway K, Horst MN. 2005. Bioaccumulation and metabolic effects of the endocrine disruptor **methoprene** in the lobster, *Homarus americanus*. Integrative & Comparative Biology 45(1):118-126.

Abstract: Methoprene is a pesticide that acts as a juvenile hormone agonist. Although developed initially against insects, it has since been shown to have toxic effects on larval and adult crustaceans. Methoprene was one of several pesticides applied to the Western Long Island Sound (WLIS) watershed area during the summer of 1999; the other pesticides were malathion, resmethrin, and sumethrin. These pesticides were applied as part of a county-by-county effort to control the mosquito vector of West Nile Virus. Subsequently, the seasonal lobster catches from the WLIS have decreased dramatically. The lethality of the pesticides to lobsters had been unknown. We studied the effects of methoprene while other investigators studied effects of the other pesticides. We questioned whether methoprene, through its effects on larvae, adults or both, could have contributed to this decline. We found that low levels of methoprene had adverse effects on lobster larvae. It was toxic to stage II larvae at 1 ppb. Stage IV larvae were more resistant, but did exhibit significant increases in molt frequency beginning at exposures of 5 ppb. Juvenile lobsters exhibited variations in tissue susceptibility to methoprene: hepatopancreas appeared to be the most vulnerable, reflected by environmental concentrations of methoprene inhibiting almost all protein synthesis in this organ. Our results indicated that methoprene concentrates in the hepatopancreas, nervous tissue and epidermal cells of the adult lobster. Methoprene altered the synthesis and incorporation of chitoproteins (cuticle proteins) into adult postmolt lobster explant shells. SDS PAGE analyses of adult post-molt shell extracts revealed changes in the synthesis of chitoproteins in the methoprene-treated specimens, suggesting that methoprene affects the normal pathway of lobster cuticle synthesis and the quality of the post-molt shell. Although it is likely that a combination of factors led to the reduced lobster population in WLIS, methoprene may have contributed both by direct toxic effects and by disrupting homeostatic events under endocrine control.

APPENDIX D

Provided below is an overview of the study to follow on page 5, Appendix D.

Toxic Synthetic Pyrethroid Pesticide Levels Found in Stream Sediments

(*Beyond Pesticides*, May 10, 2004) A family of pesticides, synthetic pyrethroids, used increasingly nationwide in place of more heavily restricted organophosphate pesticides has accumulated in many creek sediments to levels that are toxic to freshwater bottom dwellers, according to a new study. The study, "Distribution and Toxicity of Sediment-Associated Pesticides in Agriculture-Dominated Water Bodies of California's Central Valley," Weston, D. P.; You, J. C.; Lydy, M. J.; Environ. Sci. Technol. (April, 2004), is available on line to members/subscribers of the American Chemical Society.

This study is believed to be the first to evaluate the effect of synthetic pyrethroids on sediment-dwelling organisms, such as midge larvae or shrimp-like amphipods, according to University of California, Berkeley, biologist Donald P. Weston, adjunct associate professor of integrative biology and lead author. Ironically, the two organisms studied, are used by the U.S. Environmental Protection Agency (EPA) as indicators of the health of fresh water sediment, according to the author.

Dr. Weston and colleague Michael J. Lydy of Southern Illinois University (SIU) in Carbondale collected sediment samples from 42 rivers, creeks, sloughs and drainage ditches in California's Central Valley and exposed amphipods and midge larvae to the sediments for 10 days. Twenty-eight percent of the sediment samples (20 of 71) killed amphipods at an elevated rate, and in 68 percent of these sediments, the pyrethroids were at levels high enough to account for the deaths. Thus, while other pesticides may well have contributed to the amphipod deaths in some sediment samples, pyrethroids alone explain the toxicity in the vast majority of the sediment samples, Dr. Weston said.

"About one-fifth of our Central Valley sediment samples are toxic to a standard testing species due to a class of pesticides no one has tested for before, for which there are little data on their toxicology when sediment-bound, and which are being promoted as an alternative to the increasingly restricted organophosphate insecticides," Dr. Weston said.

In the tests, the midge larvae died at higher rates when exposed to sediment from 13 percent of 39 collection sites, and 40 percent of these sediment samples contained enough pyrethroids to account for the deaths. Weston notes that these midges (Chironomus tentans) are known to be about three times less sensitive to pyrethroids than are the amphipods (Hyalella azteca), which explains the difference between the species results.

"Since the levels are high enough to be toxic to the standard 'lab rat' species, the next question is: What's happening with the resident species?" Dr. Weston said. "The concern is that invertebrates, particularly crustaceans, could have reduced populations, and these organisms are an important food for a variety of bottom-feeding fish."

Alternatively, the amphipods and midge larvae from areas of intensive agricultural or urban pesticide use may have adapted to live with normally toxic levels of the pesticide. Dr. Weston and his colleagues now are sampling these organisms from the rivers, creeks, sloughs and ditches to determine if they respond the same way as lab-raised organisms. Pyrethroids are a class of compounds represented by permethrin, first marketed in 1973, and various other chemicals usually ending in the suffix -thrin. Permethrin is found in home and garden pesticides ranging from RAID to flea killers and head lice creams, but permethrin and it's kin find broad use in agriculture, such as on cotton, fruit and nut orchards, and on lettuce and rice. California's Central Valley produces more than half the nation's fruits, vegetables and nuts.

Though pyrethroids are used far less than organophosphates like diazinon and chlorpyrifos, their use in California has risen rapidly in recent years because of increased regulation of the spraying of organophosphates, due to health threats to farm workers and increased toxic runoff from fields. According to Weston, pyrethroid use in California increased 58 percent from 2001 to 2002, if account is taken of the increased potency of newer pyrethroids such as cypermethrin. Over a quarter of a million pounds of pyrethroids were spread on California farm fields in 2002, while about 500,000 pounds were used for structural and pest control and landscape maintenance.

Despite this increased use, environmental monitoring still concentrates on organophosphates, he said. Monitoring also tends to focus on concentrations in the water column, under the assumption that sediment-bound chemicals like pyrethroids are unavailable. The current study shows that to be untrue.

"It's amazing that, after 20 years of use, there is not one published study on pyrethroids in sediments in areas of intensive agriculture," Dr. Weston said.

Part of the reason for a lack of data is that analytical methods to detect pyrethroids in sediment have not been broadly available or standardized. Lydy, an environmental toxicologist with SIU-Carbondale's Illinois Fisheries and Aquaculture Center, developed such a method.

"Prior to our study, scientists in area water-monitoring programs were seeing that if they placed aquatic invertebrates in their sediment samples, the animals would die, but they didn't know why - they'd attribute it to organophosphates or organochlorines (two pesticide ingredients being phased out because of environmental concerns), or they'd put it down to 'unknown causes,'" Dr. Lydy said.

He continued: "Where our study is unique is that we looked at the toxicity and tried to figure out what was actually causing it. We detected organochlorines, such as DDT and chlordane, in the sediments, but at concentrations not high enough to cause the toxicity we noted, whereas concentrations of pyrethroids were high enough to account for that toxicity."

The samples, over 70 in all, were obtained from two major rivers - the San Joaquin and the Feather - and 19 creeks or sloughs, 17 irrigation ditches and two tailwater ponds in 10 Central Valley counties, including the ones with the greatest pyrethroid use: Fresno, Madera, Stanislaus and Sutter. Each sample was placed in a jar and left with 10 test organisms for 10 days, and the death rate compared with similar organisms raised with pristine sediment. The levels of pesticides in each sediment sample also were measured, and 75 percent contained pyrethroids.

Weston, who focuses on freshwater and marine pollution and how it gets from sediments into creatures living on the bottom, noted that another chemical sometimes applied with pyrethroids may be making the situation worse.

Piperonyl butoxide, or PBO, is a synergist that shuts down the enzymes that detoxify pyrethroids, making them last longer in an organism and increasing their killing potential.

He and his colleagues are now trying to measure the level of pyrethroid that kills amphipods, which is around 3 parts per billion in sediments, and whether levels of PBO need to be considered in order to estimate the true toxicity of pyrethroid pesticides. UC Berkeley post-doctoral researcher Erin Amweg is conducting the latter study.

"I don't want to give the impression that pyrethroids are destroying the streams, since that has not yet been shown, but if we are serious about maintaining stream health, we have to consider the sediments and not limit our sampling just to the water above," said Dr. Weston. "While pyrethroids may be preferable to the organophosphates that preceded them, our work shows that the environmental effects of pyrethroids can not be ignored and have had too little study for too long. We need to know more about pyrethroids, because if we don't, how can we regulate them?"

Dr. Lydy said, "Best management practices,' such as introducing buffer strips and wetlands, may reduce pesticide loads in aquatic systems, which would reduce the risk to non-target species."

The study by Drs. Weston, Lydy and post-doctoral researcher Jing You in the Department of Zoology at SIU appeared in the April 8 online version of the American Chemical Society's journal Environmental Science & Technology and will be published later in hard copy.

For more information, contact Robert Sanders, rls@pa.urel.berkeley.edu, 510-643-6998, University of California – Berkeley, or find the study under:

Weston, D. P., J.C. You, M.J. Lydy. 2004. Distribution and Toxicity of Sediment-Associated Pesticides in Agriculture-Dominated Water Bodies of California's Central Valley. Environ. Sci. Technol. 38(10): 2752-2759.

Distribution and Toxicity of Sediment-Associated Pesticides in Agriculture-Dominated Water Bodies of California's Central Valley

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The agricultural industry and urban pesticide users are increasingly relying upon pyrethroid insecticides and shifting to more potent members of the class, yet little information is available on residues of these substances in aquatic systems under conditions of actual use. Seventy sediment samples were collected over a 10-county area in the agriculture-dominated Central Valley of California, with most sites located in irrigation canals and small creeks dominated by agricultural effluent. The sediments were analyzed for 26 pesticides including five pyrethroids, 20 organochlorines, and one organophosphate. Ten-day sediment toxicity tests were conducted using the amphipod *Hyalella azteca* and, for some samples, the midge *Chironomus tentans*. Forty-two percent of the locations sampled caused significant mortality to one test species on at least one occasion. Fourteen percent of the sites (two creeks and four irrigation canals) showed extreme toxicity (>80% mortality) on at least one occasion. Pyrethroid pesticides were detected in 75% of the sediment samples, with permethrin detected most frequently, followed by esfenvalerate > bifenthrin >lambda-cyhalothrin. Based on a toxicity unit analysis, measured pyrethroid concentrations were sufficiently high to have contributed to the toxicity in 40% of samples toxic to *C. tentans* and nearly 70% of samples toxic to *H. azteca*. Organochlorine compounds (endrin, endosulfan) may have contributed to the toxicity at a few other sites. This study provides one of the first geographically broad assessments of pyrethroids in areas highly affected by agriculture, and it suggests there is a greater need to examine sediment-associated pesticide residues and their potential for uptake by and toxicity to benthic organisms.

Introduction

The dominance of organophosphates (OPs) among agricultural insecticides over the past several decades has led environmental monitoring programs in California to focus on dissolved phase pesticides and their toxicity (*1*, *2*). The emphasis on OPs has diverted attention from more hydrophobic pesticides associated with soils and sediments. Legacy pesticides such as some organochlorines and some currently used pesticides such as the pyrethroids are strongly hydrophobic, and monitoring suspended or bedded sediments would be more appropriate. First generation pyrethroids (e.g., permethrin) have been available since the 1970s, and many second generation pyrethroids (e.g., bifenthrin, cyfluthrin, lambda-cyhalothrin) became available in the 1980s, yet there are little data on their concentrations in aquatic sediments. There have been several mesocosm studies (e.g., refs *3* and *4*), but published field data from agricultural areas are minimal. Given recent federal restrictions on residential and some agricultural applications of OPs, and a shift to pyrethroids as replacements, data are needed on realistic environmental concentrations of these compounds.

After gradual decline throughout the 1990s, agricultural use of pyrethroids in California increased 25% from 105 171 kg in 1999 to 131 422 kg in 2002 (data from California's Pesticide Use Reporting database; www.cdpr.ca.gov). In addition, the diversity of pyrethroids used is increasing, and the newer compounds have far greater toxicity to aquatic life. About half of agricultural pyrethroid use in California occurs in the Central Valley, a region lying within the watersheds of the Sacramento and San Joaquin Rivers (Figure 1) that produces more than half of the fruits, vegetables, and nuts grown in the United States. Our goal was to determine the concentrations of pyrethroids and other hydrophobic pesticides in sediments of agriculture-dominated water bodies of the Central Valley and to determine whether toxicity to aquatic life was associated with these residues.

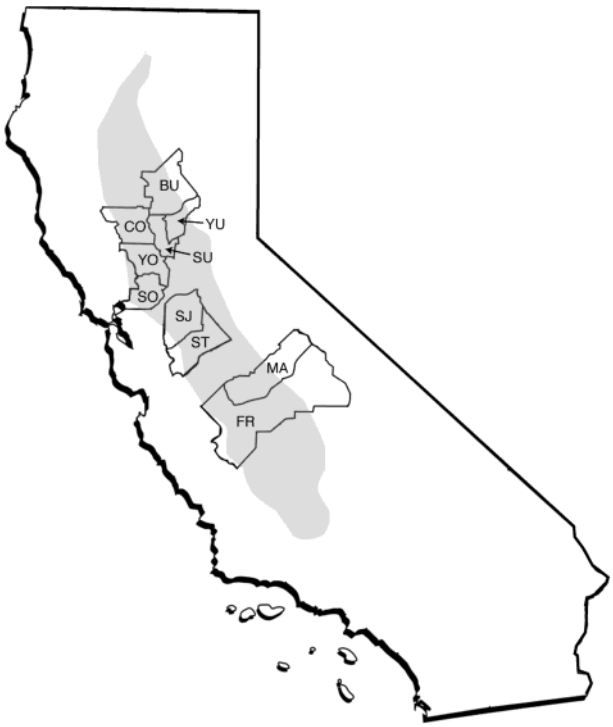

FIGURE 1. Location of California's Central Valley (shaded area) and the counties in which sampling sites were located. The counties shown are as follows: BU = Butte, YU = Yuba, SU = Sutter, CO = Colusa, YO = Yolo, SO = Solano, SJ = San Joaquin, ST = Stanislaus, MA = Madera, and FR = Fresno.

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TABLE 1. Patterns of Pyrethroid Use in Those Counties Selected for Sampling in the PUR-Guided Study[c]

county	annual agricultural pyrethroid use (kg in 2001)	crops on which most pyrethroids used (% of total pyrethroid use in county)	primary pyrethroids used on specified crop (% of total annual pyrethroid use on crop)	months of greatest pyrethroid use on specified crop (% of total annual pyrethroid use on crop)
Fresno	14927	lettuce (32%)[a]	permethrin (87%) cypermethrin (6%)	Mar (31%) Oct (37%)
		cotton (12%)[b]	cyfluthrin (77%) bifenthrin (8%) (s)-cypermethrin (7%) lambda-cyhalothrin (5%)	July (51%) Aug (38%)
		alfalfa (7%)	lambda-cyhalothrin (44%) bifenthrin (38%) permethrin (16%)	Mar (32%) July (33%)
Madera	5224	pistachios (55%)	permethrin (100%)	May (38%) June (28%) July (22%)
Stanislaus	4809	almonds (46%)	permethrin (79%) esfenvalerate (21%)	July (59%)
Sutter	3305	peaches (51%)	permethrin (89%) esfenvalerate (11%)	May (41%) June (43%)

[a] Head and leaf lettuce data combined. Use of pyrethroids on head lettuce comprises 88% of total use on lettuce. [b] Sampling site selection was based on pesticide use data from the year 2000, the most recent data available at the time. In that year, lettuce and alfalfa were the primary crops in Fresno County on which pyrethroids were used, and sample sites in the vicinity of these crops were selected. A 7-fold increase in cyfluthrin usage on cotton between 2000 and 2001 resulted in cotton moving to the second ranked crop in Fresno County in this table, based on 2001 data. [c] Data from the California Department of Pesticide Regulation's pesticide use reporting database, year 2001.

Materials and Methods

Site Selection. We combined data from two studies with different site selection approaches. The first study used the California Department of Pesticide Regulation's Pesticide Use Reporting (PUR) database to identify Central Valley counties with the greatest agricultural use of pyrethroids. Three of the four counties with the greatest pyrethroid use in the San Joaquin River watershed (Fresno, Madera, Stanislaus) and the leading county in the Sacramento River watershed (Sutter) were selected for sampling. For ease of access to some water bodies, a few samples were taken across county lines into neighboring Butte, San Joaquin, and Yuba counties. We also used the PUR database to identify crops in each county on which the majority of pyrethroids were used, months of greatest pyrethroid use, and the compounds employed (Table 1). Sampling sites were located within the regions of each county where these crops were grown. A few additional sites were added in water bodies with anecdotal evidence of sediment toxicity. Sampling sites were located in two major rivers, 11 creeks or sloughs, eight irrigation canals, and two tailwater ponds.

Most stations were sampled twice, termed "peak use" and "winter". The peak use sampling occurred in the month immediately after the peak use of pyrethroids on the target crop(s) within each county. The time of peak use sampling ranged from July 2002 to November 2002, depending on the specific crop. We sampled all sites again in March 2003 following heavy rains ("winter" sampling).

In the second study, samples were obtained from an investigation of irrigation return flows. Farms in the region typically receive irrigation water through a network of canals, and excess irrigation water that flows off the soil surface (tailwater) is returned to the canal system. Sampling stations were located within these canals, termed "agricultural drains", or in creeks to which the canal systems discharged. The principal criteria for site selection was flow dominated by irrigation return water, with only minimal consideration of local pesticide use. Sites were sampled at the beginning (March/April 2003) and toward the end of the irrigation season (August 2003).

In total, the two studies sampled 42 locations, most twice, yielding 70 samples, or 81 including replicates (see Table S1 in Supporting Information).

Sampling Procedures. All sites were sampled from the bank, using a steel trowel to skim the upper 1 cm of the sediment column. In the PUR-guided study, two replicate samples were collected on each sampling occasion, with the second sample processed only if substantial toxicity was seen in the first replicate. In the irrigation return study, a second replicate was collected at only a few sites. All sediments were homogenized by hand mixing, then held at 4 °C (toxicity samples) or −20 °C (chemistry samples).

Analytical Procedures. Sediment samples were analyzed following the methods of You et al. (*5*) for five pyrethroids: *cis*- and *trans*-permethrin (summed in data presented), esfenvalerate, bifenthrin, and lambda-cyhalothrin. Organochlorine pesticides analyzed included alpha-, beta-, delta-, and gamma-BHC, heptachlor, heptachlor epoxide, alpha- and gamma-chlordane, alpha- and beta-endosulfan, endosulfan sulfate, *p,p′*- DDE, *p,p′*- DDD, *p,p′*- DDT, aldrin, dieldrin, endrin, endrin aldehyde, endrin ketone, and methoxychlor. Chlorpyrifos was the only organophosphate insecticide quantified. Briefly, analysis was performed on an Agilent 6890 series gas chromatograph with an Agilent 7683 autosampler and an electron capture detector (Agilent Technologies, Palo Alto, CA). Two columns from Agilent, a HP-5MS, and a DB-608 were used. Qualitative identity was established using a retention window of 1% with confirmation on a second column.

Grain size distribution was determined by wet sieving. Total organic carbon was determined on a CE-440 Elemental Analyzer from Exeter Analytical (Chelmsford, MA), following acid vapor treatment to remove inorganic carbon.

Toxicity Testing. In the PUR-guided study, bulk sediments were tested with 7−10-d old *Hyalella azteca* and 10-d old larvae of *Chironomus tentans*, generally following the protocols of the U.S. Environmental Protection Agency (*6*). The irrigation return study samples were tested with only *H. azteca*. Testing was done in 400 mL beakers containing 50−75 mL of sediment and 250 mL of overlying water, with continuous aeration at 23 °C and a 16 h light:8 h dark cycle. Water was 80% replaced every 48 h using Milli-Q purified water (Millipore Corp., Billerica, MA) made moderately hard by addition of salts (*7*). Temperature, dissolved oxygen, pH, alkalinity, hardness, and ammonia were measured at days 2 and 10 prior to water replacement. Both species were fed by adding a slurry of 10 mg of Tetrafin Goldfish Flakes to

TABLE 2. Physical Properties and Pesticide Residues in the Sediments Sampled[a]

station	sampling time	% silt and clay	% organic carbon	Bif	Esf	Lam	Per	total BHC	total DDT	Diel	total Endr	total Endo	Met
AD2	Apr 2003	33.1	0.53	U	U	1.0	7.2	U	9.2	U	U	U	U
AD2, rep. 1	Aug 2003	67.2	2.35	U	9.7	U	15.1	2.2	20.1	U	U	U	U
AD2, rep. 2	Aug 2003	75.7	2.38	U	12.2	U	18.7	3.1	23.6	U	U	U	1.1
AD5	Aug 2003	68.0	1.65	U	10.9	U	129	1.3	14.3	1.1	U	U	U
AD6	Apr 2003	87.6	1.80	U	5.1	U	20.7	U	15.4	1.2	962	U	2.0
AD6	Aug 2003	91.2	1.49	U	27.5	U	U	1.3	13.5	1.2	U	U	U
AD8	Aug 2003	32.3	1.06	U	30.0	U	U	U	34.9	1.8	1.2	1.3	U
AD10	Mar 2003	14.0	0.47	U	U	U	1.3	U	1.4	U	345	U	U
AD11	Mar 2003	78.7	1.25	U	U	U	1.4	U	17.5	U	9.2	U	1.4
AD13	Aug 2003	56.0	1.81	U	U	U	U	8.5	2.1	U	U	U	1.1
AD16	Aug 2003	81.5	2.20	U	U	U	1.1	3.4	5.9	U	U	U	U
AD18	Apr 2003	69.1	0.85	U	U	U	U	U	13.8	374	U	U	190
AD19	Apr 2003	56.8	1.67	U	U	U	13.8	U	8.8	U	399	U	U
AD19	Aug 2003	66.3	0.86	U	U	U	U	U	16.2	U	U	1.1	9.0
AD21	Apr 2003	52.8	0.44	U	U	U	U	U	3.8	U	1.9	U	U
AD24	Apr 2003	69.6	0.97	U	U	U	U	U	23.6	U	1.0	U	117
AD24	Aug 2003	54.4	1.30	U	U	U	U	U	20.1	1.3	U	2.3	8.1
DC	July 2002	17.2	3.16	1.1	1.4	U	7.3	2.3	3.1	U	2.5	U	1.6
DP	Aug 2002	83.7	1.09	21.0	17.9	2.6	46.9	15.8	78.5	2.6	10.1	17.7	22.7
DP, rep. 1	Mar 2003	58.9	1.40	2.8	1.9	1.0	7.4	U	48.4	1.4	U	U	U
DP, rep. 2	Mar 2003	35.0	0.50	U	1.4	U	3.7	U	33.2	1.3	1.4	U	1.1
FA	Aug 2002	48.4	1.01	U	U	U	1.5	4.3	5.8	U	U	U	U
FL, rep.1	Nov 2002	54.7	0.48	U	U	U	224	1.1	85.6	1.9	9.8	22.3	1.7
FL, rep. 2	Nov 2002	56.5	0.65	2.6	1.3	U	133	1.3	97.4	1.7	10.3	23.2	4.3
FL	Mar 2003	72.6	0.88	U	U	U	14.1	U	76.1	1.2	1.2	12.6	U
FR, rep 2	July 2002	16.0	0.61	U	U	U	4.0	U	U	U	U	U	4.6
FS, rep. 1	Aug 2002	58.1	0.59	3.6	U	2.6	10.1	1.1	408	11.3	9.3	11.6	2.2
FS, rep. 2	Aug 2002	55.8	0.55	2.0	U	2.3	5.8	U	60.0	5.7	6.3	10.7	1.6
GS	Mar 2003	36.9	1.72	U	U	U	5.3	U	8.0	U	U	U	U
IC, rep.1	Mar 2003	77.9	0.80	1.4	2.2	1.6	6.8	U	228	2.7	3.5	1.7	U
IC, rep. 2	Mar 2003	49.8	1.25	U	7.3	1.5	14.1	U	155	5.3	9.2	2.3	U
JS	Mar 2003	55.8	2.05	U	U	U	3.2	U	4.8	4.7	U	2.7	U
LL, rep 1	Nov 2002	70.2	1.00	6.5	7.0	16.8	459	11.4	371	2.9	27.7	81.5	16.4
LL, rep. 2	Nov 2002	75.1	0.76	28.8	11.6	8.3	290	7.1	257	2.3	18.1	62.5	14.7
LL	Mar 2003	56.0	0.32	7.2	U	1.0	70.5	U	384	3.3	24.4	571	1.6
MA	Mar 2003	60.8	1.30	8.8	U	7.8	6.0	U	61.2	1.9	U	11.3	U
MS	July 2002	34.3	1.26	U	1.3	U	5.9	6.9	61.4	U	U	U	U
MS	Mar 2003	41.6	1.84	U	10.7	U	7.8	U	67.4	U	U	U	U
RC	July 2002	45.4	1.05	U	1.1	U	55.4	U	U	U	U	U	2.8
RC	Mar 2003	64.8	1.40	7.7	U	U	120	U	4.8	U	U	U	U
SJ, rep. 1	July 2002	57.4	0.78	1.2	2.7	1	U	U	54.5	U	2.2	2.2	6.3
SJ, rep. 2	July 2002	55.3		U	1.8	U	U	U	35.2	U	1.0	1.2	U
SS, rep. 2	July 2002	21.2	0.48	U	U	U	U	1.4	3.1	U	U	U	1.2
TL, rep. 1	Mar 20 03	57.6	1.36	10.4	U	U	U	U	7.5	U	U	1.0	U

[a] Pesticide concentrations as ng/g, dry weight basis, with <1 ng/g indicated by "U". The samples listed were in the highest 10th percentile for the concentrations of one or more analytes and/or were found to show toxicity to one or both test species. Analytical chemistry data for all samples is available in Table S2 of the Supporting Information. Bif = bifenthrin, Esf = esfenvalerate, Lam = lambda-cyhalothrin, Per = permethrin, Diel = dieldrin, Endr = endrin, Endo = endosulfan, and Met = methoxychlor. Total BHC = sum of alpha-, beta-, delta-, and gamma-BHC. Total DDT = sum of *p,p′*-DDT, *p,p′*-DDE, and *p,p′*-DDD. Total endrin = sum of endrin, endrin aldehyde, and endrin ketone. Total endosulfan = sum of alpha- and beta-endosulfan, and endosulfan sulfate.

each beaker daily. Survival was determined after a 10-d exposure period. Five to eight replicates per sample were tested. Sediment from San Pablo Dam Reservoir, El Sobrante, CA was used as a control. Control survival averaged 91% for *H. azteca* and 82% for *C. tentans*. Due to difficulties with *H. azteca* culturing, there was a significant delay in testing many of the PUR peak use sample set (18% of total samples) with this species. Testing could not be done for 5 months, with the sediment samples maintained in the dark at 4 °C during this time. This delay is noted below where it affects interpretation of results.

Spiked sediment tests were done with *H. azteca* and/or *C. tentans* to determine 10-d LC_{50} values for methoxychlor, endrin, and endosulfan. Control sediment containing 1% organic carbon was spiked with each pesticide and stored at 10 °C for 7 days before testing.

Data were analyzed using ToxCalc Version 5.0 (Tidepool Scientific Software, McKinleyville, CA). Dunnett's Multiple Comparison test was used to identify stations with significantly greater mortality than the control. Arcsin squareroot transformation was used when necessary to meet assumptions of normality and homogeneity of variance. Maximum likelihood regression using probit transformation was used when determining LC_{50} by dilution of test sediments.

Results

Sediment Chemistry. The tailwater ponds (stations FL and LL; Table 2) were the most contaminated of all sites, with sediments containing a wide variety of pesticides. These sediments had the highest observed concentrations of bifenthrin (28.8 ng/g), lambda-cyhalothrin (16.8 ng/g), permethrin (459 ng/g), and total endosulfan (571 ng/g), and the second highest concentrations of total BHC (11.4 ng/g) and total DDT (384 ng/g). The ponds received tailwater from adjacent lettuce fields, and their contents were recycled back onto the fields with no discharge to public waters. Many farms do not have tailwater ponds, and irrigation return flow reaches public waters either directly or indirectly via canals.

Nevertheless, since the lettuce tailwater ponds do not discharge to public waters and since sediment quality in the ponds was not typical of Central Valley surface waters in general, their data are excluded from the remainder of these sediment chemistry results.

At a detection limit of 1 ng/g, pyrethroids were detected in 75% of the samples. Permethrin was the most frequently reported pyrethroid, found in 66% of the samples. The median concentration was 1.5 ng/g, with highs of 129 ng/g in an irrigation canal (AD5); 55.4 and 120 ng/g in Root Creek adjacent to pistachio groves; and 46.9 ng/g in Del Puerto Creek, a small creek passing through orchards and diverse row crops. Bifenthrin was detectable in 18% of the samples, with a maximum of 21.0 ng/g in Del Puerto Creek. Two irrigation canals, sites MA and TL, also contained substantial amounts of bifenthrin (8.8 and 10.4 ng/g, respectively). Esfenvalerate was detectable in 32% of the samples. Highest concentrations were found in Little John Creek (30.0 ng/g), three irrigation canals (AD2, AD5, AD6; 9.7−27.5 ng/g), Del Puerto Creek (17.9 ng/g), and in Morisson Slough (10.7 ng/g) in an area of peach and plum orchards. Lambda-cyhalothrin was detectable in 12% of the samples. Maximum concentration was 7.8 ng/g in irrigation canal sediments from an alfalfa-growing area.

Total DDT was quantifiable in almost all samples. Median concentration was 6.9 ng/g and reached a maximum of 408 ng/g in an irrigation canal. DDE was the principal degradation product found, typically comprising about two-thirds of the total DDT. Dieldrin was rarely found at concentrations more than a few ng/g but reached 374 ng/g in one creek used for irrigation return. Endrin also had several atypically high concentrations (345−962 ng/g) in water bodies dominated by irrigation return flow. Total BHC reached 15.8 ng/g. Concentrations of the most toxic gamma isomer of BHC never exceeded 2 ng/g.

Endosulfan and methoxychlor are currently used organochlorines. Peak endosulfan concentrations were largely limited to the ponds adjacent to lettuce fields, but 17.7 ng/g was found in Del Puerto Creek. The most toxic form, alpha-endosulfan, typically comprised about 10% of the total endosulfan but reached 50% in some tailwater pond samples. Methoxychlor concentrations were usually low but reached 117 and 190 ng/g in two water bodies with high inputs of irrigation return flow.

Data are not presented for aldrin, alpha- and gamma-chlordane, chlorpyrifos, heptachlor, and heptachlor epoxide as they were rarely detected and were at low concentrations when measurable (<7 ng/g).

Toxicity Testing. Sediments of the tailwater ponds not only had the highest concentrations of many pesticides but also proved to be highly toxic. They were the only samples that caused statistically significant mortality in both *C. tentans* and *H. azteca*, with total or near total mortality in both species. A dilution series using sediments from LL (replicate 2, Nov 2002) and varying amounts of control sediments indicated a 10-d LC_{50} to *C. tentans* of 13% LL sediment (95% confidence interval = 10−16%). Dilution series with sediments from FL (replicate 2, Nov. 2002) indicated a *C. tentans* 10-d LC_{50} of 92% (c.i. = 89−94%) and a *H. azteca* 10-d LC_{50} of 69% (c.i. = 60−80%).

Excluding the tailwater ponds, toxicity to one of the test species was seen in 32% of the 77 samples tested (see Table S3 in Supporting Information). Five of the 39 samples tested with *C. tentans* showed toxicity, and 20 of 71 samples were toxic to *H. azteca*. No stations other than tailwater ponds were toxic to both species. Sites with particularly high or persistent mortality to *H. azteca* included Del Puerto and Ingram Creeks and 4 irrigation canals (AD2, AD6, MA, TL). A dilution series with the August AD6 sample provided a 10-d LC_{50} to *H. azteca* of 36% (c.i. = 25−49%), and the March MA sample indicated a 10-d LC_{50} of 26% (c.i. = 18−34%).

Investigating Causes of Sediment Toxicity. A toxicity unit (TU) approach was used to identify pesticides potentially responsible for observed toxicity. TU was calculated as the actual concentration divided by the LC_{50}, both on an organic carbon (oc) normalized basis. Sediment LC_{50} values (Tables 3 and 4) for both species were estimated as follows:

Pyrethroids. Cypermethrin 10-d LC_{50} values average 1.3 μg/g oc (range = 0.48−2.20) and 0.38 μg/g oc (range = 0.18−0.60) for *C. tentans* and *H. azteca*, respectively (*8*). Cypermethrin is not one of the major pyrethroids used in our study area and thus not among our analytes, but it is possible to use these data to estimate sediment LC_{50}s for other pyrethroids. Solomon et al. (*9*) plotted all water toxicity data for a wide variety of pyrethroids and noted that the 10th percentile of the toxicity distributions is a convenient criterion for characterizing relative toxicity. The 10th percentile LC_{50}s for cypermethin = 10 ng/L, lambda-cyhalothrin = 10 ng/L, bifenthrin = 15 ng/L, esfenvalerate/fenvalerate = 37 ng/L, and permethrin = 180 ng/L. Given the sediment toxicity of cypermethrin and the relative toxicity of other pyrethroids, sediment LC_{50} values for the other pyrethroids were estimated. This approach assumes that the other pyrethroids are comparable to cypermethrin in the bioavailability of particle-adsorbed residues. This assumption is reasonable, since the toxicity of pyrethroids to benthic organisms is predictable by the equilibrium partitioning-derived pore water concentration (*8*), and the pyrethroids in this study have K_{oc}'s comparable to cypermethrin (*10*).

Two published LC_{50}s are available as an independent check on the estimated LC_{50} values. The permethrin 10-d sediment LC_{50} for *C. riparius* is 21.9 μg/g oc (*11*), a value very close to our estimated permethrin 10-d LC_{50} for *C. tentans* (23 μg/g oc). The lambda-cyhalothrin 28-d EC_{50} for emergence of *C. riparius* is 6.8 μg/g oc ((*12*) given an oc content of the test sediment of 3.7% provided by J. Warinton (personal communication)), a value five times greater than our estimate of 1.3 μg/g oc.

DDE, DDD, DDT. 10-d LC_{50}s of DDT to *H. azteca* range from 100 to 470 μg/g oc and average 260 μg/g oc (*13*, *14*). DDD and DDE are 5.2 and 32 times less toxic to *H. azteca*, respectively, in water exposures (averaging results of refs *15* and *16*), suggesting the sediment LC_{50}s for these organochlorine compounds are approximately 1300 and 8300 μg/g oc, respectively.

No sediment toxicity data are available for *C. tentans*, but in water exposures the species is 12 times less sensitive to DDT than *H. azteca* and 4.3 and 1.3 times more sensitive to DDD and DDE, respectively (*16*). These factors, when applied to *H. azteca* sediment LC_{50} values, yield the *C. tentans* sediment LC_{50} estimates of Table 3.

Dieldrin. Ten-day sediment LC_{50} values for *C. tentans* have been measured at 35 and 78 μg/g oc, averaging 57 μg/g oc. Values have ranged from 1100 to 3700 μg/g oc for *H. azteca* and average 2000 μg/g oc (*17*).

Endrin. Sediment 10-d LC_{50} for *C. tentans* was measured as part of this study and found to be 4.22 μg/g oc (c.i. = 0.70−8.11). Ten-day sediment LC_{50}s to *H. azteca* range from 54 to 257 μg/g oc and average 140 μg/g oc (*13*, *14*). Information on the relative aquatic toxicities of endrin and its aldehyde and ketone degradation products was lacking, but all three compounds were summed when determining the TUs of endrin present. While the validity of this assumption is unclear, it is of little consequence since at those stations with the highest total endrin concentrations, endrin itself comprised >85% of the total.

Methoxychlor. Methoxychlor 10-d LC_{50} values were measured for this study and found to be 36.7 (c.i. = 27.2−46.8)

TABLE 3. *C. tentans* Toxicity Units (TU) of the Pesticide Analytes at All Stations Exhibiting Significant Toxicity to *C. tentans*[a]

		toxicity units of individual pesticides										
sample	Mort	Bif	Esf	Lam	Per	DDT	Diel	Endr	Met	Endo	BHC	ΣTUs
LL, Nov 2002, rep. 1	100 ± 0	0.3	0.2	1.3	2.0	*b*	*b*	0.7	*b*	4.7	*b*	9.2
LL, Nov 2002, rep. 2	100 ± 0	1.9	0.3	0.8	1.7	*b*	*b*	0.6	0.05	3.3	*b*	8.7
LL, Mar 2003	100 ± 0	1.1	*b*	0.2	1.0	*b*	*b*	1.8	*b*	74.6	*b*	78.7
FL, Nov 2002, rep. 1	98 ± 4	*b*	*b*	*b*	2.0	*b*	*b*	0.5	*b*	1.3	*b*	3.8
GS, Mar 2003	62 ± 8	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*
FL, Nov 2002, rep. 2	60 ± 17	0.2	*b*	*b*	0.9	*b*	*b*	0.4	*b*	1.0	*b*	2.5
FR, July 2002, rep. 2	58 ± 36	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*
FS, Aug 2002, rep. 2	54 ± 11	0.2	*b*	0.3	0.05	*b*	*b*	0.3	*b*	0.5	*b*	1.4
DC, July 2002	50 ± 28	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*
FS, Aug 2002, rep. 1	44 ± 24	0.3	*b*	0.3	0.07	*b*	*b*	0.4	*b*	0.6	*b*	1.7
# nontoxic samples with ≥0.5 TU (n=31)		1	0	1	0	0	0	1	0	0	0	
LC_{50} used to derive TUs (μg/g o.c.)		2.0	4.8	1.3	23	DDT = 3100 DDD = 300 DDE = 6400	57	4.2	36.7	$\alpha = 0.96$ $\beta = 3.2$ sulf = 5.2	0.73	

[a] Mort = % mortality; Bif = bifenthrin; Esf = esfenvalerate; Lam = lambda-cyhalothrin; Per = permethrin; DDT = sum TU of DDT, DDD, DDE; Diel = dieldrin; Endr = endrin; Met = methoxychlor; Endo = sum TU of alpha- and beta-endosulfan and endosulfan sulfate; BHC = gamma-BHC. [b] <0.05 TU.

and 85.8 μg/g oc (c.i. = 72.1−102.6) for *C. tentans* and *H. azteca*, respectively.

Endosulfan. *C. tentans* 10-d LC_{50} values were measured for this study and found to be 0.96 (c.i. = 0.41−1.46), 3.24 (c.i. = 1.46−4.27), and 5.22 μg/g oc (c.i. = 3.23−5.82) for alpha- and beta-endosulfan and endosulfan sulfate, respectively. *H. azteca* 10-d LC_{50} values were measured as 51.7 (c.i. = 38.6−61.6), >1000, and 873 μg/g oc (c.i. = 660−1139) for the same compounds.

BHC. The 24-h sediment EC_{50} of gamma-BHC to *C. riparius* is 0.73 μg/g oc (*18*). This estimate is shown in Table 3 as the best available data, although the actual 10-d LC_{50} for *C. tentans* is likely to be less considering our 10-d exposure and the fact that *C. tentans* is more sensitive to gamma-BHC than is *C. riparius* (*19*). No sediment LC_{50} data were available for *H. azteca*, but in 10-d water exposures, the LC_{50} of the species is 75% of that of *C. riparius* (*20*), and that conversion factor was used to derive an estimated sediment LC_{50} for *H. azteca* of 0.55 μg/g oc. In calculating TUs present at the sampling sites, only the sediment concentration of the gamma-isomer was used since other isomers of BHC have much lower aquatic toxicities (*21*).

In most of the 10 samples toxic to *C. tentans*, the TU approach suggests that several of the measured analytes were present in concentrations that could account for the observed mortality (Table 3). In the tailwater pond samples (FL and LL) where near total mortality was observed, bifenthrin, lambda-cyhalothrin, permethrin, endrin, and endosulfan were all in sufficient concentrations in most of the samples so that any one of these pesticides alone could account for the toxicity. One sample (LL, March 2003) contained 78 TUs of endosulfan.

To account for cumulative effects of multiple pesticides, the TUs of individual pesticides were summed to determine a total TU in each sample. This approach implicitly presumes additivity of toxicity as is common among pesticides (*22*), though the data do not exist to demonstrate whether specific combinations of our analytes are greater or less than additive. The default presumption of additivity is made more defensible by the fact that since the organochlorines only had appreciable TUs at a few sites, the sum TU is largely a summation of TUs of the individual pyrethroids for which a common mode of toxic action is more likely.

Outside of the tailwater ponds, the combined effects of bifenthrin, lambda-cyhalothrin, and endosulfan may have contributed to the mortality in both replicates of station FS, since they together contribute nearly 1 TU. The combined concentrations of endrin and endosulfan account for about another TU at this site. DDT, dieldrin, and BHC most likely did not contribute to the observed toxicity to *C. tentans* in any sample. In 3 of the 10 toxic samples (GS, FR, DC) the measured analytes could not account for the toxicity.

TU calculations for samples not toxic to *C. tentans* are not shown in Table 3 to conserve space, but for each analyte the number of nontoxic samples that contained at least 0.5 TU is shown. The 0.5 TU threshold is arbitrary but suggests a strong likelihood that the analyte makes a substantial contribution to the observed mortality. Bifenthrin, lambda-cyhalothrin, and endrin were the only pesticides for which mortality was expected but not seen, with only one nontoxic sample for each compound having ≥0.5 TU.

A similar TU analysis for the *H. azteca* toxicity data (Table 4) indicates bifenthrin, lambda-cyhalothrin, and permethrin concentrations were sufficiently high (≥0.5 TU) that each compound individually could have had a substantial contribution to the mortality in six of the 23 toxic samples. Esfenvalerate concentrations were ≥0.5 TU in five samples. Cumulatively, pyrethroids were likely responsible for much of the toxicity in 17 of the 23 toxic samples. The most extreme cases were the tailwater ponds where the combined effect of all four pyrethroids created up to 12.9 TUs, and 98% mortality to *H. azteca* was observed.

As was the case for *C. tentans*, the TU calculations for *H. azteca* indicated that most of the legacy organochlorine compounds were present at concentrations far too low to account for the observed toxicity. The only exception to this generality was endrin, which was found at 0.4 TU in one irrigation canal toxic to *H. azteca* and at 0.5 TU in another nontoxic canal sample. Among the current use organochlorines, methoxychlor approached toxic thresholds in one creek (Stone Corral Creek, AD18), and endosulfan may have contributed to mortality in a tailwater pond. None of the measured analytes could explain toxicity at AD11 and AD21.

Among samples without significant *H. azteca* toxicity there were only rare instances of samples containing ≥0.5 TU of any pesticide (one sample for lambda-cyhalothrin and endrin, two for permethrin). The only exception was bifenthrin for which four samples contained ≥0.5 TU of the compound but were nontoxic. Nevertheless samples containing ≥0.5 TU bifenthrin were more than three times as likely to be toxic than nontoxic, suggesting our bifenthrin LC_{50} estimate, while perhaps slightly low, is not grossly in error. Overall, the

TABLE 4. *H. azteca* Toxicity Units (TU) of the Pesticide Analytes at All Stations Exhibiting Significant Toxicity to *H. azteca*[a]

		toxicity units of individual pesticides										
sample	Mort	Bif	Esf	Lam	Per	DDT	Diel	Endr	Met	Endo	BHC	ΣTUs
MA, Mar 2003	100 ± 0	1.2	*b*	1.6	0.1	*b*	*b*	*b*	*b*	*b*	*b*	2.9
LL, Nov 2002, rep. 1	98 ± 4	1.1	0.5	4.4	6.8	*b*	*b*	*b*	*b*	0.06	*b*	12.9
FL, Nov 2002, rep. 1	97 ± 5	*b*	*b*	*b*	6.9	*b*	*b*	*b*	*b*	*b*	*b*	6.9
AD2, Apr 2003	97 ± 7	*b*	*b*	0.5	0.2	*b*	*b*	*b*	*b*	*b*	*b*	0.7
DP, Mar 2003, rep. 1	90 ± 14	0.4	0.1	0.2	0.1	*b*	*b*	*b*	*b*	*b*	*b*	0.8
IC, Mar 2003, rep. 2	90 ± 14	*b*	0.4	0.3	0.2	*b*	*b*	*b*	*b*	*b*	*b*	0.9
IC, Mar 2003, rep. 1	85 ± 13	0.3	0.2	0.5	0.1	*b*	*b*	*b*	*b*	*b*	*b*	1.1
AD6, Aug 2003	85 ± 19	*b*	1.3	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	1.3
AD2, Aug 2003, rep. 2	84 ± 9	*b*	0.4	*b*	0.1	*b*	*b*	*b*	*b*	*b*	*b*	0.5
FL, Nov 2002, rep. 2	83 ± 6	0.7	0.1	*b*	3.0	*b*	*b*	*b*	*b*	*b*	*b*	3.8
TL, Mar 2003, rep. 1	82 ± 18	1.3	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	1.3
AD2, Aug 2003, rep. 1	81 ± 18	*b*	0.3	*b*	0.09	*b*	*b*	*b*	*b*	*b*	*b*	0.4
DP, Aug 2002	78 ± 16	3.4	1.2	0.6	0.6	*b*	*b*	*b*	*b*	*b*	0.2	6.0
LL, Mar 2003	76 ± 29	4.0	*b*	0.8	3.2	0.2	*b*	0.05	*b*	0.9	*b*	9.2
MS, Mar 2003	68 ± 33	*b*	0.4	*b*	0.1	*b*	*b*	*b*	*b*	*b*	*b*	0.5
AD8, Aug 2003	67 ± 18	*b*	2.0	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	2.0
DP, Mar 2003, rep. 2	58 ± 16	*b*	0.2	*b*	0.1	*b*	*b*	*b*	*b*	*b*	*b*	0.3
AD5, Aug 2003	47 ± 27	*b*	0.5	*b*	1.2	*b*	*b*	*b*	*b*	*b*	*b*	1.7
AD6, Apr 2003	39 ± 25	*b*	0.2	*b*	0.2	*b*	*b*	0.4	*b*	*b*	*b*	0.8
AD18, Apr 2003	36 ± 28	*b*	*b*	*b*	*b*	*b*	*b*	*b*	0.3	*b*	*b*	0.3
AD11, Mar 2003	34 ± 27	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*
SJ, July 2002, rep. 2	34 ± 15	*b*	0.1	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	0.1
AD21, Apr 2003	31 ± 17	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*	*b*
# nontoxic samples with ≥0.5 TU (n=51)		4	0	1	2	0	0	1	0	0	0	
LC_{50} used to derive TUs (μg/g o.c.)		0.57	1.4	0.38	6.8	DDT = 260 DDD = 1300 DDE = 8300	2000	140	85.8	α = 52 β = >1000 sulf = 870	0.55	

[a] Mort = % mortality; Bif = bifenthrin; Esf = esfenvalerate; Lam = lambda-cyhalothrin; Per =permethrin; DDT = sum TU of DDT, DDD, DDE; Diel = dieldrin; Endr = endrin; Met = methoxychlor; Endo = sum TU of alpha- and beta-endosulfan and endosulfan sulfate; BHC = gamma-BHC.
[b] <0.05 TU.

rarity of high TU values among nontoxic samples for all analytes suggests our LC_{50} estimates are reasonable.

Discussion

There have been few measurements of pyrethroids in sediments of agriculture-influenced water bodies, and fewer still that have incorporated toxicity testing of these sediments. We found that pyrethroid residues can be widespread in sediments from regions of intensive agriculture, and in some locations are present in concentrations likely to cause toxicity to sensitive species. The tailwater ponds represented the most extreme instance, containing at least four pyrethroids that were present at concentrations that, even if considered individually, were capable of causing substantial mortality.

Sediments collected from creeks, rivers, and the irrigation canals that discharge to them did not show the extreme pesticide concentrations found in the tailwater ponds but nevertheless frequently showed toxicity to the test species. Statistically significant mortality to *C. tentans* or *H. azteca* was observed in 32% of the 77 sediment samples tested, and 42% of the locations sampled were toxic to at least one species on at least one occasion. Toxicity was seen on occasion in both major rivers sampled, eight of the 19 creeks and sloughs sampled, and seven of the 17 irrigation canals. Six sites (14% of those tested) showed >80% mortality in a test species on at least one occasion.

It appears the analytes we measured were at sufficient concentrations to explain the vast majority of observed mortality. Pyrethroids were likely to have contributed to the toxicity in 40% of samples toxic to *C. tentans* and nearly 70% of samples toxic to *H. azteca* (excluding tailwater ponds). Endrin, endosulfan, and methoxychlor may have been important in a few instances, but for the remaining toxic samples, it was not possible to determine if pesticides or other substances were responsible for the toxicity. There are over 130 pesticides used in the Central Valley, and since the concentrations of most are not measured in any monitoring program, their contribution to toxicity is unknown.

Our toxicity data are supported by an independent study that overlapped with two sampling locations. California's Central Valley Regional Water Quality Control Board sampled the Del Puerto Creek site three times from June to October of 2002 and found 39−100% mortality to *H. azteca* (J. Rowan, personal communication), compared to our observation of 78% mortality in August 2002. The same agency sampled the Orestimba Creek site in September 2002 and found 59% mortality to *H. azteca*, compared to our determination of 60% mortality in March, 2003.

In considering the frequency of toxicity and the sediment concentrations of pesticides, it should be recognized that sampling for the PUR-guided study was focused on areas of high pyrethroid use or water bodies where water quality degradation was likely. However, the irrigation return study, which made up half the total samples, targeted water bodies dominated by irrigation return flow with only minimal consideration of pesticide use or crops grown. There was a greater frequency of toxicity in the PUR-guided study (34% vs 27% in irrigation return study) and in the frequency of pyrethroid detection (85% vs 65%), but the results are still quite striking even for the return flow study with minimal site selection bias.

While our work focused on smaller tributaries, there is some indication of sediment quality impacts in the larger rivers. One sample (of three) in the Feather River proved toxic to *C. tentans* with the responsible agent unknown. Three locations were sampled on the San Joaquin River: one in July 2002 and all three in March 2003, with the July sample showing *H. azteca* mortality due to unknown causes. Further

sampling in the major rivers would be desirable to better characterize regional impacts.

An important conclusion from these data is that legacy organochlorines, while widely distributed in Central Valley sediments, were far below acutely toxic concentrations to sensitive aquatic invertebrates. The only exception to this generalization was endrin, which was found at concentrations of approximately half its LC_{50} in a few irrigation canals. Current-use organochlorine compounds (endosulfan, methoxychlor) were below acutely toxic thresholds in the majority of samples, though they may have contributed to toxicity in the tailwater ponds or a few irrigation canals where concentrations exceeded several hundred ng/g.

The extreme toxicity of sediment-associated pyrethroids indicates the need to improve the detection limits achieved in this study. The sediments tested had organic carbon contents typically about 1%, and in such sediments the *H. azteca* 10-d LC_{50} of cypermethrin is 3.6 ng/g (*8*). Based on relative toxicity among the pyrethroids, in the same sediment the LC_{50}s for bifenthrin, cyfluthrin, and deltamethrin would be on the order of 3−6 ng/g, the LC_{50}s for esfenvalerate or fenvalerate would be about 10−15 ng/g, and the LC_{50}s for permethrin and fenpropathrin would be about 60−90 ng/g. Excluding permethrin and fenpropathrin, these estimates of LC_{50} are only slightly above the one ng/g detection limit. Thus, mere detection of any of the more toxic pyrethroids at least raises the possibility of acute toxicity, even without considering that other species may be more sensitive than *H. azteca* or that chronic toxicity may occur at concentrations less than the 10-d LC_{50} used in these estimates.

The data suggest that pyrethroid concentrations in aquatic habitats of the Central Valley tend to be greater shortly after their use rather than after heavy winter rains. Though there is some dormant spraying of pyrethroids on orchard crops during winter months, most Central Valley crops treated with pyrethroids receive the greatest amounts in the summer. During this period, the mechanisms for transport of residues to aquatic systems would be irrigation return and spray drift from aerial application. Potentially pesticide-bearing soils are washed into aquatic systems by heavy rains, largely confined to December through March. However, pyrethroids typically have half-lives on the order of 1−2 months in aerobic soils (*10*), providing opportunity for substantial degradation between summer application and winter rains. In this study, 65% of the sites with measurable pyrethroids had the highest concentrations in the late summer and fall near the end of the irrigation season. At only 35% of the sites were concentrations greatest in March and April at the conclusion of the rainy season.

The prevalence of sediment toxicity in this study, and evidence that pyrethroids were likely to be responsible for much of it, clearly shows the need for greater awareness of the risks of particle-associated pyrethroids. There are considerable data on the toxicity of dissolved-phase pyrethroids to aquatic life that have been used in developing risk assessments for the compounds (*9*, *12*, *23*, *24*), but these risk assessments have generally focused more on the water column than on sediments. The bioavailability and toxicity of sediment-bound residues have received little attention, as indicated by the difficulty in locating direct sediment LC_{50} measurements for the compounds of interest in this study. The log K_{oc} for most pyrethroids ranges from 5 to 6 (*10*), and they rapidly partition on to soils or sediments (*8*). Except in close proximity to and shortly after application, pyrethroids will largely be sediment associated (*26*). It has been argued that the hydrophobicity of these compounds lessens their bioavailability (*12*, *25*), which may be the case for organisms living within the water column (e.g., daphnids widely used for toxicity testing). However, results from our study indicate a substantial risk remains to benthic organisms under realistic conditions of agricultural use. Our study did not differentiate whether the primary route of toxicity was exposure to dissolved phase pyrethroids within the pore water or ingestion and digestive desorption of particle-associated residues. Digestive routes of contaminant uptake often take on increasing importance for strongly hydrophobic compounds (*26*), and deposit-feeder digestive fluids are usually far more effective extractants of hydrophobic organics than is water (*27*). Regardless of the route of uptake, our findings of widespread sediment toxicity indicate pyrethroid uptake by and toxicity to benthic organisms, and particularly deposit-feeding species, deserves closer study.

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Supporting Information Available

Sampling location details (Table S1), analytical chemistry results for all samples (Table S2), and toxicity testing results for all samples (Table S3). This material is available free of charge via the Internet at http://pubs.acs.org.

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