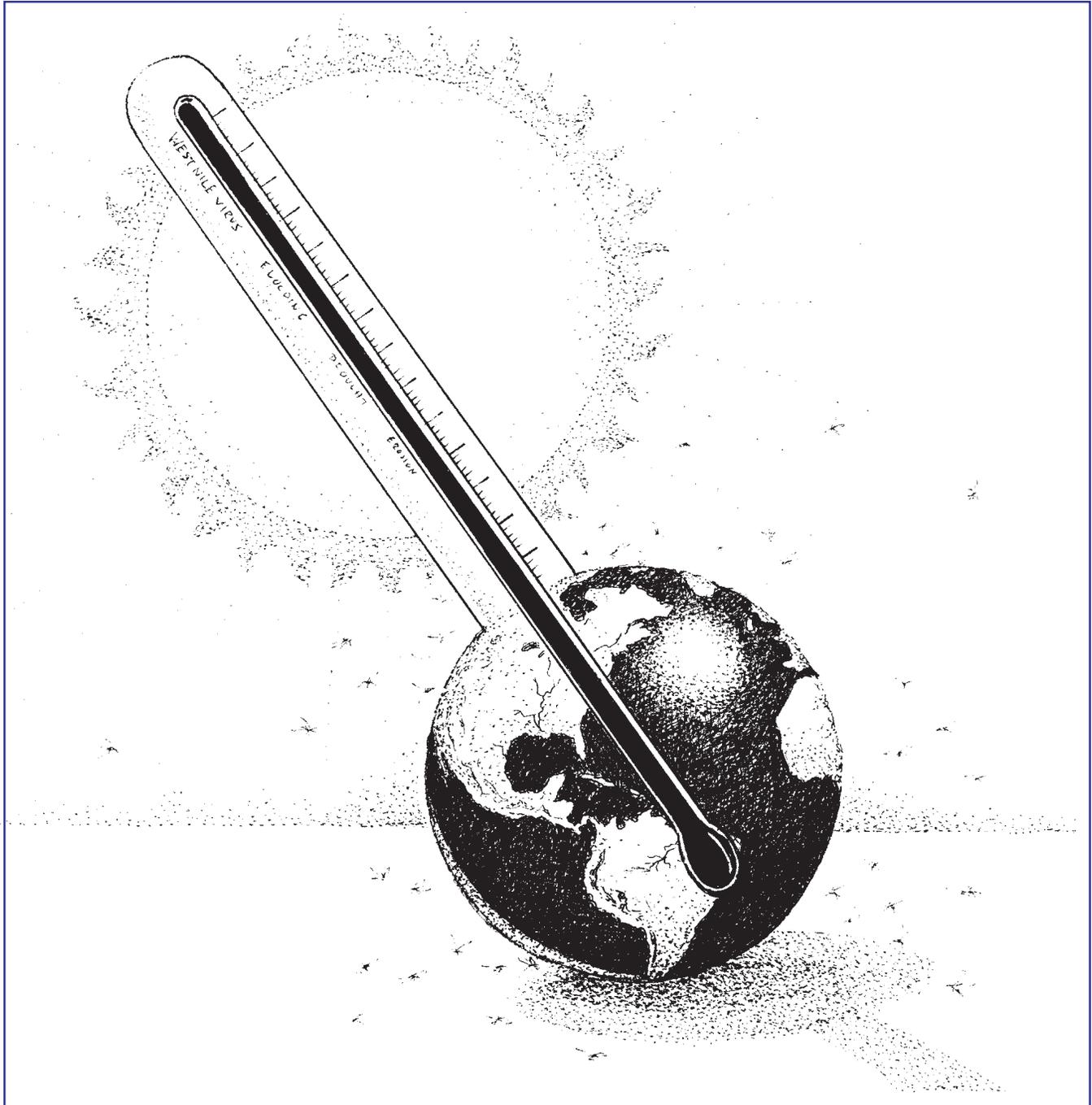


Pesticides and You

News from Beyond Pesticides / National Coalition Against the Misuse of Pesticides (NCAMP)



Global climate change and pesticides

**Pesticides Linked to Parkinson's Disease • Minimizing Mouse Madness •
ChemicalWATCH Factsheet: Rodenticides • Top Ten Myths Behind Pesticide
Dependent Pest Management in Schools • Is Global Warming Harmful to Health**

Letter from Washington

Working for Systemic Change

This issue features an article written by Paul Epstein, M.D., director of the Center for Health and the Global Environment at Harvard Medical School, about global climate change, or global warming, and the impacts that it will certainly have on increasing insect pressures worldwide. Global warming, as described by Dr. Epstein, will increase the pressure on public health officials to use more pesticides.

We can debate whether public health officials continue to overreact to the threat of West Nile Virus (WNV) in New York City and in communities from Massachusetts to Maryland. While it can be argued convincingly that WNV has limited impact on the population, compared to other public health threats, the discovery of the disease-infected mosquitoes has graphically shown us what we can expect in terms of the public health response to insect-borne disease vectors. We can expect a lot more pesticide use in our communities and a higher degree of public exposure to pesticides, given massive pesticide spray applications from aircraft and trucks. We can expect a lot more use of insect repellents, DEET and other neurotoxic materials. We can expect discussions suggesting that the benefits of pesticide use outweigh the risks of the insect-borne disease. And, just as we are beginning to see a shift away from continued use of organophosphates and other neurotoxic pesticides, we may see pressure to shift back. In fact, we are already seeing the pressure in motion.

When EPA announced its decision to phase out the use of chlorpyrifos (Dursban) in June, 2000, it retained public health uses for mosquito control. This may well have been EPA's Office Pesticide Programs first global warming decision, recognizing that a highly toxic neurotoxic chemical, otherwise too toxic to allow in widespread use, that hangs around in the environment for a long time because it contains a chlorine ring, is going to be necessary in the battle of the bugs in our new warmer world. While this is all good news for the pesticide industry, it is bad news for children, elderly, and anyone who breathes air, drinks water and eats food.

Dr. Epstein cites the fact that mosquitoes proliferate faster and bite more as the air becomes warmer. He continues, "At the same time, greater heat speeds the rate at which pathogens inside [mosquitoes] reproduce and mature. At 68 degrees F, the immature *P. falciparum* parasite takes 26 days to develop fully, but at 77 degrees F, it takes only 13 days. The *Anopheles* mosquitoes that spread this malaria parasite live only several weeks: warmer temperatures raise the odds that the parasites will mature in time for the mosquitoes to transfer the infection."

To bring the issue closer to home, Dr. Epstein cites weather and the West Nile Virus. He traces a mild winter where more mosquitoes than usual may have survived the winter in sewers, damp basements and other sources of still water. That leads to a dry spring and summer where birds congregated at dwindling water sources, and predators of mosquitoes were killed off. The summer heat caused the virus to proliferate rapidly among mosquitoes and the follow-

ing drenching rains created new breeding sites and more mosquitoes where the cycle of infected mosquitoes and birds and then people resulted.

Yes, there will be improved efforts at insect prevention and management as we learn to control insect breeding sites more effectively. However, as the fluctuations in weather conditions worsen, it becomes increasingly difficult to prevent and manage the problem.

For those most familiar with the serious problems associated with pesticide use and the real public health threat that it represents, global climate change must be of critical concern. If Dr. Epstein is right, "The conditions underlying outbreaks of [WNV] can be traced to global environmental change," we must fight for the systemic changes necessary to prevent the inevitable increase in pesticide use. We fought for the transition to organic agriculture and the adoption of national organic policy as the response to poisoning and contamination associated with pesticide-dependent conventional agriculture. Now in similar fashion, we must fight for changing our dependency on fossil fuels and reducing the emission of other heat trapping gases into the Earth's atmosphere that give rise to pesticide-dependent public health solutions. The time is now to address all the conditions that give rise to global climate change.

Diazinon: Another Phaseout

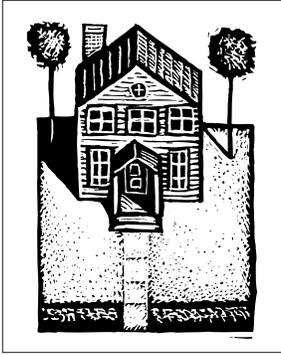
In December, 2000, EPA announced a four-year phase out of residential uses of the home and garden insecticide most widely used by homeowners. The voluntary move by the manufacturer of diazinon, while welcomed by environmentalists and public health advocates, raised concerns about continued sales to unsuspecting consumers. This is now the pattern of EPA action – identify the hazards of a pesticide and allow its manufacturer and retailers to sell off the product over an extended time period. In July, 2000 comments to EPA on its preliminary risks assessment for diazinon, Beyond Pesticides/NCAMP told EPA, "Based on the EPA's own analysis, continued use of diazinon represents an imminent hazard to the health of people and the environment and as such EPA must act to remove all uses of diazinon from the market immediately."

We can move forward successfully when we join together as an informed public interested in the health of our families, children and communities. Best wishes for a healthy new year.



— Jay Feldman is
executive director of
Beyond Pesticides/NCAMP

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National Headquarters:

701 E Street, SE,
Washington DC 20003
ph: 202-543-5450 fx: 202-543-4791
email: info@beyondpesticides.org
website: www.beyondpesticides.org
Printed on recycled paper with soy ink

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BEYOND PESTICIDES/NCAMP STAFF

Jay Feldman, *Executive Director*
Kagan Owens, *Program Director*
Greg Kidd, *J.D.*
Science & Legal Policy Director
John Kepner, *Program Associate*
Terry Shistar, *Ph.D.*, *Science Consultant*
Becky Crouse, *Public Education Associate*
Leslie Haug, *Intern*

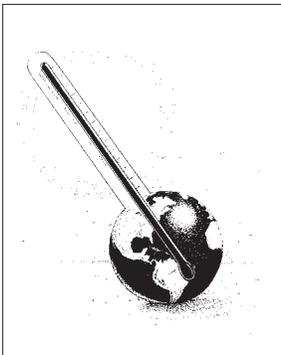
PESTICIDES AND YOU

Jay Feldman, *Publisher, Editor*
Kagan Owens, *Becky Crouse,*
Paul Epstein, *M.D.*, Greg Kidd,
John Kepner, and Terry Shistar, *Ph.D.*
Contributors
Free Hand Press, *Typesetting*

BEYOND PESTICIDES/NCAMP BOARD OF DIRECTORS

Ruth Berlin, *LCSW-C*, Maryland
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Termite Tenting Next Door Causes Concern

Dear Beyond Pesticides/NCAMP,
I just found out that our next door neighbors had their soil treated for subterranean termites yesterday, and will be having their house tented and fumigated for drywood termites this coming Friday and Saturday. The tent is scheduled to go up between 11am and 1pm on Friday, and won't be taken off until 1pm on Saturday. They won't certify that it is safe for the homeowners to return until Monday.

I have never had to be right next door to a house that was being tented. Besides keeping our windows closed and staying out of the yard, do you have any advice or recommendations for us to minimize possible exposure to the pesticides?

Please send me advice as soon as possible, as I am extremely nervous about the impending fumigation. Thanks!

Laura Blackie
Mission Viejo, CA

Dear Laura,

Whole-house fumigation is a common technique employed to eliminate a variety of structural pests. The house is covered with an impermeable covering and a toxic gas is pumped in, usually sulfuryl fluoride (Vikane™) or methyl bromide. A couple of days later, the house is vented and aired out, and the air is tested to make sure that the gas has dissipated. Generally, we would recommend that you make every effort to leave for the weekend, even to stay in a local hotel, especially if you and your neighbor's houses are in close proximity. The chemicals are extremely volatile and will travel, and could penetrate your home as well. If you cannot leave for the weekend, then try to find out when they will be venting the house after the tenting, and stay away during that period. That is when they will be flushing the chemicals out of the house and you are most likely to be exposed. Closing all of your windows is a good idea, but there have been studies that have shown that pesticides can enter your home through cracks and crevices in the structure (i.e.

around windows). You need to be careful that you don't inadvertently trap the chemical in your home. Be sure to turn off any ventilation devices that will draw in outside air, and, after they have finished tenting and venting your neighbor's house, thoroughly ventilate your own home. Run floor and ceiling fans, and open the windows and



doors. Be sure to move air that may become trapped under stairwells and near the ceiling in small areas, such as hallways. According to the Extension Toxicology Network Pesticide Information Profiles, acute effects of sulfuryl fluoride exposure include depression, slowed gait, slurred speech, nausea, vomiting, drunkenness, itching, twitching, and seizures. Chronic exposure can cause injury to lungs and kidneys, weakness, weight loss, anemia, bone brittleness, stiff joints, and general ill health. Acute toxicity of methyl bromide may cause headache; dizziness; nausea or vomiting; chest and abdominal pain; irritated eyes, nose, and throat; slurred speech; blurred vision; temporary blindness; mental confusion; sweating; lung swelling; hemorrhaging of the brain, heart, and spleen; severe kidney damage; and numbness, tremors, and convulsion. Chronic exposure to methyl bromide can include dizziness, vision and hearing disturbances, depression, confusion, hallucinations, euphoria, personality changes, irritability, and chronic pneumonia-like

symptoms. Both methyl bromide and sulfuryl fluoride are highly toxic, colorless and odorless gasses, and are mixed with chloropicrin (a pesticide registered as a grain fumigant), which acts as a warning agent to those handling either gas by irritating the eyes and nose. If you feel any health effects from the chemicals used in the tenting, find out which chemical was used, see a doctor immediately, and contact your state and regional EPA offices to report that you have had a reaction to a pesticide treatment. It should be noted that there are alternatives to toxic-chemical tenting, which include cold and heat treatments.

Beyond Pesticides/NCAMP has information about the toxicity of the chemicals used in structural fumigation and about least-toxic termite control available for \$4ppd.

Teacher Injured By School Herbicide Use

Dear Beyond Pesticides/NCAMP,

I would like to share with you the reason I am no longer teaching. I taught physical education for twenty years and also coached a variety of sports. I loved my teaching career and planned to continue teaching for many years. However, I went to school as usual on a Monday morning three years ago and was overwhelmed by pesticide fumes. Our janitors had sprayed our building with a pesticide containing diazinon, and I immediately became severely ill. My eyes burned, it became very hard to breathe, numbness spread up my arms, I got a migraine headache, and my heart was skipping beats. I had never taken many sick days, so I initially assumed I would probably be sick a few days and then quickly get back to "normal." Unfortunately, the pesticide damaged my immune system and my body will never return to normal.

I got sicker each time I tried to return to the building, but I was determined to find a way to teach. Since I taught P.E., I was able to teach for a while outdoors, calling in sick when it rained. A big shade tree became my "classroom." When cold weather came, I was forced

to start a leave of absence. Eventually, I found a doctor who specializes in the treatment of chemical injuries. She diagnosed me with an illness called Multiple Chemical Sensitivity (MCS), which means my body now reacts to a number of “everyday” chemicals: All pesticides/herbicides; perfume; gasoline; paint; glue; smoke; cleaning products; many medications; and virtually any petrochemical product. This has radically changed my life. Not only have I been forced to give up a career I loved, but also I am basically homebound because so many different products now cause me severe reactions. I used to keep my camping gear packed in my truck and twice went on 5,000-mile solo camping trips. I have never been one to sit at home, but now I am unable to leave home because of the widespread use of weed killer chemicals.

My reason for sharing this with you is to ask your help to see that this never happens to anyone else! I can't stop thinking about all the kids who may not feel well every day because they are breathing in pesticides such as diazinon. My doctor, who is one of the leading MCS researchers in the country, says HALF of all allergy and asthma patients suffer adverse affects from pesticides. How many kids are suffering needlessly? We also often hear about test scores dropping and about aggressive behavior in school. What do we expect when neurotoxic chemicals are routinely used around students? We have got to do a better job of protecting our students and staff from the health hazards associated with toxic chemicals such as diazinon.

Linda Baker

*Dear Linda,
Thank you for sharing your story with us. Unfortunately, it is similar to many oth-*



ers' personal experiences. A memo written by a Health Statistician with the Health Effects Division in the Office of Prevention, Pesticides, and Toxic Substances, Environmental Protection Agency (EPA), stated that 11,808 unintentional diazinon residential exposures were reported to Poison Control Centers from 1993-1996. That memo also stated, “diazinon is one of the leading causes of acute reactions to insecticide use reported as poisoning incidents in the U.S.” And EPA's now defunct Pesticide Incident Monitoring System reported 903 diazinon related human poisonings between 1966-1980.

You will be happy to learn that, on December 5, 2000, the EPA announced a four-year phase out of the organophosphate pesticide diazinon. Although I am sure you agree that all uses of diazinon should be halted immediately, according to the EPA Diazinon Revised Risk Assessment and Agreement with Registrants (12/5/00), products for indoor crack and crevice, and any other indoor residential or indoor non-residential uses will be canceled. Registration of these products will be canceled as of March 2001, and retailers must stop sales of the products as of December 31, 2002. For outdoor non-agricultural uses (home lawn, garden, and any other outdoor residential or outdoor non-agricultural uses), production will be phased down, with technical registrants required to reduce the amount of diazinon produced by 50% or more by 2003. Outdoor non-agricultural uses will also be phased out – formulation of products must cease by June 2003, and sale to retailers must stop by August 2003. Beginning December 31, 2004, technical registrants will start buying back existing products from retailers, and, as of the same date, product registrations will expire with no provision for ex-

isting stocks. Diazinon is the most widely used pesticide by homeowners on lawns, and is one of the most widely used pesticide ingredients for application around the home and in gardens, according to the EPA press release, EPA Announces Elimination of All Indoor Uses of Widely-used Pesticide Diazinon; Begins Phase-out of Lawn and Garden Uses (12/5/00). Allowing the sale of diazinon during this phase-out period raises concerns within the environmental community about continued sales to unsuspecting consumers. Compliance with a pesticide label does not offer the protection that consumers assume, especially in cases such as this, where the EPA has identified the pesticide as hazardous but left it in commerce. Diazinon is a member of the organophosphate family of pesticides, whose common mode of action is on the nervous system. Short-term effects of exposure could include: headaches, nausea, dizziness, swelling joints, disorientation, and respiratory problems, while long-term impacts on the nervous system can impair bodily functions.

Contact Beyond Pesticides/NCAMP for more information about diazinon (\$4 ppd), see page 5 of this issue, or see our website at www.beyondpesticides.org.

Write Us!

Whether you love us, hate us or just want to speak your mind, we want to hear from you. All mail must have a day time phone and verifiable address. Space is limited so some mail may not be printed. Mail that is printed will be edited for length and clarity. Please address your mail to:

Beyond Pesticides/NCAMP
701 E Street, SE
Washington, DC 20003
fax: 202-543-4791
email: info@beyondpesticides.org
www.beyondpesticides.org

S.C. Johnson and Son Gets Slap on the Wrist After EPA Says It Illegally Sold Pesticide

Could you imagine buying a product that actually causes the problem it is supposed to solve? Like buying cold medicine that makes you sneeze, or soap that makes your hands dirty? Certainly no company would make a product like this, right? Well, S.C. Johnson and Son did. Last January, the Environmental Protection Agency (EPA) recalled AllerCare™ Dust Mite Allergen Spray for Carpet and Upholstery, an allergy spray containing the pesticide benzyl benzoate that can actually cause allergic reactions, and charged S.C. Johnson and Son with the illegal sale and distribution of the pesticide. The

EPA recall came nearly three months after the agency began receiving adverse effect reports on the product and realized the spray was not registered as a pesticide product with the agency. (See the Spring 2000 edition of Pesticides and You (Vol. 20, No. 1) for more information on the AllerCare™ recall.) Now, after nearly nine months of waiting, EPA announced that S.C. Johnson reached a small monetary settlement for victims of AllerCare™ poisoning. Since October, over 400 incidents, ranging from severe to minor reactions, including asthma attacks, respiratory problems, burning sensations, and skin irritation, have been documented by EPA. Allergy and asthma sufferers appear to be most negatively affected by the use of the spray, with most reac-

tions occurring within 15 to 30 minutes of product application. EPA has also received some reports of reactions in pets.

AllerCare™ Dust Mite Allergen Spray for Carpet and Upholstery has been removed from store shelves and is no longer produced by the S.C. Johnson and Son, Inc. Under the agreement, the company has to pay only \$200,000 in civil penalties, but will provide funding to the Asthma and Allergy Foundation of America to purchase and staff a Mobile Asthma Clinic

“Breathmobile®” for specialized health care to Baltimore’s inner-city children. The settlement funds the “Breathmobile®” for one year of diagnosis and treatment at a cost of just under \$700,000. Many people feel this is a small price to pay for putting children and other allergy sufferers at risk.

For more information on the recall, visit EPA’s website at:

www.epa.gov/pesticides/citizens/recalls.htm. If you have had an adverse reaction to AllerCare™ products, contact Jerry Blondell, EPA, Health Effects Division (7509C), Ariel Rios Building, 1200 Pennsylvania Ave., NW, Washington, DC 20460, 1-800-858-7378, nptn@ace.orst.edu. If you have AllerCare™ Dust Mite Allergen Spray for Carpet and Upholstery in your home, contact SC Johnson, 1-877-255-3722, for instructions on where to take the product for recovery, or for a refund.

West Nile Virus Can Be Spread Bird-to-Bird Without Mosquitoes

Last summer, communities were loading their trucks with insecticides to



combat the West Nile Virus (WNV) at the first sight of a dead crow. But a new study adds more suspect to the position that spraying based only on dead birds was premature. On October 25, 2000, scientists at the U.S. Geological Survey’s (USGS) Wildlife Health Center in Madison, Wisconsin announced that WNV can be transmitted directly between birds, without the help of mosquitoes, previously thought to be the sole means of transmission. After scientists placed infected birds in the same biocontainment aviary with healthy control birds, nine infected birds died within five to eight days. Four healthy control birds died from the virus five to eight days later. The fifth control bird died eleven days after that, meaning the virus was transmitted from once healthy birds to another healthy bird. “It confirms a suspicion that we had and wanted to verify,” said Dr. Robert McLean, director of the USGS National Wildlife Health Center. “The setting was a very controlled scientific experiment and we’re not sure if or how this relates to what is happening in the wild. Mosquitoes are the primary means of transmission of the virus between birds and to humans. But this certainly opens up a host of new questions.”

Environmentalists maintain that infected crows do not indicate the presence of infected mosquitoes in the area, and should not trigger pesticide spray programs. Environmentalists also raise concerns about the effect of toxic pesticides on beneficial mosquito-eating or-



ganisms, the likelihood mosquito resistance to the pesticides, and the inaccuracy of pesticide spray trucks at reaching their target. Because of the ineffectiveness of pesticide spraying and the adverse effects on human health, many communities lobbied with these arguments following the 1999 mosquito season. Based on experience and effective management techniques, infected birds did not trigger spraying in some communities, including Nassau County, New York and Fairfield County, Connecticut. This policy drew sharp criticism from New York City Mayor, Rudolf Giuliani, who has insisted the pesticides used in New York are safe and said the reluctance of Nassau County to spray was putting New Yorkers in danger. Co-founder of Connecticut Seeking Alternatives for the Environment, Rivka Lieber, who was instrumental in stopping the Fairfield County spraying, has a different philosophy. "The current system assumes pesticides are innocent until proven guilty and that's wrong," Ms. Lieber told the *Hartford Courant*. "The presumption of innocence has made people sick and damaged the ecosystem."

According to the USGS, crows are highly susceptible to the virus, and because of their behavior, more likely than other bird species that live in close contact with one another to transmit the disease to one another. The virus attacks the crow's entire body and often affects all the major organs. Currently scientists are conducting studies to determine exactly how the virus is transmitted without mosquitoes present. "By keeping the infected and



healthy birds together in close contact, we really maximized the potential that this bird-to-bird transmission could take place. Now we know it did and we want to figure out how." The experiment was done in collaboration with the Wildlife Conservation Society, which also helped fund the study. For more information on the study contact Butch Kinerney

of the USGS at 703-648-4732 or visit www.umesc.usgs.gov/http_data/nwhc/news/westnil2.html. For an information packet on WNV send \$4 to Beyond Pesticides/NCAMP.

EPA Announces Weak Diazinon Phase-Out

The Environmental Protection Agency (EPA) announced on December 5, 2000, an agreement with the Syngenta Corporation that will allow residential uses of diazinon, the most widely used insecticide by homeowners in the U.S., to be phased out over a four-year period without any warning to consumers about its neurotoxic properties. Under the agreement, all sales of diazinon products intended for indoor use must cease by December 31, 2002, and sales for products intended for non-agricultural outdoor use must end by December 31, 2004, at which point the manufacturer will begin to buy back existing stocks. Approximately 30% of diazinon's agricultural uses will be cancelled, retaining use on over 40 crops and additional imports. Environmentalists believe that the EPA agreement is too weak, because it does not adequately protect public health. Be-

yond Pesticides/NCAMP urges homeowners, pest control companies and farmers to stop their use and retailers stop sale of diazinon immediately.

"Nothing short of a ban of diazinon will protect the public from the chemical's adverse effects to the nervous system," said Jay Feldman, executive director of Beyond Pesticides/NCAMP. "Since less toxic and non-toxic alternatives are available for all diazinon uses, it is wrong and unnecessary to allow its uses to continue during a long phase out period." In June of this year, EPA announced a similar agreement on another widely used insecticide, chlorpyrifos, and was sharply criticized for allowing sales to continue through 2001 and all stocks to be used up. A number of states' attorneys general urged retailers to stop sale of chlorpyrifos immediately when that action was announced. With an even longer phase-out period for diazinon, similar action can be expected.

Diazinon, a highly toxic organophosphate insecticide, is sold under the trade names Ortho, Spectracide and Real-Kill. It is currently registered for use in agriculture and for residential indoor and home garden use to control cockroaches, aphids, scales, mites, fleas and ticks, among others. After a long court battle in the late 1980's, uses on sod farms and golf courses were cancelled in 1988. Diazinon is neurotoxic and causes irritation to the eyes and skin. EPA's now defunct Pesticide Incident Monitoring System (PIMS) reported 903 diazinon related poisonings between 1966-80. Studies have shown birth defects in laboratory animals, and birds, especially grazing fowl like ducks and geese, are particularly susceptible to diazinon poisoning. In 1985, diazinon, applied in accordance with the label instructions, was responsible for a bird kill of over 700 Atlantic brant in New York State. EPA will be opening a public comment period on diazinon's revised risk assessment in the following weeks. Contact Beyond Pesticides/NCAMP for instructions on submitting comments and the docket number to include with your comments.



Connecticut Agrees to Investigate Lobster Deaths

Helicopters spraying pesticides were a familiar part of the New York City metro area skyline in the Fall of 1999. People ran for cover as the helicopters doused the streets, houses and waterways with insecticides used to combat the West Nile Virus (WNV). Then there was the aftermath. In the city, people were exposed to toxic pesticides and 200+ reports of poisoning were filed. In the rivers and the Long Island Sound, the water was contaminated with pesticides known to be toxic to aquatic life, and the lobsters were dying. But for one reason or another, not everyone believes the pesticides are to blame. On September 30, 2000, the Connecticut Department of Environmental Protection (DEP) announced that it has allocated \$3.5 million to study the recent die-off of lobsters in the Long Island Sound. The sound, a 90-mile inlet of the Atlantic Ocean located between Connecticut and New York, has lost virtually all of its harvestable lobster population since last year's mosquito spraying. The Connecticut DEP is among the ranks of those who do not believe pesticides are responsible for the recent decline in the

local lobster population and argues that a parasite is to blame. Other biologists believe that if parasites are responsible, the pesticides certainly played an important role, making the lobsters more susceptible to disease.

In August, local lobstermen filed suit in U.S. District Court in Brooklyn, NY, against the pesticides' manufacturers, Cheminova Inc. of Wayne, NJ, which sold malathion-based products to New



York State, and Clarke Industries of Roselle, IL, which sold pyrethoid-containing pesticides to the state of Connecticut. "I think the people who are out of business should be compensated," said Nick Crismale, a plaintiff and president of the Connecticut Lobstermen's Association told the *Stamford Advocate*. "Our second objective is to stop the spraying and allow the fishery to come back." Gladstone Jones, attorney for the Connecticut lobstermen, contends that the

pesticide manufacturers, particularly Cheminova, knew their products could harm crustaceans, which are biologically similar to insects. Both malathion and synthetic pyrethroids are extremely toxic to aquatic organisms. For more information on the toxic effects of malathion and synthetic pyrethroids, contact *Beyond Pesticides/NCAMP* or send \$4 to receive fact sheets through the mail. For more information on the lawsuit involving the lobstermen, contact *Connecticut Seeking Alternatives for the Environment (SAFE)* at 203-329-9990 or LieberR@aol.com.

Pesticide Use on the Rise in New York State

On October 18, 2000, Environmental Advocates and the New York Public Interest Research Group (NYPIRG), both based in Albany, NY, released a 44-page report, *The Toxic Treadmill: Pesticide Use and Sales in New York State 1997-1998*, which documents an enormous volume of toxic pesticide use in New York State, with disproportionately high use in urbanized areas of the state. The groups utilized data collected by the state Department of Environmental Conservation (DEC) under the 1996 *Pesticide Reporting Law*. The pesticides cited in the report pose a constellation of hazards including: health risks such as neurotoxicity, carcinogenicity, and endocrine, immune, and reproductive system damage; environmental risks such as

contamination of indoor and ambient air, water, and food; and increased pest problems due to pesticide resistance and secondary infestations. According to the report, New York is not unique in its over-reliance on hazardous pesticides, but it has the opportunity to be unique in how it faces up to this knowledge. The pesticide reporting data offer the insight to direct this effort, showing not just the greatest hazards but also the greatest opportunity for change.

In contrast with the Environmental Protection Agency's (EPA) finding that 77% of all pesticide use is in agriculture, New York urban and suburban use far exceeds agricultural use, with New York City, Long Island, and Westchester accounting for 60% of the total pesticides used by gallons in the state, while constituting only 4% of the state's geographic area. This confirms the findings in an earlier report, *Plagued by Pesticides*, by the same authors. New York City alone accounts for 36% of the total gallons of pesticides reported for the state in 1998. The report reveals that across the state, 4.5 million gallons of pesticides were reported used by commercial applicators or sold to farmers in 1998. This is a 20% increase from 1997. Nearly a third of the total amount of pesticides reported by gallons in 1998 are classified by EPA as known or suspected carcinogens. More than a quarter are suspected endocrine disruptors, and approximately one quarter belong to the highly neurotoxic chemical families of organophosphate and carbamate insecticides. The top pesticide reported by gallons in 1998 was chlorpyrifos, sold as Dursban, a broad-spectrum insecticide recently restricted by EPA in June 2000 because of its high toxicity and the danger it poses to children.

The report concludes that several steps must be taken to protect the citizens of New York. These actions include: banning the use of the most toxic pesticides and eliminating pesticide use in settings where they pose particular dangers, actively promoting safer alternatives to pesticides, assessing a variable tax for pesticide manufacturers tied to their pesticide sales to finance all pesticide programs, reducing pesticide use in New York City, allocating more resources to protect farmworkers, banning the aesthetic use of pesticides on lawns, trees, shrubs and ornamental gardens, improving the pesticide reporting data and removing barriers to justice for pesticide exposure. *For a copy of this report, visit www.envadvocates.org/public_html/Pest/Toxic_Treadmill/toxic_treadmill.htm or send \$5 to Beyond Pesticides/NCAMP for a hardcopy (44 pp.).*

Construction of Golf Course Halted Over Environmental Concerns

"Not in my backyard!" exclaimed hundreds of concerned parents, citizens and environmentalists when construction crews in Stony Point, New York broke ground on a new municipal golf course. On November 3, 2000, a state court in New York ordered officials in the Long Island town to stop construction on the golf course site pending completion of a detailed environmental impact study. The court issued a temporary restraining order in response to a lawsuit filed by the Neighborhood Network Research Center and the Stony Point Action Committee

for the Environment after the town of Stony Point granted permission for the construction of a new municipal golf course, stating, "No negative environmental impact would result from the course." The lawsuit, filed under article 78, asked the court to annul, cancel or set aside a previous decision by a municipality. "Stony Point failed to accurately fill out a detailed form seeking information about the scope of a proposal and its impact on everything from air and water to traffic and historic sites," Frank Collyer, a concerned resident involved with the lawsuit told the *Journal News*. "The town also failed to accurately outline steps that would be taken to lessen the environmental impact."

Among issues including sprawl, deforestation and poor land management, environmentalists and other citizens are concerned about the pesticide use that a new golf course would bring to the community and local ecosystem. Long Island Neighborhood Network (LINN), a partner organization of the Neighborhood

Network Research Center, believes that golf courses should be managed in such a way that there are no harmful effects on either the environment or public health. LINN currently runs an *Organic Golf* campaign, which has the ultimate goal of eliminating synthetic golf course pesticide use by implementing organic management practices at existing golf courses and supporting an organic standard for the construction of any new golf courses. *For more information on the lawsuit or the Organic Golf campaign, contact the Long Island Neighborhood Network at 516-541-4321 or visit their website at www.longislandnn.org. For a pesticides and golf packet that includes examples of golf courses that have converted to organic or drastically reduced*

their chemical dependence, send \$6 to Beyond Pesticides/NCAMP.



Wasps Used as a Biological Control for Whiteflies

They've sprayed and sprayed some more, but the pesticides used to control California's giant whitefly problem just aren't working. While this hasn't surprised environmentalists familiar with the ineffectiveness of pesticide spray programs, it has left California researchers scrambling for a new method of control. The most recent and most effective solution has come out of the University of California Cooperative Extension for Orange and Los Angeles counties, which introduced two species of parasitic wasps to control the giant whitefly population. "Applications of pesticides and removal with water are only temporary, and pesticide use is actually detrimental," John Kabashima of the University of California Coopera-

tive Extension told the *Los Angeles Times*. "Introducing pesticides into the environment disrupts other natural systems, and actually kills off beneficial predators, including the wasps, which are more fragile. The whiteflies will come back, but the beneficial parasites may not, and you'll have a worse problem than you started with." In fact, whiteflies, known as secondary pests, emerged as a serious pest problem after its natural predators were killed off by conventional synthetic pesticides. The stingerless wasps, which are imported from Mexico, control the giant whitefly population by laying their eggs inside the whitefly larvae. When the wasp eggs hatch, their larvae feed on the giant whitefly larvae, killing the host. Scientists at the University of California claim that the wasps, while not harmful to other species, will eventually have a tremendous impact on the giant whitefly, once the wasp population is established. The number of giant whiteflies has declined since the introduction of the wasps last year.

Pesticide Drifts from Farm to School, Two Students Sent Home

On the morning of November 8, 2000, children arriving at Mound Elementary School in Ventura, California walked into a cloud of Lorsban™, a pesticide containing the active ingredient chlorpyrifos, that had drifted from a neighboring lemon orchard onto school property. Two children were sent home because of symptoms of pesticide poisoning. The *Los Angeles Times* reported that dozens of students and school staff complained of headaches, nausea and dizziness associated with the pesticide exposure. Because of the incident, the citrus grove owner has agreed to halt spraying during schools hours. But that is not enough, says the community ac-



tivists group, Community and Children's Advocates Against Pesticide Poisoning (CCAAPP). CCAAPP is calling on the Agriculture Commissioner and school board officials to establish a one-mile buffer zone that prohibits the use of pesticides around schools. Beyond Pesticides/NCAMP's report, *The Schooling of State Pesticide Laws - 2000*, states that six states have recognized the importance of controlling drift by restricting pesticide applications in areas neighboring a school. These states create spray restriction zones that range from 300 feet to 2½ miles. Four of these states, Louisiana, New Hampshire, New Jersey and North Carolina, specifically restrict agricultural pesticide use either during commuting hours or regular school hours. (See the November 2000 edition of Technical Report (Vol. 15, No. 11).)

Chlorpyrifos is in the family of approximately 40 widely used organophosphate pesticides, known neurotoxic chemicals. It is the thirteenth most commonly used pesticide in agriculture, with 13 million pounds applied annually. Acute exposure can result in symptoms such as numbness, incoordination, dizziness, nausea, stomach cramps, headaches, anxiety, drowsiness, depression, and muscle twitching. In 1997, EPA Office of Pesticide Programs, Health Effects Division reported that chlorpyrifos is one of the leading causes of acute insecticide poisoning incidents in the U.S.

Chlorpyrifos has been controversial for decades. In June 2000, the U.S.

Environmental Protection Agency (EPA) and chlorpyrifos manufacturers agreed to phase-out most chlorpyrifos household uses, citing children's high risks associated with chlorpyrifos exposure. Agriculture (excluding tomatoes), golf course, mosquito control and containerized baits are not affected by the agree-

ment. The EPA chlorpyrifos agreement begins the process of getting high consumer and children exposure uses of

chlorpyrifos off the market, but continues to put people at risk by not stopping all its uses immediately. See the July 2000 edition of Technical Report (Vol. 15, No. 7). For more information on pesticides and schools, contact Beyond Pesticides/NCAMP, for a copy of *The Schooling of State Pesticide Laws - 2000* report visit www.beyondpesticides.org or send \$2 to Beyond Pesticides/NCAMP for a hardcopy.

Monsanto Says No to New GE Technologies, Environmentalists Skeptical

After years of rejecting genetically modified " Frankenfoods," consumer pressure finally paid off on November 27, 2000, when the Monsanto Corporation announced that it will back off from some controversial genetic engineering technologies and promised to be more receptive to government safety regulation. Monsanto also pledged not to use animal or human genes in modified crops and promised to sell products commercially only after they are approved for consumption by humans as well as livestock. "We were blinded by our own enthusiasm," Monsanto Chief Executive Hendrik Verfaillie conceded before a Farm Journal conference, according to the *Agribusiness Examiner*. "We focused so much on getting this technology right for the grower that we didn't fully take into account the issues and concerns it raised for other people." Despite the step forward, environmentalists remain skeptical. According to Julie Miles, a co-founder of Genetically Engineered Food Alert, "Monsanto's acknowledgement that they've rushed this technology is meaningful, but if Monsanto truly wants to respond to consumer concerns, it should support mandatory testing and mandatory labeling of genetically engineered foods." Experts believe that Monsanto's decision was most likely based on its poor performance in the stock market. For more information on genetically modified crops or the connection between genetic engineering and pesticides, contact Beyond Pesticides/NCAMP.

Pesticides Linked to Parkinson's Disease

Study Reveals Home Pesticide Use Leads to 70% Higher Disease Rate

By John Kepner

The U.S. government spends millions of dollars each year trying to find a cure for common diseases — from asthma and hyperactivity to cancer and neurological disorders. Researchers work long hours trying to develop that lucrative vaccine that will solve our medical problems. But what if reasonable changes in our use of toxic chemicals could actually prevent these life-threatening diseases? Many scientific studies link pesticides to asthma, cancer and other environmental illnesses, but we still lack the tough laws to prevent exposure to toxic chemicals, especially pesticides. After years of notably high rates in farming communities¹, recent studies have shown Parkinson's disease to be clearly linked to home-use pesticides.

Parkinson's Disease

Parkinson's disease is a neurological disorder characterized by progressively degenerative symptoms including tremors, muscle rigidity, slowness and imbalance. A loss of cells in the *substantia nigra*, a region of the brain that produces the chemical dopamine, which is essential to signal muscle cells properly, causes the symptoms of Parkinson's disease. According to medical science, the cause of Parkinson's disease is still uncertain, but recent studies suggest that it is caused by factors in the environment, rather than genetics². Other researchers believe that there is a genetic predisposition that is triggered by certain toxics in the environment. These scientists caution that because it is impossible to know your genetic disposition, all people should avoid contact with environmental toxics. Pesticides, industrial chemicals and heavy metals are the primary suspects linked to Parkinson's disease. While the disease is usually seen in people over 60, recently there has been a rise in cases for people under 40³. Today an estimated one million Americans are living with the disease. Treatments are available for the symptoms, but there is currently no cure for Parkinson's disease.

Early Findings

The suspicion that pesticides might be linked to Parkinson's disease was theorized in the 1980's following a wave of drug-induced Parkinson's-like illnesses. The drug, MPTP, which was used as a heroin substitute, is transformed in the brain after injection. The new compound, MPP+, causes the loss of dopamine producing cells and the sudden onset of a Parkinson's-like illness⁴. It was later discovered that MPP+ was not only the breakdown product of an obscure drug, but also the active ingredient of the herbicide cyperquat, and closely related to other pesticides.

This discovery sparked interest in studying the link between pesticides and Parkinson's disease. The early studies revealed a positive correlation between rural life, farming or orchard work and the incidence of the disease. A 1988 study published in *Neurology*, discovered that many people living in rural areas, with no diagnosed neurological disorders, had lower levels of dopamine producing cells than urban populations⁵. This suggested that even in the absence of the illness, some aspect of rural life was putting them at risk for Parkinson's disease. Another important study came in 1990, when Dr. William Koller at the University of Kansas interviewed 300 subjects, half of whom suffered from Parkinson's disease. He discovered that Parkinsonians were twice as likely as their healthy counterparts to have grown up in farming communities, where pesticides often contaminate ground water. Dr. Koller's study showed that people with the disease were also more likely to draw their drinking water from wells¹. In 1996, a German study linked Parkinson's disease specifically to pesticides, particularly organochlorines, alkylated phosphates and wood preservatives, and found no link to other rural factors.

Recent Findings: From Farm to Home

Through the end of the 1990's, most studies linking pesticide use to Parkinson's disease were conducted in farming communities, and the decade came to an end with no substantial link to non-agricultural pesticides. However, in May 2000, Dr. Lorene Nelson, a neuroepidemiologist at Stanford University, released the results of the first study to show a correlation between pesticides and Parkinson's disease, outside of agriculture⁶. Her study, which was also the largest ever of its kind, showed a significant link between Parkinson's disease and home pesticide use. The study questioned about 1000 subjects, half of whom were recently diagnosed with Parkinson's disease. The participants answered detailed questions about the type of pesticides used, frequency of use, and when they were first exposed to home and garden pesticides.

Dr. Nelson's study revealed that people exposed to in-home insecticides are 70 percent more likely to develop Parkinson's disease. Exposure to garden insecticides carries a 50 percent increase of developing the disease. Among herbicide users, the risk of developing Parkinson's increases as the number of days in contact with herbicides accumulates. Respondents who reported handling or applying herbicides for up to 30 days were 40 percent more likely to develop the Parkinson's, whereas respondents that reported 160 days exposure, had a 70 percent increase. Dr. Nelson's full report is expected to be published in early 2001.

¹ Koller, W. et al. 1990. Environmental Risk Factors in Parkinson's Disease. *Neurology* 40:1218-1221.

² Tanner, C. et al. 1999. Parkinson Disease In Twins: An Etiological Study. *Journal of the American Medical Association*. 281:341-378.

³ National Institute of Neurological Disorders and Stroke. *Parkinson's Disease: Hope Through Research*. National Institutes of Health. www.ninds.nih.gov/health_and_medical/pubs/parkinson_disease_htr.htm.

⁴ Langston, W.J. and P. Ballard. 1983. Chronic Parkinsonism in Humans Due to a Product of Meperidine-Analog Synthesis. *Science*. 219:979-980.

⁵ Thiessen, B. et al. 1998. Substantia Nigra Neuronal Counts in Normal Rural and Urban Population. *Neurology*. 38:348.

⁶ Stephenson, J. 2000. Exposure to Home Pesticides Linked to Parkinson Disease. *JAMA Medical News and Perspectives*. 238:3.

Minimizing Mouse Madness

A Guide to House Mouse Control

by **Becky Crouse**

A friend of mine had a pet mouse named Nickodemus. We would take him out of his cage and pet him, and let him run up and down our arms. Unfortunately, my friend's mom didn't find him so endearing. You know that "EEK!" heard in every cartoon featuring a rodent? People really do that. Poor Nickodemus was given up for adoption. Now, having awoken to something scurrying across my bed, and having found a fuzzy friend in my kitchen, I understand that it's more than a little unnerving, but I also know it's inevitable. Mice are attracted to places that provide hiding places and easy-to-access food. You are going to find them anywhere humans live, and mice have been found everywhere from penthouse apartments and upscale restaurants, to low-income neighborhoods and fast-food joints. Now, combine this food and shelter with the onset of cold weather, and suddenly you have more roommates than you ever thought possible. They're warm blooded, and they don't have a fireplace in their den. (Unless, of course, your den has become their den.) They are going to want in, and they're incredibly resourceful in their pursuit of a warm, cozy home – yours.

Can I see some ID?

The house mouse's body is brown to gray, about 3 to 4 inches long, and weighs only about 1/2 ounce. It has a semi-naked, dark tail about the length of its head and body combined, large ears and eyes in proportion to its head, and a pointed snout. Its upper incisors are flat and notched, and its feces is rod shaped, pointed at the ends, and about 1/4 inch long.

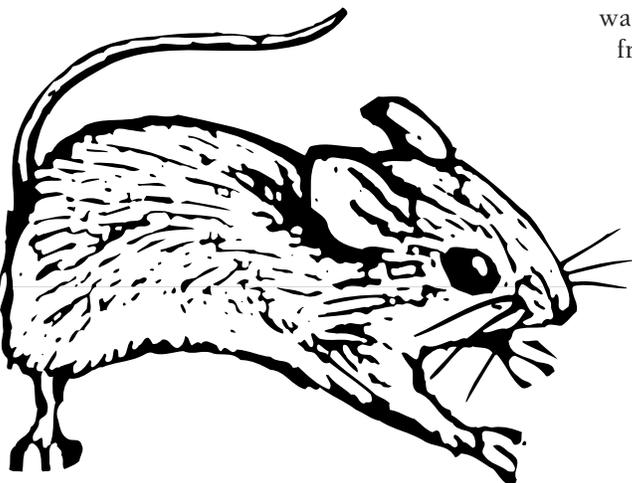
They're multiplying!

One pair of house mice can, in theory, produce 87 young per year. The female becomes sexually mature at two- to three-months old, and is sexually receptive and fertile (estrus) every four days throughout the year, but will mate at any time. Her gestation period is 20 days, and her average-sized litter is 6.7 cute, cuddly, little newborns. She can produce, depending on the availability of food, up to 10 litters per year. Mouse populations will grow as

large as their food, shelter, and other competing species will allow. No matter what method of control you choose, the only way to permanently rid yourself of a mouse problem is to remove their access to the food and shelter that you are providing.

Keeping them out.

The first defense and offense should always be prevention. A full-grown mouse can enter your house through a hole the size of a dime. They are talented climbers and able to swim, but do not need water to survive. (They get water from their food.) To minimize your house mouse magnetism:



Outside

- ▶ Stuff holes in and around the house with steel wool or copper mesh, or fill them with caulk or plaster and cover with sheet metal. Pay particular attention to the foundation and holes between the house and garage;
- ▶ Seal gaps around the doors by replacing worn thresholds and weatherstripping, and installing door sweeps;
- ▶ Raise woodpiles at least 12 inches from the ground (and pet cages, if mice find them interesting), and wrap the legs in galvanized sheet metal to prevent the mice from climbing them;
- ▶ Cut tall grass, weeds, and brush from around the foundation and dispose of the clippings;
- ▶ Discard or recycle unused clutter around the house that may be providing a home for mice;
- ▶ Pick up fallen fruit and rotting vegetables from the garden, and don't place food scraps at the top of the compost pile;
- ▶ Store birdseed in a sealed container, use a birdfeeder with a catch tray, and clean up around it regularly; and,
- ▶ Store trash in a metal container with a tight cover or fastener for the lid.

Inside

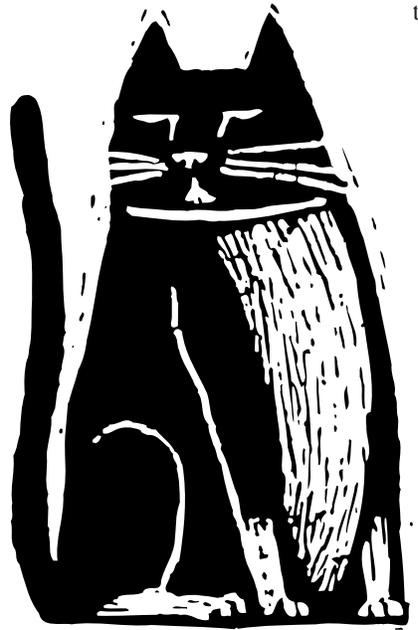
- ▶ Don't leave food on counters or dirty dishes in the sink overnight;

- ▶ Keep the stovetop, oven, broiler, and kitchen floor clean (especially under the stove and refrigerator);
- ▶ Keep the garbage in a plastic container with tight lid;
- ▶ Store grains, cereals, nuts, and pet foods in sealed plastic, metal or glass containers, or keep them in the refrigerator;
- ▶ Pick up any uneaten pet food before going to bed;
- ▶ Caulk openings around water pipes, electric wires, cables, and vents; and,
- ▶ Use hardware cloth to screen vents, floor drains, and any other openings.

How will I know?

A mouse scurries across the linoleum while you're contemplating your midnight snack, you find droppings on the counter or find a bag of barley with holes on both ends and a tunnel running between – it may be this easy to determine that you have a mouse problem. If you aren't certain that Mickey's cousins have moved in, sprinkle the surface that you suspect that they are frequenting with a light coating of flour. If correct, you'll find footprints in the flour and tracks from the flour, hopefully, to their point of entrance.

Be more aware of possible mouse activity in the fall, when the cold weather hits, paying particular attention to areas where food is stored. Watch for mouse activity outdoors, in areas adjacent to houses, which may be the first sign of an impending onslaught.



They're here!

There are a couple of tactics that you can employ to rid yourself of your uninvited guests:

Physical controls:

HAVE-A-HEART TRAPS. These "live" traps are meant to capture the mice so that you can release them instead of killing them. They are usually metal mesh with doors at either end. You can find this type of trap at your local hardware store, or contact Beyond Pesticides/NCAMP for a list of resources. Just be sure to release your little live friend far enough away from your house that he won't return, and remember to block off his point of

entry to prevent any of his friends from taking up residence in his absence.

SNAP TRAPS. If you use snap traps, purchase traps that have expanded triggers that snap when a mouse runs over them, even when unbaited, and a clothespin-like closing mechanism, which is thin enough to allow the bait pan to be bent by hand, allowing for the regulation of trigger sensitivity. Your chances of catching your mouse are greater with a more sensitive trigger.

Set baited traps out for a few days without setting the triggers, as mice are wary of new objects in their environment. You will also have a chance to see if your bait is disappearing, indicating that you have chosen a good location for your traps and bait that your mice enjoy. If there is no sign that your bait has been eaten, move your traps to a new location. If that doesn't work, then change your bait.

Mice tend to scurry along the walls, often referred to as runways. Traps should be positioned at a right angle to the wall, with the bait end towards the wall. Place five to ten traps near mouse holes, one to two feet apart. If you are the lucky host to a multitude of mice, it is more likely that they will approach from more than two directions. Try setting the traps in pairs parallel to the wall, with bait pans facing outwards. Traps should always be handled with gloves, as mice are sensitive to the odor of humans. Coating the trap with bacon grease will also help to mask your scent.

Bait should be sticky so that the mouse will disturb the trigger mechanism even if it only touches the bait lightly. Good choices include peanut butter mixed with rolled oats, raisins,

gum drops, or even a small piece of cotton that your little friends will attempt to acquire for nesting material. Various baked breads have also had great success rates, with trap shyness minimized by alternating the type of bread used.

GLUE TRAPS. Many people take issue with glue traps because they don't kill the mouse immediately and may trap nontarget species. They also get really stinky with that dead rodent odor if not checked daily. Glue traps do, however, catch both large adults and smaller mice, which frequently

escape snap traps. They also are good for those hard-to-reach places or where it is difficult to gain access to mouse runways. Glue traps come as either flat boards, or in box or tube types. While the box or tube glue trap will protect against moisture or dust, mice are more reluctant to enter an unfamiliar enclosed ob-

ject than to tread upon a flat, open object.

Mouse populations will grow as large as their food, shelter, and other competing species will allow.

Glue traps should be in place for at least five days to allow enough time for the mice to become accustomed to them before you decide they are unsuccessful. They can be baited, with the bait placed in the center of the trap to ensure that the mouse establishes full contact. Easiest clean up is often simply sweeping the successful trap into a garbage can, taking care not to touch it with your bare hands. Live animals trapped in the glue can be submerged in soapy water until dead.

With any method of trapping, be sure to block the area off where you have set your traps to prevent your children, pets or any other nontarget species from getting hurt or exposed to nasty pathogens from the dead and live mice.

REPELLENT SOUND DEVICES. Certain devices disrupt the sound communication between mice and repel rodents by generating a sound that annoys them, but at a frequency that is not heard by humans. There is little scientific proof that this is an effective method of control, though there have been reports of success using these devices. One example is a solid-state electronic unit that uses a patented method of directing variable pulsating frequencies onto a carrier, usually either the electrical wiring of a building or home, the metal gridwork within a building or the earth around the building, depending on where the unit is used. In your home, it would plug into a three-pronged electrical outlet and use the building's existing wiring to carry a variable, pulsating frequency that would distress your mouse visitors, causing them to leave. This system is designed to affect mice no matter where they are, between walls, in ceilings, and below floors. It is best used with another method of control for the first few months during the "flushing out" period, and when accompanied by habitat modification.

Biological controls:

CATS. Think you're immune to mice because you have a cat? Think again. Cats may be effective in knocking off the occasional mouse, but it is unlikely that they will be capable of suppressing an established mouse problem. If you decide to get a cat, females are more predacious than males, especially if they have a new litter or have been trained by a good mouser. Only count on your cat to prevent initial mouse entry or to detect and remove new mouse colonizers, and remember that, in the small amount of time it lives in your house, a mouse may have time to contaminate food, destroy furnishings, or spread pathogens over clean dishes.

OTHER BIOLOGICAL CONTROLS. Outside, the mouse has many natural enemies, including native hawks, owls, snakes, mites, ticks, fleas, flies, nematodes, bacteria, and viruses. Maintaining parks with wild areas within urban settings can encourage these beneficial organisms.

Specific strains of *Typhtmurium*-like salmonella bacteria are used to control outbreaks of small field rodents in Russia. This is not, however, a practical resource for consumers.

Chemical Controls:

You have options in the world of chemical mouse control — rodenticides (baits and tracking powders) and bait boxes. Mice nibble rather than eat large quantities at a time, so any rodenticide that you consider will need to be used at high concentrations, which means an increase in the hazards to nontarget species (like your pets and kids) who inadvertently happen upon your pile o' poison. If you decide to use poisons, be sure to block off the areas where you have placed them to minimize the chance of an accident.

TRACKING POWDERS AND SINGLE-DOSE BAITS: Tracking powders are extremely hazardous and should really be left to a professional pest control operator. Single-dose baits are high-concentration poisons. They are restricted materials that require a permit and can only be applied by professionals.

ANTICOAGULANTS: The most commonly used household rodenticides are multiple-dose anticoagulant baits. These chemicals are ingested in smaller doses over several days, and essentially work by preventing the mouse's blood from clotting, causing it to bleed to death internally. There is still some risk of poisoning nontarget species, even with the lower doses of poison, and are also reports of mice becoming resistant to some of the most common of

the anticoagulants — warfarin, chlorophacinone, bridufaciynm and bromadiolone. (*See our rodenticide fact sheet on pages 13-14 for more information.*)

BAIT BOXES: Bait boxes are plastic or metal boxes with the anticoagulant bait placed inside. The bait is protected from the elements, humans and pets are more protected from unintentional exposure to the bait, and the amount of bait being taken by the mice can be more carefully monitored. Bait boxes may also help increase the amount of food (and, with the food, poison) taken in by the mouse. Contact Beyond Pesticides/NCAMP for a list of resources for bait boxes.

It is extremely important to try and prevent a mouse problem before it occurs. If you have gone through the steps of prevention and still find yourself the host to mouse guests, begin utilizing your control method of choice as soon as you see signs of mouse activity. It is possible to prevent an infestation from occurring, but remember, no method is going to be a permanent fix unless you seal them out of your house and remove their access to your food.

Contact Beyond Pesticides/NCAMP for more information about house mouse control and a listing of physical control resources (\$4 ppd).

**The first defense and offense
should always be prevention.**

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Resource:

Olkowski, Helga, Daar, Shiela, and Olkowski, William. 1991. *Common-Sense Pest Control*. Newtown: The Taunton Press, Inc.



RODENTICIDES

Rodenticides, pesticides specially designed to kill rodents, pose particular risks for accidental poisoning for several reasons. Since they have been designed to kill mammals, they are also toxic to humans. Because rodents usually share human environments, use of rodenticides poses an inherent risk of exposure to people, particularly children and their pets, as well as other non-target species. In addition, as rodents have developed resistance to these chemicals, there continues to be a need to develop new and potentially more toxic rodenticides.¹

What are Rodenticides?

Rodenticides can be broken down into three categories, baits, tracking powders and fumigants. Both baits and tracking powders are rodent poisons in the traditional sense, they must be eaten to kill the pest. Baits are designed to attract the rodent to a feeding station. Baits can be used both in the field and in and around buildings. Tracking powders are placed along rodent runways in and around buildings, picked up by the fur as the animal passes by, and then ingested during grooming. Fumigants are poisonous gasses, designed to kill rodents in their burrows.

Rodenticide baits and tracking powders are the type of rodenticides that are most often encountered by homeowners with a rodent problem. There are two types of rodent poisons generally available – acute poisons (also known as single feed baits) and chronic poisons (multiple feed baits).² Acute poisons are extremely dangerous to pets and children, as one encounter can make them very sick or kill them.³

Multiple feed baits are the most commonly used type of rodent poisons. Typically these poisons act as anti-coagulants, literally causing the victim to bleed to death internally. The fact that these poisons must be made available to the pest animal over time makes them very hazardous as children, pets and other non-target animals have an extended opportunity to get into them. Current labels for rat and mouse baits used outdoors require that baits be applied in protective, tamper proof bait stations or placed in areas inaccessible to non-target wildlife.⁴

Classes of Baits

ANTI-COAGULANTS

There are two classes of anti-coagulant type rodent poisons, the coumatrans and the indandiones. Coumatrans include some very common rodent poisons such as warfarin, bromadiolone, and coumatrifuryl. Indandiones include the rodent poisons diphacinone and chlorophacinone.⁵

Both of these classes of toxic materials work by blocking vitamin K-dependent synthesis of the blood clotting substance prothrombin. Animals suffering from exposure to anti-coagulant rodenticides suffer from the following list of immediate toxic effects: nosebleeds, bleeding gums, blood in urine and feces; bruises due to ruptured blood vessels; and skin damage.⁶

Exposure to these poisons also has long-term health effects. The coumatrans, warfarin, for example, has been shown to cause paralysis due to cerebral hemorrhage⁷ and is teratogenic⁸ (causes birth defects). Long-term exposure to the indandione, diphacinone causes nerve⁹, heart, liver, and kidney damage as well as damage to skeletal muscles.¹⁰

CHOLECALCIFEROL

Also known as vitamin D₃, cholecalciferol has a unique mode of action. It is metabolized by the body into its active form, which increases the absorption of calcium and phosphorus from the gut, resulting in very high serum levels of calcium.¹¹ The prolonged hypercalcemia is delayed in onset and insidious in progression, leading ultimately to the death of the victim.¹²

BROMETHALIN

Bromethalin is a neurotoxin, unlike the other rodent poisons. The poison affects the body's ability to control muscle contraction through uncoupling oxidative phosphorylation. It can cause swelling of the brain, spinal column and nerves, leading to a loss of the myelin nerve sheath and ultimately to a reduction of nerve impulses and death.¹³ Immediate effects of exposure to bromethalin include skin and eye irritation, weakness in legs, loss of tactile sensation, and death by respiratory arrest.¹⁴

ZINC PHOSPHIDE

When zinc phosphide is ingested, it reacts with water and stomach juices to release phosphine gas, which can enter the blood stream and affect the lungs, liver, kidneys, heart and central nervous system. It is easily absorbed through skin or inhaled from fumes. With repeated exposure, it accumulates in the body to dangerous levels.¹⁵

Signs and symptoms of mild zinc phosphide poisoning include diarrhea and stomach pains. In more severe cases, nausea, vomiting, chest tightness, excitement, coldness, unconsciousness, coma and death can occur from pulmonary edema and liver damage.

Table 1. Demographic Profile of Exposure Cases to Rodenticides in 1998

SUBSTANCE	NO. OF EXPOSURES	AGE			REASON		TREATED BY DOC.	OUTCOME		
		<6	6-19	>19	Unintentional	Other		Mod	Maj	Death
Anti-coagulants	17,724	15,854	561	1,146	17,029	654	5,882	72	28	1
Strychnine	186	35	20	113	97	78	99	15	5	3
Other/unknown	2,390	1,719	158	434	2,156	219	917	35	6	1
Totals	20,300	17,608	739	1,693	19,282	951	6,898	122	39	5

From: Litovitz, T.L., et al. 1999. 1998 Annual Report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *American Journal of Emergency Medicine* 17(5). <<http://www.aapcc.org/1998.htm>>

STRYCHNINE

Strychnine causes violent convulsions because of its direct action on the central nervous system, chiefly the spinal cord. The onset of symptoms begins usually within 15 to 20 minutes of ingestion. A lethal dose of this natural toxin is as little as 15 mg in children.¹⁶

Immediate effects of exposure are irritation to the upper respiratory tract and skin, vomiting, convulsions, hyperthermia, and death due to respiratory or cardiovascular failure.¹⁷ Victims of strychnine poisoning should be placed in a warm, dark room in order to reduce the stimuli that can trigger convulsions. Medical help should be brought to the victim rather than transporting the victim to the medical center because movement will trigger convulsions.¹⁸

Classes of Fumigants

Fumigants are used to kill rodents in their burrows. As a result, homeowners are much less likely to encounter the use of these chemicals but they are worthy of mention. The two most commonly used gasses to kill rodents are phosphine gas and methyl bromide.

PHOSPHINE GAS

Available in a variety of forms including aluminum phosphide and magnesium phosphide, phosphine gas is extremely toxic. Accordingly, EPA has placed chemicals that produce phosphine gas in toxicity category I, the highest toxicity category.¹⁹

When aluminum phosphide is dropped into a rodent burrow it reacts with moisture to form phosphine gas. The signs and symptoms of exposure to phosphine gas are described above under zinc phosphide.

METHYL BROMIDE

Methyl bromide has also been placed in EPA's toxicity category I. EPA has expressed concern over methyl bromide's potential

Table 2. Ounces of Rodenticide Bait LD50s for Pets.

RODENTICIDE	DOG 10 LBS.	DOG 22 LBS.	DOG 30 LBS.	CAT 4.4 LBS.
Warfarin	13	28	38	8
Bromadiolone	35	77	105	35
Diphacinone	3	6	8	7
Chlorophacinone	160	353	481	-
Cholecalciferol	19	42	57	-
Bromethalin	8	16	22	1
Zinc phosphide	0.16	0.35	0.48	0.06

From: 1998. Rodenticide Risk to Dogs and Cats. *Techletter: For Pest Control Technicians* 4(23).

to destroy ozone.²⁰ As a result, methyl bromide is scheduled to be phased out by 2005,²¹ although there is political pressure to extend or reopen the phase out. Long-term exposure studies have found that methyl bromide is a mutagen, and neurotoxin that causes liver and kidney damage.²²

RODENTICIDE RISK TO HUMANS AND PETS

Rodenticides rank second

in the number of human exposures each year compared with the three other major categories of pesticides for which data is collected by the American Association of Poison Control Centers (AAPCC) (see table 1). According to AAPCC's latest numbers, 20,300 people were exposed to rodenticides in 1998. As mentioned above, anti-coagulant poisonings make up the vast majority of cases with 17,724 (87% of total) reported cases. Young children are the most common victims of exposure to rodenticides, 17,608 cases of exposure (87%) were children under six years of age; that is over seven times higher than the other two age groups combined. Tragically, five people died as a result of their exposure to rodenticides in 1998.

Pets and non-target wildlife also fall victim to exposure to rodenticides. Exposure to these animals can occur as a result of either feeding on the bait or eating rodents that have been killed by rodenticides. Toxicologists calculate the dose of poisons that will kill 50% of the animals that are exposed; this measurement is called an LD₅₀. It takes as little as 0.16 ounces of zinc phosphide to kill a 10 lb. dog (see table 2). Rodent poisons should be used only as a last resort. If poisons are used, homeowners need to practice extreme caution when choosing to control rodents in this way.

People dealing with a rodent problem need to consider all of the alternative, nontoxic approaches to rodent control. See pages 10-12 or contact *Beyond Pesticides/NCAMP* to find out more about nontoxic approaches to rodent control.

RODENTICIDES ChemicalWATCH Fact Sheet References

¹ Fishel, F. and P. Andre, 1999. "Pesticide Poisoning Symptoms and First Aid." University of Missouri Agricultural Engineering. <<http://muextension.missouri.edu/xplor/agguides/agengin/g01915.htm>>

² Simon, L. and W. Quarles, 1996. "Integrated Rat Management," *Common Sense Pest Control* 12(1):5-15, citing Meehan, A.P. 1984. *Rats and Mice: Their Biology and Control*. Rentokil, East Grinstead, West Sussex, United Kingdom.

³ Ibid.

⁴ U.S. EPA. 1998. *R.E.D. Facts: Rodenticide Cluster*. EPA-738-F-98-004. p. 2. <<http://www.epa.gov/oppsrrd1/REDs/factsheets/2100fact.pdf>>

⁵ Rachel Carson Council. 1992. *Basic Guide to Pesticides: Their Characteristics and Hazards*. Taylor & Francis, Washington, DC.

⁶ Ibid.

⁷ Ibid.

⁸ Extension Toxicology Network (ETN). 1995. "Warfarin." *Pesticide Information Profiles*. <<http://ace.orst.edu/cgi-bin/mfs/01/pips/warfarin.htm>>

⁹ Rachel Carson Council. 1992.

¹⁰ Extension Toxicology Network (ETN). 1995. "Diphacinone." *Pesticide Information Profiles*. <<http://ace.orst.edu/cgi-bin/mfs/01/pips/diphacin.htm>>

¹¹ Craigmill, A. 1998. *Veterinary Toxicology Notes: Hazards of New Rodenti-*

cides to Pets. *UC Davis Env. Tox. Newsletter* 8(2). <http://ace.orst.edu/cgi-bin/mfs/01/newsletters/n82_88.htm>

¹² Ibid.

¹³ Rachel Carson Council. 1992.

¹⁴ Ibid.

¹⁵ Schulze, L.D., et al. 1997. "Signs and Symptoms of Pesticide Poisoning." University of Nebraska Cooperative Extension EC97-2505-A. <<http://www.ianr.unl.edu/pubs/pesticides/ec2505.htm>>

¹⁶ Fishel, F. and P. Andre, 1999.

¹⁷ Rachel Carson Council. 1992.

¹⁸ Schulze, L.D., et al. 1997.

¹⁹ Extension Toxicology Network (ETN). "Aluminum Phosphide." *Pesticide Information Profiles*. <<http://ace.orst.edu/cgi-bin/mfs/01/pips/alumphos.htm>>

²⁰ Extension Toxicology Network (ETN). 1996. "Methyl bromide: Bromomethane." *Pesticide Information Profiles*. <<http://ace.orst.edu/cgi-bin/mfs/01/pips/methylbr.htm>>

²¹ U.S. EPA Methyl Bromide Phase Out Web Site. <<http://www.epa.gov/spdpublic/mbr/>>

²² Rachel Carson Council. 1992.

Ten Myths Behind Pesticide-Dependent Pest Management in Schools

Debunking opponents to school integrated pest management, pesticide bans and notification programs.

The pro-pesticide lobby has engaged in an all-out effort to convince local school districts that pesticides can be used safely in schools and therefore fully integrated into school pest management programs. One such group, Responsible Industry for a Sound Environment (RISE), distributed a letter containing misleading and inaccurate information on school pesticide use to 25,000+ school facility managers around the country.

To halt the pro-pesticide lobby from continuing to undercut community activists' efforts to reduce or eliminate pesticide use in favor of alternative strategies, Beyond Pesticides/NCAMP has developed this fact sheet as a guide to better understanding the issues. Notification of pesticide applications and elimination of toxic pesticide use where possible can be accomplished in our schools. Invalidate the pro-pesticide lobby's top ten myths with the facts.

MYTH #1

Pesticides are a vital ingredient to an Integrated Pest Management (IPM) program.

FACT #1:

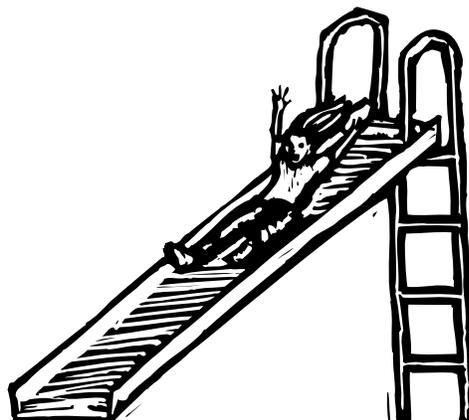
Those who argue that Integrated Pest Management (IPM) requires an ability to spray pesticides immediately after identifying a pest problem are not describing IPM. IPM is pest management that is sensitive to the health of students, school staff and the environment. Pesticide use is unnecessary because safer alternatives can successfully control pest problems. The goal of an IPM program is to minimize the use of pesticides and the associated risk to human health and the environment while controlling a pest problem. IPM does this by utilizing a variety of methods and techniques, including cultural, biological and structural strategies to control a multitude of pest problems. (See box on page 16).

Essential to the control of a pest problem are solutions based on preventing pest outbreaks to occur in the first place. For example, improving a school's sanitation can eliminate cockroaches and ants. Many techniques are relatively simple, such as mulching to prevent weeds or caulking cracks and screening openings where insects and rodents can enter a building. Constant monitoring ensures that pest buildups are detected and suppressed before unacceptable outbreaks occur.

Conventional pest control tends to ignore the causes of

pest infestations and instead rely on routine, scheduled pesticide applications. Pesticides are often temporary fixes, ineffective over the long term. Most common pests are now resistant to many insecticides. For effective pest control, it is absolutely necessary to identify the source of the problem, determine why the pest is present and modify its habitat. For example, since weeds tend to like soils that are compacted, the solution is not the temporary control achieved by killing them, but the adoption of practical strategies to make the soil less attractive to the weeds.

Alternatives to conventional hazardous pesticides are being implemented in over 100 school districts around the country and, thus, prove that alternatives work. Non-toxic and least toxic control products are a major growth area and new materials and devices are increasingly available in the marketplace.



MYTH #2:

Pesticides pose no risk to the health of children.

FACT #2

Student and staff poisoning at schools is not uncommon. Adverse health effects, including nausea, dizziness, respiratory problems, headaches, rashes, and mental disorientation, may appear even when a pesticide is applied according to label directions. Low levels of pesticide exposure can adversely affect a child's neurological, respiratory, immune and endocrine system. Of the 48 commonly used pesticides in schools, 22 can cause cancer, 26 can adversely affect reproduction, 31 are nervous system poisons and 16 can cause birth defects.¹

The General Accounting Office (GAO) in 2000 documented over 2,300 reported pesticide poisonings in schools between 1993 and 1996.² Because most of the symptoms of pesticide exposure, from respiratory distress to difficulty in concentration, are common in school children and may be assumed to have other causes, it is suspected that pesticide-related illness is much more prevalent.

EPA and Dow AgroSciences agreed in June 2000 to phase-out Dursban (chlorpyrifos), one of the most commonly used insecticides in schools, because of its high risks to children, even if used according to the label directions. The product has been marketed for the past 30 years with claims that it could be used safely. Even though EPA and the manufactures of Dursban agreed to phase-out its use

Integrated Pest Management (IPM) is a managed pest management system that: (a) eliminates or mitigates economic and health damage caused by pests; (b) uses integrated methods, site or pest inspections, pest population monitoring, an evaluation of the need for pest control and one or more pest control methods, including sanitation, structural repairs, mechanical and biological controls, other non-chemical methods, and, if nontoxic options are unreasonable or have been exhausted, least toxic pesticides.

Least toxic pesticides include: boric acid and disodium octobrate tetrahydrate, silica gels, diatomaceous earth, nonvolatile insect and rodent baits in tamper resistant containers or for crack and crevice treatment only, microbe-based insecticides, biological, living control agents, and materials for which the inert ingredients are nontoxic and disclosed. The term 'least toxic pesticides' does not include a pesticide that is determined by the EPA to be an acutely or moderately toxic pesticide, a probable, likely or known carcinogen, mutagen, teratogen, reproductive toxin, developmental neurotoxin, endocrine disrupter, or immune system toxin, and any application of the pesticide using a broadcast spray, dust, tenting, fogging, or baseboard spray application.

in many settings, including schools, it can continue to be used until existing stocks are used up. The EPA chlorpyrifos announcement begins the process of getting high consumer and children exposure uses of Dursban off the market, but puts people at risk by not stopping its uses immediately.³

MYTH #3:

Without pesticides, pests pose a serious health and safety risk to children.

FACT #3:

The pro-pesticide lobby wants people to think that if we stop using toxic pesticides, our school buildings and lawns would be overcome by disease-carrying pests and weeds. However, this is not true. School pest problems can be effectively managed without toxic pesticides, as discussed in *fact #1*. Most insect and weed pests may be a nuisance, or raise aesthetic issues, but they do not pose a threat to children's health. Children should never be exposed to potentially harmful pesticides for this reason.

Increasingly the public is calling into question the use of pesticides for cosmetic results alone. The unleashing of these

toxic chemicals into our environment for aesthetic gain is responsible for countless human suffering and untold environmental consequence. In the words of Rachel Carson, "How could intelligent beings seek to control a few unwanted species by a method that contaminated the entire environment and brought the threat of disease and death even to their own kind? Future generations are unlikely to condone our lack of prudent concern for the integrity of the natural world that supports all life."

Toxic pesticides and certain pests do pose a health risk to children,⁴ which is why schools need to implement a comprehensive school IPM program. A school IPM program is established to prevent and manage pest problems, not to let pests run rampant.

MYTH #4:

School IPM programs are too costly for schools.

FACT #4:

According to the U.S. Environmental Protection Agency (EPA), "preliminary indications from IPM programs in school systems suggest that long term costs of IPM may be less than a conventional pest control program."⁵ Because IPM focuses on prevention of the pest problem, and properly monitoring to determine the extent of the pest problem, school IPM programs can decrease the amount of money a school will spend on pest control in the long-term. Some economic investment is usually required at the outset of an IPM program. Short-term costs may include IPM training, purchasing new equipment, hiring an IPM coordinator, or making preliminary repairs to a school's buildings. Chemical-intensive methods only prove to be less expensive in the short-term. The long-term health of our children is not worth short-term economic savings that just do not add up over time.

A well-known example of school IPM is the Montgomery County, Maryland public schools. The IPM program in Montgomery County covers 200 sites and reduced pesticide use from 5,000 applications in 1985 to none four years later, saving the school district \$1800 per school and \$30,000 at the County's school food service warehouse.⁶

In Indiana, Monroe County Schools implemented an IPM program that decreased the school's pest management costs by \$6,000 in two years. Pesticide use has reportedly dropped by 90% with the IPM program, and all aerosol and liquid pesticides have been discontinued.⁷

At Vista de las Cruces School in Santa Barbara, California, pest management was contracted out with a pest control company for \$1,740 per year for routine pesticide applications. After the school switched to an IPM program, their

costs were reduced to a total of \$270 over two years.

Albert Greene, Ph.D., National IPM Coordinator for the U.S. General Services Administration (GSA), has implemented IPM in 30 million square feet, approximately 7,000 federal buildings, in the U.S. capital area without spraying toxic in-

**EPA has stated that no pesticide
can be considered 'safe.'**

secticides. Dr. Greene states that IPM, “can be pragmatic, economical and effective on a massive scale.”⁸

MYTH #5:

Pesticides are extensively tested and regulated. Before a pesticide product is approved for use, it must undergo over 120 government-mandated tests.

FACT #5:

Suggestions that pesticides in wide use have been subjected to full and adequate health and safety testing belies the widely acknowledged deficiencies in EPA’s pesticide registration process. In addition, the safety standard in pesticide law allows elevated rates of disease under a risk assessment-based standard. As a result, EPA has stated that no pesticide can be considered ‘safe.’⁹

Pesticides products contain formulations of a number of different materials, including active and inert ingredients, as well as contaminants and impurities. Additionally, pesticides, when subject to various environmental conditions, break down to other materials, known as metabolites, which are sometimes more toxic than the parent material. So-called inert ingredients can be as or more toxic than the active ingredient – active ingredients in other pesticides, toxic chemicals, chemicals regulated under other legislation, or hazardous wastes, solvents, propellants, wetting agents, petrochemicals and synergists. Inerts, often petrochemicals like toluene or xylene, are generally the largest percentage of ingredients of a pesticide product. Despite this, inert ingredients are treated as trade secret information and not disclosed on product labels. Contaminants and impurities are often a part of the pesticide product and responsible for the product hazards. Dioxin and DDT have been identified as contaminants in pesticide products.

Existing pesticide use patterns and a deficient regulatory process add up to inadequate regulation of pesticides is not protection of public health. The vast majority of all pesticide products registered for use by EPA and state gov-

ernments have never been fully tested for the full range of potential human health effects, such as cancer, birth defects, genetic damage, reproductive effects and neurological disorders, and endocrine disruption. Indeed, pesticides can be registered even when they have been shown to cause adverse health effects. Due to the numerous pesticide formulations on the market, the lack of disclosure requirements, insufficient data requirements, and inadequate testing, it is impossible to accurately estimate the hazards of pesticide products, much less lifetime exposure or risk. There is no way to predict the effects in children solely based on toxicity testing in adult or even adolescent laboratory animals, which is EPA’s procedure for evaluating adverse effects.

MYTH #6:

Each school board should only be responsible for maintaining a registry of individuals interested in being notified and not be overly burdened with providing universal notification.

FACT #6:

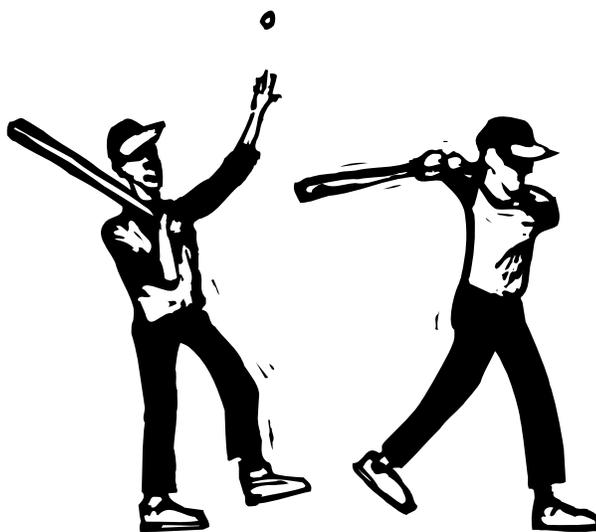
Parents are often kept in the dark about pesticide use at schools. Without notification, parents are unable to make important decisions about whether they want their children to go to school

when potentially hazardous pesticides have been applied.

Universal notification is a good way to make sure that all parents, guardians, children and staff are aware and warned about pesticide applications. Providing prior notification to all individuals attending or working at a school is less obtrusive to the school’s administrative staff. Uni-

versal notification does not require a separate database. Several school districts around the country, such as Ann Arundal County Public School system in Maryland, agree that it is much less cumbersome to provide universal notification. Many schools already send home notices and

school announcements about lice infestation, field trips, book fairs, and crime at school. Schools can simply send universal pesticide notices as they would other such announcements or they can be attached to notices already being sent home.



By providing prior written notification to all parties that would otherwise unknowingly be exposed to the chemicals and posting notification signs, affected parties can take the necessary precautions to avoid the exposure and potential harm it may cause.

Notification-based registries are a less effective means of notifying people and does not qualify as right-to-know because of its limited scope. Requiring that individuals place themselves on registries, affords only those who already know about toxic exposure the opportunity to be informed about pesticide use in the school. Registries are more costly and more resource consuming for school districts to implement. It may even require an extra staff person to keep the registry up to date and coordinate the notification.

MYTH #7:

Notification of pesticide applications are unnecessarily alarming parents and is a scare tactic by environmentalists.

FACT #7:

Parents and school staff have a basic right-to-know when pesticides are being used at school. By providing prior written notification to all parties that would otherwise unknowingly be exposed to the chemicals and posting notification signs, affected parties can take the necessary precautions to avoid the exposure and potential harm it may cause. Pro-pesticide lobbyists may be concerned that if parents and school staff know that a school is applying an EPA classified probable carcinogen, neurotoxin or other type of hazardous pesticide, they may be activated to advocate for alternative approaches that prohibit these chemicals. As discussed above, schools do not need to use toxic pesticides in the buildings or on the grounds where children spend their time learning and playing. IPM, if properly implemented, enables a safe learning environment for children, one that does not introduce unnecessary and routine use of toxic pesticides.

MYTH #8:

Parents and staff only need to be notified 24 hours prior to the use of pesticides at schools.

FACT #8:

Twenty-four hour prior notification of pesticide use does not provide enough time react. Prior notification should be made

72 hours in advance to make sure the information has been received by the student's parents or guardians and by school staff, allowing them to obtain further information regarding the pesticide application, and, if necessary, to make arrangements to avoid the exposure.

MYTH #9:

Schools should not have to notify parents and teachers prior to the use of baits, gels, pastes pesticide applications.

FACT #9:

As long as the bait, gel or paste falls under the "least toxic pesticide" definition (see box on page 16), schools do not need to provide prior notification. However, advance notification should occur for any formulation containing pesticide or other toxic ingredients that are volatile or toxic synergists. Just because a pesticide is applied in baits, gels and/or pastes does not mean these products do not contain a chemical that is a carcinogen, mutagen, teratogen, reproductive toxin, developmental neurotoxin, endocrine disruptor, or an immune system toxin.



MYTH #10:

As long as the pesticide is not applied while the area is occupied, once the students and teachers return to the area, the pesticide has dried and will not affect their health.

FACT #10:

Pesticides should never be applied when students or staff are, or are likely to be, in the area within 24 hours of the application. Pesticides residues can linger for hours, days and even months after an application is made. It all depends on the type of chemical applied and the conditions that may apply to its degradation. For example, airborne concentrations of seven insecticides were tested three days following their application in separate rooms. Six of the seven pesticides left residues behind through the third day.¹⁰ A 1998 study found that Dursban (chlorpyrifos) accumulated on furniture, toys and other sorbant surfaces up to two weeks after application.¹¹

¹ Beyond Pesticides/National Coalition Against the Misuse of Pesticides. 2000. *Health Effects of 48 Commonly Used Pesticides in Schools: A Beyond Pesticides/NCAMP fact sheet*. Washington, DC.
² U.S. General Accounting Office (GAO). 1999. *Use, Effects, and Alternatives to Pesticides in Schools*. RCED-00-17. Washington, DC.
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⁵ U.S. EPA. 1993. *Pest Control in the School Environment: Adopting Integrated Pest Management*. 735-F-93-012. Office of Pesticide Programs. Washington, DC.

⁶ Schubert, J.D., et al. 1996. *Voices for Pesticide Reform: The case for safe practices and sound policy*. Beyond Pesticides/National Coalition Against the Misuse of Pesticides. Washington, DC.
⁷ Safer Pest Control Project. 1998. *Cost of IPM in Schools: A fact sheet from the Safer Pest Control Project*. Chicago, IL.
⁸ Greene, A. 1993. "Integrated Pest Management for Buildings." *Pesticides and You* 13(2-3). Washington, DC.
⁹ U.S. General Accounting Office (GAO). 1990. *Lawn Care Pesticides: Risks Remain Uncertain While Prohibited Safety Claims Continue*. RCED-90-134. Washington, DC.
¹⁰ Wright, C., et al. 1981. "Insecticides in the Ambient Air of Rooms Following Their Application for Control of Pests." *Bulletin of Environmental Contamination & Toxicology* 26.
¹¹ Gurunathan, S., et al. 1998.

Is Global Warming Harmful to Health?

Computer models indicate that many diseases will surge as the earth's atmosphere heats up. Signs of the predicted troubles have begun to appear.

by Paul R. Epstein, M.D.

EDITOR'S NOTE: Global warming and wide fluctuations in weather patterns are projected to lead to increases in insect population and insect-borne diseases. This situation has already begun to materialize, with increasing pressure on public health officials to respond with the use of highly toxic pesticides. Dr. Epstein captures an escalating problem that deserves our attention. [Reprinted with permission. Copyright © August 2000 by Scientific American, Inc. All rights reserved.]

Today few scientists doubt the atmosphere is warming. Most also agree that the rate of heating is accelerating and that the consequences of this temperature change could become increasingly disruptive. Even high school students can reel off some projected outcomes: the oceans will warm, and glaciers will melt, causing sea levels to rise and salt water to inundate settlements along many low-lying coasts. Meanwhile the regions suitable for farming will shift. Weather patterns should also become more erratic and storms more severe.

Yet less familiar effects could be equally detrimental. Notably, computer models predict that global warming, and other climate alterations it induces, will expand the incidence and distribution of many serious medical disorders. Disturbingly, these forecasts seem to be coming true.

Heating of the atmosphere can influence health through several routes. Most directly, it can generate more, stronger and hotter heat waves, which will become especially treacherous if the evenings fail to bring cooling relief. Unfortunately, a lack of nighttime cooling seems to be in the cards; the atmosphere is heating unevenly and is showing the biggest rises at night, in winter and at latitudes higher than about 50 degrees. In some places, the number of deaths related to heat waves is projected to double by 2020. Prolonged heat can, moreover, enhance production of smog and the dispersal of allergens. Both effects have been linked to respiratory symptoms.

Global warming can also threaten human well-being profoundly, if somewhat less directly, by revising weather patterns—particularly by pumping up the frequency and intensity of floods and droughts and by causing rapid swings in the weather. As the atmosphere has warmed over the past century, droughts in arid areas have persisted longer, and mas-

sive bursts of precipitation have become more common. Aside from causing death by drowning or starvation, these disasters promote by various means the emergence, resurgence and spread of infectious disease.

That prospect is deeply troubling, because infectious illness is a genie that can be very hard to put back into its bottle. It may kill fewer people in one fell swoop than a raging flood or an extended drought, but once it takes root in a community, it often defies eradication and can invade other areas.

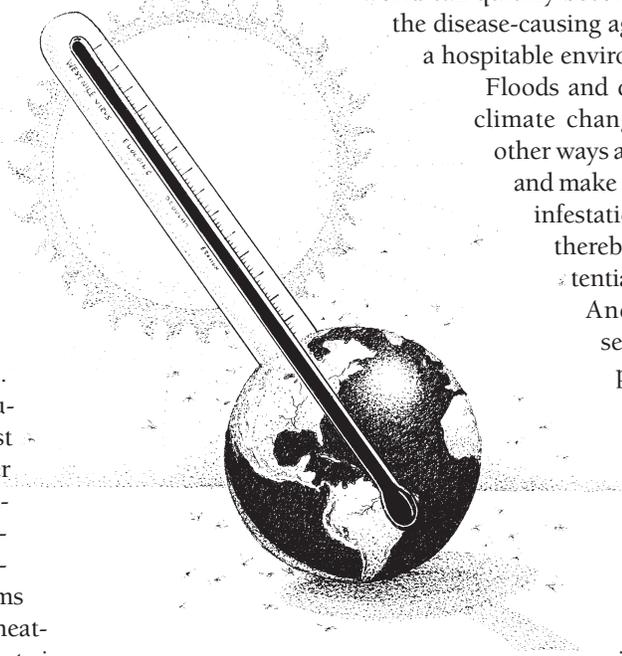
The control issue looms largest in the developing world, where resources for prevention and treatment can be scarce. But the technologically advanced nations, too, can fall victim to surprise attacks—as happened last year when the West Nile virus broke out for the first time in North America, killing seven New Yorkers. In these days of international commerce and travel, an infectious disorder that appears in one part of the world can quickly become a problem continents away if the disease-causing agent, or pathogen, finds itself in a hospitable environment.

Floods and droughts associated with global climate change could undermine health in other ways as well. They could damage crops and make them vulnerable to infection and infestations by pests and choking weeds, thereby reducing food supplies and potentially contributing to malnutrition.

And they could permanently or semipermanently displace entire populations in developing countries, leading to overcrowding and the diseases connected with it, such as tuberculosis.

Weather becomes more extreme and variable with atmospheric heating in part because the warming accelerates the water cycle: the process in which water vapor, mainly from

the oceans, rises into the atmosphere before condensing out as precipitation. A warmed atmosphere heats the oceans (leading to faster evaporation), and it holds more moisture than a cool one. When the extra water condenses, it more frequently drops from the sky as larger downpours. While the oceans are being heated, so is the land, which can become highly parched in dry areas. Parching enlarges the pressure gradients that cause winds to develop, leading to turbulent winds, tornadoes and other powerful storms. In addition, the altered pressure and temperature gradients that accompany global warming can shift the distribution of when and where storms, floods and droughts occur.



I will address the worrisome health effects of global warming and disrupted climate patterns in greater detail, but I should note that the consequences may not all be bad. Very high temperatures in hot regions may reduce snail populations, which have a role in transmitting schistosomiasis, a parasitic disease. High winds may at times disperse pollution. Hotter winters in normally chilly areas may reduce cold-related heart attacks and respiratory ailments. Yet overall, the undesirable effects of more variable weather are likely to include new stresses and nasty surprises that will overshadow any benefits.

Mosquitoes Rule in the Heat

Diseases relayed by mosquitoes—such as malaria, dengue fever, yellow fever and several kinds of encephalitis—are among those eliciting the greatest concern as the world warms.

Mosquitoes acquire disease-causing microorganisms when they take a blood meal from an infected animal or person. Then the pathogen reproduces inside the insects, which may deliver disease-causing doses to the next individuals they bite.

Mosquito-borne disorders are projected to become increasingly prevalent because their insect carriers, or “vectors,” are very sensitive to meteorological conditions. Cold can be a friend to humans, because it limits mosquitoes to seasons and regions where temperatures stay above certain minimums. Winter freezing kills many eggs, larvae and adults outright. Anopheles mosquitoes, which transmit malaria parasites (such as *Plasmodium falciparum*), cause sustained outbreaks of malaria only where temperatures routinely exceed 60 degrees Fahrenheit. Similarly, *Aedes aegypti* mosquitoes, responsible for yellow fever and dengue fever, convey virus only where temperatures rarely fall below 50 degrees F.

Excessive heat kills insects as effectively as cold does. Nevertheless, within their survivable range of temperatures, mosquitoes proliferate faster and bite more as the air becomes warmer. At the same time, greater heat speeds the rate at which pathogens inside them reproduce and mature. At 68 degrees F, the immature *P. falciparum* parasite takes 26 days to develop fully, but at 77 degrees F, it takes only 13 days. The *Anopheles* mosquitoes that spread this malaria parasite live only several weeks; warmer temperatures raise the odds that the parasites will mature in time for the mosquitoes to transfer the infection. As whole areas heat up, then, mosquitoes could expand into formerly forbidden territories, bringing ill-

ness with them. Further, warmer nighttime and winter temperatures may enable them to cause more disease for longer periods in the areas they already inhabit.

The extra heat is not alone in encouraging a rise in mosquito-borne infections. Intensifying floods and droughts resulting from global warming can each help trigger outbreaks by creating breeding grounds for insects whose desiccated eggs remain viable and hatch in still water. As floods recede, they leave puddles. In times of drought, streams can become stagnant pools, and people may put out containers to catch water; these pools and pots, too, can become incubators for new mosquitoes. And the insects can gain another boost if climate change or other processes (such as alterations of habitats by humans) reduce the populations of predators that normally keep mosquitoes in check.

Mosquitoes on the March

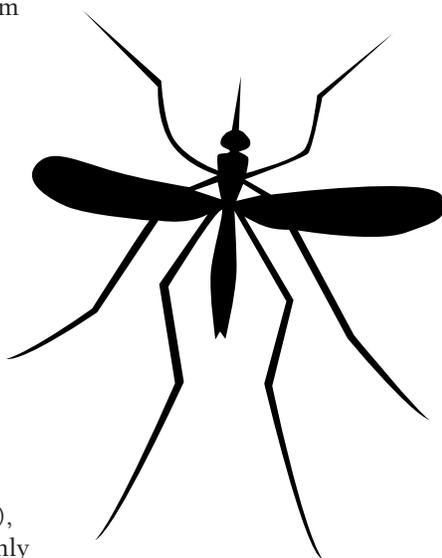
Malaria and dengue fever are two of the mosquito-borne diseases most likely to spread dramatically as global temperatures head upward. Malaria (marked by chills, fever, aches and anemia) already kills 3,000 people, mostly children, every day. Some models project that by the end of the 21st century, ongoing warming will have enlarged the zone of potential malaria transmission from an area containing 45 percent of the world's population to an area containing about 60 percent.

That news is bad indeed, considering that no vaccine is available and that the causative parasites are becoming resistant to standard drugs.

True to the models, malaria is reappearing north and south of the tropics. The U.S. has long been home to *Anopheles* mosquitoes, and malaria circulated here decades ago. By the 1980s mosquito-control programs and other public health measures had restricted the disorder to California.

Since 1990, however, when the hottest decade on record began, outbreaks of locally transmitted malaria have occurred during hot spells in Texas, Florida, Georgia, Michigan, New Jersey and New York (as well as in Toronto). These episodes undoubtedly started with a traveler or stowaway mosquito carrying malaria

parasites. But the parasites clearly found friendly conditions in the U.S.—enough warmth and humidity, and plenty of mosquitoes able to transport them to victims who had not traveled. Malaria has returned to the Korean peninsula, parts



Mosquito-borne disorders are projected to become increasingly prevalent because their insect carriers, or “vectors,” are very sensitive to meteorological conditions.

of southern Europe and the former Soviet Union and to the coast of South Africa along the Indian Ocean.

Dengue, or “breakbone,” fever (a severe flulike viral illness that sometimes causes fatal internal bleeding) is spreading as well. Today it afflicts an estimated 50 million to 100 million in the tropics and subtropics (mainly in urban areas and their surroundings). It has broadened its range in the Americas over the past 10 years and had reached down to Buenos Aires by the end of the 1990s. It has also found its way to northern Australia. Neither a vaccine nor a specific drug treatment is yet available.

Although these expansions of malaria and dengue fever certainly fit the predictions, the cause of that growth cannot be traced conclusively to global warming. Other factors could have been involved as well—for instance, disruption of the environment in ways that favor mosquito proliferation, declines in mosquito-control and other public health programs, and rises in drug and pesticide resistance. The case for a climatic contribution becomes stronger, however, when other projected consequences of global warming appear in concert with disease outbreaks.

Such is the case in highlands around the world. There, as anticipated, warmth is climbing up many mountains, along with plants and butterflies, and summit glaciers are melting. Since 1970 the elevation at which temperatures are always below freezing has ascended almost 500 feet in the tropics. Marching upward, too, are mosquitoes and mosquito-borne diseases.

In the 19th century, European colonists in Africa settled in the cooler mountains to escape the dangerous swamp air (“mal aria”) that fostered disease in the lowlands. Today many of those havens are compromised. Insects and insect-borne infections are being reported at high elevations in South and Central America, Asia, and east and central Africa. Since 1980 *Ae. aegypti* mosquitoes, once limited by temperature thresholds to low altitudes, have been found above one mile in the highlands of northern India and at 1.3 miles in the Colombian Andes. Their presence magnifies the risk that dengue and yellow fever may follow. Dengue fever itself has struck at the mile mark in Taxco, Mexico.

Patterns of insect migration change faster in the mountains than they do at sea level. Those alterations can thus serve as indicators of climate change and of diseases likely to expand their range.

Opportunists Like Sequential Extremes

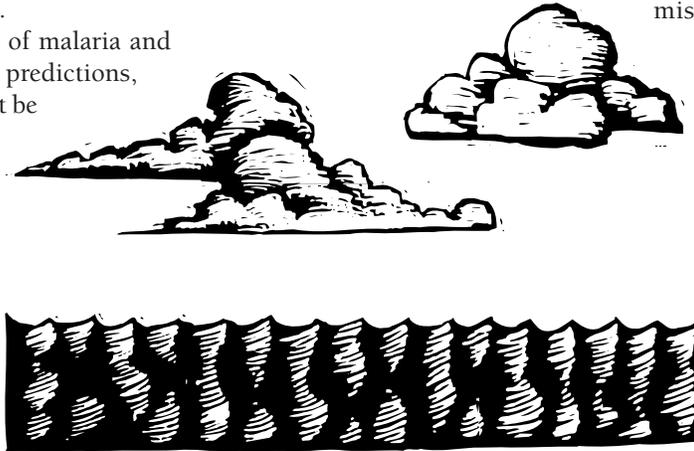
The increased climate variability accompanying warming will probably be more important than the rising heat itself in fueling unwelcome outbreaks of certain vector-borne illnesses. For instance, warm winters followed by hot, dry summers (a pattern that could become all too familiar as the atmosphere heats up) favor the transmission of St. Louis encephalitis and other infections that cycle among birds, urban mosquitoes and humans.

This sequence seems to have abetted the surprise emergence of the West Nile virus in New York City last year. No one knows how this virus found its way into the U.S. But one reasonable explanation for its persistence and amplification here centers on the weather’s effects on *Culex pipiens* mosquitoes, which

accounted for the bulk of the transmission. These urban dwellers typically lay their eggs in damp basements, gutters, sewers and polluted pools of water.

The interaction between the weather, the mosquitoes and the virus probably went something like this: The mild winter of 1998–99 enabled many of the mosquitoes to survive into the spring, which arrived early. Drought in spring and summer concentrated nourishing organic matter in their breeding areas and simultaneously killed off mosquito predators, such as lacewings and ladybugs, that would otherwise have helped limit mosquito populations. Drought would also have led birds to congregate more, as they shared fewer and smaller watering holes, many of which were frequented, naturally, by mosquitoes.

Once mosquitoes acquired the virus, the heat wave that accompanied the drought would speed up viral maturation inside the insects. Consequently, as infected mosquitoes sought blood meals, they could spread the virus to birds at a rapid clip. As bird after bird became infected, so did more mosquitoes, which ultimately fanned out to infect human beings. Torrential rains toward the end of August provided new puddles for the breeding of *C. pipiens* and other mosquitoes, unleashing an added crop of potential virus carriers.



Dengue, or “breakbone,” fever (a severe flulike viral illness that sometimes causes fatal internal bleeding) is spreading as well... Neither a vaccine nor a specific drug treatment is yet available.

Like mosquitoes, other disease-conveying vectors tend to be “pests”—opportunists that reproduce quickly and thrive under disturbed conditions unfavorable to species with more specialized needs. In the 1990s climate variability contributed to the appearance in humans of a new rodent-borne ailment: the hantavirus pulmonary syndrome, a highly lethal infection of the lungs. This infection can jump from animals to humans when people inhale viral particles hiding in the secretions and excretions of rodents. The sequential weather extremes that set the stage for the first human eruption, in the U.S. Southwest in 1993, were long-lasting drought interrupted by intense rains.

First, a regional drought helped to reduce the pool of animals that prey on rodents—raptors (owls, eagles, prairie falcons, red-tailed hawks and kestrels), coyotes and snakes. Then, as drought yielded to unusually heavy rains early in 1993, the rodents found a bounty of food, in the form of grasshoppers and piñon nuts. The resulting population explosion enabled a virus that had been either inactive or isolated in a small group to take hold in many rodents. When drought returned in summer, the animals sought food in human dwellings and brought the disease to people. By fall 1993, rodent numbers had fallen, and the outbreak abated.

Subsequent episodes of hantavirus pulmonary syndrome in the U.S. have been limited, in part because early-warning systems now indicate when rodent-control efforts have to be stepped up and because people have learned to be more careful about avoiding the animals’ droppings. But the disease has appeared in Latin America, where some ominous evidence suggests that it may be passed from one person to another.

As the natural ending of the first hantavirus episode demonstrates, ecosystems can usually survive occasional extremes. They are even strengthened by seasonal changes in weather conditions, because the species that live in changeable climates have to evolve an ability to cope with a broad range of conditions. But long-lasting extremes and very wide fluctuations in weather can overwhelm ecosystem resilience. (Persistent ocean heating, for instance, is menacing coral reef systems, and drought-driven forest fires are threatening forest habitats.) And ecosystem upheaval is one of the most profound ways in which climate change can affect human health. Pest control is one of nature’s underappreciated services to people; well-functioning ecosys-

tems that include diverse species help to keep nuisance organisms in check. If increased warming and weather extremes result in more ecosystem disturbance, that disruption may foster the growth of opportunist populations and enhance the spread of disease.

Unhealthy Water

Beyond exacerbating the vector-borne illnesses mentioned above, global warming will probably elevate the incidence of waterborne diseases, including cholera (a cause of severe diarrhea). Warming itself can contribute to the change, as can a heightened frequency and extent of droughts and floods. It may seem strange

that droughts would favor waterborne disease, but they can wipe out supplies of safe drinking water and concentrate contaminants that might otherwise remain dilute. Further, the lack of clean water during a drought interferes with good hygiene and safe rehydration of those who have lost large



amounts of water because of diarrhea or fever.

Floods favor waterborne ills in different ways. They wash sewage and other sources of pathogens (such as *Cryptosporidium*) into supplies of drinking water. They also flush fertilizer into water supplies. Fertilizer and sewage can each combine with warmed water to trigger expansive blooms of harmful algae. Some of these blooms are directly toxic to humans who inhale their vapors; others contaminate fish and shellfish, which, when eaten, sicken the consumers. Recent discoveries have revealed that algal blooms can threaten human health in yet another way. As they grow bigger, they support the proliferation of various pathogens,

among them *Vibrio cholerae*, the causative agent of cholera.

Drenching rains brought by a warmed Indian Ocean to the Horn of Africa in 1997 and 1998 offer an example of how people will be affected as global warming spawns added flooding. The downpours set off epidemics of cholera as well as two mosquito-

borne infections: malaria and Rift Valley fever (a flulike disease that can be lethal to livestock and people alike).

To the west, Hurricane Mitch stalled over Central America in October 1998 for three days. Fueled by a heated Caribbean, the storm unleashed torrents that killed at least 11,000 people. But that was only the beginning of its havoc. In the aftermath, Honduras reported thousands of cases of cholera, malaria and dengue fever. Beginning in February of this year, unprecedented rains and a series of cyclones inundated large

And ecosystem upheaval is one of the most profound ways in which climate change can affect human health.

parts of southern Africa. Floods in Mozambique and Madagascar killed hundreds, displaced thousands and spread both cholera and malaria. Such events can also greatly retard economic development, and its accompanying public health benefits, in affected areas for years.

Solutions

The health toll taken by global warming will depend to a large extent on the steps taken to prepare for the dangers. The ideal defensive strategy would have multiple components.

One would include improved surveillance systems that would promptly spot the emergence or resurgence of infectious diseases or the vectors that carry them. Discovery could quickly trigger measures to control vector proliferation without harming the environment, to advise the public about self-protection, to provide vaccines (when available) for at-risk populations and to deliver prompt treatments.

This past spring, efforts to limit the West Nile virus in the northeastern U.S. followed this model. On seeing that the virus had survived the winter, public health officials warned people to clear their yards of receptacles that can hold stagnant water favorable to mosquito breeding. They also introduced fish that eat mosquito larvae into catch basins and put insecticide pellets into sewers.

Sadly, however, comprehensive surveillance plans are not yet realistic in much of the world. And even when vaccines or effective treatments exist, many regions have no means of obtaining and distributing them. Providing these preventive measures and treatments should be a global priority.

A second component would focus on predicting when climatological and other environmental conditions could become conducive to disease outbreaks, so that the risks could be minimized. If climate models indicate that floods are likely in a given region, officials might stock shelters with extra supplies. Or if satellite images and sampling of coastal waters indicate that algal blooms related to cholera outbreaks are beginning, officials could warn people to filter contaminated water and could advise medical facilities to arrange for additional staff, beds and treatment supplies.

Research reported in 1999 illustrates the benefits of satellite monitoring. It showed that satellite images detecting heated water in two specific ocean regions and lush vegetation in the Horn of Africa can predict outbreaks of Rift Valley fever in the Horn five months in advance. If such assessments led to vaccination campaigns in animals, they could

potentially forestall epidemics in both livestock and people.

A third component of the strategy would attack global warming itself. Human activities that contribute to the heating or that exacerbate its effects must be limited. Little doubt remains that burning fossil fuels for energy is playing a significant role in global warming, by spewing carbon dioxide and other heat-absorbing, or "greenhouse," gases into the air. Cleaner energy sources must be put to use quickly and broadly, both in the energy-guzzling industrial world and in developing nations, which cannot be expected to cut back on their energy use. (Providing sanitation, housing, food, refrigeration and indoor fires for cooking takes energy, as do the pumping and purification of water and the desalination of seawater for irrigation.) In parallel, forests and wetlands need to be restored, to absorb carbon dioxide and floodwaters and to filter contaminants before they reach water supplies.

The world's leaders, if they are wise, will make it their business to find a way to pay for these solutions. Climate, ecological systems and society can all recoup after stress, but only if they are not exposed to prolonged challenge or to one disruption after another. The Intergovernmental Panel on Climate Change, established by the United Nations, calculates that halting the ongoing rise in atmospheric concentrations of greenhouse gases will require a whopping 60 to 70 percent reduction in emissions.

I worry that effective corrective measures will not be instituted soon enough. Climate does not necessarily change gradually. The multiple factors that are now destabilizing the global climate system could cause it to jump abruptly out of its current state. At any time, the world could suddenly become much hotter or even much colder. Such a sudden, catastrophic change is the ultimate health risk—one that must be avoided at all costs.



**Climate, ecological systems and society
can all recoup after stress, but only if they
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to one disruption after another.**

The Author

PAUL R. EPSTEIN, an M.D. trained in tropical public health, is associate director of the Center for Health and the Global Environment at Harvard Medical School. He has served in medical, teaching and research capacities in Africa, Asia and Latin America and has worked with the Intergovernmental Panel on Climate Change, the National Oceanic and Atmospheric Administration, and the National Aeronautics and Space Administration to assess the health effects of climate change and to develop health applications for climate forecasting and remote-sensing technologies.

A Review of *Making Better Environmental Decisions*

Mary O'Brien (MIT Press, Cambridge, MA, 2000)

by Terry Shistar, Ph.D.

For five years, I have taught a seminar on risk assessment at the University of Kansas without a textbook. None of the available options met my needs—until now, with the publication of Mary O'Brien's *Making Better Environmental Decisions—An Alternative to Risk Assessment*.

Making Better Environmental Decisions is to environmental policy what *Getting to Yes* is to negotiation. That is, it teaches not only the flaws of risk assessment, but also—more importantly—how to turn the risk assessment setting into an opportunity to push for something better, alternatives assessment.

Since risk assessment sometimes masquerades as a consideration of alternatives, it is a good idea to have some general rules for determining which is which. Here are three:

- A risk assessment estimates the risks associated with a single course of action, or a tightly constrained selection of options, while an alternatives assessment compares a wide range of different alternatives for achieving a desired end.
- In a risk assessment, if you can't estimate a certain kind of risk, it is assumed to be zero. In an alternatives assessment, a choice with a risk of unknown proportions is considered dangerous compared to alternatives without that risk.
- The question a risk assessment seeks to answer is, "How much is safe (or, at least, acceptable)?" This question is unanswerable since we can never measure all the risks. An alternatives assessment doesn't try to find a safe amount of exposure to a danger. Instead, it provides information that allows us to choose among alternatives based on the hazards they pose as well as their relative benefits.

I don't want to give the impression that the value of *Making Better Environmental Decisions* is only (or even mostly) as a textbook. This is a book I would like to see in the hands of every activist who has ever faced a pesticide risk assessment, a water quality standards guidance document, or an environmental impact statement. When faced with a flood of data and formulas, our immediate reaction is often to plunge in and find hidden assumptions, missing data, and ignored risks. Guess what? As soon as we do that, we're giving up on our game and agreeing to play *their* game.

As long as unknown risks are counted as zero, we're looking for "safe" exposure levels, and we've quit demanding that

non-toxic alternatives be considered, we lose. We know that alternatives work. We need to change the game by demanding that they be considered. This, by the way, is what Beyond Pesticides/NCAMP has been doing for years, and *Making Better Environmental Decisions* can help us do it better.

My favorite chapter is titled "We Know How to Push for Alternatives Assessments." This chapter gives several examples of how citizens have been able to turn situations in which risk assessments were being used to justify environmental damage into opportunities to force consideration of alternatives. For example, the book cites some victories of Greenpeace:

Greenpeace has followed through on its commitments to clean production and the precautionary principle in its various campaigns, thereby changing the nature of each debate. For instance, Greenpeace has:

- helped develop the precautionary principle-based UN resolution to ban drift-net fishing on the high seas ...
- worked with African governments to produce the Bamako Convention, which prohibits imports of banned or unregistered pesticides from outside of Africa and greatly restricts imports of hazardous wastes into Africa...
- worked with the International Joint Commission on Great Lakes Water Quality to call for the phaseout of industrial uses of chlorine...
- worked with German and British refrigeration engineers to develop butane- and propane-based refrigerators that avoid the use of hydrochlorofluorocarbons and hydrofluorocarbons, which are ozone-depleting substitutes for the more potent ozone-depleting chlorofluorocarbons...

Greenpeace opposes risk assessment of dangerous options, and works to bring alternatives to the table.

Making Better Environmental Decisions is available at bookstores in hardback (\$55.00) and paperback (\$22.95) editions. Get yourself a copy and give one to a friend. This book is also available through the Beyond Pesticides/NCAMP website (www.beyondpesticides.org) where, for no additional cost, your purchase triggers a donation to our organization.

Terry Shistar, Ph.D., teaches a class on risk assessment at the University of Kansas in Lawrence, KS and is Secretary of the Board of Beyond Pesticides/NCAMP.



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While living in the United States from 1977 to 1986, she founded the Physicians for Social Responsibility, an organization of 23,000 doctors with a strong record of working to prevent environmental illness. On trips abroad, she helped start similar organizations in many other countries. The international umbrella group, International Physicians for the Prevention of Nuclear War, won the Nobel Peace Prize in 1985. She also founded the Women's

Action for Nuclear Disarmament (WAND) in the U.S. in 1980.

Dr. Caldicott has received many prizes and awards for her work, including 17 honorary degrees, and was personally nominated for the Nobel Peace Prize by Linus Pauling—himself a Nobel Laureate. She has written for numerous publications and has authored several books, *Nuclear Madness* (1979), *If You Love This Planet: A Plan to Heal the Earth* (1992) and *A Desperate Passion: An Autobiography* (1996). She also has been the subject of several films, *Eight Minutes to Midnight*, nominated for an Academy Award in 1982, and *If You Love This Planet*, which won the Academy Award for best documentary in 1983.

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