OTHER CHEM
Piperonyl Butoxide

chemicalWATCH

Mevinphos, *continued from page 5*
mevinphos, including the potential for groundwater contamination, mevinphos' persistence in the environment, and the need for crop rotational label restrictions." Despite an expressed concern for the significant number of mevinphos poisonings, EPA "is reserving consideration of special review for mevinphos until data become available that more clearly identify the risks posed by mevinphos use."

Sources:

1. Thomson, W.T. (1984). *Agricultural Chemicals: Insecticides.* Thomson Publications, Fresno, CA.

2. U.S. EPA. (1988). *Guidance for the reregistration of pesticide products containing mevinphos as the active ingredient.* Office of Pesticides Programs. Washington, DC.

Piperonyl Butoxide

Some pesticide formulations contain pesticide synergists, in addition to the active ingredients and inerts. Synergists are chemical agents included to enhance the killing power of the active ingredients. Examples include piperonyl butoxide and N-Octyl bicycloheptene dicarboximide (MGK 264). A range of products from repellents to foggers to pediculicides (lice killers) to garden sprays contain these commonly used synergists. They appear to increase the potency of pyrethrin, pyrethroids, rotenone, carbamates, and occasionally other classes of insecticides. Formulations generally contain 5-20 times more synergist than active ingredient.

Piperonyl butoxide inhibits important liver enzymes responsible for breakdown of some toxins, including pesticidal active ingredients. Specifically, it has been shown to inhibit hepatic microsomal oxidase enzymes in laboratory rodents and by inference in humans. A related group of enzymes is inhibited in insects. Because these enzymes act to detoxify many drugs and other chemicals, a heavy exposure to an insecticidal synergist may make a person temporarily vulnerable to a variety of toxic insults that would normally be easily tolerated.

The acute toxicity of piperonyl butoxide is low, far lower than the active ingredients it accompanies. The acute oral LD_{50} for rats is 11.5 g/kg and dermal absorption is limited. Poisoning symptoms include anorexia, vomiting, diarrhea, intestinal inflammation, pulmonary hemorrhage, and perhaps mild central nervous system depression. Repeated contact may cause slight skin irritation.

Rat metabolism studies submitted to EPA and considered supplementary, demonstrate that piperonyl butoxide is slowly absorbed from the gastrointestinal tract, with blood levels peaking after 4-6 hours. Excretion occurs via the urine and feces, peaking after 12-24 hours and 24-48 hours, respectively. No evidence of specific tissue retention is described, and additional testing to determine the structure of metabolites is required.

EPA's chronic toxicity database is incomplete. A single teratology (birth defects) study was submitted by IBT laboratories and is considered invalid. Mutagenicity testing indicates piperonyl butoxide is not mutagenic with the exception of one study which yielded "equivocal" results. A rat reproduction study noted no effect on reproductive performance or pup development, however decreases in weight gain were observed at the highest dose level. A rat chronic feeding/cancer study was negative for tumor formation, however, a variety of liver effects were observed. At the lowest dose level, 30 mg/kg/day, liver weights were increased. At the highest dose level, 500 mg/kg/day, there were increased liver weights and liver cell enlargement in males and females. Increased hepatic "focal mixed cells" and increased serum cholesterol were observed in females. In addition, increases in "pigment in follicles" and abnormal overgrowth of follicular cells of the thyroid were noted in both sexes.

Sources:

Gosselin, et al. 1984. *Clinical Toxicology of Commercial Products,* Williams & Wilkins, Baltimore, MD

Morgan, D.P. 1982. Recognition and Management of Pesticide Poisonings, 4th Edition. U.s. Environmental Protection Agency, Washington, DC.

U.S.-EPA. 1988 Memorandum from John Doherty to Phil Hutton Re: Piperonyl Butoxide chronic feeding/oncogenicity study. April 28, 1988. Office of Pesticides and Toxic Substances (OPTS), Washington, DC.

U.S.-EPA. 1988. Memorandum from John Doherty to Geraldine Werdig Re: Piperonyl Butoxide metabolism and pharmacokinetic studies. February 25, 1988. OPTS. Washington, DC.

U.S.-EPA. 1987. Memorandum from John Doherty to Joseph Tavano Re: Piperonyl Butoxide rat multi-generation reproduction study. October 30, 1987. OPTS. Washington, DC.

U.S.-EPA. 1985. Memorandum from Irving Mauer to T. Gardner/P. Schroeder Re: Piperonyl Butoxide mutagenicity study. June 27, 1985. OPTS. Washington, DC.

U.S.-EPA. 1988. Toxicology One-Liner. Office of Pesticide Programs. Washington, DC.

chemBRIEFS

Mercury in Paint, Partial Action

EPA announced the cancellation of mercury for use in indoor latex paints. The June 29, 1990 action followed a case of acrodynia, a severe form of mercury poisoning, in a MI child whose home was painted in 1989 with paint containing 930ppm mercury, three times the legal level of 300ppm. In response, the federal Centers for Disease Control, MI Depts. of Agriculture and Health, and NCAMP called for a ban on mercury fungicides in paint (PAY, June 1990). Following negotiations between EPA and mercury registrants, it was agreed that as of August 20, 1990, manufacturing interior paint containing mercury will be unlawful, although **all** stocks may be sold. By July 23, 1990, the mercury registrants will label products for outdoor use only and deliver similar labels to paint manufacturers. NCAMP Staff Toxicologist Catherine Karr noted that the "action would reduce an important component of public exposure to hazardous levels of mercury. However, in light of widely available alternatives, allowing continued public exposure and environmental degradation from mercury use in exterior paint is imprudent and inappropriate." EPA is requiring additional data to assess the risk to painters and residents from exterior paint containing mercury.