ChemicalWatch Factsheet

A Beyond Pesticides/ NCAMP Factsheet

Aldicarb

First registered in 1970 for use on cotton by Union Carbide, which has since sold its interests to RhonePoulenc, aldicarb is now registered for use on a number of crops including citrus, potatoes and peanuts. Over 5 million pounds are used annually in the U.S. to battle insects, mites and nematodes.

Aldicarb (Temik™) is both highly acutely toxic and readily leachable. Widespread public concern about aldicarb began in July, 1985 with an epidemic of poisoning that affected approximately 1000 people who had eaten aldicarb tainted watermelon. To date, aldicarb has been detected in thousands of wells, and it is this potential for groundwater contamination that has led to recent state and federal regulation of aldicarb use.

Like other carbamate pesticides, aldicarb, and its metabolic products aldicarb sulfoxide and aldicarb sulfone, reversibly inhibit cholinesterase (ChE), an important nervous system enzyme. The rat oral LD_{50}=0.9mg/kg, the dose required to kill half the test population, making it one of the most acutely toxic pesticides registered. Dermal and inhalation routes are also highly toxic.

A wide range of symptoms may result from cholinesterase inhibition including gastrointestinal disturbances, unconsciousness, blurred vision, excessive salivation, seizures, and disorientation. Extensive ChE inhibition may result in death.

Anecdotal reports have linked symptoms with a range of calculated doses as low as 0.0026 mg/kg. This both suggests a range of sensitivity to aldicarb's acute effects and indicates that the difference between a dose causing no or mild clinical signs and one causing severe symptomatic response may be extremely small in some cases.

***chemicalWATCH* Stats:**

Chemical Class: Carbamate insecticide
Use: Agriculture
Toxicity Rating: Toxic
Signal Word: Danger
Health Effects: Suspected endocrine disruptor, linked to neurotoxic and reproductive effects, asthma, and learning behavior problems.
Environmental Effects: Detected in groundwater, potential leacher, toxic to birds and fish/aquatic organisms.

In its Special Review process, initiated in 1986, EPA maintained that aldicarb does not cause any chronic injury in laboratory animals, including immunologic, oncogenic, teratogenic, reproductive, delayed neurotoxic, or mutagenic effects, and concluded that the only reliably demonstrated toxic effect of aldicarb is acute cholinesterase inhibition. However, Fiore et a1. described immune suppression among Wisconsin women exposed to aldicarb in drinking water. Although independent reviewers have called the study "tantalizing enough to warrant some follow-up immune studies," EPA notes that the biological significance of the irnmunological effects observed is yet to be determined.

Revised EPA risk estimates for aldicarb consumption in food and water, based on field residue data and subpopulation consumption patterns, indicate that children and infants are most likely to be at risk. As many as 55% of these subpopulations are exposed to dietary levels greater than 0.001 mg/kg, the Reference Dose below which clinical signs or depressed ChE activity is unlikely. Indeed EPA estimated that when drinking water is at the current health advisory level of 10 ppb, 13% of consuming infants could be exposed to doses greater than or equal to the reference dose.

So far, aldicarb has been detected in the groundwater of 48 counties in 16 states at levels ranging from 1 to over 500 ppb. Levels exceeding the 10 ppb health advisory level were found in 25 of those counties

in some 11 states. EPA responded to this widespread contamination by instituting use restriction measures which it claims are sufficient to substantially reduce risks. The label measures include prohibition of use within 300 feet of any drinking water well, and monitoring requirements for areas assessed to have medium leaching potential. In addition, approved state management plans will be required for aldicarb use in areas considered highly vulnerable to ground water contamination.

Wildlife is also affected by aldicarb exposure. It is highly toxic to mammals, birds, estuarine/marine organisms, and freshwater organisms. Aldicarb has been found to pose a threat to the endangered Attwater's Greater Prairie Chicken, and use is prohibited if this species is located in or adjacent to the treatment area.

Reprinted from Pesticides and You, Vol. 9, No. 5, December 1989

Update, *March 2007*:

A 1999 animal study by Jaeger et al. found that mixtures of aldicarb, atrazine,and nitrate at concentrations allowable by EPA are capable of altering immune, endocrine, and nervous system functions in mice.

In March 2003, the European Union signed an agreement to phase out use of aldicarb. Eight EU member states will continue to use aldicarb through 2007. U.S. EPA has scheduled an interim reregistration eligibility decision for March 2007.

Aldicarb *chemicalWATCH* Factsheet Bibliography

Fiore, M. et al. 1986. Chronic exposure to aldicarb contaminated groundwater and human immune function. Environmental Research, December.

Jaeger, JW, IH Carlson, and WP Porter. 1999. Endocrine, immune, and behavioral effects of aldicarb(carbamate), atrazine (triazine) and nitrate (fertilizer) mixtures at groundwater concentrations. Toxicology and Industrial Health 15:133-151.

Morbidity and Mortality Weekly Reports: Aldicarb food poisoning from contaminated melons-California. Journal of the American Medical Association 256(2):175-176.

Pesticide and Toxic Chemical News. 1986. Aldicarb human immune suppression effect doubted by experts. November 19.

U.S. EPA. 1988. Aldicarb Special Review Technical Support Document. Office of Pesticide Programs. Washington, DC.

U.S. EPA. 1988. Preliminary determination to cancel registrations of aldicarb products and availability of technical support document, Federal Register, 53:24630, June 29.

BEYOND PESTICIDES
701 E Street, S.E., Suite 200, Washington DC 20003
202-543-5450 (v) • 202-543-4791 (f)
info@beyondpesticides.org • www.beyondpesticides.org