chemicalWATCH Factsheet

CARBARYL

Regulatory History
Carbaryl (Sevin™) is said by EPA to be " one of the most widely applied insecticides in the US," since use began in 1959, with 10-15 million pounds used annually. This widespread use is a major reason why carbaryl may pose more dangers than most other pesticides. Manufactured by Union Carbide, this broad-spectrum insecticide is used on a variety of crops, in forestry and on ornamentals, in home gardens, and on livestock and pets. One manufacturing method uses methyl isocyanate (MIC), the agent responsible for thousands of deaths in Bhopal, India when a Union Carbide plant leaked the deadly gas.

In 1980, five years after the special review process was initiated due to concerns regarding teratogenicity, fetotoxicity, mutagenicity, oncogenicity, neurotoxicity, and viral enhancement, the review was abruptly withdrawn. Criticizing the action, Janette Sherman, M.D., then a member of EPA's Advisory Committee on Toxic Substances, commented that, "it was a political and economic decision." In April 1991, during an assessment of data needs for the reevaluation process (reregistration), EPA concluded that there were serious data gaps in all disciplinary areas, including chronic toxicity studies, carcinogenicity studies in rats and mice and a teratogenicity study in dogs. Nonetheless, there is an extensive body of literature on the toxicity of carbaryl.

Carbaryl, a carbamate, is a contact nerve toxin that inhibits the enzyme cholinesterase, with a resulting disruption of nerve impulse transmissions.

Acute Toxicity
The routine use of insecticides, such as carbaryl, in the home where they contaminate indoor air and do not readily dissipate, exposes a substantial proportion of the population, particularly the young and the elderly, to considerable risk. Carbaryl is considered very toxic, with a rat oral LD50 (dose needed to kill 50% of the test population) of 225 mg/kg. The World Health Organization has established an acceptable daily intake (ADI) of 0.01 mg/kg/day, while EPA's ADI is 0.1 mg/kg/day. Carbaryl is readily absorbed through the skin, with almost total absorption through the forearm and scrotum. A skin sensitizer in guinea pigs, it causes allergic dermatitis in dogs, and dermatitis and acute irritations in humans. Acute signs and symptoms of carbaryl poisoning include blurred vision, nausea, headache, salivation, breathing difficulties, muscle twitching, and ataxia.

Carbaryl causes an array of serious neurotoxic effects in animals, including irreversible neurological damage and behavioral disturbances. Humans and cats develop aggressive behavior with irritability, paranoia and physical assaults, while learning and response rates are slowed in rats given small doses. Physicians may often fail to diagnose carbaryl poisoning. In one case, despite the "opinions of an internist, cardiologist, neurologist and orthopedist, it was an observation by the patient's wife that established the diagnosis of carbaryl poisoning.

Chronic Toxicity
As long ago as 1969, a Health Education and Welfare Department (HEW) report recommended that the use of carbaryl "should be immediately restricted to prevent risk of human exposure," because low doses cause birth defects when given to pregnant dogs. So far, the HEW warning has been ignored. Carbaryl is also a mutagen, toxic to the kidney and liver, damages ovaries and testes, and causes behavioral problems in humans and animals. Children, women, pregnant women, older persons, and those whose health is compromised are particularly susceptible to carbaryl's effects.

In 1975, EPA concluded that "carbaryl has been shown to produce birth defects in guinea pigs, rabbits and dogs; and fetotoxicity in mice, rats and gerbils." Defects included lack of intestines and failure to form skeletal tissues. EPA felt that carbaryl was not a potent teratogen because the effects generally occurred at levels that were toxic to the pregnant animal. Carbaryl causes abortions in monkeys. It causes severe effects on the formation of sperm in male rats and on the ovary of female rats. Sperm abnormalities have been described in carbaryl production workers as well. According to EPA, "The results of cytogenetic tests imply that carbaryl may induce chromosomal effects in mammalian cells in culture, in whole animals, and in plants, and

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carbaryl has been shown to cause primary DNA damage in cultured human cells." Despite this and the human evidence of spermatogenic effects, the agency concludes that it "probably only acts as a weak mutagen." EPA's Scientific Advisory Panel recommended that carbaryl carry a warning regarding the hazard to pregnant women. However, only a warning not to use carbaryl on pregnant dogs appears on the label.

For years, EPA accepted invalid rat and mice carcinogenicity studies as showing that carbaryl was not carcinogenic. The agency stated that although individually the studies were unacceptable, collectively they provided sufficient evidence of non-carcinogenicity. In 1991, EPA reversed itself and issued a data call-in on both carcinogenicity studies. The breakdown product nitrosocarbaryl, formed in the stomach, is highly mutagenic and carcinogenic.

Ecological Effects

Carbaryl is highly toxic to bees, killing more bees in California than any other pesticide. It is highly toxic to aquatic invertebrates, LC_{50} is 6.4 ppb (parts per billion) for Daphnia (the common water flea), and its breakdown product, alpha-naphthol, is quite toxic to mollusks and other estuarine organisms. Moderately toxic to fish (LC_{50} is 1.95-6.76 ppm), it may also bioaccumulate. Carbaryl acts synergistically to increase the toxicity of other pesticides such as 2,4-D, rotenone or pentachlorophenol in trout.

Carbaryl *chemicalWATCH* Factsheet Bibliography

Anger, W.K. 1981. "Effects of carbaryl on variable interval response rates in rats." *Neurobehavioral Toxicology* 2:21-24.

Bear, D. 1986. "Aggression in cat and human precipitated by a cholinesterase inhibitor." *Psychosomatics* 27:535-536.

Branch, R.A. 1986. "Is carbaryl as safe as its reputation? Does it have a potential for causing chronic neurotoxicity in humans?" *American Journal of Medicine* 80:659-664.

Carpenter, C.P. et al. 1961. "Mammalian toxicity of 1-naphthyl-N-methyl carbamate (Sevin insecticide)." *Journal of Agriculture Food* Chemistry 9:30-39.

Dougherty, W.J. 1971. "The effect of carbaryl on reproduction in the monkey." *Toxicology Applications in Pharmacology* 19:365.

Ellenhorn, M.J. et al. 1988. "Medical toxicology: diagnosis and treatment of human poisoning." Elsevier Science Publishing Co., Inc. New York.

Extension Toxicology Network (EXTOXNET) Pesticide Information Profiles. 2001. "Carbaryl." <http://pmep.cce.cornell.edu/profiles/extoxnet/carbaryl-dicrotophos/carbaryl-ext.html>.

Lijinsky, W. 1978. "Carcinogenesis by nitroso derivatives of methylcarbamate insecticides and other nitrosamides in rats and mice. In: Environmental aspects of N-nitroso compounds." IARC Scientific Publishing (19).

Shtenberg, A.I. et al. 1971. *Vopr. Pitan.* 30:42-49.

Singh, J.M. 1973. "Decreased performance behavior with carbaryl-an indication of clinical toxicity." *Clinical Toxicology* 6:97-108.

Smalley, H.E. et al. 1968. "Teratogenic action of carbaryl in Beagle dogs." *Toxicological Applications in Pharmacology* 13:392-403.

Smalley, H.E. et al. 1969. "The effects of chronic carbaryl administration on the neuromuscular system of swine." *Toxicological Applications in Pharmacology* 14:490-494.

Statham, C.N. et al. 1975. "Potentiation of the acute toxicity of several pesticides and herbicides in trout by carbaryl." *Toxicological Applications in Pharmacology* 34:83-87.

Wills, J.H. et al. 1968. "Effects of oral doses of carbaryl on man." *Clinical Toxicology* 1:265-271.

Wyrobek, A.J. et al. 1981. *Environmental Health Perspective* 40:255-265.

U.S. Department of Health Education and Welfare. 1969. "Report of the secretary's commission on pesticides and their relationship to environmental health." Washington D.C.

U.S. EPA. 1991. "Reregistration Eligibility Document: carbaryl." Office of Pesticide Programs. Washington D.C. <http://www.epa.gov/oppsrrd1/REDs/>.

U.S. EPA. 1991. "Data call-in chemical status sheet: carbaryl."

U.S. EPA. 1991. "Personal communication with OPP/RD product manager."

U.S. EPA. 1984. "Chemical Factsheet for carbaryl." Office of Pesticide Programs. Washington D.C.

U.S. EPA. 1983. "Guidance for the reregistration of manufacturing-use and certain end-use pesticide products containing carbaryl." Office of Pesticide Programs. Washington D.C.

U.S. EPA. 1980. "Determination not to initiate a rebuttable presumption against registration of pesticide products containing carbaryl." 45 FR 81869-81876.

U.S. EPA. 1975. "Pesticide Fact Sheet: carbaryl." Washington D.C.

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