

chemicalWATCH Factsheet

CHLORPYRIFOS

On June 8, 2000, the U.S. Environmental Protection Agency (EPA) and Dow AgroSciences, reached an agreement to stop the sale of most home, lawn and garden uses for chlorpyrifos because of its health risks to children.

What is Chlorpyrifos?

Chlorpyrifos (trade names include Dursban™ and Lorsban™), one of the most widely used insecticides in the U.S. with 20 to 24 million pounds applied annually, has been linked to thousands of pesticide poisoning incidents. This Dow AgroSciences, previously DowElanco, product is a broad-spectrum chlorinated organophosphate insecticide.

Chlorpyrifos is registered for the control of cutworms, corn rootworms, cockroaches, grubs, flea beetles, flies, termites, fire ants, mosquitoes, and lice. It is used as an insecticide on grain, cotton, fruit, nut, and vegetable crops, as well as on lawns and ornamental plants. It is also registered for direct use on sheep and turkeys, for horse site treatment, dog kennels, domestic dwellings, farm buildings, storage bins, and commercial establishments.

Chlorpyrifos Toxicity and Health Effects

Chlorpyrifos is acutely toxic to bees, birds, mammals, aquatic life, and certain species of algae, the rat LD₅₀ is 135 mg/kg.

Poisoning from chlorpyrifos may affect the central nervous system, the cardiovascular system, and the respiratory system as well as a skin and eye irritant. Acute exposure can result in such symptoms as numbness, tingling sensation, incoordination, dizziness, vomiting, sweating, nausea, stomach cramps, headache, vision disturbances, muscle twitching, drowsiness, anxiety, slurred speech, depression, confusion and in extreme cases, respiratory arrest, unconsciousness, convulsions, and death. Persons with respiratory ailments, recent exposure to cholinesterase inhibitors, cholinesterase impairment, or liver malfunction are at increased risk from exposure to chlorpyrifos. Chlorpyrifos has also been linked to Multiple Chemical Sensitivity (MCS).

In animals, chlorpyrifos transforms to chlorpyrifos-oxon, which is about 3000 times as potent against the nervous system as chlorpyrifos itself.

Chlorpyrifos is linked to delayed peripheral neuropathy (degenerative lesions of sensory, motor, or reflex nerves). Italian researchers published a disturbing report of an acute chlorpyrifos poisoning episode, resulting in delayed peripheral neuropathy. There are also reports of EEG (brainwave) pattern, sleep pattern and behavioral changes lasting over a year following exposure to organophosphate insecticides.

Organophosphates are cholinesterase inhibitors. Organophosphates bind irreversibly to the active site of an essential enzyme for normal nerve impulse transmission, acetylcholine esterase (AChE), inactivating the enzyme. A common diagnostic for poisoning is to assay for blood AChE depression. It can take many weeks for new enzyme to be resynthesized. Repeated or prolonged exposure to organophosphates may result in the same effects as acute exposure including delayed symptoms.

A 1996 study of children exposed to chlorpyrifos in utero found that extensive and unusual patterns of birth defects, including brain, nervous system, eyes, ears, palate, teeth, heart, feet, nipples, and genitalia. Published literature and EPA documents contain reports that identify similarities in defects found in test animals and children exposed to chlorpyrifos.

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.

A U.S. News & World Report investigation, "The stuff in the backyard shed," (November 8, 1999, page 64-68) reports that since 1992, Dow AgroSciences and predecessor manufacturers have sent approximately 7,000 reports of chlorpyrifos-induced reactions to EPA. The agency, according to the report, suspects chlorpyrifos in 17,771 incidents reported to the U.S. Poison Control Centers between 1993 -96.

In 1999, EPA's Office Pesticide Programs, Health Effects Division, reported that four pesticides, phosmet, proetamphos, chlorpyrifos, and dimethoate, had consistently high rankings in being responsible for symptoms, health care facility visits, hospitalizations, and fatal outcomes in adults and children. These four organophosphate pesticides are responsible for 90% of pesticide exposures reported in children under six to the Poison Control Centers around the country from the 1993-1996. The report also stated that "children, under six exposed to organophosphates, were three times more likely to be hospitalized, five times more likely to be admitted for critical care, and four times more likely to have experienced a major medical outcome or death, than if exposed to some other, non organophosphate, pesticide."

There are also a wide range of adverse environmental effects linked to chlorpyrifos, include toxicity to: beneficial insects, freshwater fish, other aquatic organisms, bird, a variety of plants, soil organisms, and domestic animals. It has been shown to bioaccumulate in fish and synergistically react with other chemicals. Chlorpyrifos may be toxic to some plants, such as lettuce. Residues remain on plant surfaces for approximately 10 to 14 days. Data indicate that this insecticide and its soil metabolites can accumulate in certain crops.

Chlorpyrifos Residues / Persistence

There are few data available on air levels or surface residues following application either as a termiticide or for indoor pest control. The American Conference of Governmental Industrial Hygienists recommends an occupation air level guideline of 200 micrograms/cubic meter ($\mu\text{g}/\text{m}^3$) for a forty hour work week. The National Academy of Sciences proposed a $10\mu\text{g}/\text{m}^3$ air level for the general public, while EPA has proposed an air limit of $0.49\mu\text{g}/\text{m}^3$ for children, and $1\mu\text{g}/\text{m}^3$ for adult exposures.

Work by Fenske *et al.* found that air levels 24 hours after a proper application were as high as $30\mu\text{g}/\text{m}^3$ in the infant breathing zone, 60 times EPA's limit. Furthermore, Fenske calculated that

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701 E Street, S.E., Suite 200, Washington DC 20003

202-543-5450 (v) • 202-543-4791 (f)

info@beyondpesticides.org • www.beyondpesticides.org

infant exposure through inhalation and skin absorption may be more than five times the human threshold for acute effects (No Observable Effect Level). The researchers state that, "Exposures to cholinesterase inhibiting compounds following properly conducted broadcast applications could result in doses at or above the threshold of toxicological response in humans."

In common with most organophosphates, chlorpyrifos has a relatively short biological half-life, roughly 24 hours in blood, and 60 hours in fat (assuming that multiple or continuous exposure does not occur) and it has shown no potential to bioaccumulate in mammals. Its half-life indoors is estimated to be 30 days. Various studies of different treatment methods show chlorpyrifos present up to eight years post application. A 1998 study found that chlorpyrifos accumulated on furniture, toys, pillowcases, and other sorbant surfaces up to two weeks after indoor application.

Chlorpyrifos is sensitive to light, alkaline substances such as bleach, and microbial degradation. Eventually, it degrades completely to car-

bon dioxide and water. The half-life of chlorpyrifos in water is relatively short, from a few days to two weeks. It adsorbs readily to sediments and organic matter, its half-life in soil is usually between 60 and 120 days, but can range from 2 weeks to over one year, depending on the soil type, climate, and other conditions.

The granular formulation of chlorpyrifos has been found to be more persistent and may persist as long as 180 days. The major biological metabolite and environmental breakdown product is 3,5,6-trichloro-2-pyridinol (TCP). According to an EPA memorandum, groundwater monitoring at a Cape Cod golf course detected TCP in samples. Reports from the USDA Southern Forest Experimental Station note that the termiticide formulation is effective against termites for more than 15 years.

Chlorpyrifos History

In 1997, EPA and Dow AgroSciences agreed to restrict several uses of chlorpyrifos, including a ban of the chemical for indoor broadcast flea control, indoor total release fogging, paint additives,

and direct application pet care products. Concentrated forms of chlorpyrifos no longer be sold to consumers, and the ready-to-use products now include labels which forbid use in dangerous areas, such as on/around furniture and toys.

In 1994, an unapproved formulation of chlorpyrifos was sprayed on General Mills, Inc., oats used to make 160 million boxes of Cheerios and Lucky Charms. According to EPA, most of the cereals had been consumed before the adulteration was detected. This is especially disturbing due to the pervasive nature of Cheerios and its consumption by young children.

In an unrelated incident, Dow AgroSciences agreed to pay \$876,000 for failing to disclose adverse effects incidents involving chlorpyrifos under a consent agreement. The agreement covers 337 violations over ten years of FIFRA §6(a)(2) reporting requirements.

In June 2000 EPA and Dow AgroSciences agreed to a stop sale of many uses of chlorpyrifos due to its health risk. *For information on the recent phase-out agreement, please contact us.*

Chlorpyrifos *chemicalWATCH* Factsheet Bibliography

- Aspelin, A., 1997. *Pesticide Industry Sales and Usage: 1994 and 1995 Market Estimates*. U.S. EPA, Office of Prevention, Pesticides and Toxic Substances, Office of Pesticide Programs, Biological and Economic Analysis Division, Washington, DC.
- Berteau, P., et al. 1985. "An assessment of the hazard from absorption from surfaces." CA Department of Health Services. Sacramento, CA.
- Blondell, J. 1999. "Review of Poison Control Center data for residential exposures to organophosphate pesticides, 1993-1996." U.S. EPA Memorandum, February 11.
- California Department of Health Sciences, Hazard Evaluation Section, Office of Environmental Health Hazard Assessment. 1987. "Hazards of indoor pesticides under investigation" *Tox-Epi Review* (September). Berkeley, CA.
- Chambers, J., et al. 1993. "Inhibition of patterns of brain acetylcholinesterase and hepatic and plasma alesterases following exposures to three phosphorothionate insecticides and their oxons in rats." *Fund. Appl. Toxicol.* 21:111-119.
- Chambers, J. et al. 1989. "Bioactivation and detoxification of organophosphorus insecticides in rat brains." In J. Caldwell, et al. *Intermediary Xenobiotic Metabolism: Methodology, Mechanisms, and Significance*. Taylor and Francis, Basingtoke, U.K.
- Cox, C. 1994, 1995. "Chlorpyrifos, part I-III: toxicology, human exposure, ecological effects." *Journal of Pesticide Reform* 14(4):15-20, 15(1):14-20, 15(2):13-19.
- Cremllyn, R. 1991. "Synthetic insecticides II organophosphorous and carbamate compounds." *Agrichemical: Preparation and Mode of Action*. John Wiley and Sons, Chichester, U.K.
- Dunphy, J., et al. 1980. "Respiratory arrest in an infant." *CA Morbidity Weekly Report*. April 11, 1980.
- Ellenhorn, M., et al. 1988. *Medical Toxicology: Diagnosis and Treatment of Human Poisoning*. Elsevier Science Publishing Co., New York, NY.
- Extension Toxicology Network. 1996. "Chlorpyrifos." *Pesticide Information Profiles*. <<http://ace.orst.edu/cgi-bin/mfs/01/pips/chlorpyr.htm>>.
- Fenske, R., et al. 1990. "Potential exposure and health risks of infants following indoor residential pesticide applications." *Amer. J. Public Health* 80(6):689-693.
- Fikes, J. 1992. "Clinical, biochemical, electrophysiologic, and histologic assessment of chlorpyrifos induced delayed neuropathy in the cat." *NeuroToxicology* 13:663-378.
- Gurunathan, S. et al. 1998. "Accumulation of chlorpyrifos on residential surfaces and toys accessible to children." *Environmental Health Perspectives* 106(1).
- Hayes, W. 1982. *Pesticides Studied in Man*. Williams & Wilkins, Baltimore, MD.
- Hodgson, M. et al. 1986. "Organophosphate poisoning in office workers." *J. Occup. Med.* 28(6):434-437.
- Horowitz, J. 1981. "Exposure to pesticides on the job." *Los Angeles Times*, April 30th.
- Kim, N. 1984. Testimony on chlorpyrifos. New York Department of Health, Albany, NY.
- Lotti, M. et al. 1986. "Inhibition of lymphocytic neuropathy target esterase predicts the development of organophosphate-induced delayed polyneuropathy." *Arch. Toxicol.* 59(30):176-179.
- Naffziger, D. et al. 1985. "Indoor environmental monitoring of Dursban L.O.™ following broadcast application." *Down to Earth* 41(1):7-10.
- Nelson, M. et al. 1990. "Genotoxicity of the organophosphorus insecticide chlorpyrifos based on human lymphocyte culture." *Cytologia* 55:589-592. Cited in Cox, C. 1994. "Chlorpyrifos, part I: Toxicology." *Journal of Pesticide Reform* 14(4):15-20.
- NY State Department of Conservation. 1986. *Draft Environmental Impact Statement on Amendments to 6 NYCRR Part 326 Relating to the Restriction of the Pesticides Aldrin, Chlordane, Chlorpyrifos, Dieldrin, and Heptachlor*. Bureau of Pesticides Management, Albany, NY.
- Roueché, B. 1988. "Annals of medicine; the fumigation chamber." *The New Yorker Magazine*. January 4th.
- Sherman, J. 1996. "Chlorpyrifos (Dursban) associated birth defects." *Archives of Environ. Health* 51(1):5-8.
- Sultatos, L. 1991. "Metabolic activation of the organophosphorus insecticides chlorpyrifos and fenitrothion by refused rat liver." *Toxicol.* 68:1-9. Cited in Cox, C. 1994. "Chlorpyrifos, part I: Toxicology." *Journal of Pesticide Reform* 14(4):15-20.
- Thomson, W. 1992. *Agricultural Chemicals: Insecticides*. Thomson Publications, Fresno, CA.
- Thrasher, J., et al. 1993. "Immunologic abnormalities in humans exposed to chlorpyrifos: preliminary observations." *Arch. Environ. Health* 48(2):89-93.
- U.S. EPA, Office of Prevention, Pesticides and Toxic Substances. 1997. "Review of Chlorpyrifos Poisoning Data." Memorandum, January 14.
- U.S. EPA. 1986. Information sheet: Cape Cod golf course monitoring project. Office of Pesticide Programs, Washington, DC.
- U.S. v. George Y. Roggy* (D.C. Minn. 1994).

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701 E Street, S.E., Suite 200, Washington DC 20003

202-543-5450 (v) • 202-543-4791 (f)

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