



ChemicalWatch Factsheet

PROPOXUR

Despite its known toxic hazards to children, resulting in its cancellation for indoor residential uses in 2007, the insecticide propoxur (2-isopropoxyphenyl methylcarbamate) has emerged as the chemical that exterminators want to use in the war on bedbugs. To allow this to happen, the U.S. Environmental Protection Agency (EPA) would have to change its regulatory restrictions. Many states are calling on EPA to do just that.

Known by the trade name Baygon™ and found in numerous registered products, propoxur (2-isopropoxyphenyl methylcarbamate) was first registered as an insecticide by Bayer AG in 1963. It is used to control ticks, fleas, and a variety of insects including crickets, ants, wasps, cockroaches and silverfish. It is an N-methyl residual carbamate insecticide, a family of toxic chemicals that includes carbaryl, aldicarb and carbofuran. Propoxur currently has registered uses for in-and-around industrial, commercial (including food handling establishments and food processing plants), and residential facilities.¹ Indoor residential uses of sprays for cracks and crevices were voluntarily cancelled in 2007, but formulations such as bait traps, pastes, and impregnated shelf paper are still registered for residential use. The primary outdoor uses are structural perimeter bait applications, as well as liquid and aerosol spot treatments. There are no agricultural uses.

Mode of Action

As a member of the neurotoxic N-methyl carbamate family of chemicals, propoxur shares a common mechanism of toxicity with other chemicals in this class. This means that hazards associated with multiple exposures to carbamate pesticides are cumulative. Carbamates share structural characteristics and an ability to inhibit acetylcholinesterase (AChE), an enzyme important for the transmission of nerve impulses. When AChE is inhibited, acetylcholine accumulates and cholinergic toxicity results due to

continuous stimulation of cholinergic receptors throughout the central and peripheral nervous systems, leading to overstimulation of neurotransmitters.²

While EPA and much of the scientific literature suggest that those exposed to carbamates who experience cholinesterase inhibition typically recover rapidly (minutes to hours), there is considerable

information that links exposure of this sort to long-term neurological damage. In insects paralysis occurs eventually leading to death.

Acute Toxicity

According to EPA's acute toxicity classification, propoxur is moderately toxic (Toxicity Category II) for oral exposure and slightly toxic (Toxicity Category III) via the dermal and inhalation routes of exposure. Propoxur and other carbamates are easily absorbed through breathing, eating or skin contact. Malaise, muscle weakness, dizziness, and sweating are commonly reported early symptoms of

poisoning. Headache, nausea, and diarrhea are often prominent. In rare cases, death may result from respiratory system failure. The potential for acute irritant effects to the skin and eyes is low. It is not considered to be a skin sensitizer. In rats, propoxur poisoning resulted in brain pattern and learning ability changes at lower concentrations than those which caused cholinesterase-inhibition and/or organ weight changes.³

Propoxur is reportedly less toxic when absorbed through the skin, than when it is ingested. Acute animal tests in rats, mice, and guinea pigs demonstrate propoxur to have high acute toxicity by inhalation exposure and high to extreme acute toxicity by ingestion.⁴

Chronic Toxicity

Chronic inhalation exposure has resulted in depressed

ChemicalWatch Stats

Chemical Class: Carbamate insecticide

Use: Control of ants, roaches and hornets indoors (institutional, industrial, commercial buildings) and limited outdoor applications

Prohibition: Residential uses that expose children

Toxicity rating: Highly toxic

Signal Word: Caution, Warning, or Danger

Health Effects: Probable human carcinogen (Group B2), Cholinesterase inhibitor

Environmental Effects: Toxic to bees, fish and other aquatic organisms, birds and mammals

cholinesterase levels, headaches, vomiting, and nausea in humans. Ingestion studies in animals have reported depressed cholinesterase levels, depressed body weight, effects to the liver and bladder, and slight increase in neuropathy.⁵

Carcinogenicity

Propoxur is classified as a B2 probable human carcinogen for which the carcinogenic potential has been quantified at 3.7×10^{-3} based on male rat bladder tumors. Tumors in bladders are observed in rats but not in other species.⁶ The state of California lists propoxur as a known human carcinogen.⁷ Research from the National Cancer Institute shows that propoxur might increase the risk of developing non-Hodgkin's lymphoma (NHL).⁸ Propoxur has also been identified as being highly correlated with leukemia in successive generations.^{9,10} Other studies indicate that propoxur could act through some epigenetic mechanisms, such as tumor promotion or cell proliferation.¹¹

Endocrine Disruption

A study from Tulane University found that propoxur and other carbamates weakly activated estrogen- or progesterone-responsive reporter genes in breast and endometrial cancer cells, and decreased estradiol- or progesterone-induced reporter gene activity in the breast and endometrial cancer cells.¹²

Reproductive and Developmental Toxicity

In reproductive studies, rats fed propoxur have a reduced mean number of fetal implantation sites and a reduced mean number of pups. The developmental data for propoxur indicate that there is no evidence of an increased sensitivity to propoxur from pre- or post-natal exposures. In the rat developmental toxicity study, no developmental effects were noted at the highest dose tested at which significant maternal effects were noted. However, in a rabbit study, developmental toxicity was noted at the highest dose tested with animals exhibiting a slight increase in post implantation loss with corresponding reductions in mean fetuses per dam in the presence of significant maternal toxicity, including mortality, dyspnea, restlessness and slight decreases in mean food consumption and mean body weight.¹³

Occupational Exposure

Occupational exposure to propoxur may occur through inhalation and dermal contact with this compound at workplaces where propoxur is produced or used. Signs of intoxication among spray workers and residents include lethargy, sweating, nausea, and headache; among those applicators with gross dermal and inhalation exposures, tachycardia, emesis, and vertigo ensued. Workers recovered within 30 minutes of cessation of operations and after washing the compound from their skin.¹⁴ According to EPA, workers handling propoxur (e.g., pest control operators) should wear long-sleeved shirts, long pants, chemical resistant gloves, shoes, and socks.¹⁵

Pets

Propoxur is formulated as impregnated pet collars. As a result, residential dermal exposure and non-dietary ingestion are of concern for individuals, especially children, coming into contact with (hugging, petting) treated pets. Residues of propoxur are expected on pet skin and fur, which pose exposure risks via the oral and dermal routes, especially for young children who have high incidences of hand-to-mouth activity. Pet collars continuously expose pet owners to propoxur residues since these products are designed to continually emit residues throughout their active period. Pet collars can contaminate indoor air as propoxur volatilizes from pets. EPA is currently considering a petition to ban propoxur-based pet collars.



Indoor Residential Data and Exposures

The main route of exposure to propoxur is through the dermal and inhalation route. Propoxur is detected and persists in indoor air¹⁶ and dust.¹⁷ On surfaces, pesticide residues can persist for 60 hours or longer.¹⁸ One study found that airborne concentrations are still detectable 33.5 hours after spraying propoxur indoors.¹⁹ Another determined that the volatilization of propoxur from treated surfaces increases with humidity, resulting in higher air concentrations.²⁰ In a study investigating indoor-air insecticide levels in inner-city residences, propoxur is found in over 90% of indoor air samples of homes with pregnant women.²¹ Inhalation studies have found that propoxur exposure effects a depression of plasma by 20 to 30 percent and erythrocyte and brain cholinesterase activities.²² Dermal absorption studies,²³ which have shown that propoxur is absorbed through the skin, also find that skin moisture (affected by high temperatures and humidity) influences the dermal uptake of propoxur.²⁴ A dermal LD50 study with laboratory rats found that on the day of propoxur application to the skin, muscular fasciculations suggestive of cholinesterase inhibition were observed along with decreased motor activity.²⁵

Various monitoring studies have confirmed that indoor residential pesticide applications increase the exposure and health risks of residents, especially infants. Indoor residues of pesticides have been detected in carpets, hard surfaces, walls and dust. In a study with inner city underserved mothers and newborns, propoxur levels were significantly higher in the personal air of women reporting use of an exterminator, can sprays, and/or pest bombs during pregnancy, compared with women reporting no pesticide use or use of lower toxicity methods.²⁶

Metabolites

Propoxur breaks down relatively quickly in mammalian systems

as well as the environment. Metabolism studies indicate a number of urinary metabolites (such as O-isopropoxyphenyl and 2-hydrophenyl methylcarbamate) are rapidly excreted after exposure to propoxur. In a rat metabolism study, 85% of radioactively labeled propoxur was eliminated in 16 hours, 25-35% as the volatile compounds CO₂ and acetone, and 50% in urine as conjugates.

Ecological Effects and Environmental Fate

In the environment, propoxur is subject to degradation via hydrolysis and microbial action. If released to air, a vapor pressure of 9.68×10^{-6} mmHg (millimeters of mercury) at 20 deg C indicates propoxur will exist in both the vapor and particulate phases in the ambient atmosphere. The half-life in air is approximately 12

hours. Hydrolysis is both pH and temperature dependent. Under acidic conditions, propoxur is relatively stable. At neutral pH (7), half-life is 30 days at 30°C, and drops to 23 hours at 50°C. Although propoxur is subject to rapid leaching and may be expected to move laterally through runoff, except under acid conditions, residues would be subject to hydrolysis. Bacterial degradation of propoxur is the same under aerobic or anaerobic soil conditions, with half-lives ranging from 80 to 210 days (depending upon soil type). Degradation products in soil and water are O-isopropoxyphenol, CO₂, and methyl amine, all of which volatilize into the atmosphere or enter the metabolic pools of plants and microflora. A non-specific poison, propoxur is highly toxic to non-target, beneficial species, such as bees, and is of very high toxicity to crustaceans, fish, aquatic insects, and aquatic worms.

Citations

- ¹ USEPA. 2010. Propoxur Final Work Plan for Registration Review. Office of Pesticide Programs. Docket number EPA-HQ-OPP-2009-0806.
- ² USEPA. 2007. Revised N-Methyl Carbamate Cumulative Risk Assessment. Office of Pesticide Programs. Washington DC.
- ³ EXTTOXNET. 1993, 1996. Pesticide Information Profile- Propoxur.
- ⁴ USEPA. 2000. Propoxur (Baygon) Hazard Summary. Available at <http://www.epa.gov/ttn/atw/hlthef/propoxur.htm>.
- ⁵ USEPA. 2000.
- ⁶ Cohen, S et al. 1998. Mitogenic effects of propoxur on male rat bladder urothelium. *Carcinogenesis* 15 (11): 2593-2597.
- ⁷ OEHHA. Proposition 65 List of Chemicals. CA Environmental Protection Agency. Available at http://www.oehha.ca.gov/prop65/prop65_list/Newlist.html.
- ⁸ Colt, J. et al. 2005. Residential insecticide use and risk of non-Hodgkin lymphoma (NHL). *Proc Amer Assoc Cancer Res*, Volume 46.
- ⁹ Birnbaum, L.S, and Fenton, S. E. 2003. Cancer and Developmental Exposure to Endocrine Disruptors. *Environ Health Perspect* 111:389–394.
- ¹⁰ Zahm, S. H. and Ward, M.H. 1998. Pesticides and Childhood Cancer. *Environ Health Perspect* 106(Suppl 3):893-908.
- ¹¹ Wang, T.C. et al. 2009. Multiple factors conferring high radioresistance in insect Sf9 cells. *Mutagenesis* 24(3): 259-269.
- ¹² Klotz, D.M. et al. 1997. Inhibition of 17 beta-estradiol and progesterone activity in human breast and endometrial cancer cells by carbamate insecticides. *Life Sciences*: 60(17):1467-1475.
- ¹³ USEPA. 1997. Reregistration Eligibility Decision (RED) Document. Office of Prevention, Pesticides and Toxic Substances. Washington DC.
- ¹⁴ Hazardous Substances Database. Propoxur. <http://toxnet.nlm.nih.gov/cgi-bin/sis/search/f?./temp/~Qwj05W:1>.
- ¹⁵ USEPA. 2010.
- ¹⁶ Esteve-Turrillas FA, et al. 2009. Use of semipermeable membrane devices for monitoring pesticides in indoor air. *J AOAC Int.* 92(5):1557-65; Leva P et al. 2009. Evaluation of the fate of the active ingredients of insecticide sprays used indoors. *J Environ Sci Health B.* 44(1):51-7.
- ¹⁷ Colt JS et al. 2004. Comparison of pesticide levels in carpet dust and self-reported pest treatment practices in four US sites. *J Expo Anal Environ Epidemiol.* 14(1):74-83.
- ¹⁸ Class, TJ and Kintrup, J. 1991. Pyrethroids as household insecticides: analysis, indoor exposure and persistence. *Fresenius' Journal of Analytical Chemistry.* 340:446-453.
- ¹⁹ Kuo HW and Lee HM. 1999. Volatility of propoxur from different surface materials commonly found in homes. *Chemosphere.* 38(11):2695-705.
- ²⁰ Miller, C. W. and Shafik, T. M. 1974. Concentrations of propoxur in air following repeated indoor applications. *Bull World Health Organ.* 51(1): 41–44.
- ²¹ Whyatt, RM et al. 2007. Within- and between-home variability in indoor-air insecticide levels during pregnancy among an inner-city cohort from New York City. *Environ Health Perspect.* 115(3):383-9.
- ²² Kimmerle G and Iyatomi, A. 1976. Toxicity of propoxur to rats by subacute inhalation. *Sangyo Igaku.* 18(4):375-82; Pauluhn J, Machemer L and Kimmerle G. 1987. Effects of inhaled cholinesterase inhibitors on bronchial tonus and on plasma and erythrocyte acetylcholine esterase activity in rats. *Toxicology.* 46(2):177-90.
- ²³ van de Sandt, J. et al. 2000. Comparative in Vitro–in Vivo Percutaneous Absorption of the Pesticide Propoxur. *Toxicological Sciences.* 58:15-22; Brouwer, R. et al. 1993. Skin contamination, airborne concentrations, and urinary metabolite excretion of propoxur during harvesting of flowers in greenhouses. *Amer. J. of Industrial Medicine.* 24(5):593-603.
- ²⁴ Meuling, W.J.A, et al. 1997. The influence of skin moisture on the dermal absorption of propoxur in human volunteers: a consideration for biological monitoring practices. *Science of The Total Environment* 199 (1-2):165-172.
- ²⁵ USEPA. 1997. Reregistration Eligibility Decision (RED) Document. Office of Prevention, Pesticides and Toxic Substances. Washington DC.
- ²⁶ Whyatt, RM, et al. 2003. Contemporary-use pesticides in personal air samples during pregnancy and blood samples at delivery among urban minority mothers and newborns. *Environ Health Perspect.* 111(5): 749–756.