

Chlorpyrifos

Trade names

Dursban, Lorsban and many others.

Uses

Broad-spectrum organophosphate insecticide, acaricide, and nematicide.

Used on nuts, fruit, grain, seeds, vegetables, fodder, ornamentals, buildings, ships, homes, turf, fence posts, cattle ear tags, sheep dip, etc.

Classifications and risk statements

WHO: Class II moderately hazardous

EU: R25 – Toxic if swallowed; R50/53 – very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

US EPA: Category II moderately toxic

Regulatory status

International

No international regulatory action.

National

Yemen: banned.

Jamaica: restricted.

EU: approved until June 30th 2016, but not authorised in Denmark, Finland, Latvia, Lithuania, Sweden.

South Africa: household, home garden, and domestic use banned.

USA: use on tomatoes, and in homes, schools and children's parks, with the exception of child-proof baits, are banned.

International standards

On PAN International's list of Highly Hazardous Pesticides (2013) for global phase-out, because of high bee toxicity.

Manufacture

Dow AgroSciences, Cheminova, Gharda, Makhateshim, SumiAgro and numerous producers in China.

Exposure

Residues in food

Widespread in fruit and vegetables; also in dairy products, nuts, cottonseed, wheat and wheat-based products such as bread and pasta, rice, maize, chickpeas, fish, muesli, jam, olive oil, pizza, hamburgers, raisins; also soft drinks and drinking water.

Other exposures

Pervasive in the environment. Adults and children are widely exposed through occupational use, contact with treated household surfaces, ingestion or inhalation of contaminated dust including

house dust, breathing air in treated buildings or near treated fields or orchards, contact with flea collars on pets. It is a common residue in the dust of rural houses and farmworker vehicles.

Has high potential for adverse effects in occupational applications, especially in developing countries. High levels of exposure have been recorded in Vietnam, Thailand, and Egypt.

Health effects

Mechanism of toxicity

At least three main modes of action: it inhibits the enzyme acetylcholinesterase (AChE) causing overstimulation of the nervous system; it causes oxidative stress, a process involved in many human diseases, including cancer, Parkinson's disease, Alzheimer's disease, diabetes, and heart failure; and it causes endocrine disruption.

Poisonings

Occupational and non-occupational poisonings have occurred in a number of countries including India, Jamaica, Nicaragua, and USA. Suicides have been reported from France, India, Iran, Israel, Italy, Nepal, Nicaragua, South Africa, Spain, Sri Lanka, Taiwan, Turkey, USA, and Venezuela.

Residues in people

Found in cervical fluid, sperm fluid, cord blood, meconium, breast milk, and maternal and infant hair. Biomonitoring in the US showed that 94% of people had chlorpyrifos in their bodies in 1999–2000.

Acute toxicity

Inhibition of AChE leads to increased secretions, sensory and behavioural disturbances, incoordination, depressed motor function, respiratory depression, tremors, convulsions, and death. Seizures, lethargy, and coma are common in children.

Early symptoms of poisoning include headache, nausea, dizziness, sweating, salivation, lacrimation, and rhinorrhea; muscle twitching, weakness, tremor, incoordination, vomiting, abdominal cramps, and diarrhoea; constriction of pupils, blurred and/or dark vision; anxiety, restlessness, depression, memory loss, confusion, and toxic psychosis manifested as confusion or bizarre behaviour.

Chronic toxicity

General: liver damage, inhibition of cytochrome P450 metabolism of testosterone, oestrogen, other pesticides, drugs, etc.

Neurotoxicity: potent neurotoxin at



Photo by Ilang Ilang Quijano

Chlorpyrifos, an organophosphate, has been found in human cord blood and meconium. It is known to cause foetal damage leading to neurodevelopmental disorders.



Pesticide Action Network Asia and the Pacific

P.O. Box 1170

10850 Penang, Malaysia

Tel: (604) 657 0271 / 656 0381

Fax: (604) 658 3960

Email: panap@panap.net

Homepage: www.panap.net

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Meriel Watts, PhD
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low levels of exposure, below those that trigger foetal AChE inhibition, demonstrated in numerous laboratory and epidemiological studies. Exposures *in utero* and in early childhood can lead to behavioural anomalies in adolescence and adulthood.

Epidemiological studies have found delayed cognitive and psychomotor development, reduced IQ, attention-deficit/hyperactivity disorder (ADHD) and pervasive developmental disorder, smaller head circumference, and altered brain structure. It may have long-term consequences for social adjustment and academic achievement. The effects reported are regarded as being comparable to those seen with exposures to other neurotoxicants such as lead and tobacco smoke.

Chlorpyrifos has a greater adverse effect on neural cell replication and is inherently more toxic to the developing brain than the more acutely toxic organophosphates such as diazinon and parathion. It is toxic to children at doses that are not toxic to adults, and tests using adult animals cannot predict the long-term delayed effects of chlorpyrifos in offspring.

There are sex-related differences in effects on the brain, and subsequent cognitive function in adolescence and adulthood, with females affected more than males by prenatal exposures and vice versa for postnatal exposures.

Epidemiological studies show both acute and chronic exposures result in neuropsychological effects, including peripheral and central neuropathy, affective disorders, and neurocognitive deficits.

It can cause organophosphate induced delayed polyneuropathy (OPIDP), and organophosphate induced chronic neuropathy (OPICN), involving degeneration of the peripheral and central nervous systems, which may result in weakness, numbness and paresthesia, and even life-threatening paralysis. It can cause chronic neuropsychological effects like anxiety, depression and suicide.

There is evidence from laboratory and epidemiological studies of an association with Parkinson's disease.

Cancer: US EPA reported no evidence of carcinogenicity in animal studies, but recognised that a number of epidemiological studies indicate chlorpyrifos may be carcinogenic in humans, the association being strongest for lung and rectal cancers.

It acknowledged preliminary associations with breast and prostate cancer. Studies also indicate possible associations with non-Hodgkin's lymphoma, lung, kidney, and brain cancer. Chlorpyrifos is a breast cancer risk through its endocrine actions.

Genotoxicity and mutagenicity: Independent studies have found chlorpyrifos to be

mutagenic or genotoxic in human, rat, mouse, Chinese hamster, toad, fish, fruitfly, and plant cells. US EPA stated chlorpyrifos is not mutagenic, but California EPA stated it may be genotoxic.

Endocrine disruption: inhibits metabolism of testosterone and oestradiol, and testosterone synthesis; reduces serum levels of cortisol and thyroid hormone T4; induces alterations in thyroid and adrenal glands, and differentially affects levels of thyroid-stimulating hormones in men and women. It causes breast cancer cells to grow.

Reproductive and developmental toxicity: teratogenic effects observed in rats include skeletal malformations, small hind and fore limbs, lack of spinal development, absence of thoracic vertebrae and cleft palate; in humans, defects of the brain, eyes, ears, palate, teeth, heart, feet, nipples, and genitalia have been associated with gestational exposure to chlorpyrifos. Neuroteratogenicity has also been found in both animal and human studies.

Reproductive effects in animals include decreased foetal weight and viability; increased foetal death and early resorption; decreased sperm motility and count; and decline in viability and developmental competence of oocytes.

In humans, chlorpyrifos exposure is associated with decreased birth weight and birth length, DNA damage in sperm, and decreased sperm concentration and sperm motility.

Immunotoxicity: there is evidence from both laboratory and epidemiology studies of immune toxicity, including effects on lymphocytes, thymocytes, T cells, tumour necrosis factor, and autoimmunity.

Metabolic: early life exposure may predispose a person to obesity, diabetes, and cardiovascular problems later in life.

Environmental and agroecological effects

Toxicity

Aquatic: very toxic to fish, and moderately to very highly toxic to amphibia and aquatic invertebrates. It causes endocrine disruption in fish, frogs, and mussels. In fish, it has caused mutagenicity, spinal deformities, and altered swimming behaviour.

Some species of amphibia are very highly sensitive to chlorpyrifos and scientists are concerned about its potential involvement in declining frog populations. Sublethal effects include teratogenicity, genotoxicity, and altered swimming of tadpoles.

Residues have been found in sea otters, frogs, fish, and oysters. It can accumulate in areas of intense biological productivity, such as littoral zones and river deltas, posing a long-term threat to aquatic ecosystems. It can have significant effects on aquatic community structure. There have been large fish kills reported in

several countries.

Birds: very highly toxic to some birds; sublethal effects include reduced flying ability. There have been a number of reported bird kills.

Mammals: very toxic to mammals.

Agroecological disruption

Bees: extremely toxic to bees.

Beneficials: extremely toxic to beneficial insects, incompatible with IPM.

Soil organisms: moderately toxic to earthworms; has an inhibitory effect on soil microbial functional diversity, reducing microbial biomass by as much as 50% after application, inhibiting nitrogen mineralisation, and reducing bacterial, fungal and actinomycetes populations.

Resistance: resistance is now widespread, involving 65 species in at least 47 countries.

Environmental fate and contamination

Soil: meets the Stockholm Convention criteria for persistence in soil; increased persistence with increased organic matter, decreased temperature, decreased pH, and decreased UV light. Reduces the availability of nitrogen and phosphate in the soil. Soil residues found in Argentina, Ghana, India, New Zealand, Philippines, Serbia, and South Africa.

Aquatic: meets the Stockholm Convention criteria for persistence in sediment and water; residues found in groundwater, surface waters and sediments, wastewater, and marine sediments in numerous countries including Australia, Bangladesh, China, India, Indonesia, Iran, Israel, Japan, Malaysia, and Philippines.

Bioaccumulation: described as moderate to high. The Kow ranges from 4.7 to 5.11, meeting the Stockholm Convention criteria of 5. Reported bioaccumulation factors vary, but some are higher than the criteria of 5,000.

Other: residues found in air, rain, snow, and fog in some countries. Found in indoor air up to 8 years after application. Chlorpyrifos volatilises, undergoes long-range transport, and distils out of the air in cold climates far from its site of application. It has been measured consistently in the Arctic, in ice, snow, fog, air, seawater, lake sediment, fish and vegetation. It is amongst the pollutants with the highest concentrations present in the Arctic, in excess of most legacy POPs pesticides.

Alternatives

Numerous alternatives exist, including biopesticides such as neem, biological controls such as Btk, and predatory and parasitic insects.

There are also numerous agroecological pest management practices that focus on preventing pest build up.

Sources

refer to Chlorpyrifos monograph at <http://www.panap.net/sites/default/files/monograph-chlorpyrifos.pdf>.