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Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade
Chemical Review Committee
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Item 7 (f) of the provisional agenda*

Inclusion of chemicals in Annex III of the Rotterdam Convention: review of notifications of final regulatory actions to ban or severely restrict a chemical: methyl parathion

Methyl parathion : supporting documentation from Brazil

Note by the secretariat

The secretariat has the honour to provide, in the annex to the present note, the supporting documentation supplied by Brazil in support of its final regulatory action on methyl parathion.

* UNEP/FAO/RC/CRC.1/1.

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Annex

Methyl parathion

(Brazil summary data for CRC - based on the notification of final regulatory action of emulsifiable concentrates and dusts containing active ingredient)

Properties
Description of physico-chemical properties of the chemical

Pure Methyl parathion is a colorless crystalline solid. The technical product is light to dark tan, with about 80% purity.

Class: organophosphate pesticide;
Molecular Formula: C8H10NOSPS;
CAS Number: 298-00-0;
Molecular Weight: 263.21;
Water Solubility: 55-60 mg/L (25 oC)
Solubility in Other Solvents: s. in dichloromethane, 2-propanol, toluene, and most organic solvents;
Melting Point: 35-36 oC
Vapor Pressure: 1.3 mPa (20 oC)
Partition Coefficient (Log **Pow): 3.5185 - 3.8388**
Adsorption Coefficient: 5100

Description of toxicological properties of the chemical

Acute Toxicity: Methyl parathion is highly toxic via the oral route, with reported oral LD50 values of 6 to 50 mg/kg in rats, 14.5 to 19.5 mg/kg in mice, 420 mg/kg in rabbits, 1270 mg/kg in guinea pigs and 90 mg/kg in dogs. It is highly toxic via the dermal route as well, with reported dermal LD50 values of 67 mg/kg in rats, 1200 mg/kg in mine, and 300 mg/kg in rabbits. The 1-hour inhalation LC50 for methyl parathion in rats is 0.24 mg/L. Effects associated with acute exposure to methyl parathion are similar to those associated with exposure to other organophosphate pesticides. Symptoms of acute exposure to organophosphate or cholinesterase-inhibiting compounds may include the following: numbness, tingling sensations, incoordination, headache, dizziness, tremor, nausea, abdominal cramps, sweating, blurred vision, difficulty breathing or respiratory depression, and slow heartbeat. Very high doses may result in unconsciousness, incontinence, and convulsions or fatality. Persons with respiratory ailments, recent exposure to cholinesterase inhibitors, cholinesterase impairment, or liver malfunction is at increased risk from exposure to methyl parathion.

Chronic Toxicity: Studies with human vuluntenrs have found that of 1 to 22 mg/person/day has no effect on cholinesterase activity. In a 4-week study of volunteers given 22, 24, 26, 28 or 30 mg/person/day, mild cholinesterase inhibition appeared in some individuals in the 24, 26 and 28 mg dosage groups. In the 30 mg/persorn/day (about 0.43 mg/kg/day) group, red blood cholinesterase activity was depressed by 37%. When methyl parathion was fed to dogs for 12 weeks, a dietary level of 1.25 mg/kg caused a significant depression of red blood cell and plasma cholinesterase.

A dietary level of O. 12-5 mg/kg produced no effects.

Teratogenic Effects: In a three-generation study with rats fed dietary levels of 0.5 or 1.5 mg/kg/day, there were no compound-related teratogenic effects. Single injections of 5 to 10 mg/kg in rats on day 12 of pregnancy and single injections of 20 mg/kg on day 10 of pregnancy in mice caused no statistically significant changes in the offspring. Oral administration of 4 to 6 mg/kg on day 9 or 15 of pregnancy in rats resulted in no fetal anomalies. Available evidence indicates that methyl parathion does not cause teratogenic effects.

Mutagenic Effects: no signs of mutagenicity were seen in mice given dosages of 5 to 100 mg/kg, or in mice fed methyl parathion for 7 weeks. Available evidence suggests that methyl parathion is nonmutagenic.

Carcinogenic Effects: Available evidence suggests that methyl parathion is not carcinogenic.

Organ Toxicity: Methyl parathion primarily affects the nervous system.

Fate in Humans and Animals: Methyl parathion is rapidly absorbed into the bloodstream through all normal routes of exposure. Following administration era single oral dose, the highest concentration of methyl parathion in body tissues occurred within 1 to 2 hours. Methyl parathion does not accumulate in the body, and is almost completely excreted by the kidneys (urine) within 24 hours as phenolic metabolites.

Reference: Extension Toxicology Network (Pesticide Information Profiles)

Description of ecotoxicological properties of the chemical

Breakdown of Chemical in Soil and Groundwater:
Methyl parathion is of low persistence in the soft environment, with reported field half-lives of I to 30 days. A representative value is estimated to be 5 days. The rate of degradation increases with temperature and with exposure to sunlight. Methyl parathion is moderately adsorbed by most soils, and is slightly soluble in water. Due to its low residence time and soil binding affinity, it is not expected to be significantly mobile. 4-Nitrophenol, a breakdown product of methyl parathion, does not adsorb well to soil particles and may contaminate groundwater. When large concentrations of methyl parathion reach the soil, as in an accidental spill degradation will occur only after many years, with photolysis being the dominant route. Some volatilization of applied methyl parathion may occur.

Breakdown of Chemical in Surface Water:
Methyl parathion degrades rapidly in seawater, lake, and river waters, with 100% degradation occurring within 2 weeks to 1 month or more. Degradation is faster in the presence of sediments, and is faster in fresh water than in salt water. Complete breakdown occurs at a rate of 5 to 11% in 4 days in rivers, and more slowly in marine waters. In water, methyl parathion is subject to photolysis, with a half-life of 8 days during the summer and 38 days in winter

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Breakdown of Chemical in Vegetation:

Uptake and metabolism of methyl parathion in plants is fairly rapid. Within 4 days after applying methyl parathion to the leaves of corn, it was almost completely metabolized. Reference: Extension Toxicology Network (Pesticide Information Profiles)

Risk and hazard evaluation
The action was based on information on environmental persistence and toxicity of methyl parathion.
Pesticide poisonings, some resulting in death, have become a serious public health problem, requiring intervention across a number of different areas. On the other hand, samples collected from actual users in the countryside, hospital records, programmes for recognition and treatment of poisoning from exposure to pesticides, provide crucial information about danger caused by pesticide use in the field, like the SINITOX (National System of Toxic-Pharmacological Information).

Reference to the relevant documentation: Extension Toxicology Network (Pesticide Information Profiles)

Reasons for the final regulatory action

The reason for the final regulatory action relevant to the human health.
Summary of the known hazards and risks presented by the chemical to human health, including the health of consumers and workers: Methyl parathion is a highly toxic insecticide in EPA toxicity class I. Some or all formulations of methyl parathion may be classified as Restricted Use Pesticides (RUPs). RUPs may be purchased and used only by certified applicators. Labels for products containing methyl parathion must bear the Signal Word DANGER.
Reference to the relevant documentation
WHO, WHOPES
EPA
IPCS, INTOX, INCHEM
PANNA
EXTOXNET
OIT
IPCS/WHO
ECO/PAHO
IRPTC/UNEP
OECD/EC
US EPA
Academic studies and researches

Expected effect of the final regulatory action

Total control for the uses specially authorized; better environment quality and health due the safety use and of this chemical.

Reason for the final regulatory action relevant to the environment

Summary of the known hazards and risks to the environment: Methyl parathion is very toxic to birds. Reported acute oral LDS0 values are 3 mg/kg in American kestrels, 7.5 mg/kg in European starlings, 6 to 10 mg/kg in mallards, 8 mg/kg in northern bobwhites, 10 to 24 mg/kg in red-wing blackbirds, and 8 mg/kg in ring-neck pheasants. The 5- to 8-day dietary LC50 values reported for methyl parathion include 69 ppm in Japanese quail, 330 to 680 ppm in mallard, 90 ppm in northern bobwhite, and 91 ppm **in** ring-neck pheasant. Methyl parathion is moderately toxic to fish and to animals that eat fish. Reported 96-hour LCS0 values are from 1.9 to 8.9 mg/L in the following fish species: coho salmon, cutthroat trout, rainbow trout, brown trout, lake trout, goldfish, carp, fathead minnow, black bullhead, channel catfish, green sunfish, bluegill, largemouth bass: and yellow perch. Reported 96-hour LCS0 values indicate very high toxicity for aquatic invertebrates such as Daphnia spp., scuds, and sideswimmers. Methyl parathion is toxin to bees.

Reference to the relevant documentation
FAO WHO EPA PANNA EXTOXNET
And others (see notification at the 2.4. I)

Expected effect of the final regulatory action: Use controlled of this chemical by Federal Body &Environment, Health and Agriculture.