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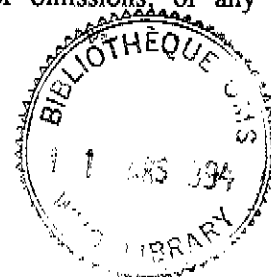
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WHO/FAO DATA SHEET ON PESTICIDES

No. 92

FENAMIPHOS

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CLASSIFICATION:

Primary use: Nematicide
 Secondary use: Insecticide
 Chemical group: Organophosphorus compound

1.0 GENERAL INFORMATION

1.1 **COMMON NAME:** fenamiphos (E-ISO); phénamiphos (F-ISO)

1.1.1 **Identity:**

IUPAC name: ethyl 4-methylthio-m-tolyl isopropylphosphoramidate

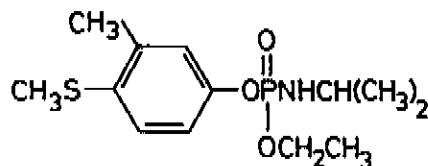
CAS name: ethyl 3-methyl-4-(methylthio)phenyl (1-methylethyl)phosphoramidate

CAS registry number: 22224-92-6

RTECS number: TB3675000

Molecular formula: C₁₃H₂₂NO₃PS

Relative molecular mass: 303.4

Structural formula:

Synonyms or trade names: Bay 68138; Nemacur^R; Nemacur P^R.

1.2 **SYNOPSIS:** Fenamiphos, an organophosphorus nematicide, is extremely toxic to mammals. The technical product is listed in the WHO Recommended Classification of Pesticides by Hazard under class Ia, Extremely hazardous. Readily absorbed through foliage and roots of plants, it has a fairly long-acting systemic activity in plants. Residues are generally moderately well adsorbed on soil, reducing the amount of leaching through soil into ground waters. No indications of toxicity other than through inhibition of acetylcholinesterase activity are reported in the limited amount of published data available.

1.3 SELECTED PROPERTIES

1.3.1 **Physical characteristics:** The technical product is a tan, waxy solid with a melting point around 46 °C. Pure fenamiphos is a white crystalline product with a melting point of 49.3 °C. The technical material has a minimum purity of 87%. Fenamiphos is not corrosive.

1.3.2 **Solubility:** In water at 20 °C, 700 mg/L. Slightly soluble in most organic solvents.

1.3.3 **Stability:** Hydrolysed by strong acids and alkalis. No degradation in propanol/water (1:1) at 40 °C, pH 7, after 50 days.

1.3.4 **Vapour pressure:** 0.12 mPa (20 °C)

1.4 AGRICULTURE, HORTICULTURE AND FORESTRY

1.4.1 **Common formulations:** Granular 5-15%, emulsifiable concentrate 40%, 250 g/L EW. In combination with carbofuran, disulfuron, isofenphos or fensulfothion in granule and emulsifiable concentrate formulations.

1.4.2 **Susceptible pests:** Effective against ecto- and endo-parasitic, free-living, cyst-forming and root-knot nematodes. May also control mites, aphids, thrips, fleahoppers and mealy bugs.

1.4.3 **Use pattern:** Used as a soil treatment, with or without incorporation, as a root dip, as a seed treatment or as a foliar application. Fenamiphos is readily absorbed through roots and leaves to give a systemic nematicidal action. Controls nematodes in a wide variety of field, vegetable and fruit crops; good water solubility makes it particularly useful in wet or heavy soils.

1.4.4 **Unintended effects:** Toxic to fish. Some phytotoxicity has been reported following foliage application to alfalfa, squash, tomatoes, and some ornamentals.

1.5 **PUBLIC HEALTH USE:** No recommended usage reported.

1.6 **HOUSEHOLD USE:** No recommended usage reported.

2.0 TOXICOLOGY AND RISKS

2.1 TOXICOLOGY - MAMMALS

2.1.1 **Absorption route:** Fenamiphos may be absorbed from the gastrointestinal tract; by inhalation or through intact skin.

2.1.2 **Mode of action:** Direct inhibition of cholinesterases. The sulfoxide and sulfone metabolites are more potent inhibitors than fenamiphos itself.

2.1.3 **Excretion products:** Fenamiphos is extensively metabolized by the rat to sulfoxides and sulfones, N and O dealkylation products and conjugates. Excretion of a 2 mg/kg b.w. radio-labelled oral dose was predominantly via the urine and expired air and was essentially complete within 15 hours.

2.1.4 Toxicity, single dose:

Oral LD₅₀

Rat (M & F)	2.3 - 19.4 mg/kg b.w.
Mouse (M)	22.7 mg/kg b.w.
Mouse (F)	8.3 mg/kg b.w.
Rabbit	5.0 mg/kg b.w.
Rabbit (M)	10 - 17.5 mg/kg b.w.
Guinea pig (M)	56 - 100 mg/kg b.w.
Dog (M)	ca. 10 mg/kg b.w.
Cat (M)	ca. 10 mg/kg b.w.

Dermal LD₅₀

Rat (M)	73 - 500 mg/kg b.w.
Rat (F)	84 - 154 mg/kg b.w.
Rabbit	178 - 225 mg/kg b.w.

Inhalation LC₅₀

1 hour	Rat	110 - 175 mg/m ³
4 hour	Rat	91 - 100 mg/m ³

Intraperitoneal LD₅₀

Rat (M & F)	3.0 - 4.9 mg/kg b.w.
Mouse (M & F)	3.4 mg/kg b.w.
Guinea pig (M)	17.3 mg/kg b.w.

Primary irritation: Application of 0.25 ml of a liquid formulation (equivalent to 0.085 mg a.i.) to intact or abraded rabbit skin did not cause primary irritation. Instillation of the same formulation into the conjunctival sac of rabbits caused irritation at 0.034 mg a.i. equivalent.

2.1.5 Toxicity, repeated dose:

Oral or intraperitoneal administration of 1.7 or 1.5 mg/kg b.w./day respectively, five days/week for sixty days, was not lethal to male rats, and no evidence of cumulative toxicological effects was observed. Female rats survived daily intraperitoneal administration of 1 mg/kg for sixty days while 40% of those administered 2 mg/kg did not survive. All rats died at 3 mg/kg.

No effects on growth, haematology, clinical chemistry, gross or microscopic pathology were observed in rats exposed to aerosol concentrations of up to 3.5 mg/m³ for three weeks. Plasma cholinesterase activity was depressed at 3.5 mg/m³ but no effect was observed on erythrocyte and brain cholinesterase.

Dermal application 0.5, 2.5 or 10 mg/kg b.w. to rabbits for three weeks caused no adverse effects other than a slightly decreased plasma cholinesterase activity at 2.5 mg/kg b.w. and above.

Cumulation of effect: Fenamiphos does not accumulate in body tissues but cumulation of effects was demonstrated during exposure. Plasma and erythrocyte cholinesterase activity remain inhibited but clinical symptoms such as behavioural changes and tremors appear only during the early period of exposure.

2.1.6 Dietary studies:

Short term: Administration of 0, 4, 8, 16 or 32 mg/kg/diet to rats for three months caused a slight increase in absolute liver weights in males at the two highest concentrations. Relative liver weights were unaltered, however, and no abnormal histopathology of this, or any other organ, was observed. Evidence of cholinergic stimulation was apparent in both sexes at 32 mg/kg/diet but was only observed for the first two months of the study. Plasma cholinesterase activity was depressed at 8 mg/kg/diet and above. A no-effect-level (NOEL) based upon plasma cholinesterase was found to be 4 mg/kg/diet.

Groups of beagle dogs were fed fenamiphos in the diet at 0, 2, 6 and 18 mg/kg/diet for three months. Administration of 18 mg/kg/diet to male and female dogs caused behavioral abnormalities, evidenced by signs of cholinergic stimulation and a decreased growth rate in the females. At 2 mg/kg/diet plasma cholinesterase activity was marginally decreased in both sexes while erythrocyte cholinesterase activity was unaffected at this dose. The NOEL for dogs was considered to be 2 mg/kg/diet.

In a two year feeding study in dogs at concentrations of 0, 0.5, 1.0, 2.0, 5.0 and 10.0 mg/kg/diet no adverse effects other than inhibition of blood cholinesterase were observed. The NOEL for plasma cholinesterase was 1 mg/kg/diet.

Long term: Signs of cholinergic stimulation were observed in Wistar rats receiving 30 mg/kg/diet (maximum dose tested) but were apparent only for the first six weeks of the two year study. Inhibition of plasma cholinesterase was observed at concentrations above 3 mg/kg/diet. Increased absolute and relative thyroid weights were observed in females receiving 30 mg/kg/diet, but goitre, tumours or other abnormal histopathological findings were absent. No adverse effects were observed on growth, mortality, haematologic or clinical chemistry evaluations, or in histopathologic examinations. A NOEL was set at 3 mg/kg/diet (equivalent to 0.17, to 0.23 mg/kg b.w./day) on the basis of plasma cholinesterase inhibition.

Groups of male and female Fischer 344 rats were fed diet containing mean effective concentrations of 0, 1.7, 7.8 and 37 mg/kg/diet (equivalent to 0, 0.1, 0.5 and 2.5 to 3.4 mg/kg b.w./day) of technical fenamiphos (89.3% pure) for two years. Erythrocyte cholinesterase activity was significantly inhibited in both sexes at all dose levels (approximately 6%, 30% and 70% at 1.7, 7.8 and 37 mg/kg/diet respectively). Brain cholinesterase activity was also significantly decreased in high dose males at termination and in both sexes after one year of treatment only. No treatment-related neoplastic lesions were observed.

2.1.7 Supplementary studies of toxicity:

Carcinogenicity: No evidence of carcinogenicity was observed in two two-year chronic/carcinogenicity studies in rats (see Long term above) or in mice fed fenamiphos for 18 months at 25 and 50 mg/kg/diet.

Teratogenicity:

Rats: Pregnant female FB30 rats were orally administered (gavage) fenamiphos (92.5%) at doses of 0, 0.3, 1.0 and 3.0 mg/kg b.w. day from day 6 to day 15 of gestation.

Cholinergic signs were observed in 18 out of 25 dams receiving 3 mg/kg b.w./day. No signs of toxicity were observed at 0.3 and 1.0 mg/kg b.w./day.

It was concluded that fenamiphos at doses up to and including 3.0 mg/kg b.w./day was not embryotoxic or teratogenic in FB30 rats.

Rabbits: Pregnant female Chinchilla rabbits were orally administered (gavage) fenamiphos (91% pure) at doses of 0, 0.1, 0.5 and 2.5 mg/kg b.w./day from day 6 through day 18 post coitum.

Maternal toxicity including death was observed at 5 mg/kg b.w./day.

Based on the maternal toxicity observed at the high dose level, the NOEL for the females was 0.5 mg/kg b.w./day. Fenamiphos was not considered to be embryotoxic or teratogenic to Chinchilla rabbits.

Reproduction: No adverse effects on reproduction were observed in a three-generation study in rats receiving fenamiphos at 0, 3, 10 and 30 mg/kg of diet.

Mutagenicity: Fenamiphos showed no mutagenic activity in a micronucleus test, in the Ames test and in *Escherichia coli*. In a dominant lethal test in mice, fenamiphos did not induce alterations in male germinal cells.

Neurotoxicity: No evidence of delayed neurotoxicity was observed in hens following a single gavage dose of 5.0 mg/kg b.w. (LD₅₀) or thirty days dietary administration of 1, 3, 10 or 30 mg/kg/diet.

2.1.8 **Modification of toxicity:** No potentiation occurred when fenamiphos in combination with disulfoton was administered to male rats.

2.2 TOXICOLOGY - MAN

2.2.1 **Absorption route:** Fenamiphos may be absorbed from the gastrointestinal tract, from the lungs and through intact skin.

2.2.2 **Dangerous doses:** No published information available.

2.2.3 **Observations on occupationally exposed workers:** No published information available.

2.2.4 **Observations on exposure of the general population:** No published information available. With good agricultural practice the general public should not be exposed to hazardous amounts of fenamiphos.

2.2.5 **Observations on volunteers:** No published information available.

2.2.6 **Reported mishaps:** No published information available.

2.3 TOXICITY TO NON-MAMMALIAN SPECIES

2.3.1 **Fish:** Toxic to fish

LC₅₀ (96 hour)

Rainbow trout	0.07 - 0.11 mg/L
Goldfish	3.2 mg/L
Bluegill sunfish	0.01 - 0.017 mg/L

2.3.2 **Birds:****Oral LD₅₀**

Mallard duck (M)	1.7 mg/kg b.w.
Mallard duck	0.9 - 1.2 mg/kg b.w.
Bobwhite quail	0.7 - 1.6 mg/kg b.w.

Dermal LD₅₀

Mallard duck (M)	2.3 mg/kg b.w.
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LC₅₀ - 5 day

Mallard duck	316 mg/kg/diet
Japanese quail	59 mg/kg/diet
Bobwhite quail	38 mg/kg/diet

Reproduction: In a 19 week dietary study with mallard ducks, fertility, egg fragility and brain cholinesterase activity were not affected at the highest dose tested (16 mg/kg/diet) while egg production and hatchling survival were reduced. The NOEL was reported to be 8 mg/kg/diet.

2.3.3 **Other species:** No published information available. As an organophosphate with insecticidal activity, toxicity to bees would be anticipated.

3.0 **FOR REGULATORY AUTHORITIES - RECOMMENDATIONS OF COMPOUND**3.1 **RECOMMENDED RESTRICTIONS ON AVAILABILITY**

[For definition of categories see the 'Introduction to Data Sheets'].

Solid formulations > 30% and liquid formulations > 7.5%: Category 2

All other formulations: Category 3

3.2 **TRANSPORTATION AND STORAGE**

All formulations: Should be transported in clearly labelled, leakproof containers, away from food and drink. Storage should be under lock and key and secure from access by children and other unauthorized persons.

3.3 **HANDLING**

All formulations: Protective clothing (see part 4) should be worn for the handling of all formulations. Adequate washing facilities should be available in the immediate area. Eating, drinking and smoking should be prohibited during handling and before washing after handling.

3.4 **DISPOSAL AND/OR DECONTAMINATION OF CONTAINERS**

All formulations: Whenever possible containers should be either returned to the supplier, or safely disposed of in an approved manner. Care must be taken to avoid subsequent contamination of water sources. Decontamination of containers in order to use them for other purposes should not be permitted.

3.5 SELECTION, TRAINING AND MEDICAL SUPERVISION OF WORKERS

All formulations: Pre-employment and periodic medical examination of workers is necessary and should include blood cholinesterase tests. Special account should be taken of the workers' ability to comprehend and follow instructions. Training of workers in techniques to avoid contact is essential.

3.6 ADDITIONAL REGULATIONS RECOMMENDED IF DISTRIBUTED BY AIRCRAFT

No recommended aerial usage reported.

3.7 LABELLING

DANGER - POISON
(Skull and cross bones insignia)

Fenamiphos is an organophosphorus compound which inhibits cholinesterase enzymes. It is of very high toxicity. Contact with the skin, inhalation of dust or spray, or swallowing may be fatal. Wear protective gloves, clean protective clothing, and a respirator of the organic-vapour type when handling this material. Bathe immediately after work. Ensure that containers are stored under lock and key. Empty containers must be disposed of in such a way as to prevent all possibility of accidental contact with them. Keep the material out of reach of children and well away from foodstuffs, animal feed and their containers. In case of contact, immediately remove contaminated clothing and wash the skin thoroughly with soap and water; for eyes, flush with water for 15 minutes. If poisoning occurs, call a physician. Atropine sulfate is a pharmacological antidote. Artificial respiration may be needed.

3.8 RESIDUES IN FOOD

Maximum Residue Levels (MRLs) have been recommended by the FAO/WHO Joint Meeting on Pesticide Residues (JMPR). The Acceptable Daily Intake (ADI) has been estimated by the JMPR at 0 - 0.0005 mg/kg b.w.

4.0 PREVENTION OF POISONING IN MAN AND EMERGENCY AID

4.1 PRECAUTIONS IN USE

4.1.1 **General:** Fenamiphos is an organophosphorus pesticide of high toxicity. It is readily absorbed through intact skin from the gastrointestinal tracts and by inhalation. Repeated exposure may have a cumulative effect on cholinesterase activity. Fenamiphos formulations should only be handled by trained personnel wearing protective clothing. Its use is severely restricted in several countries.

4.1.2 **Manufacture and formulation:** TLV - 0.1 mg/m³. This time-weighted average value has been determined from risks associated with skin exposure and subsequent dermal absorption.

Dust and aerosol formation must be controlled, preferably by mechanical means. Protective clothing (see 4.1.3) and respiratory equipment is necessary to reduce dermal and inhalation exposures.

4.1.3 **Mixers and applicators:** When opening the container and when mixing, protective impermeable boots, clean overalls, neoprene gloves and respirator should be worn. Mixing, if not mechanical, should always be carried out with a paddle of appropriate length. When spraying tall crops or during aerial application, a face mask should be worn, as well as an impermeable hood, clothing, boots, and neoprene gloves. The applicator should avoid working in spray mist and avoid contact

with the mouth. Particular care is needed when equipment is being washed after use. All protective clothing should be washed immediately after use, including the insides of gloves. Splashes must be washed immediately from the skin, or eyes with large quantities of water. Before eating, drinking, or smoking, hands and other exposed skin should be washed.

- 4.1.4 **Other associated workers:** Persons exposed to fenamiphos and associated with its application should wear protective clothing and observe the precautions described above in 4.1.3 under "Mixers and applicators".
- 4.1.5 **Other populations likely to be affected:** With good agricultural practice and subject to section 4.2 and 4.3 below, other populations should not be exposed to hazardous amounts of fenamiphos.

4.2 ENTRY OF PERSONS INTO TREATED AREAS

Unprotected persons should be kept out of treated areas for at least four days.

4.3 SAFE DISPOSAL OF CONTAINERS AND SPILLAGE

Residues in containers should be kept to a minimum and emptied in a diluted form into a deep dry pit (depth over 0.5 m), taking care to avoid contamination of ground waters. The empty containers should be disposed of in an approved manner. If not returned to the producer re-use of containers should not be permitted for any purpose.

Spillage of liquid fenamiphos formulations should be covered with absorbent material. This material or spillage of dry residues should be collected and burned or buried as described above. Residues should be removed by scrubbing with detergent and then rinsing with large quantities of water.

Impermeable gauntlets and protective overalls should be used for all handling procedures.

4.4 EMERGENCY AID

- 4.4.1 **Early symptoms of poisoning:** Early symptoms of poisoning may include excessive sweating, headache, weakness, giddiness, nausea, vomiting, increased salivation, stomach pains, diarrhoea, blurred vision, slurred speech and muscle twitching. Later there may be shortness of breath, convulsions and coma.
- 4.4.2 **Treatment before person is seen by physician, if these symptoms appear following exposure:** The person should stop work immediately, remove contaminated clothing and wash contaminated skin with soap and water and flush the area with large quantities of water. If swallowed, and if the person is conscious, vomiting should be induced. Artificial respiration should be given when necessary bearing in mind that if mouth-to-mouth resuscitation is used, vomit may contain toxic amounts of pesticide. Call a physician immediately or organize immediate transport to a physician or hospital.

5.0 FOR MEDICAL AND LABORATORY PERSONNEL

5.1 MEDICAL DIAGNOSIS AND TREATMENT IN CASES OF POISONING

- 5.1.1 **General information:** Fenamiphos is an organophosphorus pesticide of high mammalian toxicity. It is readily absorbed through intact skin and by inhalation. It acts by direct inhibition of acetylcholinesterase affecting nerve transmission.

- 5.1.2 **Symptoms and signs:** Poisoning is due to excessive stimulation by acetylcholine of all cholinergic innervation. Thus initial symptoms and signs of poisoning may include excessive sweating and salivation, headache, weakness, miosis, dyspnoea, nausea, vomiting and diarrhoea, blurred vision and muscle fasciculations. More severe poisoning leads to respiratory failure due to a combination of bronchorrhea, bronchoconstriction (muscarinic effects), paralysis of respiratory muscles (nicotinic effects) and respiratory centre paralysis (central effects). The latter includes, in severe cases, coma and convulsions.
- 5.1.3 **Laboratory:** Diagnosis is confirmed by finding inhibition of erythrocyte or whole blood acetylcholinesterase. However, treatment must start immediately and cannot be delayed until confirmation from the laboratory. This test cannot be used to control the effectiveness of the treatment nor is it of help for prognosis.
- 5.1.4 **Treatment:** Patients with respiratory failure must be given artificial ventilation, then diazepam (10 mg intravenously) to control convulsions. When vital functions are controlled, atropine sulfate is given (initial dose is usually 2 mg intravenously) followed by pralidoxime (1000 mg) or toxogonin (250 mg) by slow intravenous infusion.

If the pesticide has been ingested, gastric lavage might be needed or vomiting induced. Protection of airways (intubation) is required if inducing vomiting in unconscious patients.

For skin contact, the skin should be washed with soap and large amounts of water. Precautions should be taken by medical personnel during these decontamination procedures to prevent their own overexposure. If the compound has entered the eyes, they should be washed with large quantities of saline or water.

Atropine treatment might be required for several days after poisoning. Only clinical assessment determines atropine dose, i.e. evident signs of atropinization (dry mouth, tachycardia, vasodilation, mydriasis) should be maintained. Total amounts of atropine given to these patients might be extremely high because they are tolerant to the effects of atropine.

Caution should be taken when doses of atropine are reduced because reappearance of symptoms might occur, due to redistribution processes in the body. Cholinesterase reactivators such as pralidoxime and toxogonin are usually only effective during the first few days of poisoning, unless the slow disposal of the chemical within the body suggests that some acetylcholinesterase is newly inhibited. Indications for the continuing use of reactivators might derive from measurements of erythrocyte cholinesterase before and after treatment with such reactivators.

- 5.1.5 **Prognosis:** Unless brain hypoxia has occurred, full recovery is expected.
- 5.1.6 **References to previously reported cases:** No published information available.

5.2 SURVEILLANCE TESTS

Any fall in erythrocyte cholinesterase activity to 70% of the pre-exposure value, requires an investigation of working methods and hygiene and more frequent cholinesterase tests. Symptoms of poisoning may appear when the erythrocyte cholinesterase activity is less than 35% of pre-exposure value. If erythrocyte cholinesterase activity is less than 50% of normal, the worker must be suspended from all contact with organophosphorus or carbamate pesticides until the level rises above 70% of the pre-exposure value. Pseudocholinesterase activity in the plasma can fall to very low levels without evidence of symptoms. This only indicates undesirable exposure.

5.3 LABORATORY METHODS

5.3.1 Detection and assay of compound:

A specific gas-chromatographic procedure for the determination of residues of fenamiphos and sulfoxide and sulfone is available. Some basic references are listed as follows:

Luke MA, Froberg JE, Doose GM, Masumoto HT (1981), *J Assoc Off Anal Chem* **64(5)**: 1187-1195.

Peterson D, Winterlin W (1986), *J Agric Food Chem*, **34(2)**: 153-156.

Thornton JS, (1971), *J Agric Food Chem* **19**: 890-893.

5.3.2 Other tests in case of poisoning: Activity of cholinesterase in the blood or plasma provide the most useful diagnosis of poisoning.

Ellman GL et al (1969), *Biochem Pharmacol* **7**: 88-95.

Wilhelm K, & Reiner E (1973), *Bull Wld Health Org*, **48**: 235-238.

REFERENCES

1. FAO/WHO (1988), *Pesticide Residues in Food - 1987 Evaluations, Part II - Toxicology*, FAO Plant Production and Protection Paper 86/2, Rome, Food and Agriculture Organization of the United Nations.
2. *The Pesticide Manual, A World Compendium* (9th edition 1991), Worthing, C.R. and Hance, eds., British Crop Protection Council, 20 Bridport Road, Thornton Heath, CR4 7QG, United Kingdom.
3. WHO (1975), *1974 Evaluations of Some Pesticide Residues in Food*. WHO Pesticide Residue Series No. 4, 295-333, Geneva, World Health Organization.
4. WHO (1986), *Environmental Health Criteria No. 63, Organophosphorus Insecticides: A General Introduction*. UNEP/ILO/WHO Geneva.
5. WHO (1994), *The WHO Recommended Classification of Pesticides by Hazard and Guidelines to Classification 1994-1995*, Geneva, World Health Organization mimeographed document (WHO/PCS/94.2).

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