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READ THE ENTIRE LABEL BEFORE USING THIS PRODUCT.

USE ONLY IN ACCORDANCE WITH INSTRUCTIONS.

KEEP OUT OF REACH OF CHILDREN

STEADFAST

INGREDIENTS

Fenitrothion
Other ingredients

STEADFAST is an organophosphorous insecticide. It is a non-systemic insecticide with contact and stomach action. Its active ingredient is fenitrothion.

STEADFAST is a cholinestrase inhibitor. It is used to control insects in stored cereals and hides and skins, pasture pests and to control locusts. It is also used as STRIKE ULV as an outdoor fogger for control of insect pests (flies, mosquito etc).

Trade Names Of Other Firms: Trade names for products containing Imidacloprid include Accothion, Agrothion, Bay 41831, Cyfen, Cytel, Dicofen, Fenstan, Folithion, Kaleit, Mep, Metathion, Micromite, Novathion, Nuvanol, Pestroy, Sumanone, Sumithion, and Verthion (112, 22, 138, 111, 116).

What is STEADFAST and how does it work?

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It works by interfering with the transmission of stimuli in the insect nervous system. Specifically, it causes a blockage in a type of neuronal pathway (nicotinic) that is more abundant in insects than in warm-blooded animals (making the chemical selectively more toxic to insects than warm-blooded animals). This blockage leads to the accumulation of acetylcholine, an important neurotransmitter, resulting in the

insect's paralysis, and eventually death. It is effective on contact and via stomach action.

Key Benefits of STEADFAST:

1. Quick knockdown effect.
2. Highly effective
3. Available in ULV for application flexibility

PRECAUTIONS

Harmful if swallowed, inhaled or absorbed through skin. Causes eye irritation. Avoid contact with skin, eyes or clothing. Avoid breathing dust or vapor. Wash thoroughly with soap and water after handling. Remove contaminated clothing and wash before reuse. Keep children or pets away from treated area until dry.

SYMPTOMS OF POISONING

Irritation on skin or eyes.

MEDICAL TREATMENT

Treatment is symptomatic.

FIRST AID

If on skin, remove contaminated clothes. Rinse and then rinse skin immediately with plenty of water and soap for 15-20 minutes. Call a poison control centre or doctor for treatment advice. If inhaled, move person for fresh air. If person is not breathing, call for an ambulance, then give artificial respiration, preferably mouth-to-mouth if possible. Call a poison control centre or doctor for further treatment advice. If in eyes, first hold eye open and rinse with plenty of water for 15-20 minutes (remove contact lenses if easily possible). Call poison control center or doctor for treatment advice. If ingested, call a poison control centre or doctor immediately for treatment advice. Have person sip a glass of water if able to swallow. Do NOT induce vomiting unless told to do so by poison control center or doctor. Do not give anything to an unconscious person.

DIRECTIONS OF USE

Poultry Sheds

STEADFAST is one of two chemicals registered for the control of lesser mealworm (Litter **Beetle**, **Darkling Beetle** or Black **Beetle**) infestations in broiler sheds.

Advice from growers indicates that the recommended dose is usually applied twice, 4-6 days apart by boom sprayers and, in some cases, airblast orchard sprayers (for walls). The spray is applied to the cleaned floors of sheds, walls and in a band around the outside of the

shed wall and the shed left to dry and air for a couple of days.

Litter (usually wood shavings) is then placed in the shed and the shed left to stand for a few more days. Chickens are then introduced and no more chemical is applied during the growing period. A period of 7-10 days normally elapses between the removal of a finished batch of chickens and the introduction of a new batch.

Occasionally, farmers will keep part of the litter from the previous batch and this litter is treated in the same operation as the shed floors and walls at the same rate of use and by the same method. Under normal circumstances, there is no treatment of litter at all.

Poultry Sheds and Hides and Skins

Hand held wands, boomsprayers and orchard sprayers are used in poultry sheds to cover walls and floors, while hand held wands appear to be used for hides and skins.

Poultry Sheds

Although adult lesser meal worm may lodge in the throats of young chicks and thereby cause physical harm to birds, the main problems are the damage larvae do to the building insulation and the potential for tapeworm transmission.

Physical barriers and decoy pupation sites (to avoid larvae tunneling into insulation) are possible alternative control strategies but these have not been taken up by producers. Because fenitrothion has proved satisfactory in this situation, there does not appear to have been any trial work undertaken to find alternative chemical treatments.

However, advice has been received during the review that the Queensland Department of Primary Industries has commenced a project to investigate control of lesser mealworm in poultry housing (including use of fenitrothion) which is scheduled to be completed in mid-2000.

DISPOSAL METHODS

Do not dispose of undiluted chemicals on site. If recycling, replace cap and return clean containers to recycler or designated collection point. If not recycling, break, crush, or puncture and bury empty containers in a local authority landfill. If no landfill is available, bury the containers below 500 mm in a disposal pit specifically marked and set up for this purpose clear of waterways, desirable vegetation and tree roots. Empty containers and product should not be burnt.

STORAGE CONDITION

Store in the closed, original container in a cool, well-ventilated area. Do not store for prolonged periods in direct sunlight. Store in a locked room or place away from children, animals, food, feedstuffs, seed and fertilizers. Triple or preferably pressure rinse containers before disposal. Add rinsing to spray tank.

For More Details including effects on environment email contact@ivorychem.com with Subject "STEADFAST DETAILS"

More Details:

TOXICOLOGICAL EFFECTS

- **Acute Toxicity:** The acute toxicity of fenitrothion to mammals is considered to be low (120, 2, 125). Typical symptoms of acute poisoning are observed in rats at doses considerably higher than those applied for parathion-methyl, a structural analogue of this substance (146). The acute oral LD50 for rats ranges between 250-800 mg/kg; 715-870 mg/kg for mice; and 500 mg/kg for guinea pigs. The acute dermal LD50 for rats is >890 mg/kg and >3,000 mg/kg for mice. The acute inhalation LC50 in rats was reported to be 5.0 mg/l (13, 111, 112, 113, 111, 116, 148). Other lethal dose values for rats were given as: 378 mg/m³/4-hour inhalation LC50; 950 mg/kg intratracheal LD50; 33 mg/kg intravenous LD50; 300 mg/kg intraperitoneal LD50 (113). Another source reported the dermal LD₅₀ for rats to be 300 mg/kg (116). Mice had acute toxicity values of 2500 mg/kg dermal LD50; 229 mg/kg oral LD50; 1,000 mg/kg subcutaneous LD50; 280 mg/kg intraperitoneal LD50; and 1,000 mg/kg intracerebral LD50 (113). Guinea pigs were reported to have acute toxicity values of 500 mg/kg oral LD50; and 112 mg/kg intravenous LD50 (113). The oral acute toxicity for cats was 142 mg/kg (113). Studies reported primary dermal irritation; mild dermal irritation was reported in a rabbit study. Primary eye irritation was also reported; mild irritation was seen after a single application of 0.1 ml of fenitrothion into unwashed eyes of albino rabbits (148). The acute oral toxicity reported for a human female was a TD₀₁ of 800 mg/kg (113).
- **Chronic Toxicity:** Chronic symptoms in

humans include: general malaise, fatigue, headache, loss of memory and ability to concentrate, anorexia, nausea, thirst, loss of weight, cramps, muscular weakness and tremors. Fenitrothion at sufficient dosage produces typical cholinergic poisoning (2, 125). In a study with rats, a dietary level of 500 ppm for 90 days was tolerated. They grew normally, and cholinesterase in plasma, red cells and tissues was decreased. A dietary level of 30 ppm for six months decreased the red cell and brain cholinesterase of female but not male rats; neither sex showed any sign of toxicity. A dietary level of 5 ppm for 92 weeks was a no-effect-level (NEL) (2, 125). Mice that received fenitrothion at a dietary level of 1000 ppm developed symptoms within a week and at the end of a 20-day feeding period had cholinesterase activity in brain, red cells, and plasma reduced to 45, 26 and 5% of normal, respectively. Monkeys are more susceptible than dogs. A dosage of 2 mg/kg/day produced no effect on serum or erythrocyte cholinesterase in dogs but after 2 months of administration, did cause a reduction of erythrocyt enzyme activity in monkeys. A dietary concentration of 5 ppm was found to be a NEL in calves (2, 125). Adverse effects and death were observed in rats given a diet containing 400 ppm for 63 weeks. Some of the animals survived, although at this level there was a 100% drop in erythrocyte cholinesterase. In 1.77-year feeding trials, the NEL for rats was 5 mg/kg diet (111). In dogs, doses of 0, 2, 9 and 40 mg/kg body weight/day of fenitrothion were administered for 98 days; at 40 mg/kg/day signs of poisoning and cholinergic stimulation were observed (146). Rats receiving a diet containing 10 ppm showed a slight drop in erythrocyte cholinesterase activity after 5 weeks of treatment; activity returned to normal 2 weeks after treatment stopped; with a 20 ppm level dose there was a reduction in erythrocyte and brain cholinesterase activity. No significant effect on cholinesterase activity was observed in plasma or erythrocytes at a dietary level of 20 ppm, and it was only with 100 ppm or more that effects were observed; enzyme activity returned to normal 30-40 days after the end of treatment (146). Rats fed on a diet containing 400 ppm for two years showed a 100% drop in erythrocyte cholinesterase activity. At 100 ppm, 10-

30% depression of brain and 30-65% depression of erythrocyte and plasma cholinesterase activity occurred (146). In dogs, a slight depression in blood plasma and erythrocyte cholinesterase activity was observed after 60 days with a dosage level of 9 mg/kg/day. Moderated depression occurred with 40 mg/kg/day for 29 days (146). Daily feeding of 100 mg/kg body weight/day over 60-90 days to dairy cows and sheep does not result in its excretion in milk (13). Other studies indicated that there is a significant inhibition of growth and various cholinergic signs for 2 to 3 weeks following administration of 500 ppm fenitrothion in rats (120). The no-observable-effect-level (NOEL) for brain and red blood cell cholinesterase is 10 ppm, while the systemic NOEL for plasma inhibition in dogs is 5 ppm (148). Sumithion 50EC (a product containing fenitrothion) has been shown to cause delayed neurotoxicity in adult rats, as well as humans (143).

- **Reproductive Effects:** Damage to the nuclear membrane, decreases in staining capacity of cells, and an increase in anomalous mitoses were reported in monolayer cultures of fibroblasts taken from rats that received 0.1 or 0.2 of the LD50 level of fenitrothion daily during the first 15 days of pregnancy. However, the results were not dosage related, nor was there any change in rate of proliferation or mitotic phase distribution from that of the controls (2, 125). Behavioral deficits have been noted in newborn mammals (115). Results from a study where pregnant rats were treated with 0, 5, 10 and 15 mg/kg of the product Sumithion 50EC daily through gestation days 7 to 15, showed the following results: There were no significant differences in number of pups born per litter, weight per litter or day of eye and ear opening. There was a significant difference in mortality up to day 16 postpartum: at the 15 mg/kg dose, 17.5% of the pups died; at the 10 and 5 mg/kg dose, 16.0% of the pups died; at the 0 mg/kg dose, 5% of the pups died. One pup in the 15 mg/kg group was anophthalmic and one developed tremor and ataxia on day 16, and thus both were excluded from the study. The remaining pups gained weight normally and showed no overt signs of intoxication. No significant behavioral effects could be measured at the lowest dose of 5 mg/kg/day. At the 10 and 15 mg/kg/day doses, while

- several of the behavioral outcomes were significantly different from controls, there seemed to be a difference between the "simple" behavioral measures such as motor activity and motor coordination and the more "complex" measures such as conditioned escape and social interactions. Behavioral measures showed significant alterations as long as 104 days following birth, indicating that prenatal intoxication with Sumithion had persistent effects that showed the offspring to be different from untreated animals. The lack of effect at the 5 mg/kg/day dose indicates that this chemical has a steep dose-response function and that exposure of agricultural workers should be carefully monitored (143).
- **Teratogenic Effects:** No teratogenic effects were observed in albino rabbits dosed with 0, 0.3 or 1 mg fenitrothion/kg/day in gelatine capsules on gestation days 6 through 18 (149, 151).
 - **Mutagenic Effects:** No mutagenic effects were seen in *Drosophila melanogaster* or mice (2, 125).
 - **Carcinogenic Effects:** In a two-year feeding study in rats (50 males and 50 females), no dose-related increase in tumor incidence was found upon histopathological examinations of all groups (149, 151, 152). Fenitrothion was administered in the diet to groups of 50 male and 50 female ICR Swiss mice at dose levels of 0, 30, 100 and 200 ppm for 78 weeks. There was no evidence of compound-related effects on appearance and behavior, body weight or mortality. Gross necropsies revealed no consistent compound-related changes in any organs or tissues. The histopathological examinations revealed no consistent treatment-related increase in tumor incidences (149, 152).
 - **Organ Toxicity:** One of the contaminants of fenitrothion, O,O,S-trimethyl phosphorothioate, has a distinct cytotoxic effect on the lungs of rats and is known to modulate immune responses in mice (2, 125). Fenitrothion is an immunotoxin (115). In patients who died of pesticide poisoning, 240 ppm fenitrothion were found in the liver (2). Fenitrothion is considered a suspect viral enhancer, implicated in Reye's syndrome (115).
 - **Fate in Humans and Animals:** Fenitrothion is oxidized by mono-oxygenases in animals, insects and plants and is thereby changed to derivatives containing the P=O group, which are more powerful inhibitors of cholinesterase than was the original thiophosphate. After that, further degradation occurs by rupture of a P-O-CH₃ linkage which is more quickly metabolized in the liver than the P-O phenyl linkage rupture occurring with parathion, which could contribute to fenitrothion's low mammalian toxicity (2). Studies in the mouse, rat and guinea pig have shown that fenitrothion is rapidly absorbed from the mammalian gastrointestinal tract. The presence of the oxygen analogue has been demonstrated in all tissues examined and this oxygen analogue has been detected in blood one minute after an intravenous injection of fenitrothion (146). Daily fenitrothion doses of 2.5 and 5 mg/man/day for 5 days were excreted within a 12 hour period and there was no indication of accumulation. Fenitrothion applied to the skin of rats disappeared most rapidly in the first hour, suggesting an absorption rate of slightly over 1%. After 31 hours, the highest concentration, other than on the skin, was found in the cartilaginous part of the bones (113, 2, 125). When volunteers were given single oral doses ranging from 2.5 to 20 mg/person, the maximal concentration of p-nitro-m-cresol in the urine was reached within 12 hours, and nearly the entire amount discharged was eliminated during the first 24 hours. Although the amount recovered was directly dosage-related, the proportion recovered was inversely dosage-related. With one exception, cholinesterase activity remained normal following these doses (2, 125). The half-life of fenitrothion was noticeably longer after 10 doses of 30 mg/kg/day than after a single dose of 300 mg/kg. It was concluded that this effect was caused by suppression of the metabolism of the compound during its repeated administration and was associated with inhibition of demethylation and hydrolysis by microsomal enzymes (2, 125). Fenitrothion is decomposed rapidly in tissues to desmethylsumition, dimethyl-phosphorothioic acid and phosphorothionic acid (120). The oxygen derivative of fenitrothion is formed in the microsomal fraction of the cell, the main metabolizing organs being the liver and kidneys. The major excretion product is 3-methyl-4-nitrophenol, which can be further oxidized to 3-carboxy-4-nitrophenol.

Another metabolite is the desmethyl-derivative (146). Eighteen people were subjected to clinical examination while spraying fenitrothion. The level of blood plasma cholinesterase was determined at regular intervals but no abnormalities were found. Blood cholinesterase was analysed in a large number of inhabitants of Nigeria where fenitrothion had been sprayed. After spraying for one week, a 50% reduction in blood cholinesterase in 20 spraymen was recorded. Rapid return to normal levels subsequently took place (146). Single oral doses of between 2.5 and 20 mg fenitrothion (approximately 0.042 to 0.33 mg/kg body weight) were administered orally to 24 human subjects. The urinary excretion of the metabolite 3-methyl-4-nitrophenol was almost complete in 24 hours, the excretion peak occurring after 12 hours. The plasma cholinesterase level did not decline, except in one of the subjects who had received 0.33 mg/kg fenitrothion (146).

ECOLOGICAL EFFECTS

- **Effects on Birds:** Negative results were observed in studies on delayed neurotoxicity in hens (113, 148). The oral LD50 for chickens was reported as 28 mg/kg (116). Fenitrothion was found to be highly toxic to upland gamebirds and slightly toxic to waterfowl (acute oral toxicity value to bobwhite quail and mallards was determined to be 23.6 mg/kg and 1,190 mg/kg, respectively) (112, 148). The LC50 for pheasants was 450 to 500 ppm in diets of 2-week-old birds when fed fenitrothion-treated feed for 5 days, followed by untreated feed for 3 days (27).
- **Effects on Aquatic Organisms:** The time for achieving the highest levels of uptake and the extent of retention of organophosphate residues by fish was directly related to the extent of persistence of a compound in water. Mutsugo fish exposed to 0.6-1.2 mg/l of fenitrothion attained the highest body concentrations (162 mg/kg) after 3 days. Fenitrothion (4.9 mg/kg) persisted longer than 4 weeks in fish (153). Fenitrothion is considered somewhat toxic to fish (22). The 96-hour LC50 was 1.7 ppm for brook trout and 3.8 ppm for bluegill sunfish; moderately toxic to both warmwater and coldwater fish (112, 148). The 96-hour LC50 to various species of North American freshwater fish has also been reported as 2-12 micrograms/l. The chronic toxicity of fenitrothion to fish is considered low (122). The 48-hour LC50 values for carp ranged between 2.0 mg/l and 4.1 mg/l (13, 111). One source stated that aerial spraying of fenitrothion at 2 or 3 oz/acre, on New Brunswick forests has been reported to have no deleterious effect on fish in streams in the treated area (153). In a study on the acute toxicity of fenitrothion to rainbow trout, embryos were found to be the least sensitive, the sac fry stage was intermediate, and fingerlings and adults were the most sensitive. The toxicity of fenitrothion to rainbow trout increased with increasing temperature. The sublethal effects of fenitrothion exposure on fish include:
 - **Morpho Anatomical Changes:** Swelling of the abdomen of fathead minnows occurred. Young Atlantic salmon exposed to 1 mg/l swam with distended fins.
 - **Behavioral Changes:** There was a pronounced decline in various agonistic behaviors (chasing, vacating, nipping, etc.) within 2 hours of exposure to several concentrations of fenitrothion. Comfort behaviors (flicks, thrusts, etc.) increased with increasing concentration of toxicant, but declined at higher concentration. Altered station selection occurred. At higher concentrations, some fish were unable to maintain position and were swept downstream. After a 5-hour exposure, fish swam near the surface with bloated stomachs and heads pointing downward. Movement was slowed so much that Atlantic salmon did not attempt to avoid capture with a dipnet. Salmon parr exposed to 1 mg/l fenitrothion were more vulnerable to predation by brook trout.
 - **Biochemical Changes:** Acetylcholinesterase activity was inhibited 13% to 25% after various sublethal concentrations of fenitrothion. Cholinesterase activity in the erythrocytes, gills, heart, and serum of rainbow trout was reduced within 1 hour after exposure to fenitrothion.
 - **Respiratory Effects:** Oxygen

consumption of *Labeo rohita* exposed to fenitrothion progressively decreased with increasing concentrations of insecticide. Exposure caused increased ventilation rate and buccal amplitude at concentrations slightly higher than the 48-hour LC50.

- **Effect on Growth:** Orally administered fenitrothion had no effect on the growth of rainbow trout (122). The compound is considered very toxic to crustaceans and aquatic insects and has a medium toxicity to aquatic worms (115). A freshwater invertebrate toxicity (48-hour or 96-hour EC50) reported fenitrothion to be very highly toxic to aquatic invertebrates (3 ppb for *Gammarus fasciatus*) (148).
- **Effects on Other Animals (Nontarget species):** There is sufficient information to characterize fenitrothion as highly toxic to honeybees (acute toxicity value = 0.383 micrograms/bee) when bees are exposed to direct treatment or to dried residues on foliage (13, 22, 147). Fenitrothion is considered toxic to spider mites with long residual action (120). Fenitrothion, applied to host eggs at field rates in the laboratory were found to be highly toxic to *Trichogramma orasiliensis* released on the eggs, causing 84-100% mortality in 24 hours (121). The long-term effects of fenitrothion and phosphamidon were evaluated on predaceous carabid beetles and lycosid spiders one year after treatment of Northwestern Ontario forests at 6 oz/A and 4 oz/A, respectively. The populations of these predators were clearly suppressed in the treated area. The results "did not imply a one year persistence of the insecticides, but rather a persistent disturbance of the ecosystem" (27). The acute oral toxicity of fenitrothion to mule deer was reported to be 727 mg/kg (27).

ENVIRONMENTAL FATE

In studies of lesser date moth control, fenitrothion was added to a 1:1 mixture of wheat flour and pollen grains. This mixture was dusted on female clusters of date palms at the time of pollination. Not only did it prove to be effective, but this method of application was less environmentally polluting than the use of high-pressure sprays (141).

- **Breakdown of Chemical in Soil and Groundwater:** Preliminary data indicates fenitrothion degrades fairly rapidly in soil with a half-life of less than one week in non-sterile muck, sandy loam soils. The compound is intermediately mobile in a variety of soils ranging from sandy loam to clay (112, 148).
- **Breakdown of Chemical in Surface Water:** Surface foam on lakes acts as a scavenger and a trap for organic pollutants. Following aerial spraying of fenitrothion, 701 micrograms/l of fenitrothion was recorded in a surface slick formed by wind actions, compared to 9.5 micrograms/l in the subsurface water (153). Another study indicated the half-life for the disappearance of fenitrothion at 23 degrees C and pH 7.5 in buffered lake water and natural lake water in the dark (10 ppm sol.) was 21.6 and 49.5 days, respectively. In a field experiment (pH 7.0-7.5, 19-23 degrees C), the half-life of fenitrothion was 1.5-2 days upon spraying of a 10% fenitrothion EC-formulation at a rate of 4 oz/A to a model water system (149).
- **Breakdown of Chemical in Vegetation:** Damage to cabbage and fruit is possible only if the application dose is exceeded. Fenitrothion has been known to be phytotoxic to cotton, Brassica crops, and certain fruit crops when high rates were applied. Certain apple varieties may be russeted (13, 22). In a study conducted by FAO/WHO, about 50% of ³²P-labelled fenitrothion sprayed on rice plants penetrated into the tissues in 24 hours. At the end of this period only 10% was left, indicating rapid decomposition. Some fenitrothion oxon was formed but it disappeared from the tissues more rapidly than fenitrothion. Rice grains harvested 46 days after treatment contained 0.0007 ppm fenitrothion and less than 1 ppm of p-nitrocresol and dimethyl phosphorothioic acid (149). Although the oxon may form in plants, it occurs only during the first few days after treatment and in proportions (ca 1%) smaller than those in animals. Desmethyl compounds occur only in minor amounts in plants. The half-life of fenitrothion in green plants ranges between the values established for Parathion and Parathion-Methyl, i.e. between one and two days; the half-life of the oxon is estimated to be only a few hours (FAO/WHO) (149).
- **Breakdown of Chemical in Air:** An experiment was carried out in a vacant

dormitory building in order to establish the airborne residue of concentrations of seven pesticides used for cockroach control. Airborne concentrations of fenitrothion on the day of application were 3 micrograms/cubic meter. All were below 0.7 micrograms/cubic meter by the third day after application. The airborne concentrations correlated well with the vapor pressures of the various pesticides (2).

PHYSICAL PROPERTIES AND GUIDELINES

Physical Properties:

- **Appearance:** pure material forms a yellowish brown liquid with an unpleasant odor (2, 125)
- **Chemical Name:** O,O-dimethyl O-4-nitro-m-tolyl phosphorothioate (IUPAC), O,O-dimethyl O-(3-methyl-4-nitrophenyl) phosphorothioate (CA), O,O-dimethyl O-(3-methyl-4-nitrophenyl) thiophosphate (13)
- **CAS Number:** 122-14-5 (13, 113)
- **Molecular Weight:** 277.25 (116, 2, 125)
- **Water Solubility:** In water at 20 degrees C, 30 mg/l (13, 144, 149); at 30 degrees C, 14 mg/l water (111); nearly insoluble in water (141, 112); insoluble in water (2)
- **Solubility in Other Solvents:** Readily soluble in common organic solvents, e.g. acetone, alcohol, benzene and chlorinated hydrocarbons (13). dichloromethane, 2-propanol, toluene (112). Hardly soluble in n-hexane (112). Soluble in ethers, methanol, xylene, ketones, esters, and aromatic hydrocarbons. Low solubility in aliphatic hydrocarbons (113, 2). At 20 -25 degrees C, > 1 kg/kg dichloromethane, methanol and xylene, 42 g/kg hexane, 0.1 - 1.0 kg/kg propan-2-ol. It is hydrolyzed by alkali; at 30 degrees C, 50% loss occurs in 4.5 hours in 10M sodium hydroxide (111)
- **Melting Point:** 0.3 degrees C (148)
- **Vapor Pressure:** 7 x 10 to the minus 5 mbar at 20 degrees C (13); 18 mPa at 20 degrees C (111)
- **Partition Coefficient:** 2380 (149)
- **Adsorption Coefficient:** Not Available
- **Stability:** Fenitrothion is completely stable for two years if stored at temperatures between 20 and 25 degrees C. Storage temperature should

not exceed 40 degrees C. It is unstable in alkaline media (2, 125). The thermal stability of this compound is low, and when it is heated above 100 degrees C it undergoes Pishchemuka isomerization and may decompose explosively. It must be stored in enameled, aluminum or glass containers. Iron promotes decomposition of fenitrothion (144).

- **Specific gravity:** 1.3227 (113); 1.32-1.34 (120, 148); 1.3084 at 20 degrees C (144, 2, 125)
- **Boiling point:** 109 degrees C at 0.13 mbar; 164 degrees C at 1.3 mbar (13). 140-145 degrees C/0.1 mmHg (111, 2, 125, 149). 244 degrees F (118 degrees C) at 0.05 mmHg (113). 118 degrees C at 0.01 mmHg (148)
- **Flashpoint:** 166 degrees C (closed cup) (149)
- **Volatility:** 0.09 mg/m³ (144)

Exposure Guidelines:

- **ADI:** temporary for man 0.003 mg/kg (until 1986) (111) 0.005 (146)
- **MCL:** Not Available
- **RfD:** Not Available
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** Not Available



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