

**APPENDIX 1
(OF APPENDIX B)**

CARBOFURAN

Trade Name: Furadan

Chemical name: 2,3-dihydro-2,2-dimethyl-benzofuranyl methyl carbamate

Other Names: Bay 70143; Coraterr; D1221

Melting Point: 150-152°C

Vapor Pressure: 2×10^{-5} mm Hg

Solubility: 700 ppm

Introduction:

Carbofuran was first produced by the FMC Corporation in Middleport, New York and was released under the trade name Furadan in 1969. It is a broad spectrum systemic insecticide, acaricide, and nematocide coming in several formulations, including Furadan granules and Furadan 4 flowable. EPA has proposed a Maximum Contaminant Level (MCL) and Suggested No Adverse Response Level (SNARL) of 36 ppb.

Registered Uses:

Carbofuran is registered for use on a wide variety of crops ranging from corn to grapes. It controls corn rootworm, alfalfa weevil, aphids, nematodes, leafhoppers and phloxera in grapes and most other foliar and soil pests. In rice cultivation it is applied to control rice water weevils.

Degradation:

Carbofuran degrades into CO₂, methylamine, and the corresponding 7-phenol (Figure 1). Neither carbofuran nor any of its metabolites have potential for biomagnification. Furthermore, all breakdown products are less toxic than the parent compound (FMC Co. 1969). Repeated applications of carbofuran in successive years does not apparently result in an increase in soil or crop residues (FMC Corp., 1969) as 7-phenol binds strongly and irreversibly to soil where it slowly degrades.

Environmental Fate:

Insensee and Tayaputch (1986) evaluated the environmental fate of C¹⁴ carbofuran by applying it as a wettable powder to test soils at concentrations of 6 and 12 ppm. Twenty five days after treatment and seven days after flooding of the experimental chambers, less than 20% of the ¹⁴C remained in the treated soil while 50% had leached down and out the bottom of the sampling tubes (approximately 15-20 cm). The peak carbofuran concentration in the overlying water was reached 9 days after flooding and amounted to about 12% of the amount initially applied. The level of carbofuran in the overlying water

decreased with a half life of 22 days. Nicosia (1989) also determined that the half life of carbofuran dissolved in California rice irrigation water ranged between 18 and 25 days. It took about 70 days for carbofuran concentrations to degrade to about 1 ppb.

Zayed et al., (1988) employed a ¹⁴C label to elucidate the distribution of carbofuran in a rice-fish model ecosystem. Most of the carbofuran was found a month after application in plant roots and shoots (15-30%), soil (30 to 35%), and fish (25-30%). Eight to 10% remained in the water phase.

Biological Effects and Toxicity:

Carbofuran is a temporary acetylcholinesterase inhibitor. Acetylcholine is formed at the end of autonomic preganglionic fibers and is the chemical responsible for transmission of electrical signals across the synaptic gaps, the space between adjoining nerve cells. Immediately after transmission, acetylcholine is normally destroyed by the enzyme acetylcholinesterase so that the stimulated organ may return to a normal state. Carbofuran out-competes acetylcholine for available binding sites on acetylcholinesterase. This leaves insufficient enzyme to denature the acetylcholine with the end result that the organ continues to be stimulated. Carbofuran's acetylcholinesterase binding is not permanent and after a period of time normal nerve function is restored (FMC Corp., 1969). However, in the interim, the organ's function is disrupted.

Carbofuran toxicity is listed in Table 1. The LD50 for rats is 11 mg/kg (Farm Chemicals Handbook 1988). Toxicity in other animals varies widely from 190 ppm for mallard ducks (FMC Corp.) to 48 ppb for Daphnia magna (Johnson, 1985). Carbofuran is highly toxic to some estuarine organisms, like pink shrimp, whose 48 hour LC50 is 4.6 ppb (EPA 1976). Fish toxicity ranges from 117 ppm (bluegill) to 1758 ppm (fathead minnows, Mayer and Ellersieck, 1986).

Courtney et al. (1985) administered carbofuran to rodents at 0.05 to 5 mg/kg daily on the 7 to 19th day of gestation. Carbofuran did not produce fetotoxicity, fetal lethality or malformations in rats or mice at doses which were not maternally lethal.

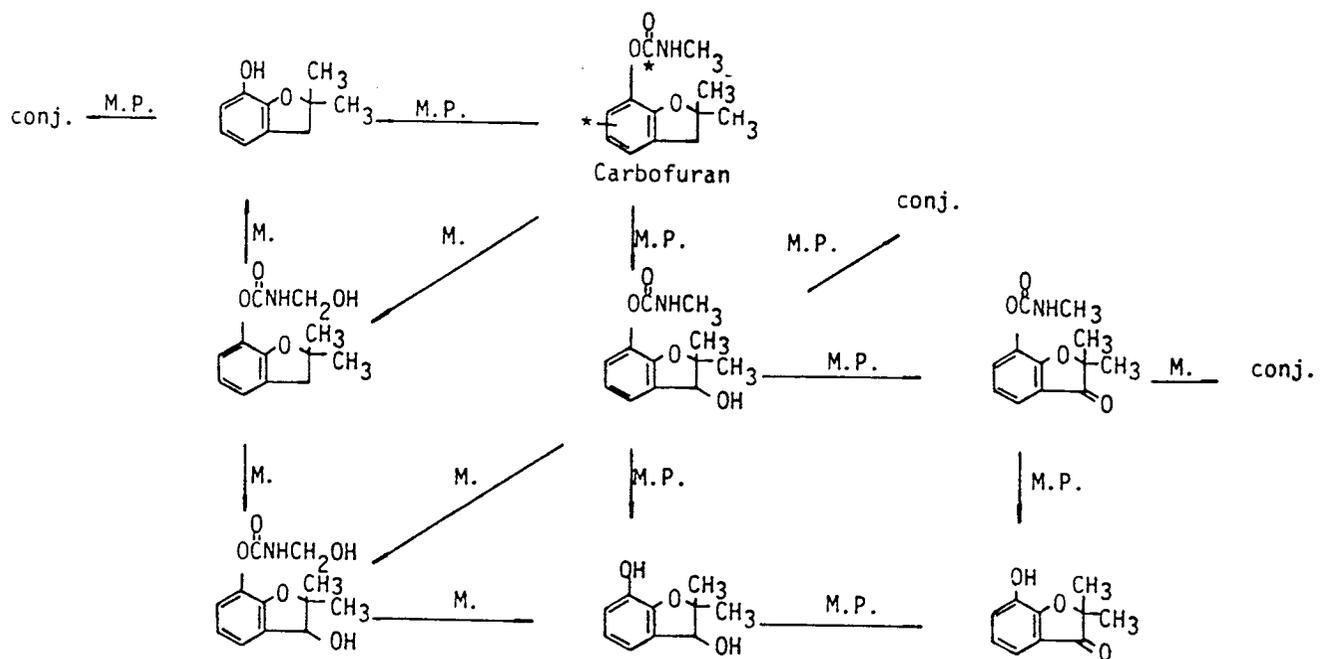


Figure 1. Proposed degradation pathway for carbofuran (Aizawa and Hiroyasu, 1982).

METHYL PARATHION

Trade Name: Metacide

Chemical Name: 0,0-dimethyl 0-p-nitrophenyl phosphorothioate

Other Names: Dimethyl Parathion, Metaphos, Devthion, Wofatox, and Cekumethion

Melting Point: 37-38°C.

Solubility: 50 ppm

Introduction:

Methyl Parathion is classified as restricted use organophosphate insecticide in California. Methyl Parathion has low solubility in water (50 ppm) and as such is formulated into dust, emulsifiable concentrates, and wettable powders.

Registered Uses:

Methyl Parathion is registered for use on many insects and is especially effective on boll weevils. It is also effective against green leafhoppers, stem borers, armyworm, leaffolders and in California rice fields on tadpole shrimp larvae.

Degradation and Environmental Fate:

The degradation rate of methyl parathion is a function of pH. Wolfe et al. (1986) found, using sediment samples titrated with acid or base, that the half-life of methyl parathion in the aqueous phase was 8 and 89 days at pH 10 and 2, respectively. Stephenson and Kane (1984) report the half-life of methyl parathion to be between 9.9 and 13.3 days with higher pHs increasing the degradation rate.

In the environment methyl parathion can be readily hydrolyzed by both natural and enzymatic catalysis. Hydrolysis of the sulfur/phosphate bond (Figure 2) creates a more potent chemical called methyl paraoxon. Synthesis of methyl paraoxon is the favored metabolic pathway for insects and this explains the chemical's particular effectiveness as an insecticide. The rate of hydrolysis to paraoxon in mammals is slower but still fast enough to make it highly toxic (Stephenson and Kane, 1984). Methyl parathion also undergoes reduction in soil to amino-methyl parathion, a less toxic compound than its parent.

Biological Effects and Toxicity:

As described above, methyl parathion is metabolized to methyl paraoxon. Methyl paraoxon is a permanent acetylcholinesterase inhibitor. As such, its mode of action is similar to that previously described for carbofuran. Since both compounds act by the same biochemical mechanism, they are predicted to exhibit an additive type of toxicity when found together.

Methyl parathion is highly toxic to mammals, birds and invertebrates (Table 1). The acute oral and dermal LD₅₀ for rats is 14-24 ppm and 67 mg/kg. The concentration of methyl

parathion producing toxicity to the green alga Chlorella vulgaris was 50 ppb. Acute toxicity to Lemma minor was 7 ppb. Crossland (1984) found the 96 hour LC50 for Daphnia magna to be 0.14 ppb. It was also found that there was no observed effect on fathead minnow at concentrations less than 310 ppb. When 100 ppb of methyl parathion was sprayed on a test pond, Daphnia populations were eliminated and there was no repopulation until 10 weeks after treatment. In the same study the weight of fish exposed to 400 ppb methyl parathion for 28 days was less than that of fish in the control treatment. The weight of these fish doubled while the experimental one did not change. This was thought due to the reduction in food for experimental fish caused by methyl parathion toxicity to invertebrates.

Methyl Parathion showed positive mutagenic activity in a study by Rashid and Mumma (1984). This activity included increased frequency of chromosome aberrations in bone-marrow cells after a high dose administration to mice, direct mutations in Pseudomonas aeruginosa, and induced chromosomal aberrations and chlorophyll mutations in barley plants. Salmonella tykphimurium (Ta100) showed an increase in revertants (> 2 fold) after activation of methyl parathion by the rat's S9 factor. There was also a significant number of reversions occurring in E. coli. This effect could be due to the capability of methyl parathion or its metabolites to methylate nucleic acid.

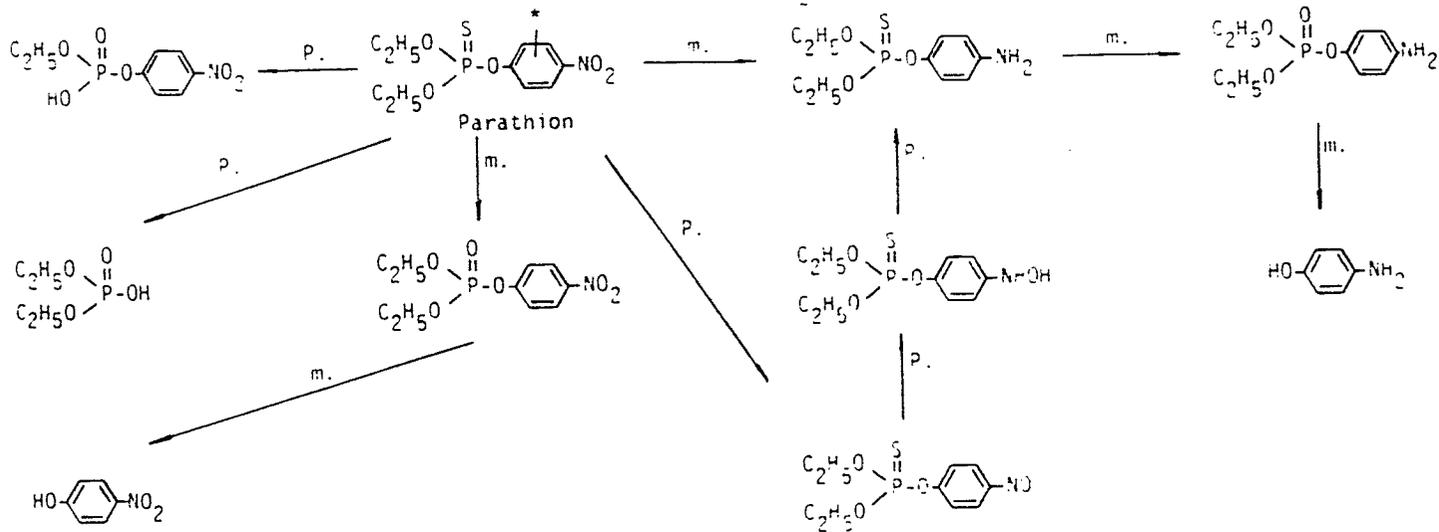


Figure 2. Proposed degradation pathway for methyl parathion (Aizawa and Hiroyasu, 1982).

MALATHION

Trade Name: Cythion

Chemical Name: 0,0-dimethylphosphorodithoate of diethylmercaptosuccinate

Other Names: Mercaptothion, Carbofos, Mercaptotion, and Maldison

Solubility: 145 ppm

Vapor pressure: 4×10^{-5} mm Hg.

Introduction:

Malathion was first produced by American Cyanamid in 1950 as a non-systemic insecticide and acaricide acting as both a contact and a stomach poison. Malathion is marketed as 99.6% technical grade liquid. Available formulations include wettable powders (25 or 50%), emulsifiable concentrates, dusts, and aerosols. There are several regulatory water quality guidelines for malathion. The Department of Health Services action level and NAS health advisory level are both 160 ppb while EPA's "maximum" water quality criteria to protect aquatic life is 0.1 ppb.

Registered Uses:

Malathion is registered for use on many insects including aphids, spider mites, scale insects, house fly, and mosquitos, as well as a large number of other sucking and chewing insects attacking fruits vegetables and stored products.

Environmental Fate and Degradation:

The persistence of malathion, like many pesticides, depends largely upon the temperature and pH of the environment. Mulla (1963) amended 2 ppm malathion into tap water with a pH of 8 and found that the degradation rate was rapid with 60, 14 and 0% remaining after 24, 48, and 72 hours, respectively. Eichellenger and Lichtenberg (1971) used river water with a pH of 7.3 to 8.0 and fortified it with 10 ppb malathion. Their findings demonstrate 25, 10, and 0% of the spike remained after 7, 14, and 28 days, respectively. The calculated pesticide half-life for the latter experiment was 4.3 days. In another study, Devine (1987) reported that the half-life of malathion in water was 6 and 8 days at pH 7 and 8, respectively.

The degradation of malathion in soil is dependent upon its type. Narayana et al. (1987) determined that 8% of the malathion applied to loam soil remained one day after application, while 13% remained in clay and 70% in a sandy loam mixture. Approximately 5-23% of this degradation was attributed to chemical breakdown while 77-99% was due to microbial action. Arthrobacter soil bacteria can utilize malathion as a sole carbon source (Walker and Stojanovic, 1974).

Several studies demonstrate that aquatic marsh and estuarine microorganisms increase malathion degradation in water. For example, water inoculated with fungi and bacteria

degraded 50% of the amended malathion in 2.5 hours (Paris et al., 1975). In tests by Narayana et al. (1987), the uptake of malathion by blue green algae was rapid (8-16 hours) and inversely related to concentration. In the above study, malathion accumulated in Anabaena to maximum levels of 348, 488, and 850 ppm at water borne concentrations of 1, 5, and 10 ppm, respectively. Thereafter a steady decline in intracellular concentrations were observed with increasing algal biomass. Malathion bioconcentration factors were 348, 98, and 85 times at each of the above concentrations. The algae in this study, unlike fungi and other microorganisms, did not metabolize malathion, but rather appeared to concentrate it. The toxicity of malathion contaminated algae when fed upon by herbivores is, apparently, unknown.

Biological Effects and Toxicity:

Malathion is selective in its toxicity due to the presence of carbaryl groups, which are readily hydrolyzed to non-toxic compounds by mammalian enzymes (Figure 3). In contrast, insects actively metabolize malathion to malaoxon, a more potent insecticide. Malaoxon is a neural toxin. Its mode of action is similar to that previously described for carbofuran with the exception that it is a permanent acetylcholinesterase inhibitor. Malathion, carbofuran, and methyl parathion all have similar toxicological modes of action and, as such, are expected to exhibit additive toxicity when present together.

Salmonella typhimurium was used to test the mutagenicity of malathion (Pednekar et al., 1987). No mutagenic activities were observed either before or after activation with rat liver. In contrast, other studies that have been conducted have ascribed genotoxic properties to malathion. Yoder et al. (1973) and Van Bao et al. (1974) showed that malathion caused chromosomal breaks in humans. Sylianco (1978) found a significant increase in micronuclei in mice.

Malathion toxicological data is listed in Table 1. The oral LD₅₀ for rats is between 100 and 1300 ppm (Windholz, 1976). The avian oral LD₅₀ ranges from 165 ppm for Pheasant to 1485 ppm for Mallard ducks. The LC₅₀ for striped bass and shrimp is 40 and 300-500 ppb, respectively. Daphnia magna has one of the lowest reported LC₅₀'s at 2 ppb. (Devine 1987).

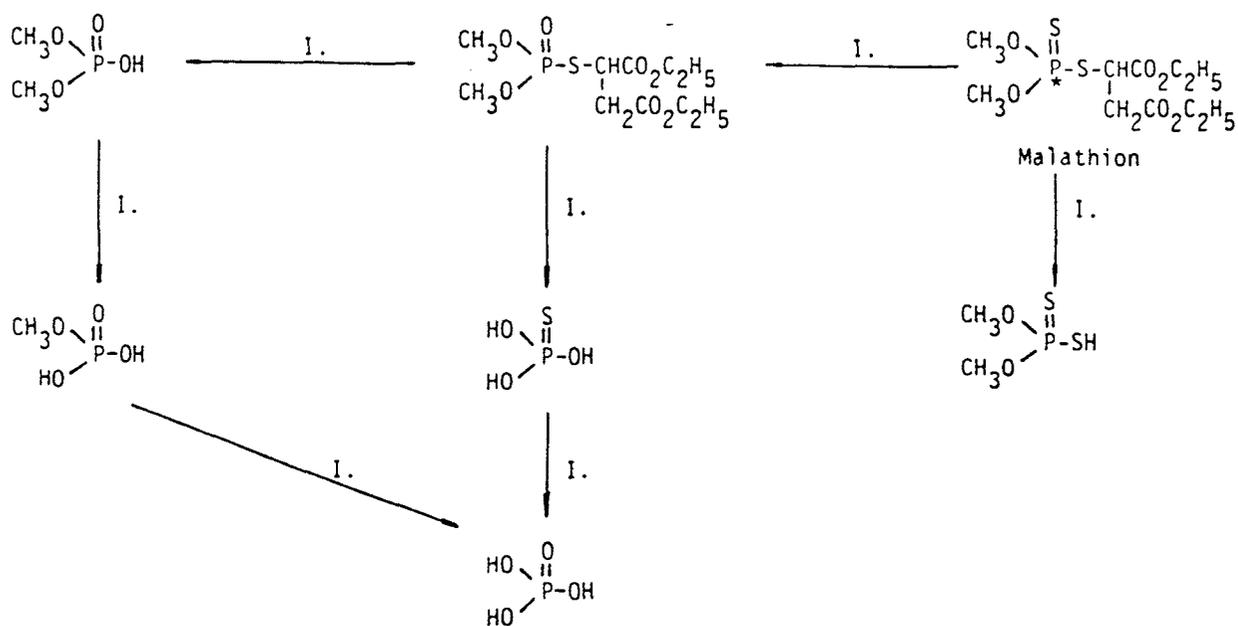


Figure 3. Proposed degradation pathway for malathion (Aizawa and Hiroyasu, 1982).

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