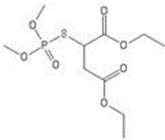
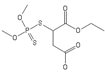
**APPENDIX 1-9. Degradate line of evidence**

***Identification of degradates of concern***

Malathion is known to form Malaoxon (**Figure 1-9.1**). Malathion may oxidize in the environment to form the biologically active compound, malaoxon. The primary route for malathion dissipation is metabolism to the less toxic malathion dicarboxylic (DCA) and monocarboxylic acids (MCA).

**Figure 1-9.1. Structures of malathion, malaoxon, and mono- and di- carboxylic acid, respectively (from left to right).**

Data are available for fish, aquatic and terrestrial invertebrates, aquatic-phase amphibians, birds and mammals for malaoxon (**Tables 1-9.1 and 1-9.2**). For invertebrates and some species of fish and birds (dietary-based), the toxicity values between malathion and malaoxon are similar. However, the available avian (dose-based), mammalian, and aquatic-phase amphibian data indicate that malaoxon is of greater toxicity compared to the parent up to about 2 orders of magnitude (aquatic-phase amphibians).

**Table 1-9.1. Comparison of acute mortality toxicity data available for aquatic animals for malathion and malaoxon.**

|  |  |  |  |
| --- | --- | --- | --- |
| **Species** | **Malathion Toxicity Value**  **(Reference)** | **Malaoxon Toxicity Value**  **(Reference)** | **Ratio (malathion:malaoxon)** |
| **Aquatic** | | |  |
| Rainbow trout  (*Oncorhynchus mykiss*) | 96-hr LC50 (µg/L) = | 96-hr LC50 = 68 µg/L  (48571802) | 0.5-2.9 |
| 33 (48078003) |
| 152 (E12182) |
| 170 (47540302) |
| 66, 80, 94, 100, 138, 200  (40098001) |
| Bluegill sunfish  (*Lepomis macrochirus*) | 96-hr LC50 (µg/L) = | 96-hr LC50 = 57.9 µg/L  (48571801) | 0.35-5.8 |
| 20, 30, 40, 55, 84, 87, 103, 110 (40098001) |
| 48 (47540304) |
| 336 (E77525) |
| Yellow-legged frog  (*Rana boylii*) | 96-hr LC50 = 2137 µg/L  (E92498) | 96-hr LC50 = 23.8 µg/L  (E92498) | 90 |
| Carp  (*Cyprinus carpio*) | 96-hr LC50 =  6590 & 23180 µg/L (40098001; E14861) | 48-hr LC50 =  1600 µg/L (E86) | 4.1-14.5 |
| Medaka  (*Oryzias latipes*) | 48-hr LC50 = 1800 µg/L  (E18398) | 48-hr LC50 = 280 µg/L  (E18398) | 6.4 |
| Blue catfish  (Ictalurus farcatus) | 96-hr LC50 = 17000 µg/L  (E112921) | 96-hr LC50 = 3100 µg/L  (E112921) | 5.5 |
| Water flea  (*Daphnia magna*) | 48-hr EC50 (µg/L) = | 48-hr EC50 = 0.31 µg/L  (48571803) | 3-258 |
| 0.9 (E104559) | 48-hr LC50 = 11.3 µg/L  (E156795) | 0.07-7.1 |
| 1 (40098001) |
| 1.58 (E5370) |
| 1.6-2.2 (E6449) |
| 1.8 (E96171) |
| 3.53 (E80724) |
| 10.7 (E156795) |
| 80 (E94536) |
| Midge  (*Chironomus riparius*) | 1 day EC50 (µg/L) = | 1 day EC50 = 5.4 µg/L (E6830) | 0.04-67 |
| 1.9 (E6830) |
| 0.42 &108 (E14897) |
| 0.21 -362 (E89488, 89, 90) |
| Mosquito (Culex tarsalis) | 1 day LC50 = 10 & 1502 µg/L (E94524) | 1 day LC50 = 42 & 247 µg/L (E94524) | 0.04-36 |

**Table 1-9.2. Comparison of acute mortality toxicity data available for terrestrial animals for malathion and malaoxon.**

|  |  |  |  |
| --- | --- | --- | --- |
| **Species** | **Malathion Toxicity Value**  **(Reference)** | **Malaoxon Toxicity Value**  **(Reference)** | **Ratio (malathion:malaoxon)** |
| **Terrestrial** | | |  |
| Northern bobwhite (*Colinus virginianus*) | 14-d LD50 = 361 mg/kg-bw  (48153114) | 14-d LD50 = 42.6 mg/kg-bw (48153104) | 8.5 |
| 8-d LC50 = 3497 mg/kg-diet  (00022923) | 14-d LC50 = 870 mg/kg-diet  (48153105) | 4 |
| Ring-necked pheasant (*Phasianus colchicus*) | 14-d LD50 = 167 mg/kg-bw  (00160000) | 14-d LD50 = 22.1 mg/kg-bw  (49024601) | 6.2 & 7.6 |
| 14-d LD50 = 136 mg/kg-bw  (48963305) |
| 8-d LC50 = 2639 mg/kg-diet  (00022923) | 8-d LC50 = 1759 mg/kg-diet  (48963302) | 1.5 & 1.4 |
| 8-d LC50 = 2505 mg/kg-diet  (48963301) |
| Canary  (*Serinus canaria*) | 14-d LD50 >2400 mg/kg-bw  (48571805) | 14-d LD50 = 52.6 mg/kg-bw  (48571806) | >46 |
| Mallard duck (*Anas platyrhynchos*) | 14-d LD50 = 1485 mg/kg-bw  (00160000) | 14-d LD50 = 87 mg/kg-bw  (48963306) | 17 & > 26 |
| 14-d LD50 >2250 mg/kg-bw  (48963307) |
| 8-d LC50 >5000 mg/kg-diet  (00022923) | 8-d LC50 > 2880 mg/kg-diet  (48963302) | NA |
| 8-d LC50 >5850 mg/kg-diet  (48963303) |
| Parasitic Wasp (*Campoletis sp)* | 1-d LD50 = 0.0064 µg/org (E113287) | 1-d LD50 = 0.0046 µg/org (E113287) | 1.4 |
| Rust-Red Flour Beetle (*Tribolium castaneum*) | 1-d LC50 = 0.46 & 116 mg/eu (E68165) | 1-d LC50 = 0.37 & 8.15 mg/eu (E68165) | 0.05-313 |
| Rat (*Rattus norvegicus* ) | Acute LD50s = 207-8150 mg/kg/bw | 14-d LD50 = 50 mg/kg/bw  (48571808) | 4-163 |

For the two degradates, DCA and MCA, data are available for fish, aquatic invertebrate and mammals (**Table 1-9.3**). These degradates are orders of magnitude less toxic compared to the parent. Therefore DCA and MCA are not of toxicological concern and will not be considered further.

**Table 1-9.3. Comparison of acute mortality toxicity data available for aquatic and terrestrial animals for dicarboxylic and monocarboxylic acid.**

| **Test Material /**  **Test Species** | **%**  **AI** | **Duration** | **Toxicity Value** | **Reference MRID or ECOTOX** |
| --- | --- | --- | --- | --- |
| dicarboxylic acid | | | | |
| Bluegill sunfish (*Lepomis macrochirus*) | 98.8 | 96 hr | >87 mg/L | 47540306 |
| Waterflea (*Daphnia magna*) | 98.8 | 48 hr | 66.9 mg/L  (48.1 - 93.0) | 47540305 |
| Rat | 98.8 | 14-day | > 2000 mg/kg/bw | 49252803 |
| monocarboxylic acid (α and β mixture) | | | | |
| Bluegill sunfish (*L. macrochirus*) | 92.2 | 96 hr | 77 mg/L  ( 51-151) | 47540309 |
| Waterflea (*D. magna*) | 92.2 | 48 hr | 3.1 mg/L  (1.7 - 7.0) | 47540310 |
| Rat | 92 | 14-day | >2000 mg/kg/bw | 49252804 |

**Mechanism of Oxon Formation**

The chemical transformation process of OPs involves the substitution of the sulfur atom in the P–S bond of the organophosphate pesticide with an oxygen atom. While several studies have been conducted that indicate that OP and organodithiophosphate chemicals that have sulfur double bonds to the central phosphorus atom generally form oxons during chemical disinfection by chlorine compounds (Magara et al., 1994, Duirk and Collette, 2006; Wu and Laird, 2003), much less information is available on how the oxons form in the natural environment. The transformation occurs via oxidative desulfonation, which could potentially occur through photolysis and aerobic metabolism, as well as other oxidative processes (*e.g.,* reaction with hydroxyl radicals and ozone).

A number of studies have documented atmospheric transport and deposition of pesticides apparently from the Central Valley to the Sierra Nevada Mountains (Fellers *et al*., 2004, Sparling *et al*., 2001, LeNoir *et al*., 1999, and McConnell et al., 1998). Prevailing winds blow across the Central Valley eastward to the Sierra Nevada Mountains, transporting airborne industrial and agricultural pollutants into Sierra Nevada ecosystems (Fellers *et al.*, 2004, LeNoir *et al*., 1999, and McConnell *et al*., 1998). Available literature has also documented oxon detections in air, rain, fog (Majewski and Capel, 1995) and surface waters in the United States (USGS, 2011). Although these studies provide evidence that the oxon is present in the environment, these studies do not provide sufficient, consistent information on the levels of the oxon degradate relative to the parent, nor what conditions favor the oxon formation and/or persistence in the environment. As a result the actual transport pathway(s) of the oxon are unclear (*e.g.*, is it formed in the treated areas and transported via volatilization/runoff to waterbodies, or does the parent compound volatilize and then transform to oxon in the atmosphere or at the receiving waterbody) and how environmental estimated concentrations of the oxon could best be modeled.

Malathion metabolizes readily in moist, microbially active soils. However, if malathion is in contact with metabolically inactive surfaces such as dry soils or impervious surfaces common in non-agricultural settings, photo-oxidation to the toxic degradate malaoxon can occur. Field data indicate that up to 10% of malathion can be transformed to malaoxon in these conditions (see **APPENDIX 3-1**). Oxidation to malaoxon can also be catalyzed in the presence of chlorine. To a much more limited degree than in chlorine catalyzed or photo-oxidation conditions, malathion can metabolize to malaoxon under other circumstances, but malaoxon is not a major degradate (*i.e.*, occurs at more than 10% of malathion applied in environmental fate studies) in that pathway. Malaoxon dissipates and degrades similarly to malathion with rapid metabolism in aerobic conditions and rapid hydrolysis in alkaline conditions. Therefore, short duration malaoxon concentration peaks (less than one day) may be expected in non-agricultural streams during run-off events. Stream monitoring efforts associated with the discontinued Mediterranean Fruit Fly Eradication Program found malaoxon concentrations as high as 328 ppb. NAWQA surface water monitoring detected malaoxon in 0.1% of samples for which it was tested with a peak detection of 0.2 ppb.

**Potential Effects of Malaoxon**

Available monitoring data indicate that, when detected, malaoxon is generally detected at lower concentrations compared to the parent. Given that malaoxon is of similar or somewhat greater toxicity compared to the parent, the effects of malaoxon on mortality to exposed non-target organisms are likely to be similar to those of the parent.

The likelihood of exposure to malaoxon may be less than that of the parent. This is because 1) malathion is generally less persistent in the environment when compared to malathion and 2) if oxon formation occurs in air, only a fraction of the applied malathion would be transported to the air and subject to transformation. Lower exposure to malaoxon is suggested by the available monitoring data in which malaoxon was detected less frequently than the parent (when considering samples where both chemicals were quantified).

For birds and mammals, the uncertainty associated with not quantifying malaoxon may be less because of the conservative nature of the foliar dissipation half-life that is used for malathion. Available half-lives range over an order of magnitude, i.e., 0.9-10.9 days. The dissipation of malathion is conservatively represented by a 90th percentile value of 6.1 days. Appreciable malaoxon formation relative to malathion concentrations occurred on sand (6%) and dry soil (10%). Residues of malaoxon on steel, concrete, and plastic were very low or nondetectable. In a laboratory photolysis study, malaoxon air concentrations were at their maximum between 24 and 48 hours after application which falls within the range of the foliar dissipation half-life for malathion. In two field studies measuring malathion and malaoxon residues in ground or forage-dwelling invertebrates and ground vegetation after a foliar application to either oilrape seed (at 0.785 lb a.i./A) or alfalfa (1.34 lb a.i./A), the maximum measured concentration of malaoxon was 3.7 or 11%, 0.6 or 2.9%, and 0.2 or 0.3% of the measured concentration for malathion, respectively (ratio of malaoxon to malathion concentration at the maximum measured malaoxon concentration) (MRID 49086410 and 49086411). The most sensitive avian species is the ring-necked pheasant for which there is a 7-fold difference between malathion and malaoxon. For mammals (using the rat), the most sensitive acute mortality value is 207 mg/kg/bw, which is 4 times greater than the malaoxon toxicity value. Therefore, while malaoxon is more toxic than parent to birds and mammals, given the environmental fate, formation and persistence of malaoxon, effects due to malaoxon, are anticipated to be potentially captured by using a conservative half-life for malathion and the toxicity endpoints based on exposure to malathion.

The most conservative assumption related to mortality to aquatic organisms would be that malaoxon is 90 times greater than the parent (based on the yellow-legged frog data from ECOTOX 92498). It is noted that in this study, neither control mortality nor measured test concentrations were reported. For other aquatic species, the toxicity difference is generally within one order of magnitude. The hydrolysis half-life for malaoxon is more rapid than malathion, but like malathion, is also pH dependent (8.8 days at pH 7, 0.16 days at pH 9). The highest levels of aquatic malaoxon found in a search of available data were a result of medfly control efforts in California (CDFG 1982) (see **APPENDIX 1-10**). In this monitoring program, in some cases, while the concentration of malaoxon was greater than malathion, generally the concentrations of malaoxon were similar or approximately one-half of malathion. It is noted that residential uses that are no longer supported also potentially contributed to these concentrations. These runoff concentrations are much higher than agricultural runoff levels or non-targeted residential runoff levels. In an aquatic field dissipation study in Missouri for which malathion was applied to a flooded rice paddy, malathion residues detected in water samples collected after the first and second application dissipated to below the detection limit (10 μg/L) in samples taken prior to the second and third applications. In water samples collected one day after the last application, malathion concentrations averaged 17 μg/L and had decreased to 10 μg/L by the second sampling day. Malaoxon residues were <10 μg/L at all sampling dates. Other field data indicate that up to 10% of malathion can be transformed to malaoxon which may then be available during runoff. Additional details on the monitoring data are also presented in Chapter 3.

When the range of toxicity differences between malathion to malaoxon across taxa (less to similar to more) along with the formation rate and variable detection concentrations in water for malaoxon are taken together, the range of uncertainty may be already incorporated into the uncertainty in the mortality threshold for malathion for fish and aquatic phase amphibians[[1]](#footnote-1). Therefore, a lack of quantification of potential increases of mortality due to the potential presence of malaoxon does not represent greater uncertainty compared to what is already inherent in the mortality thresholds for malathion. For these reasons, in most cases, risks of mortality associated with the oxon are not expected to materially influence (*i.e*., change) any species-specific effects determination that is based on malathion parent. Therefore, the potential risks associated with the oxon will not be quantitatively assessed. However, because of the uncertainties associated with the potential risks of the oxon, the oxon will be qualitatively considered in the weight-of-evidence approach used for making effects determinations.

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1. for the HC05: SE = 15.7, CV = 0.36; for the all aquatic vertebrate threshold, the uncertainty bound due to slope values represents an additional order of magnitude [↑](#footnote-ref-1)