Environmental Fate and Toxicology of Dimethoate

April Van Scoy, Ashley Pennell and Xuyang Zhang

Department of Pesticide Regulation, California Environmental Protection Agency, Sacramento, CA, 95812, USA

Contents

Corresponding Author
April R. Van Scoy-DaSilva
California Department of Pesticide Regulation
1001 I Street
Sacramento, CA 95814
Phone: (916) 445-0113
Email: april.dasilva@cdpr.ca.gov

Abstract

The insecticide dimethoate, an organophosphate, was first introduced in1962 for broad spectrum control of a wide range of insects including mites, flies, aphids, and plant hoppers. It is known to inhibit AChE activity like other organophosphates, resulting in nerve damage which may lead to death. In the environment, hydrolysis represents a major degradation pathway under alkaline conditions, whereas volatilization is not a major route of dissipation from either water or moist soils. Dimethoate is also degraded by microbes under anaerobic conditions and the major degradation product, omethoate, has been identified. Dimethoate has been found to adversely impact many organisms. In plants, photosynthesis and growth are highly impacted, whereas birds exhibit inhibition in brain enzyme activity, thus sublethal effects are apparent. Furthermore, aquatic organisms are expected to be highly impacted via direct exposure and display changes in swimming behavior. This insecticide has been found to be less toxic than other organophosphates.

1 Introduction

Dimethoate ([O,O-Dimethyl S-(N-methylcarbamoylmethyl) phosphorodithioate]) is an organophosphorous insecticide that is used worldwide in agriculture and urban areas due to its high efficacy and rapid environmental degradation. It was registered in 1962 and has been used to control a wide range of insects including mites, flies, aphids, and plant hoppers (Mirajkar et al. 2005). Dimethoate can be applied to many crops such as, fruit, vegetables, grain and ornamentals, in addition to non-agricultural applications for landscape maintenance and structural pest control. However, in 2000, all non-agriculture uses of dimethoate including residential uses were cancelled. Roughly 816,466 kg of active ingredient is applied annually on agricultural sites with the highest applications being on alfalfa, wheat, cotton, and corn (US EPA 2009). In California, its use has decreased approximately 90% on alfalfa, oranges, and grapes between the years 1990 and 2011 (CDPR 2014a).

Dimethoate is highly water soluble and has low soil persistence. Due to these two factors, the potential to runoff into surface waters and/or leaching into groundwater is high. However, a thorough understanding on the environmental fate of dimethoate is needed in order to mitigate

the negative impacts it has on the environment. This paper reviews the relevant literature and addresses the environmental fate, chemistry and toxicology of dimethoate.

2 Chemistry

Dimethoate (Fig. 1) is an organophosphorous insecticide that is highly water soluble. When pure, it is a white crystalline solid with a mercaptan odor. At room temperature, it is stable in aqueous solutions of pH 2-7; however, it is unstable under alkaline conditions. It has a low affinity for soils and a moderate affinity for organic matter. It is susceptible to hydrolysis under acidic conditions, is moderately stable to microbial degradation and is non-volatile as reflected by its low vapor pressure (US EPA 2008). Additional physiochemical properties of dimethoate are presented in Table 1.

3 Chemodynamics

3.1 Soil

Due to its strongly hydrophilic nature, surface and groundwater contamination must be considered. The adsorption to soils is weak; however, studies have found organic matter (OM) content to impact its retention in soils.

The adsorption and desorption processes of dimethoate was investigated by Vagi et al. (2010) using three Greek soils from the Mytilene Island region, each with different pH, clay and organic matter content. Majority of the adsorption isotherms followed an L-shape; transformation into L-shaped isotherms resulted as OM content increased. Desorbed amounts of the pesticide were only available when the soil was washed with water due to its hydrophilic nature. Furthermore, results indicate hysteresis and was observed in soils with higher OM content; however, dimethoate weakly sorbed to all three soils. Al Kuisi (2002) reported similar results. Dimethoate adsorption was measured on eight soil types (pH from 8.0-8.45; OM from 0.73-2.95%; clay content from 5.9-14.9%) in which resulting isotherms also followed an L-shape. Adsorption coefficients (K_{ads}), determined via the Freundlich equation, ranged from 1.01 to 10.36 suggesting dimethoate is weakly adsorbed and is influenced by a change in organic matter content.

Loam soil half-lives, determined from two field trials held in 1989 and 1990, were approximately 5.1 to 7.1 days, respectively (Wu and Fan 1997). In addition, measured soil residues after each field trial showed a decline over time; however, measurable residues were still present up to 31 days post application. Bohn (1964) studied the accumulation of dimethoate in a sandy loam soil. The insecticide was measured in the top 3 inches of the soil and its half-lives were determined to be 4 days under drought conditions and 2.5 days following a moderate rainfall, respectively. Kolbe et al. (1991) observed a quick decrease in dimethoate concentrations following a retardation phase of 1-2 days, when applied to clay loam soil; this may be attributed to biodegradation. Half-lives were determined for the three soils. For both the humus rich sandy soil and heavy clay soil, at 10°C and 20°C, the half-lives were 15 and 9 days. However, the half-life in clay loam soil was determined to be 10 and 5 days at 10°C and 20°C, respectively. This study also confirms a faster decrease in the pesticide due to the increased organic matter.

The mobility of dimethoate from amended and unamended soil was studied by Antonious et al. (2007). Broccoli plants were grown under three soil managements: native unamended soil, native soil amended with sewage sludge, and native soil amended with yard waste compost. Dimethoate 4E was applied to the broccoli foliage and the amount of pesticide that reached the soil was a result of spray drift or runoff from rain or irrigation. The pesticide residue was higher in the unamended soil (134.5 ng/g soil) compared to the amendments with sewage sludge (30.5 ng/g soil) and yard waste compost (46.1 ng/g soil); runoff concentrations followed the same trend. Thus, an increased amount of OM in the soil decreased the amount of dimethoate in collected runoff. Overall, studies have indicated that organic matter content in the soil increases dimethoate's sorptive ability and consequently decreases its chances of being transported in runoff water or percolating down into the groundwater.

3.2 Water

Transport through soil via leaching, adsorption, or volatilization are affected by factors such as water solubility, volatility, and stability. Due to dimethoate's high water solubility and low soil adsorption coefficient, its retention in soil will be low and its dispersion and transport in soils will be affected by soil type and soil moisture content. Under simulated field conditions (19-21

°C; 500 g of soil), El Beit et al. (1977b) found that soil type played a major role in leaching; leaching increased in the following soils: clay < clay loam < loam < sandy clam loam < sand. The retention within these soils is thought to be impacted by physical forces and hydrogen bonding.

Losses of dimethoate due to soil water can lead to high amounts of chemical leaching into groundwater or transported off soil surfaces into nearby water bodies. El Beit's (1977a) study showed that an increase in the initial soil moisture resulted in an increase in dimethoate's ability to leach. Furthermore, reduction in organic matter content not only reduces the potential for biodegradation, but also accelerates pesticide loss through processes such as evaporation and leaching.

Pesticide leaching through a haplic acrisol soil (rich in clay; classified by the FAO) found in northern Thailand was assessed by Ciglasch et al. (2005). Dimethoate was applied (2,860 g/ha) to plots on a 10-year-old lychee orchard and leachate was monitored for 8 wk; borosilicate suction lysimeters were installed. The fields received rainfall following application and pesticide residues were found to translocate to a depth of 55 cm in a single flush, thus this movement is independent of soil sorption coefficients. Of all the pesticides applied to these fields, dimethoate was detectable in the leachate up to one month (Ciglasch et al. 2005). However, due to the rainstorm in this study, it is suggested that further studies be conduct to identify a range of dissipation rates. Another study monitored dimethoate concentrations in the Mae Sa watershed in northern Thailand due to its frequent use (Sangchan et al. 2014). A total of 370 water samples were collected and analyzed from three gauging stations along the watershed; a maximum concentration of 0.4 μg/L was measured. Sangchan et al. (2014) compared the measured contamination level to environmental quality standards set forth by the Canadian Council of Ministers of the Environment. Overall, none of the samples containing dimethoate exceeded the Canadian limit of 0.62 μg/L thus the measured concentrations are thought to be of little concern for this watershed.

This insecticide has been detected in surface waters throughout California. Ensminger et al. (2009) collected water samples from streams throughout the Central Valley of California which

is dominated by agricultural land. Dimethoate was detected in 2 of 21 samples during the irrigation season at 0.074 and 0.190 μg/L. In California (CDPR 2014b), measurable dimethoate concentrations were found in many monitored waterways with the highest residue detected at 11.5 μg/L (Table 3). The maximum measured residue level was above the chronic aquatic life benchmark value for invertebrates (0.5 μg/L) set forth by the US EPA, thus suggesting a higher exposure risk than fish (chronic aquatic benchmark of 430 μg/L).

The threat posed by pesticides leaching is often groundwater contamination; however, it is unknown if measured concentrations are an environmental concern. To investigate this, Loewy et al. (2003) sampled 30 groundwater wells over three years. Among the detected pesticides, dimethoate was found at concentrations up to 10.9 μg/L with a mean concentration of 0.219 μg/L; overall detections were in 14.1% of collected samples. Dimethoate's groundwater ubiquity score (GUS) index value of 3.51 indicates that it has a high potential to leach. A monitoring study conducted in Saudi Arabia, on the persistence of pesticides in ground water, found high concentrations of dimethoate in 87% of the total regions sampled (El-Saeid et al. 2011), whereas in China detections were positive in 37% of collected water samples (Gao et al. 2009). In California (CDPR 2003), measurable dimethoate concentrations were found in 3 of 5542 groundwater samples with the highest residue detected at 24.0 μg/L (Table 3).

3.3 Air

The volatilization rate of dimethoate from both wet and dry surfaces is low as suggested by its low vapor pressure and Henry's law constant (Table 1). In California (CDPR 2013), a study conducted in 2012 did not measure dimethoate in any of the 156 collected air samples (Table 3). El Beit (1977b) determined the loss of dimethoate from soil via evaporation was impacted by soil type, but independent of soil depth. Furthermore, they note that evaporation was greatest in experiments using sand and less volatilization occurred in experiments using loam. Volatilization is considered a minor route of dissipation; however, if other dissipation routes are found to be minor, volatilization may play a larger role in removing the pesticide from soil over time.

4 Environmental degradation

4.1 Abiotic Processes

Hydrolysis

The rate of dimethoate hydrolysis is dependent on pH, soil type, temperature and other weather conditions. To demonstrate pH dependency, Ruzicka et al. (1967) carried out hydrolysis studies (70 °F; 20% ethanol present) using river waters of varying pH and hardness. They found the hydrolytic half-lives to decrease as pH lowered from 8.0 to 7.5 (Thames River water $t_{1/2}$= 22 h and Irthing River water $t_{1/2}$= 18 h, respectively). Further investigation using an ethanol buffer solution, at pH 6, resulted in a half-life of 12 h.

Temperature dependency was illustrated by Lartiges and Garrigues (1995). Using four different water types, hydrolysis studies were conducted at 6 and 22 °C, in addition to three pHs (6.1, 7.3 and 8.1). In ultrapure water, at the lowest pH and temperature, dimethoate was stable to hydrolysis ($t_{1/2}$= 423 days); however, when the temperature increased, hydrolysis was observed ($t_{1/2}$= 193 days). Hydrolysis in seawater was determined to increase as both the pH and temperature were increased; a half-life of 36 days was measured at 22 °C and pH 8.1. These results indicate hydrolysis occurs more rapidly under alkaline conditions. Druzina and Stegu (2007) reported similar findings. The dissipation of dimethoate in river and groundwater at varying pH and temperature resulted in different half-lives. In groundwater, at pH 6, the half-life was 94.9 days, whereas at pH 8.5 the half-life was 66 days. When compared to river waters (pH 8), hydrolysis was more rapid as temperature increased from 4 to 25 °C (i.e., $t_{1/2}$= 169 days to $t_{1/2}$= 74.5 days, respectively).

In northern Vietnam, dimethoate was applied to a combined rice paddy-fish pond farming system to determine dimethoate's environmental fate (Anyusheva et al. 2012). When applied to paddy water (approx. 22 g total; water pH 8.1; sandy loam soil) during the spring crop season it disappeared rapidly within 14 days and a DT_{50} of 0.3 days was determined. When compared to the summer-autumn crop season, it also disappeared rapidly with a DT_{50} of 0.8 days.

El Beit et al. (1978) looked at the degradation of dimethoate in soil leachates (of distilled water) varying in pH. At the lowest pH (4.2) the pesticide was stable for 19 days; however, as pH

increased to 11.0, degradation occurred within 20 h. Further studies looked at the impact of solutions incubated with either urea or lime ($Ca(OH)_2$). In the presence of either, the pesticide was observed to decrease; however, degradation was greater in the lime solution due to a possible change in to an alkaline solution.

Photolysis

The photocatalytic oxidation of dimethoate was observed by Evgenidou et al. (2006). Using a high-pressure mercury lamp (125 W) and a low amount of titanium dioxide as a catalyst (TiO_2; 100 mg/L), dimethoate (20 mg/L in distilled water) degraded into nine by-products, which are listed in Table 2. Based on the intermediates formed, it is likely that this pesticide degrades via oxidation and dealkylation reactions that proceed simultaneously. Decay of dimethoate forms the secondary intermediates, *O*,*O*-dimethyl phosphoric ester, *O*,*O*,*O*-trimethyl phosphoric ester and *O*,*O*,*S*-trimethylphosphorothiate. In addition, Microtox toxicity tests using the irradiated solutions revealed that the transient intermediates (oxon derivative, disulfide, and *O*,*O*,*S*-trimethyl thiophosphorothioate) were more toxic than dimethoate itself.

Photocatalysts are often utilized in order to advance oxidation processes. Under simulated solar irradiation (300 W xenon lamp), the use of the catalyst 2,4,6-triphenylthiapyrilium cation ($TPTP^+$; 10 mg/L) reduced dimethoate concentrations by 20% after 60 min of irradiation; this reduction suggests an electron transfer mechanism (Gomis et al. 2012). Chen et al. (2007) identified an increase in the efficiency of dimethoate degradation as TiO_2 catalyst concentrations increased to approximately 0.6 g/L. In particular, irradiating dimethoate (500 W UV-lamp; 120 min) with UV radiation alone resulted in a 3.22% degradation efficiency whereas in the presence of the catalyst (0.6 g/L) degradation was 80.15% efficient. This is attributed to an increase in the total surface area available for the pesticide to adsorb to; however, higher concentrations of the catalyst may reduce overall efficiency due to a light scattering effect.

Using a photoreactor containing a Hg lamp (12 W; UV at 254.7 nm), the pesticide, in an aqueous solution, was irradiated in the presence of an oxidizing agent, hydrogen peroxide (H_2O_2) and a catalyst, iron (III) chloride hexahydrate (Nikolaki et al. 2005). Oxidation reactions resulted in the by-products, dimethyl phosphite, N-methyl-acetamide, and formic acid. The products were

detectable up to 45 min following test initiation and were further oxidized into carbon dioxide, sulfate, phosphate, and ammonium ions. Furthermore, the hydrolysis intermediate omethoate was detected prior to irradiation of the pesticide solution.

4.2 Biotic Processes

Microbial degradation of pesticides is often a major player in reducing pesticide residues within the environment. To determine the potential for dimethoate to be degraded by microbes, two bacterial strains, *Pseudomonas aeruginosa* W171 (isolated from water) and *Bacillus licheniformis* F102 (isolated from *Labeo rohita* intestine) were used. DebMandal et al. (2008) found both strains to degrade the pesticide. In addition, four metabolites resulted from the degradation by the *P. aeruginosa* strain, whereas the *B. licheniformis* was found to completely degrade the pesticide within three days. The bacterium *Raoultella* sp. X1 has been found to degrade dimethoate, but environmental and nutritional conditions were found to be important. Using dimethoate as the sole carbon source resulted in poor degradation; however, 75% of the initial concentration was removed via co-metabolism (Liang et al. 2009).

Microbes found in sewage sludge or wastewater often have the ability to use pesticides as carbon sources for survival, thus leading to decreases in pesticide concentrations. Multiple bacterial strains were isolated from the wastewater treatment pool of a factory that manufactured dimethoate. Out of all of the strains, strain Lgjj-3, having similar lineage to the *Paracoccus* sp., had the highest degrading capabilities, ultimately reducing dimethoate (100 mg/L) to below detection levels within 6 h. Li et al., (2010) also identified seven degradation products and proposed the mechanism shown in Figure 2. It is suggested that this strain degrades dimethoate via hydrolysis, decarboxylation, oxidation and an additional hydroxylation reaction. Isolated from sewage and soil from cotton fields, *Aspergillus niger* ZHY256 has been found to degrade the pesticide by approximately 87% via cleavage of the phosphorus-sulfur (P-S) linkage (Liu et al. 2001).

Using an expanded granular sludge bed reactor, Monsalvo et al. (2014) investigated the biodegradability of dimethoate. Under anaerobic conditions, the pesticide was added to the reactor at concentrations up to 500 mg/L and incubated for 21 days. Within the incubation

period, dimethoate did not degrade; however, it was noted that an acclimation period of 50 days was sufficient to observe a complete removal of the pesticide.

Deshpande et al. (2001) tested the ability of 25 bacterial strains to degrade dimethoate. After an 8-day incubation, only two strains, *Pseudomonas aeruginosa* MCMB-427 and *Bacillus megaterium* MCMB-428 were efficient enough to degrade the pesticide by 95%. Furthermore, they identified the degradation by *Pseudomonas aeruginosa* MCMB-427 to be plasmid-mediated and thus transferable amongst other strains. This group concluded that in order to understand the genetic basis of this degradation, additional studies are warranted.

5 Ecotoxicology

5.1 Mode of Action

Like other organophosphates, dimethoate inhibits acetylcholinesterase (AChE) which is present in mammals, fish, birds and insects. AChE is a class of enzymes that initiate the hydrolysis of acetylcholine (ACh), a neurotransmitter, into inactive choline and acetic acid (Fukuto 1990). The inhibition creates a buildup of acetylcholine at the nerve synapses disabling the enzyme cholinesterase that is vital for a functioning central nervous system (Lundebye et al. 1997). The concentration of ACh in the synapses results in continuous stimulation of the muscles eventually leading to seizures, exhaustion and possibly death.

5.2 Insects

Systemic insecticides such as dimethoate enter plant tissues and can be translocated into the plant's nectar. For instance, alfalfa treated with the insecticide (at 304 mg/L a.i.) contained 16 mg/L of dimethoate in the nectar one day post-application and within two weeks it was at 1 mg/L (Barker et al. 1980). Barker et al. (1980) investigated the toxicity of the measured dimethoate concentrations within nectar to honeybees. Worker bees (*Apis mellifera* L.) were fed contaminated and uncontaminated nectar for 7 days; mortality and cholinesterase inhibition resulted. Observations showed that dimethoate is not considered a repellent and will be consumed by bees. Further studies observed the impact on bee survival, colony development and comb building. Sucrose solutions, with (up to 5 mg/L) and without dimethoate were provided to

bees for 3 weeks. Bees were highly impacted by the highest dose resulting in death within the first week, no new comb and little sugar honey stored. Those dosed with 0.2 mg/L did not show signs of toxicity until the third week of the study where both comb and egg production were reduced (Waller and Barker 1979); colonies were impacted at each tested concentrations.

Jepson et al. (1995) investigated the toxicity of topically applied dimethoate to adult coccinellids (*Coccinella septempuncta)* and carabids (*Bembidion obtusum*, *Nebria brevicollis*, *Trechus quadristriatus* and *Demetrias atricapillus*). LD_{50} (48-h) values ranged from 17.7-98.8 ng/insect and as body size increased, insect susceptibility decreased.

Midge 4th instar larvae were exposed to a wide range of concentrations up to 4.52 mg/L for *Chironomus riparius* and 7.12 mg/L for *Kiefferulus calligaster*. Both species exhibited significant cholinesterase inhibition; however, glutathione S-transferase (GST) activity was not significantly impacted in *K. calligaster* compared to inhibition in *C. riparius; C. riparius* were more sensitive with a 48-h LC_{50} of 0.481 mg/L, compared to that of *K. calligaster* (1.747 mg/L). In addition, 3rd instar larvae were exposed (up to 0.455 mg/L) to assess the effects on growth and emergence. At the highest concentration, a cholinesterase inhibition of 66% was observed, whereas each concentration delayed emergence time (Domingues et al. 2007).

Over time, insecticide resistance may occur. Vontas et al. (2001) compared the dimethoate-resistant strain of the olive fruit fly (*Bactrocera oleae*) with a colonized parental strain and field-collected population. Topical applications of the insecticide were placed on the abdominal sternum of the insect; after 24 h, bioassays were conducted. Results identified that oxidative metabolism was not the major factor in resistance, but an altered acetylcholinesterase with poor catalytic efficiency was the major component.

5.3 Aquatic organisms

Due to dimethoate's hydrophilic nature, its potential to bioaccumulate is insignificant as suggested by its high water solubility and low log K_{ow}, however, it is still possible that adverse effects may result. Toxicity values are presented in Table 4.

Beusen and Neven (1989) investigated the toxicity related to high purity and emulsifiable concentrate (10% a.i.) dimethoate exposure to freshwater fish and *Daphnia magna*. Zebrafish, guppy and *Daphnia magna* exposed to both were found to be more susceptible to the emulsifiable concentrate with 48-h LC_{50} values of 7.5, 15.7 and 0.83 mg/L, respectively. This may have been a direct result to the solvent within the concentrate. Exposure to the high purity dimethoate (99%) did not result in mortality of either the zebrafish or guppy within 96-h; however, a measured 48-h LC_{50} (1.7 mg/L) for *D. magna* was determined. Exposure studies (concentrations from 2.5-4.0 mg/L) using catfish (*Heteropneustes fossilis*) observed altered swimming behavior, increased gulping for air and increased mucus secretion over the body. In addition, the fish were highly sensitive to low concentrations with a 96-h LC_{50} of 2.98 mg/L (Pandey et al. 2009).

Further studies investigated the biochemical responses resulting from dimethoate exposure. Adult male rainbow trout (*Oncorhynchus mykiss*) were exposed to concentrations of dimethoate under semi-static condition for either 5, 15, or 30 days. Blood and liver samples were taken. Tests revealed that dimethoate did not significantly impact testosterone levels; however, 17β-estradiol levels increased in the 5 and 15-day tests leading to the belief that it has estrogen mimic capabilities. In addition, liver tissues showed impaired membrane permeability (Dogan and Can 2011).

Freshwater rotifers, *Brachionus calyciflorus* and *Asplanchna brightwelli*, were exposed to four dimethoate concentrations (0.4, 0.8, 1.2 and 1.6 mg/L) and their swimming responses were recorded. Chen et al. (2014) found dimethoate to significantly inhibit the rotifer's swimming angular and linear speed and this response was dependent on pesticide concentration. Similar results were reported by Guo et al. (2012) with *Brachionus calyciflorus*, exposed to dimethoate concentrations ranging from 0.18 to 1.59 mg/L. In addition to speed inhibition, swimming behavior, particularly direction, was negatively impacted suggesting inhibition of AChE.

The acute toxicity of the insecticide to Australian freshwater shrimp, *Paratya australiensis*, was determined by Kumar et al. (2010). Shrimp, collected from a pristine site of the Finniss River area, were exposed to seven nominal concentrations ranging from 0.05 to 20 mg/L; the 96-h

LC_{50} was determined to be 800 µg/L. In addition, the authors predicted a 21-day chronic lethality value for shrimp based on a log-log model to be 89 µg/L. Mysid shrimp (*Neomysis integer*) were exposed to concentrations of dimethoate up to 5,000 µg/L. Mortality was recorded and a 96-h LC_{50} of 540 µg/L was calculated (Roast et al. 1999).

The freshwater prawn, *Macrobrachium rosenbergii*, at the post-larval stage was used to study the effects of pesticide exposure and its impact of feeding rates. Five concentrations of dimethoate (78.12 to 1,250 µg/L) were used for lethality tests; surviving prawns were placed into freshwater to assess feeding behavior. Satapornvanit et al. (2009) determined both a 24 and 48-h LC_{50} for dimethoate to be 142.1 and 102.7 µg/L, respectively. Post-exposure feeding tests, measuring sublethal effects, resulted in a 24-h EC_{50} of 269.3 µg/L. Due to the sublethal effects concentration being greater than that of the lethal test, the authors conclude that post-exposure feeding tests cannot be used to detect this pesticide's toxicity.

5.4 Plants

Dimethoate residues on foliar surfaces following application and its residual toxicity were investigated by Chowdhury et al. (2005). They found that as the plant's surface wax increased, the insecticidal efficacy was not impacted. Thus, dimethoate will likely be found in low concentrations in plant waxes due to its hydrophilic nature and its potential affinity for the plant cuticle.

The degradation of the insecticide within yerba mate (*Ilex paraguariensis*) plants was studied; field samples were randomly collected. Dimethoate residues on dry leaves were found to decrease from samples collected one day to 31 days post application; half-lives of the pesticide within yerba mate plants ranged from 9.8-11.8 days, respectively (Schmalko et al. 2002).

Wheat plants at 6 days of germination were treated with dimethoate at 50, 100 or 200 mg/L; after 10 days plant leaves were analyzed. At the lowest dose, plants exhibited an increase in shoot and root length, whereas higher doses decreased growth. Furthermore, an increase in chlorophyll and carotenoids resulted from the 50 mg/L dose. A decrease in the photosynthetic activity and

inhibition in growth indicates dimethoate may be hazardous to wheat plants at high concentrations (Pandey and Gopal 2011). Similar results were observed by Mishra et al. (2008). , Dimethoate at 50 mg/L stimulated growth and photosynthesis in cowpea (*Vigna unguiculata*); however, higher concentrations lead to a reduction in photosynthetic electron transport activity and damage to pigments.

5.5 Mammals

Although dimethoate targets insects, studies have shown mammalian impacts as well. Dose-response studies were conducted by Long at al. (2006) using laboratory mice. Single or daily doses were administered intraperitoneally and brain and serum AChE activity were measured. Single doses of dimethoate did not cause a significant inhibition in AChE activity; however, the daily doses did decline overall activity. Besides a response in AChE, cytochrome P450 (CYP2B) activity was found to be inhibited as well.

Adult Wistar rats were exposed for 30 days to dimethoate in water or diet alone or co-administered with selenium or vitamin E to assess lung damage. Changes in animal behavior, in dimethoate only tests, were observed and included depression, dyspnea and diarrhea among others. Extracted lung tissue revealed lipid peroxidation; however, in the presence of selenium and/or vitamin E, malondialdehyde concentrations were restored to levels similar to those in the controls. Further observations included histopathological changes such as hemorrhages, increases in glutathione peroxidase and superoxide dismutase and a decrease in acetylcholinesterase (Amara et al. 2012). However, in the presence of antioxidants, such as those used in this study, there is potential to alleviate damage from dimethoate exposure.

Developmental toxicity was investigated by Farag et al. (2006). Pregnant Fischer-344 rats were dosed via oral gavage with concentrations of 0, 7, 15 and 28 mg/kg/day dimethoate of gestation days 6-15. At the higher doses, clinical signs of toxicity, such as tremors and weakness, occurred, in addition to reduced cholinesterase activity in both maternal and fetal brains. Furthermore, the number of living fetuses and mean fetal weight was reduced indicating fetotoxicity results from exposure to the highest dose in this study.

Human exposures are possible due to dimethoate's high use. Six workers were exposed dermally and through inhalation when spraying the pesticide onto tomato crops enclosed in plastic houses. Each sprayman applied dimethoate as a 40% emulsifiable concentrate in two applications which were 15 days apart. Gauze sponges were placed on the workers to assess dermal exposure and blood samples were collected to identify cholinesterase inhibition. Overall, a reduction in plasma cholinesterase was observed and dermal exposure to the forearms, hands and upper legs was greatest resulting in a mean exposure dose of 914 mg/day (Al-Jaghbir et al. 1992), thus precautionary measures to limit exposure are necessary for applicators.

5.6 Birds

Field studies assessing bird exposure to spray drift was conducted by Cordi et al. (1997). To do so, four hedgerows which bordered fields sprayed with the pesticide were chosen and nest boxes containing both nestlings and adult great tits (*Parus major*) were placed into the hedges. Application of dimethoate (1 L/ha) occurred 59 ft from two of the four hedges, on both sides, by using a boom sprayer; wind direction was approximately at right angles to the hedges at speeds of 8.5 ft/s from the west and 11.8 ft/s from the east-south-east, respectively. Responses of exposure by adults included inhibition in serum butyrylcholinesterase (BChE), whereas nestlings experienced significant decreases in BChE and carboxylesterase (CbE) activity; nestling growth rates were also negatively impacted. Japanese quail (*Coturnix coturnix japonica*) dosed with the pesticide displayed inhibition in both AChE and cholinesterase (ChE) activity; overall brain AChE activity was reduced by 85% when dosed at 75 mg/kg (Westlake et al. 1981).

Martin et al. (1996) fed pesticide treated grasshopper carcasses to 3-day-old ring-necked pheasant chicks (*Phasianus colchicus*) in order to assess the effects of birds consuming treated insects. They determined a dimethoate LD_{50} of 28.9 mg/kg body weight which was approximately 0.2 LD_{50} doses per day given the body weight of the birds (approximately 30 g). Lower AChE activity was measured in birds consuming treated feed compared to those fed untreated feed.

6 Summary

The insecticide dimethoate, an organophosphate, was first introduced in1962 for broad spectrum control of a wide range of insects including mites, flies, aphids, and plant hoppers. It inhibits AChE activity like other organophosphates, resulting in nerve damage, which may lead to death. It is considered highly toxic to insects; however, dimethoate resistance has been observed.

Dimethoate has both a low vapor pressure and Henry's law constant, thus volatilization is not a major route of dissipation from either water or moist soils. Photolysis is a minor dissipation pathway; however, studies have shown that in the presence of a catalyst, the rate of photolysis does increase. The insecticide has high water solubility, and under alkaline conditions, hydrolysis predominates and represents a major degradation pathway. It has a low soil sorption capacity which varies by soil type and organic matter content. Dimethoate is degraded by microbes under anaerobic conditions, and bacterial species have been identified that are capable of using dimethoate as a carbon source. Although many intermediate by-products have been identified by abiotic and biotic processes, the major degradation product is omethoate.

Dimethoate has been found to adversely impact many organisms. In plants, photosynthesis and growth are highly impacted, whereas birds exhibit inhibition in brain enzyme activity, thus sublethal effects are apparent. Furthermore, aquatic organisms are expected to be highly impacted via direct exposure. Overall, aquatic organisms display changes in swimming behavior; however, dimethoate is not as toxic as other organophosphates. Consistent toxicity results include inhibition in growth and more importantly, acetylcholinesterase activity.

Although dimethoate has been widely used on many field crops and has high water solubility, it has only been infrequently detected in groundwater samples. However, to reduce the potential for surface and groundwater contamination, care should be taken when applying dimethoate-containing products for agriculture or other uses.

References

Al-Jaghbir MT, Salhab AS, Hamarsheh FA (1992) Dermal and inhalation exposure to dimethoate. Arch Environ Contam Toxicol 22:358-361

Al Kuisi M (2002) Adsorption of dimethoate and 2,4-D on Jordan Valley soils and their environmental impacts. Environ Geol 42:666-671

Amara IB, soudani N, Troudi A, Hakim A, Zeghal KM, Boudawara T, Zeghal N (2012) Dimethoate induced oxidative damage and histopathological changes in lung of adult rats: Modulatory effects of selsnium and/or vitamin E. Biomed Environ Sci 25(3):340-351

Antonious GF, Ray ZM, Rivers Jr. L (2007) Mobility of dimethoate residues from spring broccoli field. J Envrion Sci Heal B 42:9-14

Anyusheva M, Lamers M, La N, Nguyen VV, Streck T (2012) Fate of pesticides in combined paddy rice- fish pond farming systems in northern Vietnam. J Environ Qual 41:515-525

Barker RJ, Lehner Y, Kunzmann MR (1980) Pesticides and honey bees: Nectar and pollen contamination in alfalfa treated with dimethoate. Arch Environ Contam Toxicol 9:125-133

Beusen JM, Neven B (1989) Toxicity of dimethoate to *Daphnia magna* and freshwater fish. Bull Environ Contam Toxicol 42:126-133

Bohn WR (1964) Disappearance of dimethoate from soil. J Economic Entomology 57:798-799

CDPR (2003) Sampling for pesticide residues in California well water. 2003 Well inventory database Cumulative report 1986-2003. http://www.cdpr.ca.gov/docs/emon/pubs/ehapreps/eh0308.pdf

CDPR (2013) Air monitoring network results for 2012, Vol. 2. Report AIR 13-02. http://www.cdpr.ca.gov/docs/emon/airinit/amn_2012_final_report_1013.pdf Accessed Oct 14, 2014

CDPR (2014a) Pesticide Use Reporting. Database. http://www.cdpr.ca.gov/docs/pur/purmain.htm Accessed Aug 10, 2014

CDPR (2014b) Surface water protection program database. http://www.cdpr.ca.gov/docs/emon/surfwtr/surfcont.htm Accessed Oct 14, 2014

Chen J, Wang Z, Li G, Guo R (2014) The swimming speed alteration of two freshwater rotifers *Brachionus calyciflorus* and *Asplanchna brightwelli* under dimethoate stress. Chemosphere 95:256-260

Chen JQ, Wang D, Zhu MX, Gao CJ (2007) Photocatalytic degradation of dimethoate using nanosized TiO_2 powder. Desalination 207:87-94

Chowdhury ABMNU, Jepson PC, Ford MG, Frampton GK (2005) The role of cuticular waxes and surface roughness in determining the insecticidal efficacy of deltamethrin and dimethoate applied as emulsifiable concentrates to leaf surfaces. Int J Pest Manage 51(4):253-263

Ciglasch H, Amelung W, Totrakool S, Kaupenjohann M (2005) Water flow patterns and pesticide fluxes in an upland soil in northern Thailand. European Journal of Soil Science 56:765-777

Cordi B, Fossi C, Depledge M (1997) Temporal biomarker responses in wild passerine birds exposed to pesticide spray drift. Environ Toxicol Chem 16(10):2118-2124

DebMandal M, Mandal S, Pal NK, Aich A (2008) Potential metabolites of dimethoate produced by bacterial degradation. World J Microbiol Biotechnol 24:69-72

Deshpande NM, Dhakephalkar PK, Kanekar PP (2001) Plasmid-mediated dimethoate degradation in Pseudomonas aeruginosa MCMB-427. Lett Appl Microbiol 33:275-279

Dogan D, Can C (2011) Endorcirne disruption and altered biochemical indices in male *Oncorhynchus mykiss* in response to dimethoate. Pestic Biochem Phys 99:157-161

Domingues I, Guilhermino L, Soares AMVM, Nogueira AJA (2007) Assessing the dimethoate contamination in temperate and tropical climates: Potential use of biomarkers in bioassays with two chironomid species. Chemosphere 69:145-154

Druzina B, Stegu M (2007) degradation study of selected organophosphorus insecticides in natural waters. Intern J Environ Anal Chem 87(15):1079-1093

El Beit IOD, Wheelock JV, Cotton DE (1977a) Factors affecting the accumulation of dimethoate in soil. Intern J Environ Studies 11:187-196

El Beit IOD, Wheelock JV, Cotton DE (1977b) Factors affecting the fate of dimethoate in soils. Int J Environ Stud 11:113-124

El Beit IOD, Wheelock JV, Cotton DE (1978) Factors influencing the degradation of dimethoate in soils and solutions. Int J Environ Stud 11:253-260.

El-Saeid MH, Al-Turki AM, Al-Wable MI, Abdel-Nasser G (2011) Evaluation of pesticide residues in Saudi Arabia ground water. Res J Environ Sci 5(2):171-178

Evgenidou E, Konstantinou I, Fytianos K, Albanis T (2006) Study of the removal of dichlorvos and dimethoate in a titanium dioxide mediated photocatalytic process through the examination of intermediates and the reaction mechanism. J Hazard Mater B137:1056-1064

Farag AT, Karkour TAZ, El Okazy A (2006) Developmental toxicity of orally administered technical dimethoate in rats. Birth Defects Res B 77:40-46

Fukuto TR (1990) Mechanism of action of organophosphorus and carbamate insecticides. Environ Health Persp 87:245-254

Gao J, Liu L, Liu X, Zhou H, Lu J, Juang S, Wang Z (2009) The occurrence and spatial distribution of organophosphorus pesticides in Chinese surface water. Bull Environ Contam Toxicol 82:223-229

Getenga ZM, Jondiko JIO, Wandiga SO, Beck E (2000) Bull Environ Contam Toxicol 64:359-367

Gomis J, Arques A, Amat AM, Marin ML, Miranda MA (2012) A mechanistic study on photocatalysis by thiapyrylium salts. Photodegradationof dimethoate, alachlor and pyrimethanil under simulated sunlight. Appl Catal B-Environ 123-124:208-213

Guo R, Ren X, Ren H (2012) Assessment the toxic effects of dimethoate to rotifer using swimming behavior. Bull Environ Contam Toxicol 89:568-571

Jepson PC, Efe E, Wiles JA (1995) The toxicity of dimethoate to predatory coleopteran: Developing an approach to risk analysis for broad-spectrum pesticides. Arch Environ Contam Toxicol 28:500-507

Kolbe A, Bernasch A, Stock M, Schutte HR, Dedek W (1991) Persistence of the insecticide dimethoate in three different soils under laboratory conditions. Bull Environ Contam Toxicol 46:492-498

Kumar A, Correll R, Grocke S, Bajet C (2010) Toxicity of selected pesticides to freshwater shrimp, *Paratya australiensis* (Decapoda: Atyidae): Use of time series acute toxicity data to predict chronic lethality. Ecotox Environ Saf 73:360-369

Lartiges SB, Garrigues PP (1995) Degradation kinetics of organophosphorus and organonitrogen pesticides in different waters under various environmental conditions. Environ Sci Technol 29:1246-1254

Li R, Zheng J, Wang R, Song Y, Chen Q, Yang X, Li S, Jiang J (2010) Biochemical degradation pathway of dimethoate by Paracoccus sp. Lgjj-3 isolated from treatment wastewater. Int Biodeter Biodegr 64:51-57

Liang Y, Zeng F, Qiu G, Lu X, Liu X, Gao H (2009) Co-metabolic degradation of dimethoate by Raoultella sp. X1. Biodegradation 20:363-373

Liu YH, Chung YC, Xiong Y (2001) Purification and characterization of a dimethoate-degrading enzyme of *Aspergillus niger* ZHY256, isolated from sewage. Appl Environ Microb 67(8):3746-3749

Loewy RM, Carvajal LG, Novelli M, Pechen de D"Angelo AM (2003) Effect of pesticide use in fruit production orchards on shallow ground water. J Environ Sci Heal B B38(3):317-325

Long SM, Dawson A, Shore RF (2006) A comparison of the effects of single and repeated exposure to an organophosphate insecticide on acetylcholinesterase activity in mammals. Environ Toxicol Chem 25(7):1857-1863

Lundebye AK, Curtis TM, Braven J, Depledge MH (1997) Effects of the organophosphorous pesticide, dimethoate, on cardiac and acetylcholinesterase (AChE) activity in the shore crab *Carcinus maenas*. Aquat Toxicol 40:23-36

Martin PA, Johnson DL, Forsyth DJ (1996) Effects of grasshopper-control insecticides on survival and brain acetylcholinesterase of pheasant (*Phasianus colchicus*) chicks. Environ Toxicol Chem 15(4):518-524

Mirajkar N, Pope CN (2005) Dimethoate. Encyclopedia of Toxiocology pp. 47-49

Mishra V, Srivastava G, Prasad SM, Abraham G (2008) Growth, photosynthetic pigmants and photosynthetic activity during seedling stage of cowpea (Vigna unguiculata) in response to UV-B and dimethoate. Pestic Biochem Phys 92:30-37

Monsalvo VM, Garcia-Mancha N, Puyol D, mohedano AF, Rodriguez JJ (2014) Anaerobic biodegradability of mixtures of pesticides in an expanded granular sludge bed reactor. Water Sci Technol 69(3):532-538

Nikolaki MD, Oreopoulou AG, Phillippopoulos CJ (2005) Photo-fenton assisted reaction of dimethoate in aqueous solutions. J Environ Sci Heal B 40:233-246.

PAN (2014) The pesticide action network pesticide database. http://www.pesticideinfo.org/Detail_Chemical.jsp?Rec_Id=PC33349. Accessed Aug 10, 2014.

PPDB (2014) The pesticide properties database. http://sitem.herts.ac.uk/aeru/ppdb/en/Reports/244.htm. Accessed Aug 10, 2014.

Pandey JK, Gopal R (2011) Laser-induced chlorophyll fluorescence: A technique for detection of dimethoate effect on chlorophyll content and photosynthetic activity of wheat plant. J Fluoresc 21:785-791

Pandey RK, Singh RN, Singh S, Singh NN, Das VK (2009) Acute toxicity bioassay of dimethoate on freshwater airbreathing catfish, *Heteropneustes fossilis* (Bloch). J Environ Biol 30(3):437-440

Roast SD, Thompson RS, Donkin P, Widdows J, Jones MB (1999) Toxicity of the organophosphate pesticides chlorpyrifos and dimethoate to *Neomysis integer* (Crustacea: Mysidacea). Wat Res 33(2):319-326

Ruzicka JH, Thomson J, Wheals BB (1967) The gas chromatographic determination of organophosphorous pesticides. Part II. A comparative study of hydrolysis rates. J Chromatogr 31:37-47

Sangchan W, Bannwarth M, Ingwersen J, Hugenschmidt C, Schwadorf K, Thavornyutikarn P, Pansombat K, Streck T (2014) Monitoring and risk assessment of pesticides in a tropical river of an agricultural watershed in northern Thailand. Environ Monit Assess 186:1083-1099

Satapornvanit K, Baird DJ, Little DC (2009) Laboratory toxicity test and post-exposure feeding inhibition using the giant freshwater prawn *Macrobrachium rosenbergii*. Chemosphere 74:1209-1215

Schmalko ME, Ramallo LA, Ferreira D, Berlingheri RD (2002) Dimethoate degradation in plants and during processing of yerba mate leaves. Braz Arch Biol Techn 45(4):419-422

United States Environmental Protection Agency. Office of Pesticide Programs. Revised interim reregistration eligibility decisions for dimethoate (2008) US Environmental Protection Agency Office of Prevention, Pesticides and Toxic Substances: Washington, D.C.

Vagi MC, Petsas AS, Kostopoulou MN, Lekkas TD (2010) Adsorption and desorption processes of the organophosphorus pesticides, dimethoate and fenthion, onto three Greek agricultural soils. Intern J Environ Anal Chem 90:369-389

Vontas JG, Cosmidis N, Loukas M, Tsakas S, Hejazi MJ, Ayoutanti A, Hemingway J (2001) Altered acetylcholinesterase confers organophosphate resistance in the olive fruit fly *Bactrocera oleae*. Pestic Biochem Phys 71:124-132

Waller GD, Barker RJ (1979) Effects of dimethoate on honey bee colonies. J Econ Entomol 72:549-551

Westlake GE, Bunyan PJ, Martin AD, Stanley PI, Steed LC (1981) Organophosphate poisoning. Effects of selected organophosphate pesticides on plasma enzymes and brain esterases of Japanese quail (*Coturnix coturnix japonica*). J Agric Food Chem 29:772-778

World Health Organization (2004) Dimethoate in drinking-water. Background document for the development of WHO Guidelines for drinking-water quality.

Wu J, Fan D (1997) Degradation of dimethoate in chrysanthemums and soil. Bull Environ Contam Toxicol 59:564-569

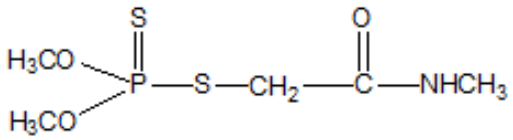

Figure 1. Structure of Dimethoate

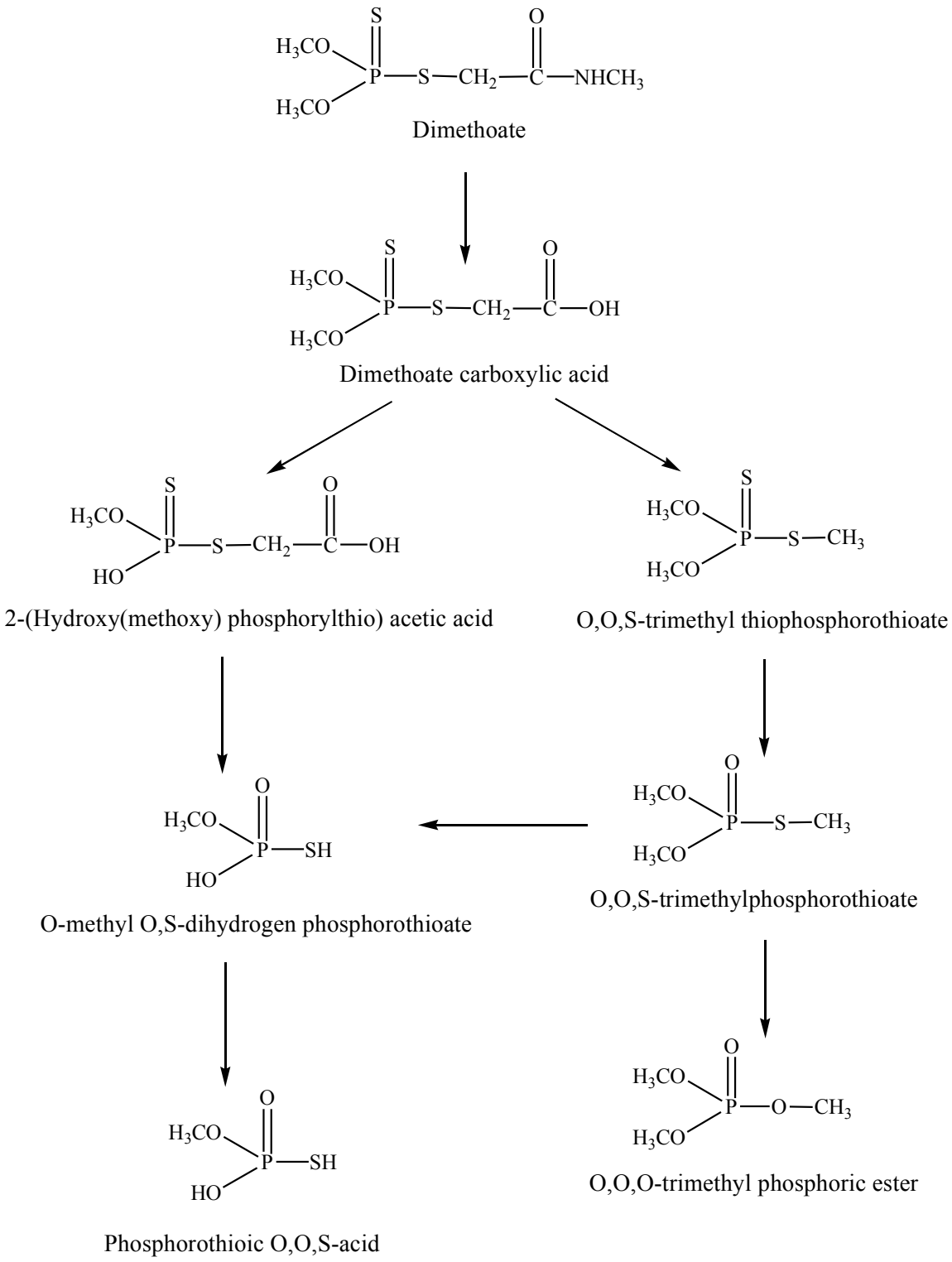

Figure 2. Suggested microbial transformation pathway for dimethoate by *Paracoccus* sp. Lgjj-3 (adapted from Li et al. 2010).

Table 1. Physiochemical Properties of Dimethoate.

CAS Number [a]	60-51-5
Molecular Formula [a]	$C_5H_{12}NO_3PS_2$
Molecular Weight (g/mol) [a]	229.3
Density (g/ml) [a]	1.31
Henry's law constant at 25°C (Pa m^3 mol^{-1}) [a]	1.42 X 10^{-6}
Vapor pressure at 25°C (mPa) [a]	0.247
Octanol-water partition coefficient at pH 7, 20°C (log Kow) [a]	0.704
Soil Adsorption Coefficient (Koc) [b]	11
Water Solubility at 21°C (mg/L) [a]	39,800
Half-lives in aqueous solutions (days)[c] pH 2-7 pH 9	 Stable 12

[a)]PPDB (2014), [b)]PAN (2014), [c)] WHO (2004)

Table 2. Suggested photocatalytic transformation by-products for dimethoate using TiO2 as a catalyst (adapted from Evgenidou et al. 2006).

Dimethoate by-products
O,*O*-dimethyl phosphonic ester
O,*O*,*O*-trimethyl phosphoric ester
N-methyl-2-sulfanylacetamide
O,*O*,*S*-trimethylphosphorothiate
2-*S*-methyl-(*N*-methyl) acetamide
O,*O*,*S*-trimethyl thiophosphorothioate
1-Methyl-2-(acetyl-*N*-methyl-) methane disulfide
omethoate
1,2-Bis(acetyl-*N*-methyl-) methane disulfide

Table 3. Dimethoate concentrations measured throughout California [a, b, c]

Media	Number of samples	Number of detections	Percent detection (%)	Minimum concentration	Maximum concentration
Surface water	5945	531	9	0.007 µg/L	11.5 µg/L
Ground water	5542	3	0.05	0.38 µg/L	24 µg/L
Air	156	0	0	ND	ND

a) Data from CDPR, 2014b
b) Data from CDPR, 2003
c) Data from CDPR, 2013

Table 4. Toxicity of Dimethoate to Aquatic Organisms. These values exceed the aquatic life benchmark values set forth by the US EPA (fish= 3.1 mg/L and invertebrates= 0.0215 ug/L). [a]

Aquatic organism	Scientific name	Test	Concentration (mg/L)
Rainbow trout	*Oncorhynchus mykiss*	96-h LC_{50}	6.2
Stonefly	*Pteronarcys californica*	48-h LC_{50}	0.043
Water flea	*Daphnia magna*	96-h LC_{50}	3.32
Mysid shrimp	*Mysidopsis bahia*	96-h LC_{50}	15

a) Data from US EPA RED, 2008