Environmental Fate and Toxicology of Methomyl

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Contents

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1 Introduction

The insecticide methomyl (*S*-methyl *N*-(methylcarbamoyloxy)thioacetimidate; CAS 16752-77-5; Fig. 1) was first introduced by E.I. du Pont de Nemours in 1968 (US EPA, 1998b). In 1978, the US Environmental Protection Agency classified methomyl as a restricted-use pesticide (RUP; US EPA 1998a); currently 15 registered products are categorized as such (US EPA 1998b). Further restrictions were implemented in 1995, limiting use to certain agricultural production areas, requiring addition of an embittering agent during formulation and requiring the use of bait stations (US EPA 1998a). Within the USA, approx. 262,000 kg of methomyl (a.i.) was applied on agricultural crops annually from 1999 to 2004 (US EPA 2010). However, estimates for the period between 2001 and 2007 show annual average usage of approx. 363,000 kg (a.i.); major crop uses included sweet corn, lettuce, onions, and tomatoes (US EPA 2010). In 2007, some 227,711 kg of active ingredient was applied in California alone (CDPR 2007).

Methomyl is an oxime carbamate insecticide that controls a broad spectrum of arthropods such as spiders, ticks, moths, flies, beetles, aphids, leafhoppers, and spider mites often found on various field crops, ranging from fruits to tobacco (Kidd and James 1991). Methomyl is formulated as a soluble concentrate, a wettable powder or a water-soluble powder (Kidd and James 1991) and is the active ingredient of Du Pont 1179™, Flytek™, and Kipsin™, among other trade formulations (Kamrin and Montgomery 1999). Furthermore, the main formulated water-soluble products contain approx. 25–90% methomyl, whereas the water-miscible products only contain some 12.5–29% (IPCS 1995). Methomyl is weak-to-moderately persistent, with a soil half life ($t_{1/2}$) ranging from a few to more than 50 days; however, under ideal field conditions the $t_{1/2}$ should be no longer than 1 week (IPCS 1995).

Human exposures to methomyl fall into three toxicity categories defined by the US EPA that depend on the route of exposure: I, oral exposure (highly toxic); II, inhalation (moderately toxic); and III, dermal exposure (slightly toxic; US EPA 1998b). Furthermore, methomyl is considered to be highly toxic to mammals, fish and aquatic invertebrates (Farre et al. 2002). To illustrate, the acute oral LC_{50} given for rats was 17–45 mg/kg (Mahgoub and El-Medany 2001), the LC_{50} values for bluegill sunfish and rainbow trout were 0.9–3.4 mg/L, and the LC_{50} values for *Daphnia magna* were from 0.022 to 0.026 mg/L (Yi et al. 2006; Periera et al. 2009). Because methomyl's water solubility and toxicity to non-targeted aquatic organisms is high (Table 1), concerns exist for its potential impact on surface water, groundwater, and aquatic organisms. Therefore, the most up to date information may be useful
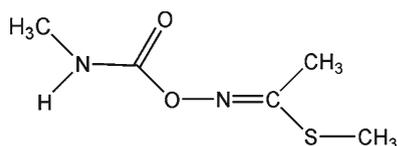

Fig. 1 Methomyl structure

Table 1 Physicochemical properties of methomyl

Chemical Abstract Service registry number (CAS#)[a]	16752-77-5
Molecular formula[a]	$C_5H_{10}N_2O_2S$
Molecular weight (g/mol)[a]	162.2
Density at 25°C (g/mL)[a]	1.29
Melting point (°C)[a]	78–79
Octanol-water partition coefficient (log K_{ow})[b]	1.24
Organic carbon normalized partition coefficient (K_{oc})[b]	72
Vapor pressure at 25°C (mmHg)[b]	5.6×10^{-6}
Henry's law constant (Pa m^3 mol^{-1})[a]	2.13×10^{-6}
Solubility at 25°C (g/L)[a]	
Water	57.9
Methanol	1,000
Acetone	730
Ethanol	420
Isopropanol	220
Toluene	30

[a]Data from Tomlin (2000), [b]Data from US EPA (1989)

in characterizing any potential environmental effects attributable to methomyl. To that end, we have reviewed the relevant literature, and in this chapter address methomyl's chemistry, environmental fate, and toxicology.

2 Chemistry

Methomyl is an *O*-(methylcarbamoyl)oxime carbamate; as such, its structure is similar to both aldicarb and thiocarboxime (Kuhr and Dorough 1976). When pure, methomyl is a white crystalline solid with a slight sulfurous odor. At room temperature, it is moderately to highly soluble in water and alcohols and has a low affinity for both soils (e.g., illite) and organic matter. Methomyl is denser than water, is susceptible to hydrolysis under alkaline conditions, and is subject to degradation via photocatalytic reactions and by microbes at various rates. Methomyl's physicochemical properties are presented in Table 1.

3 Chemodynamics

3.1 Soil

Because of its strongly hydrophilic nature, there is concern that methomyl may contaminate both surface and groundwater. Although increased soil organic matter

and clay content (both amount and type) can influence methomyl's retention by soil, its overall adsorption to soil is generally weak-to-moderate at best.

Several researchers have assessed the adsorption of methomyl by various soil types and organic compositions. For example, Cox et al. (1993) investigated the role that clays (smectites, illites, and kaolinites) and humic acids (saturated with cations) play in methomyl sorption. In general, perhaps because of its surface area, the sorption to smectites (K_d = 4.5–9.58) was greater than to both illites (K_d = 1.56) and kaolinites (K_d = 0.5). Methomyl was shown to also possess a higher sorption affinity for humic acid (K_d = 399.5) than clays (Cox et al. 1993).

Leistra et al. (1984) calculated sorption coefficients to model the extent of methomyl leaching in greenhouse soils (sandy, loamy sand, and loam soil), under different transformation (degradation) and irrigation rates. They found only 0.03% of the original mass had leached after 110 days, under both low transformation (first-order rate coefficient k_r = 0.0495 day^{-1}; $t_{1/2}$ = 14 day) and high irrigation (4 mm/day) rates; thus minimal leaching of the insecticide was predicted from these results. Furthermore, adsorption coefficients for soil/liquid partitioning (K_d) were determined. The resulting coefficients, 0.46×10^{-3} m^3/kg (sandy), 0.43×10^{-3} m^3/kg (loamy sand), and 1.30×10^{-3} m^3/kg (loam) indicate that methomyl has a weak-to-moderate affinity for soils (Leistra et al. 1984).

Jones et al. (1989) reported methomyl to have a $t_{1/2}$ of 2 days in surface soils and 0.5–1.6 months in subsoils. However, values reported in other studies were different; under laboratory conditions Kahl et al. (2007) reported an average $t_{1/2}$ of 15.5 days in topsoil, whereas under field conditions the $t_{1/2}$ was approx. 0.97–1.25 days for cropped soil (Aktar et al. 2008). To summarize, although predictions vary with soil type and organic matter content, they all indicate that methomyl is not very persistent in complex soils.

Variations in reported adsorption coefficients and half-lives indicate that environmental conditions are important in influencing this pesticide's transport (i.e., leaching) and degradation. Because methomyl has been widely used in agriculture, it is important to understand its transport and fate within field soils. It is known to be rapidly degraded into CO_2 by soil microbes (Nyakundi et al. 2011); however, trace amounts of the parent insecticide and its hydrolytic product (*S*-methyl-*N*-hydroxythioacetamidate) are also detectable (Harvey and Pease 1973). Furthermore, Nyakundi et al. (2011) demonstrated the potential of white rot fungi to remediate the insecticide in contaminated soils. Kahl et al. (2007) investigated the depth to which methomyl can leach in soil. The highest concentrations appeared at an 80 cm depth, with degree of transport dependent on water flow and degree of soil porosity.

3.2 Water

Methomyl has high water solubility and a weak-to-moderate adsorption to soils, and therefore poses a contamination risk to surface and groundwater (Table 1). The US

Geological Survey's National Water-Quality Assessment (NAWQA) Program monitored eight US urban surface waters for residues of herbicides and insecticides (Hoffman et al. 2000), methomyl residues were detected only in Las Vegas Wash (Las Vegas, NV); the probable source of these detections was sewage treatment plant effluent and urban runoff (Hoffman et al. 2000). NAWQA also analyzed for pesticide residues in groundwater between 1992 and 1996. They sampled 2,485 sites and detected residues of 67 pesticides. The maximum methomyl concentration detected in this study was <17 ng/L (Kolpin et al. 2000). In California (CDPR 2011), measurable methomyl concentrations were found in many monitored urban and agricultural waterways. The highest residue detected (55.3 μg/L) was from Chualar Creek (Monterey County), whereas the highest agricultural-related detection (0.343 μg/L) was in Orcutt Creek (Santa Barbara County; CDPR 2011). The risks posed in these and other locations can be established from aquatic life benchmarks that have been set by the US EPA; invertebrates are considered the most sensitive species and have the lowest chronic aquatic life benchmark. The residue levels found in Orcutt Creek were below the chronic aquatic invertebrate benchmark (0.7 μg/L), which suggests a low exposure risk. In contrast, exposure risks in Chualar Creek were relatively high, thus increasing the potential for nontarget species toxicity.

The leaching of methomyl and its degradation products into water sources has also been investigated. To evaluate the effects of both irrigation and rainfall, the insecticide thiodicarb was applied to two sites containing sandy clay loam and sandy loam soils, respectively; each site was regarded to posses the potential for groundwater contamination (Jones et al. 1989). Since methomyl is the principal degradation product of thiodicarb (which is also hydrophobic), there is a presumption that it will be detected at higher levels within water sources. Jones et al. (1989) found that soil collected 2 months after application contained low residue levels; however, methomyl was detected 1 month after application in groundwater at depths of 1.8 and 3.2 m. Carbo et al. (2008) also examined the potential of methomyl to contaminate shallow aquifers (<4.5 m deep) in Mato Grosso, Brazil. Water samples collected from monitoring wells placed in cotton fields contained measurable concentrations, ranging from less than the limit of detection (LOD=0.10 μg/L) to 22.81 μg/L (Carbo et al., 2008).

During any rainfall event, methomyl has the potential to run off of application sites and into adjacent uncontaminated sites. To investigate this phenomenon, Harvey and Pease (1973) studied a loamy sand soil that had been cleared of vegetation. The levels of insecticide detected in the runoff (<0.01 mg/L) and in the soil from both treated and untreated plots (<0.04 mg/L, 15 days after application), suggested that little surface runoff or leaching occurs providing the agent is applied at recommended rates. Furthermore, Kahl et al. (2008) detected dissolved concentrations (i.e., 11.4% of applied mass; water-pesticide suspension applied via spraying) in stream water that were four times greater than that of the fungicide chlorothalonil (which is strongly hydrophobic; detected at levels of 3.5% of applied mass in the same stream water).

3.3   Air

The volatilization rate of methomyl from both dry surfaces and water is relatively low, as predicted by its low vapor pressure and Henry's law constant values (Table 1). Hence, volatilization does not significantly contribute to methomyl's dissipation from soil (either moist or dry) or water. Yeboah and Kilgore (1984) determined that the methomyl concentrations (from liquid concentrate) measured in the ambient air of a pesticide storage building were minimal (13.7 ng/m^3), compared to its threshold limit value (TLV = 2,500 μg/m^3; ACGIH 1982; Yeboah and Kilgore 1984). When compared to other monitored pesticides, Baker et al. (1996) reported that concentrations in ambient air near methomyl-treated crops were non-detectable.

## 4   Environmental Degradation

4.1   Abiotic Processes

Hydrolysis
Methomyl is potentially subject to hydrolysis via cleavage of the ester bond to form its main degradates methomyl oxime and CO_2 (Kuhr and Dorough 1976; US EPA 1998a).

However, environmental hydrolysis does not readily occur to a significant degree. To illustrate, Malato et al. (2002) found that methomyl solutions at either pH 2.7 or pH 5 did not significantly degrade via hydrolysis after 20 days; Tamimi et al. (2006) later verified this result at pH 6 as well. The authors of both studies concluded that hydrolysis does not occur to any significant extent in the field—at least under mild-to-strong acidic conditions.

In the presence of Cl$^-$ (simulating the chlorination of drinking water), the rate of methomyl's hydrolytic breakdown varies; changes in pH between 7.6 and 8.9 produced half-lives differing by 30-fold (0.4–12 min; Miles and Oshiro 1990). Further investigation at pH 7.3 produced the degradation products acetic acid, methanesulfonic acid, and dichloromethylamine (all resulting from free chlorine reactions with methomyl); rates of product formation increased with increased Cl$^-$ concentrations and temperature (Miles and Oshiro 1990). Breakdown products produced at various pHs are shown in Fig. 2; at near neutral pH levels sulfoxidation occurs, whereas *N*-chlorination predominates at higher pHs (Miles and Oshiro 1990).
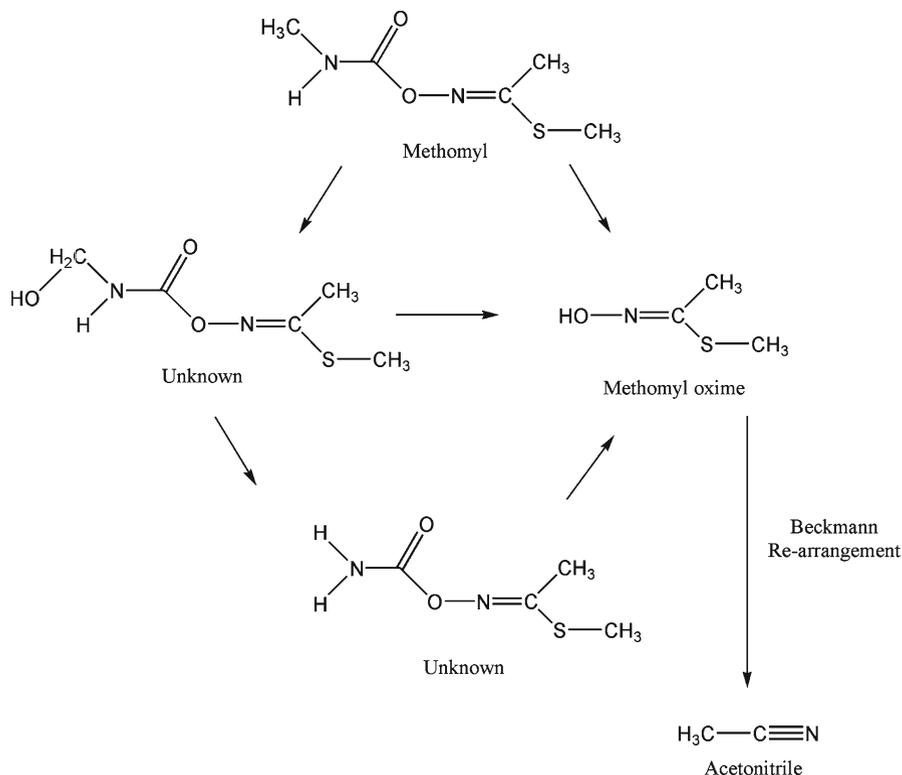
Photolysis
The degradation of methomyl by direct photolysis represents a minor degradation pathway; Tamimi et al. (2006) confirmed that direct photolysis occurs, but observed <4% degradation following 45 min of UV irradiation. Direct photolysis is negligible because methomyl's molar extinction coefficient is low for wavelengths higher than 290 nm (Tamimi et al. 2006); the wavelength spectrum for methomyl in aqueous
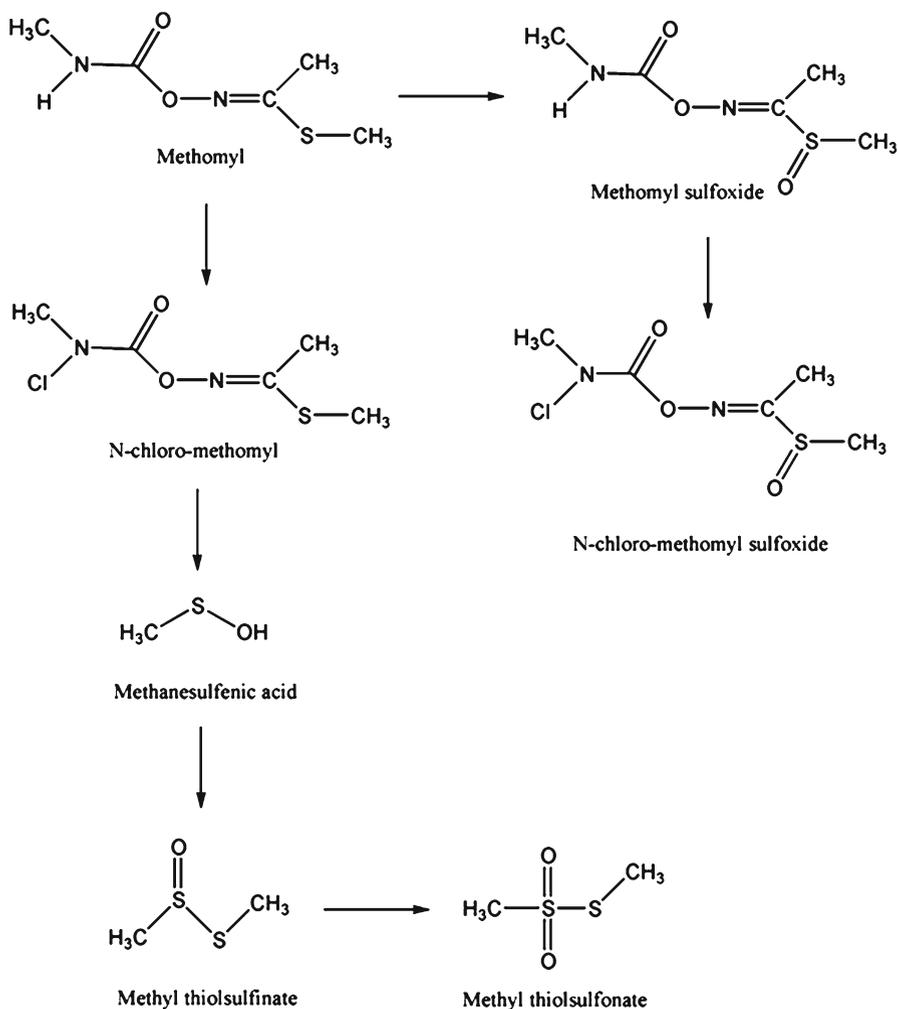
Fig. 2 Proposed reaction mechanism for methomyl under chlorinated water conditions. Additional breakdown products include acetic acid, bicarbonate, and methanesulfonic acid (adapted from Miles and Oshiro 1990)

solution is in the range of 200–300 nm, whereas the solar spectrum ranges from 300 to 600 nm (Malato et al. 2002). Tomasevic et al. (2010) investigated the influence of water quality on photolytic degradation (at 254 nm) and found that the $t_{1/2}$ of technical grade methomyl in distilled water (79.7 min; pH 5.5) was lower than that in either seawater (123 min; pH 7.9) or deionized water (97.6 min; pH 5.2). Furthermore, degradation appears to be governed by pseudo-first order kinetics, whether alone or in the presence of photosensitizers such as TiO_2 or ZnO (ZnO proved to be a better catalyst; Tomasevic et al. 2010).

Fig. 3 Proposed photocatalytic degradation pathway for methomyl. Additional breakdown products include acetamide, acetic acid, glycolic acid, oxalic acid, formic acid, and CO_2 (adapted from Tamimi et al. 2006)

Indirect photolysis is the more efficient degradation pathway for methomyl, particularly under catalytic conditions (Fig. 3). Tamimi et al. (2008) conducted various photocatalytic experiments and found that photo-Fenton and Fenton reactions more efficiently degraded methomyl (100% and 86.1%, respectively), than did direct photolysis and UV+H_2O_2-catalyzed reactions (<4% and 60%, respectively). However, Mico et al. (2010) concluded that oxidation via ozonation (10.5 mg/L O_3; pH 4.5) occurs more rapidly than the photo-Fenton reaction, with complete degradation occurring within 60 min.

Chen et al. (1984) measured the photodegradative rates of various carbamate insecticides. Methomyl was degraded most rapidly when placed on a glass slide (film thickness of 0.67 μg/cm^2) and irradiated in a photochemical reactor at an environmentally relevant wavelength (300 nm; 33–36°C). They also found that carbamates containing an electron-donating aliphatic group were more completely degraded than were those with an electron-withdrawing aromatic group (Chen et al. 1984).

The authors identified the photodegradative $t_{1/2}$ for methomyl to be approx. 48 h when it was applied as a thin film.

4.2 Biotic Processes

Microbial digestion of methomyl appears to be the most effective means by which it is degraded. Such degradation in two soils was investigated by Fung and Uren (1977), who employed perfusion experiments to study both soil sorption and microbial transformation; loss of the insecticide from a fine sandy loam (58%) soil was greater than that from fine sandy clay loam (38%) soil. In addition, since adsorption was minimal, the observed rapid loss of methomyl was attributed to microbial transformation (Fung and Uren 1977).

The influence of pH on the soil degradation of methomyl was studied by Harvey and Pease (1973). Under laboratory conditions (42-day exposure), methomyl applied to soil collected from the San Joaquin Valley of California (pH 7.9) degraded rapidly; upon termination of the experiment, some 45% of the radiolabeled parent compound had been converted to $^{14}CO_2$ and another 31% of the parent was retained within the soil extract (Harvey and Pease 1973).

Farre et al. (2002) evaluated the aerobic digestion of methomyl by using activated sludge populated with *Vibrio fischeri*. They found the insecticide and its major metabolite (*S*-methyl-*N*-hydroxythioacetamidate) to be completely degraded within 12 and 28 days, respectively; neither parent nor metabolite was toxic to the microbe. The gram-negative bacterium *Stenotrophomonas maltophilia* M1, isolated from contaminated irrigation sites in Egypt, was also found to significantly degrade methomyl (Mohamed 2009). Furthermore, *S. maltophilia* M1 contains a methomyl-degrading gene within plasmid PMb that is believed to be responsible for the observed digestion, and this gene is potentially transferable among other bacterial strains (Mohamed 2009).

Xu et al. (2009) recently isolated a gram-negative bacterium (*Paracoccus sp.* mdw-1) from activated sludge; methomyl appears to be used as its sole source of carbon and/or nitrogen, and warm alkaline conditions (30°C, pH 7–9) were optimal for both growth and degradation (Xu et al. 2009). Complete degradation of the insecticide (within 10-h incubation) by mdw-1 produced an unknown metabolite, which was speculated to be *S*-methyl-*N*-hydroxythioacetamidate (Xu et al. 2009). Furthermore, a microbial isolate of *Pseudomonas* spp. (EB20), cultured from a mineral slat medium (pH 7; 30°C), was observed to degrade 77% of methomyl (10 mg/L) within 2 weeks (El-Fakharany et al. 2011).

The white rot fungi isolate WR2 has also been shown to degrade methomyl and its metabolite—both in less than 42 days (Nyakundi et al. 2011). However, when combined with an additional isolate (WR9), degradation occurred more rapidly (22–25 days); accelerated degradation has generally occurred when microbe mixtures are present (Nyakundi et al. 2011).

5 Toxicology

5.1 *Mode of Action*

Similar to other carbamate insecticides, methomyl inhibits acetylcholinesterase (AChE), which is contained within synaptic junctions between neurons (Kuhr and Dorough 1976). When AChE is inhibited, the hydrolytic deactivation of acetylcholine (ACh) is reduced, so that it continues to stimulate the postsynaptic receptors to eventually cause nerve and/or tissue failure. In mammals, many vital functions are controlled by the peripheral nervous system, and any inhibition of these functions may lead to fatality. However, arthropods lack a peripheral nervous system (nerves outside of the central nervous system), so inhibition of AChE is not immediately fatal to them; arthropod fatality, however, may result indirectly from a secondary response caused by interrupting nerve signaling (Kuhr and Dorough 1976). Xuereb et al. (2009) exposed the amphipod *Gammarus fossarum* to various concentrations of methomyl and observed no significant mortality at 65% AChE inhibition; however, at inhibition rates of higher than 50%, significant alterations to feeding rates and locomotion were observed. Methomyl is known to cause toxicity by systemic action via direct contact or ingestion (Kuhr and Dorough 1976).

5.2 *Insects*

Methomyl is designed to target a broad range of insects, from immature stages to adults. Its penetration is thought to occur through the integument of the tracheal system, whereas penetration into the hemolymph is insignificant (Gerlot 1969). When radiolabeled forms of methomyl or methomyl oxime were applied to the abdomen of female face and house flies and black cutworm larvae, they both rapidly penetrated the bodies of fly species but much more slowly in cutworm larvae (Gayen and Knowles 1981). Four unknown metabolites were formed. However, the yield of $^{14}CO_2$ varied among the three species. A minimal amount of $^{14}CO_2$ was detected from insects treated with ^{14}C-methomyl oxime; this metabolite, therefore, is not considered to be a precursor for $^{14}CO_2$ formation (Gayen and Knowles 1981). Methomyl is also toxic to bulb mites (LC_{50}, 2.0 mg/L), some 15 times more so than bendiocarb. However, production of volatile degradation products such as acetonitrile and methylamine may have contributed to this toxicity (Gencsoylu et al. 1998).

Although many parasites and insects are beneficial to crops, they may also attract damaging predators that insecticidal formulations may target as well. Plapp and Bull (1978) studied the toxicity of methomyl to the tobacco budworm, its parasite *Campoletis sonorensis* and larvae of its predator *C. carnea* (a common green lacewing). The agent was highly toxic to all three species, when compared to endosulfan. In addition, toxicity data showed methomyl to be more potent towards the tobacco budworm, a pest (LC_{50}, 2.29 µg/vial), than the predator *C. carnea*

(LC_{50}=2.69 µg/vial; Plapp and Bull 1978). Hagley et al. (1981) exposed the adult parasite *Apanteles ornigis*, collected from infested apple tree leaves, to various insecticides under laboratory conditions and reported methomyl and permethrin to be equally toxic; however greater potency to adults than larvae was observed. Furthermore, methomyl was more toxic than synthetic pyrethroid insecticides to the adults of five parasitic species, when exposed to insecticide-treated filter papers for 5 days (Waddill 1978).

Davis and Kuhr (1974) investigated the toxicity of topically applied methomyl to three strains of 4th-instar cabbage looper (*Trichoplusia ni;* susceptible, DDT- and parathion-resistant). Following 48-h exposure, LD_{50} values of 0.029, 0.057, and 0.34 ug/larvae, respectively, were produced. These three strains of 5th-instar cabbage loopers were injected with 2 µg of ^{14}C-methomyl and were observed to possess variable degradation rates $t_{1/2s}$ of 60, 44, and 15 min, respectively (Kuhr 1973). When compared with other tissues, metabolic activity was highest in fat body tissue homogenates. The presence of oxygen and NADPH contributed to maximum activity and results suggest that methomyl is metabolically degraded by mixed-function oxidase systems (Kuhr 1973).

Methomyl was identified as being simultaneously present with other pesticides in honey bees and brood combs in Connecticut from 1983 to 1985 (Anderson and Wojtas 1986). Pooled dead bees had measurable methomyl residues ranging from 0.04 to 3.4 mg/L; however it was less frequently detected than either methyl parathion or carbaryl, indicating that insecticide combinations may be highly detrimental to bees from additive or synergistic actions (Anderson and Wojtas 1986). However, methomyl alone is highly toxic to honey bees on contact (LD_{50} <0.5 µg/bee; US EPA 1998a).

5.3   Aquatic Organisms

The bioaccumulation potential for methomyl is considered to be relatively insignificant as predicted by its log K_{ow} and water solubility values (Table 1). For example, methomyl did not significantly accumulate (<0.02 mg/L) in fish tissue following a 28-day exposure to a concentration of 0.75 mg/L (Kaplan and Sherman 1977). Although methomyl bioaccumulates only minimally, it is acutely toxic to many aquatic species. For example, it is highly toxic to *Daphnia magna* and pink shrimp (*Penaeus duorarum),* and somewhat less toxic to bluegill sunfish (*Lepomis macrochirus*) and sheepshead minnow (*Cyprinodon variegates*; Table 2). The sensitivity of *Daphnia longispina* genotypes to methomyl was compared to that of *D. magna*; the toxicity to *D. magna* (EC_{50}, 24.17 µg/L) was lower than the highly sensitive *D. longispina* M (EC_{50}, 4.71 µg/L) and *D. longispina* T (EC_{50}, 9.78 µg/L; Periera and Goncalves 2007).

Li et al. (2008) found that the potency of methomyl to topmouth gudgeon increased as exposure time increased; LC_{50} values were 1.228 mg/L at 24 h, 0.782 mg/L at 48 h, 0.538 mg/L at 72 h, and 0.425 mg/L at 96 h, respectively.

Table 2 Toxicity of methomyl to aquatic organisms[a]

Aquatic organism	Scientific name	Test	Concentration (mg/L)
Channel Catfish	*Ictalurus punctatus*	96-h LC_{50}	0.53
Bluegill Sunfish	*Lepomis macrochirus*	96-h LC_{50}	1.05
Sheepshead Minnow	*Cyprinodon variegatus*	96-h LC_{50}	1.16
Waterflea	*Daphnia magna*	48-h EC_{50}	0.0088
Pink Shrimp	*Penaeus duorarum*	96-h LC_{50}	0.019
Mysid	*Mysidopsis bahia*	96-h LC_{50}	0.23

[a]Data from US EPA (1998a)

The insecticide significantly inhibited brain AChE activity in this species at 96 h of exposure to various concentrations. However, hepatic glutathione *S*-transferase (GST) activity showed more than a 40% decline when exposed to various concentrations for 96 h (Li et al. 2008). Yi et al. (2006) investigated the inhibition of brain AChE in both male and female carp (*Carassius auratus*). Measurement of the bimolecular carbamylation and decarbamylation rates showed that methomyl inhibited AChE in both males and females at similar rates; thus AChE sensitivity was similar between genders (Yi et al. 2006).

5.4   Birds

Methomyl was toxic to terrestrial game birds on an acute oral basis (ring-necked pheasant LD_{50}, 15.4 mg/kg; mallard LC_{50}, 15.9 mg/kg); however, it is slightly toxic on a subacute dietary basis (5-day LC_{50} ranges from 1,100 to 2,883 mg/L; Tomlin 2000; US EPA 1998a). Recently, contaminated corn kernels have been linked to the death of hundreds of pigeons in Medellin, Colombia; detection of methomyl in the bodies of the birds exceeded the median lethal dose for other avian species of 10–20 mg/kg (Villar et al. 2010). Pigeons exposed to methomyl suffered decreased plasma cholinesterase (ChE) levels in brain homogenates; however, ChE levels reserged, which indicates that the effects of this carbamate insecticide are reversible (Villar et al. 2010).

5.5   Mammals

Although methomyl targets insects, studies have shown mammals to suffer adverse effects after methomyl exposure. On an acute oral basis methomyl is highly toxic to rats, with LD_{50} values of 17–24 mg/kg and a reproductive-based No Observable Effect Concentration (NOEC) of 75 mg/L (US EPA 1998a). Erythrocytes, collected from male Wistar rats, and then exposed to methomyl underwent hemolysis, a decline in both AChE and GST activities and an increase in lipid peroxidation

levels; such exposure suggests induction of oxidative damage (Mansour et al. 2009). A susceptible Chinese hamster cell line (6TG-S V79) was exposed to a log-dose range of methomyl, resulting in an LD_{50} of 959.6 μg/mL; *N*-nitroso methomyl was some 260 times more potent in the same test system (LD_{50} of 3.64 μg/mL; Wang et al. 1998). In another observation, significant inhibition of gap-junctional intercellular communication (GJIC) occurred at concentrations exhibiting little cytotoxicity (Wang et al. 1998).

Although methomyl poisoning in humans has not been widely studied, poisoning cases are known to have occurred. In one such case, a 60-year-old man was exposed via inhalation and transdermal absorption while spraying methomyl in his greenhouse; upon hospitalization his blood concentration was 1.6 mg/L (Tsatsakis et al. 2001). Fatalities have resulted from methomyl poisonings—both accidental and suicidal. For example, Driskell et al. (1991) reported the crash of a crop dusting plane as it sprayed methomyl onto grape seed fields. The methomyl level in the pilot's blood was 570 ± 9 ng/mL; the effects of methomyl on the pilot's nervous system were regarded to have resulted in the loss of control and crash. Miyazaki et al. (1989) reported a double suicide attempt, in which both spouses ingested methomyl powder; only one succumbed. The insecticide was measured in both the deceased spouse's serum (44 μg/g) and blood (0.2 μg/g), and an autopsy revealed multiple miliary hemorrhages in the brain—suspected to be the result of induced asphyxiation.

Human fatalities have also resulted from additive or synergistic interactions between methomyl and other chemicals. In one case, a 35-year-old male was discovered to have measurable blood concentrations of methomyl (3–8 ng/mL) and nicotine (222–733 ng/mL); both insecticides were also detected at high concentrations in the stomach. The adverse stimulatory actions of methomyl, in combination with nicotine on the nervous system resulted in death (Moriya and Hashimoto 2005).

6 Summary

The insecticide methomyl, an oxime carbamate, was first introduced in 1968 for broad spectrum control of several insect classes, including Lepidoptera, Hemiptera, Homoptera, Diptera, and Coleoptera. Like other carbamates, it inhibits AChE activity, resulting in nerve and/or tissue failure and possibly death. Considered highly toxic to insects (larval and adult stages), methomyl is thought to be metabolically degraded via mixed-function oxidase(s).

Methomyl has both a low vapor pressure and Henry's law constant; hence, volatilization is not a major dissipation route from either water or moist or dry soils. Photolysis represents a minor dissipation pathway; however, under catalytic conditions, degradation via photolysis does occur. Methomyl possesses a moderate-to-high water solubility; thus hydrolysis, under alkaline conditions, represents a major degradation pathway. Methomyl has a low-to-moderate sorption capacity to soil. Although results may vary with soil type and organic matter content, methomyl is

unlikely to persist in complex soils. Methomyl is more rapidly degraded by microbes, and bacterial species have been identified that are capable of using methomyl as a carbon and/or nitrogen source. The main degradation products of methomyl from both abiotic and biotic processes are methomyl oxime, acetonitrile, and CO_2.

Methomyl is moderately to highly toxic to fishes and very highly toxic to aquatic invertebrates. Methomyl is highly toxic orally to birds and mammals. Methomyl is classed as being highly toxic to humans via oral exposures, moderately toxic via inhalation, and slightly toxic via dermal exposure. At relatively high doses, it can be fatal to humans.

Although methomyl has been widely used to treat field crops and has high water solubility, it has only infrequently been detected as a contaminant of water bodies in the USA. It is classified as a restricted-use insecticide because of its toxicity to multiple nontarget species. To prevent nontarget species toxicity or the possibility of contamination, as with all pesticides, great care should be taken when applying methomyl-containing products for agricultural, residential, or other uses.

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