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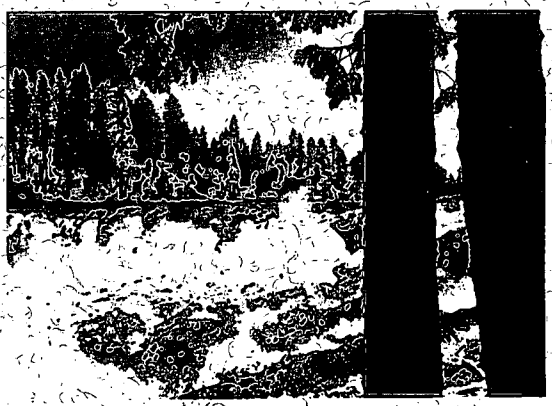
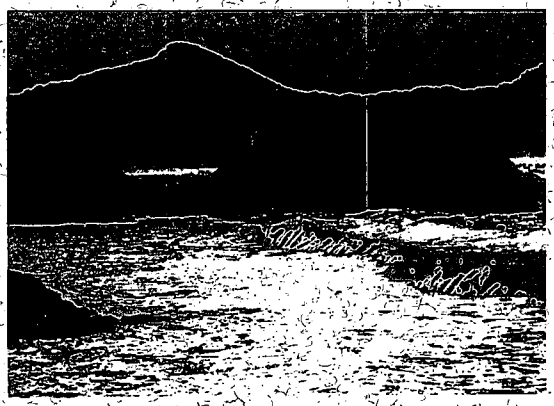
Canadian Water Quality Guidelines for Metolachlor

R.A. Kent, B.D. Pauli, D.M. Trotter and J. Gareau



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WATER QUALITY BRANCH
OTTAWA, ONTARIO, 1991

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***Monenco Consulting Ltd.**
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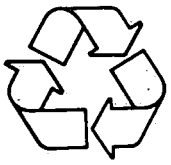

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Abstract

A literature review was conducted on the uses, fate, and effects of metolachlor on raw water for drinking water supply, freshwater aquatic life, agricultural uses, recreational water quality and aesthetics, and industrial water supplies. The information is summarized in this publication. From it, water quality guidelines for the protection of specific water uses are recommended.

Résumé

On a examiné la documentation relative aux utilisations, à l'évolution et aux effets du métolachlore sur les sources d'approvisionnement en eau potable, sur les organismes aquatiques d'eau douce, sur l'utilisation de l'eau pour l'agriculture, sur la qualité de l'eau pour les loisirs et l'esthétique, ainsi que sur l'eau utilisée à des fins industrielles. Ces renseignements sont résumés dans cette publication. À partir de cette étude, on recommande des concentrations limites de métolachlore afin de protéger les diverses utilisations de l'eau.

Canadian Water Quality Guidelines for Metolachlor

R.A. Kent, B.D. Pauli, D.M. Trotter and J. Gareau

SOURCES, OCCURRENCE, AND CHARACTERISTICS

Uses and Production

Metolachlor, the common name for the chloroacetamide herbicide 2-chloro-6'-ethyl-N-(2-methoxy-1-methylethyl)-acet-o-toluidide (IUPAC), is a colourless, odourless liquid. It has the Chemical Abstracts Service (CAS) name 2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl)-acetamide and the CAS Registry No. 51218-45-2 (Worthing and Walker, 1987). It was introduced in 1974 by Ciba-Geigy AG under the code name CGA-24705 and marketed as a herbicide under the trade name "Dual®."

The technical-grade metolachlor product marketed in Canada is Dual® Ciba-Geigy 960E. This product is an emulsifiable concentrate or emulsion containing 960 $g \cdot L^{-1}$ of the active ingredient (ai). Formulations include Primextra, a mixture of 300 $g \cdot L^{-1}$ metolachlor and 200 $g \cdot L^{-1}$ atrazine, and Galex 500 EC, a mixture of 200 $g \cdot L^{-1}$ metobromuron and 300 $g \cdot L^{-1}$ metolachlor (both marketed by Ciba-Geigy Canada) (Agriculture Canada, 1989).

Metolachlor is a germination inhibitor used mainly for weed control of grasses. Agricultural applications are made using preemergence (Chesters *et al.*, 1989) or preplant incorporated treatments (Thomson, 1979; WSSA, 1983). Application rates are 1.4–4.5 kg ai·ha^{-1} for crop and noncrop areas, depending on soil and climatic conditions (U.S. EPA, 1988). Metolachlor can be used for weed control in corn, soybeans, potatoes, snap beans, dry beans, sorghum, lima beans, sugar beets, and rutabagas. It is used in combination with atrazine for weed control in corn. Weeds controlled by metolachlor include crab grass, goosegrass, witch grass, barnyard grass, fall panicum, pigweed, foxtails, yellow nutsedge, and eastern black nightshade (Chesters *et al.*, 1989; Ontario Ministry of Agriculture and Food, 1989). Metolachlor has recently been recommended for use in a winter barley/no-till grain sorghum rotation (Diawara and Banks, 1990). In the United States, metolachlor is applied using ground spray equipment, aircraft, or centre pivot irrigation systems (U.S. EPA, 1988).

Metolachlor is not manufactured in Canada and was first registered in Canada in 1977 (Agriculture Canada, 1989). Reported imports of metolachlor for Canada in 1985, 1986, and 1987 were 4839, 4522, and 4322 t, respectively (Statistics Canada, 1986, 1988). In New Brunswick, 221 kg of metolachlor were sold in 1985 (Shanks, 1985). In 1986 and 1987, 230 and 182 kg, respectively, were sold (Shanks, 1986, 1987). Since the withdrawal from general use of the similar chloroacetamide herbicide alachlor in 1985 (Frank *et al.*, 1990), the consumption of metolachlor has increased significantly. In Ontario, for instance, 842 t of the metolachlor active ingredient were used on field crops, fruits, vegetables, and roadsides in 1983 (McGee, 1984). By 1988, this value had risen to over 1724 t (Moxley, 1989). Over the same years, alachlor use decreased from 1060 t in 1983 (McGee, 1984) to 2.2 t in 1988 (Moxley, 1989), which made metolachlor the most used herbicide in Ontario in 1988.

Physical and Chemical Properties

The structural formula for metolachlor is shown in Figure 1. Selected physical and chemical properties of metolachlor are presented in Table 1.

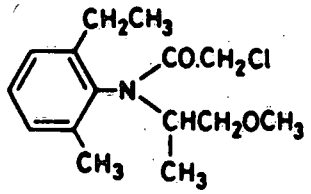

Figure 1. Structural formula for metolachlor.

Although various authors stated that metolachlor was soluble in most organic solvents, details of its solubility in these solvents were not provided. For instance, it is "very" soluble in benzene, dichloromethane, hexane, methanol, and octan-1-ol

(Worthing and Walker, 1987), but it is insoluble in ethylene glycol and propylene glycol (WSSA, 1983). Its aqueous solubility is reported to be 530 $mg \cdot L^{-1}$ (Worthing and Walker, 1987).

Table 1. Physical and Chemical Properties of Metolachlor

Parameter	Value
Chemical formula	$C_{15}H_{22}ClNO_2$[1]
Molecular weight	283.8[1]
Physical state	Colourless, odourless liquid at 25°C[2]
Boiling point	100°C at 0.001 mmHg[1]
Specific gravity	1.085 ± 0.005 at 20°C[1]
Vapour pressure	1.3×10^{-5} mmHg at 20°C[1] 1.7 mPa[3]
Henry's Law constant	3.7×10^{-7}[6]
Aqueous solubility	530 $mg \cdot L^{-1}$ at 20°C[3]
Aqueous stability	Half-life of a 0.25% solution at 100°C was 30 h at pH 3, 18 h at pH 7, and 1.5 h at pH 10[4]
Adsorption coefficients	
K_{oc}	0.21–0.47 $m^3 \cdot kg^{-1}$ for soil organic matter content from 1.3 to 34.5 $g \cdot kg^{-1}$[5]
K_d	0.76×10^{-3} to 1.75×10^{-3} $m^3 \cdot kg^{-1}$ for soil organic matter content from 1.3 to 34.5 $g \cdot kg^{-1}$[5]
Half-life in soil	30–50 d (northern U.S.)[1] 15–25 d (southern U.S.)[1] 22–531 d (laboratory rates with various moisture regimens)[4]
Elemental analysis	C, 63.48%; H, 7.83%; Cl, 12.49%; N, 4.93%; O, 11.27%
K_{ow}	$\log(P_{ow}) = 3.13$[2]

[1] WSSA, 1983
[2] LeBaron *et al.*, 1988
[3] Worthing and Walker, 1987
[4] U.S. EPA, 1987b
[5] Wood *et al.*, 1987
[6] Chesters *et al.*, 1989

A summary of analytical techniques and detection limits for quantifying metolachlor residues in soil and water is presented in Table 2.

Mode of Action

Metolachlor, along with the general class of chloroacetamide herbicides, is a plant growth inhibitor. Although its specific biochemical mode of action is unknown, metolachlor's general mode of action appears to be the inhibition of protein synthesis, terpenoid synthesis (specifically the inhibition of the incorporation of the amino acid leucine into protein) (Pillai *et al.*, 1979), and gibberellic acid synthesis (LeBaron *et al.*, 1988; Wilkinson, 1988). Metolachlor is also reported to inhibit fungal RNA synthesis and thus would appear to interfere with the assembly of nucleic acids (Fisher and Hayes, 1985). The primary site of metolachlor uptake is the coleoptile region (Braverman *et al.*, 1985). This usually allows susceptible species to germinate, but the seedlings either do not emerge from the soil or emerge with stunted or abnormal growth (LeBaron *et al.*, 1988). Early seedling growth is probably restricted as a result of inhibition of cell division and enlargement, cortical cell expansion, and mitotic activity (Chesters *et al.*, 1989). Details of the histologic and morphologic symptoms of metolachlor toxicity in sorghum were reported by Ebert (1980) and Paradies *et al.* (1981).

Metolachlor is metabolically deactivated by tolerant plant species (WSSA, 1983; Chesters *et al.*, 1989). Pathways for the metabolism of metolachlor in corn begin with the conjugation of the chloroacetyl side chain with glutathione. Subsequently, the glutathione tripeptide is broken down to the cysteine conjugate, which then undergoes oxidative deamination. Reduction of the transient α-ketoacid to the thiolacetic acid conjugate is followed by oxidation to the corresponding sulfoxide derivatives. These represent the terminal products of the glutathione-dependent metabolic system (LeBaron *et al.*, 1988). These derivatives may have the side chain ether group cleaved for final conjugation with glucose. Similar metabolic pathways occur in lettuce and potatoes (Szolics *et al.*, 1981a, 1981b).

Resistance to the toxic effects of metolachlor in some plants is conferred by the action of the enzyme glutathione S-transferase, which has the ability to conjugate the herbicide with glutathione to form a nontoxic complex (Edwards and Owen, 1986). Chemical seed protectants or safeners protect nontarget plants such as grain sorghum against injury by stimulating the spontaneous and enzymatic conjugation of metolachlor and glutathione (Zama and Hatzios, 1986). In addition, chemicals used for seed treatments to protect seedlings from metolachlor toxicity can enhance the

plant's ability to metabolize metolachlor (Fuerst and Gronwald, 1986).

Metolachlor in the Environment

Metolachlor may enter surface waters as a result of accidental spills or application to watercourses, and by surface or subsurface movement from treated fields. Metolachlor has also been found in rainwater as a result of direct evaporation and recondensation from treated soil and plants (maximum concentration of 2.4 $\mu g \cdot L^{-1}$); its presence in rainwater coincided with agricultural applications (Baker, 1986; Richards *et al.*, 1987).

Table 2. Analytical Techniques for Determining Metolachlor Residues in Soils and Water

Matrix	Extraction, solvent, cleanup	Apparatus	Recovery (%)	Parent and/or metabolites	Detection Limits (μg^{-1} [soil] or $\mu g \cdot L^{-1}$ [water])	Reference
Soil	Methylene chloride	GLC/FID (M) TLC (Metab) MS (Metab)	NR	M & Metab	NR	Liu *et al.*, 1988
Soil	Methanol and partitioned into hexane/alumina columns/elution with hexane:ether, 2:1 v/v	GLC/ECD	90±2.0	M	NR	Walker and Zimdahl, 1988
Soil	Methanol and partitioned into hexane	GC/ECD	97±7	M	NR	Braverman *et al.*, 1986
Soil	Dichloromethane reflux in Soxlet/ evaporation/ethyl acetate	GC/TSD	84±5 (extraction)	M	1.0	Harvey, 1987
Water (culture media)	Methylene chloride/ redissolved in methanol	GC/FID HPLC GC/MS	NR	M & Metab	NR	Krause *et al.*, 1985
Water	Methylene chloride, sonification if needed	GC/N-P	80 (avg.)	M	35–44.7	Kramer and Baker, 1985
Water	Freon 113	GLC/FID GC/MS	NR	M & Metab	NR	McGahen and Tiedje, 1978
Water	Methylene chloride, sonification if needed	GC/N-P	67 (avg.)	M	0.25	Richards *et al.*, 1987
Water	Chloroform/ evaporation, redissolve in mentanol	GLC/ECD	70-95	M	0.1	Frank *et al.*, 1987a, 1987b
			80–90	M	<0.02	Frank and Logan, 1988
			80–90	M	0.05	Frank *et al.*, 1990
Water	Dichloromethane	GC/ECD	NR	M	5	Pionke *et al.*, 1988

ECD = electrolytic conductivity detector
FID = flame ionization detector
HPLC = high-pressure liquid chromatography
GC = gas chromatography
GLC = gas-liquid chromatography
M = metolachlor
Metab = metolachlor metabolites
MS = mass spectrometry
N-P = nitrogen-phosphorus detector
NR = not reported
TLC = thin-layer chromatography
TSD = thermionic specific detector (the nitrogen-phosphorus detector is equivalent to the TSD)

Although only limited surveys for metolachlor in surface waters have been conducted, most records of water contamination in Canada appear to involve wells contaminated by spillage or back-siphoning from tanks used to mix metolachlor and water (Frank *et al.* 1987a, 1987b). For instance, Frank *et al.* (1987a) found 1 of 91 wells in southern Ontario contaminated with metolachlor. The herbicide had been used on 25 farms in the summer before the survey. The metolachlor concentration was 112 $\mu g \cdot L^{-1}$ in the first sample taken from the well and had been reduced to only 29 $\mu g \cdot L^{-1}$ 225 d after pumping out the well. The authors concluded that the well became contaminated through introduction of the chemical from the surface while mixing and loading spray equipment.

Concentrations in Water, Sediment, and Biota

Metolachlor has been found in surface and subsurface waters (Appendix A). Extensive sampling at the mouths of the Grand, Saugeen, and Thames rivers in southern Ontario between January 1981 and December 1985 showed metolachlor to be present in 21 of 454 samples (Frank and Logan, 1988); over 339 t of metolachlor were used on the total crop area of just over 1 million hectares (0.34 $kg \cdot ha^{-1}$) in 1983. Mean metolachlor concentrations for this period in the Grand, Saugeen, and Thames rivers were 0.9, 0.7, and 3.6 $\mu g \cdot L^{-1}$, respectively (Frank and Logan, 1988). In each river, metolachlor was found during only 1 or 2 of the 5 sampling years.

When the Ontario Ministry of the Environment (OMOE, 1987a, 1987b) sampled 15 municipal waterworks in 1985, 6 of 31 samples contained metolachlor with a concentration range of 0.4–5.1 $\mu g \cdot L^{-1}$. None of the treated water samples contained metolachlor. Also in 1985, the OMOE (1987a) sampled 351 private wells. These wells were not selected at random but were chosen because of their perceived susceptibility to pesticide contamination. A total of 52 wells (15%) showed metolachlor contamination, and 4 of these wells had metolachlor in concentrations above 105 $\mu g \cdot L^{-1}$. The high concentration was probably the result of infiltration of contaminated surface runoff into poorly constructed or sited wells. In 1986 (OMOE, 1987b), 42 groundwater sampling sites, consisting of 37 domestic wells and 5 municipal groundwater supply wells in areas of intense corn and soybean production, were sampled. Metolachlor was detected in 3 domestic wells with a concentration range of 1.2–3.2 $\mu g \cdot L^{-1}$. During the same year, 25 municipal surface waterworks were monitored for pesticide levels in raw and treated water. Metolachlor was detected at 8 of the 25 locations in 40 of 417 (10%) samples collected. The concentration range was from 0.51 to 15 $\mu g \cdot L^{-1}$. Metolachlor was also found in the treated water at 5 locations; 23 of 150 samples (15%) contained metolachlor with a range of concentrations between 0.47 and 5.97 $\mu g \cdot L^{-1}$. In the May to August sampling period of 1987, 7 of 12 samples from the Sydenham River in this area contained metolachlor (maximum concentration of 14 $\mu g \cdot L^{-1}$), and 6 of 12 drinking water samples were contaminated (maximum concentration of 16 $\mu g \cdot L^{-1}$) (Frank *et al.*, 1990).

In the United States, metolachlor was detected in 1644 of 1997 (82%) surface water samples tested, with a maximum concentration of 138 $\mu g \cdot L^{-1}$ (U.S. EPA, 1987a). The 85th percentile for all detectable concentrations was 11.5 $\mu g \cdot L^{-1}$. According to the U.S. EPA (1987a), metolachlor was detected in 45 of 239 groundwater samples in the United States, with a maximum concentration of 0.25 $\mu g \cdot L^{-1}$. Chesters *et al.* (1989), however, reported that metolachlor was found in 49 of 442 groundwater samples, with a maximum concentration of 680 $\mu g \cdot L^{-1}$. This maximum concentration was the result of mishandling of the herbicide around a well. A concentration of 12 $\mu g \cdot L^{-1}$ was found in a monitoring well in Wisconsin following normal agricultural applications.

Pionke *et al.* (1986) tested water from 18 wells and two springs in agricultural areas of Pennsylvania. Metolachlor was not detected in any of the samples. Fishel and Lietman (1986) also sampled groundwater in Pennsylvania and detected a maximum concentration of 3.4 $\mu g \cdot L^{-1}$ during the fall. In Wisconsin, from a total of 1508 analyses involving 358 wells, metolachlor was detected in 1 sample (the actual concentration was not reported but was below 25 $\mu g \cdot L^{-1}$) (Krill and Sonzogni, 1986).

Information on metolachlor concentrations in sediments or biota was not found in the literature.

Environmental Fate, Persistence, and Degradation

Soil

A major factor controlling movement of metolachlor in the environment is adsorption to soil. Organic matter, clay content, and cation exchange capacity are the most important soil characteristics

in terms of increased metolachlor adsorption (Obrigawitch *et al.*, 1981; Strek and Weber, 1981: Weber and Peter, 1982; Kozak *et al.*, 1983; Peter and Weber, 1985; Braverman *et al.*, 1986; Wood *et al.*, 1987). Adsorption is lower in alkaline soil (Jordan, 1978), but changes in pH below pH 7 have little effect on adsorption (Chesters *et al.*, 1989).

Adsorption to clay may result in the retention of metolachlor in surface soils. A sandy loam soil that had previously received several metolachlor treatments over a period of years (quantities not given) contained 29.3 $\mu g \cdot kg^{-1}$ metolachlor in the surface layer (4% clay) and only 8.4 $\mu g \cdot kg^{-1}$ at 2.5–3.5 cm depth (0.3% clay) (Huang and Frink, 1989). Generally, soil adsorption increases with increasing soil organic matter content (Peter and Weber, 1985). However, because soil adsorption does not always strictly parallel increases in soil organic matter and clay content, Chesters *et al.* (1989) suggested that the type of organic matter may influence adsorption.

Soil distribution coefficients (K_d), which measure the amount of metolachlor adsorbed to soil in a metolachlor–soil–solvent solution at equilibrium, ranged from 0.5 to 10.9 (ratio of the amount adsorbed [in $nmol \cdot g^{-1}$] to the equilibrium concentration [in μM]) in a study conducted by Peter and Weber (1985). The K_d values corresponded to a range in soil organic matter from 0.5% to 8.7%, and adsorption was positively correlated to organic matter and clay content.

Within the soil organic fraction, humic substances are the most important components influencing adsorption. Adsorption is thought to occur as a result of multifunctional hydrogen bonding between the carbonyl oxygen of the metolachlor molecule and hydrogen atoms of the carboxyl and hydroxyl groups of humic substances (Kozak *et al.*, 1983). Charge transfer bonding between the aromatic nucleus of metolachlor and aromatic rings of organic matter are also thought to play a part in the adsorption mechanism (Peter and Weber, 1985).

Various field trials have demonstrated the effect of soil composition on metolachlor leaching. Leachability, as measured by R_f values, was negatively correlated with organic matter, cation exchange capacity, and K_d values (Jordan, 1978), emphasizing the influence of adsorption on mobility. Metolachlor applied at rates of 3 and 6 $kg \cdot ha^{-1}$ to a tropical soil containing 1.9% organic matter and 13.2% clay was found to have leached to a depth of 30 cm 84 d after treatment. Over the same period, the same application of metolachlor leached to a depth of only 20 cm in a similar soil with increased organic matter (2.1%) and clay (17.2%) content (Utulu *et al.*, 1986). The organic material in hardwood tree bark (species not given) is more efficient than a soil–peat mixture in retarding the movement of metolachlor in greenhouse studies (Fine *et al.*, 1982; Kuhns *et al.*, 1982). The environmental significance of this particular retarding effect of tree bark was not discussed by the authors.

In field studies using a light-textured Ontario Plainfield sand soil (91.5% sand; 1.5% silt; 7% clay; 0.7% organic matter), metolachlor residues exhibited limited downward movement (to only 10 cm) after 386 mm of rainfall (Bowman, 1988). In other experiments also conducted by Bowman (1988, 1989), lysimeters that were 15 cm in diameter and 75 cm in length were buried in a sand-filled enclosure with 5 cm of the lysimeter cylinder projecting above the soil surface. The lysimeters received rainfall totalling 707 mm from 14 May to 8 October in 1986 and 526.6 mm in 1987, including supplementary artificial watering. Effluents were removed from a 1-L Pyrex beaker beneath the lysimeters via a 0.48-cm (i.d.) stainless steel tube. Dual® 960 E was applied to the surface of each lysimeter as a 10-mL aqueous emulsifiable concentrate to provide 5.27 mg per lysimeter. This was equivalent to 2.75 $L \cdot ha^{-1}$, the maximum recommended field application rate for metolachlor. With the supplemental watering, metolachlor was leached to only 40 cm in the lysimeter.

Metolachlor was not detected at depths greater than 30 cm in a field study near Ottawa during a year in which rainfall was unusually heavy (Patni *et al.*, 1987). The authors assumed that all rainfall reaching tile drains (0.6–0.9 m below the soil surface) and drainage ditches percolated through the soil because of the lack of slope (<0.02%) for surface water runoff from the plots. Metolachlor concentrations in the drainage water ranged from not detected (detection limit of 0.05 $\mu g \cdot L^{-1}$) to 12 $\mu g \cdot L^{-1}$ after a metolachlor application of 2.6 kg $ai \cdot ha^{-1}$.

In a Hagerstown silty clay loam soil in Pennsylvania, Hall *et al.* (1989) bored horizontal channels 1.2 m under conventionally tilled (CT) and no-tillage (NT) corn fields and installed plastic

gutters to collect water percolating to this depth after rainfall events. A preemergence metolachlor application of 2.2 kg ai·ha^{-1} was made in May. In 1984, a total of 109 cm of rainfall was recorded in this area. The mean concentration of metolachlor in NT percolates was higher (1.4 $\mu g \cdot L^{-1}$) than in CT percolates (0.6 $\mu g \cdot L^{-1}$). The maximum concentration of metolachlor in NT percolates was considerably higher (21.5 $\mu g \cdot L^{-1}$) than in CT percolates (2.5 $\mu g \cdot L^{-1}$). The percentage of applied herbicide reaching the gutters 1.2 m below the soil surface was less than 0.1% for CT and 0.17% for NT. As metolachlor residues were not detected in soil cores below 61 cm but were detected in soil leachates at 1.2 m, the authors concluded that macropore transport of the herbicide in the soil was occurring. (Patni *et al.* [1987] arrived at a similar conclusion after their field study.) Although approximately the same amount of rain (100 cm) fell in 1985, mean herbicide concentrations in drainage were much greater under both tillage systems. The loss for 1985 was 0.43% for CT and approximately 1.5% for NT. The authors concluded that the yearly differences were related to the number of leaching events and their proximity to the herbicide application date.

Chesters *et al.* (1989) reported that no field studies concerning metolachlor concentrations in surface runoff were found during their extensive review of the literature. In a simulation study, a plot of loamy sand soil with 1.5% organic matter and 8% slope was treated with 1.1 kg·ha^{-1} metolachlor. On days 1, 3, and 7 after treatment, 3.8 cm of rain was applied at 1.3–2.5 cm·h^{-1}. Of the applied metolachlor, 4.5% was lost in surface runoff and sediment (Dynamac Corporation, 1986).

The primary factor affecting metolachlor degradation in soil is microbial activity (Table 3). Aerobic soil microorganisms produced $^{14}CO_2$ from ring-labelled metolachlor during 84 d of incubation in a clay loam soil (Ellgehausen, 1976a). After a short lag phase, 4.8% of the applied metolachlor was converted to $^{14}CO_2$. The remaining metabolic products consisted mainly of the oxalic acid derivatives of metolachlor (18% of total radioactivity). Less than 8% of the 5 mg·kg^{-1} treatment remained unchanged after 84 d. By contrast, in soil sterilized using an autoclave, 65% of the applied metolachlor remained unchanged after the same period. A dechlorinated derivative of metolachlor accounted for 30% of the applied dose in the sterilized soil. In the nonsterile soil, this same compound comprised only about 1% of the total radioactivity in the soil extracts. The imposition of anaerobic conditions greatly reduced $^{14}CO_2$ liberation from nonsterile soils, and sterilization of anaerobic soils almost completely prevented production of $^{14}CO_2$. Further experiments with soils containing insoluble residues from aerobic and aged anaerobic soils indicated that the nonextractable residues are primarily formed by microbial activity and are susceptible to further microbial degradation (Ellgehausen 1976a, 1976b). During a 28-d incubation, no $^{14}CO_2$ was liberated from a soil sterilized with γ-irradiation that had been given a ^{14}C-metolachlor treatment (Liu *et al.*, 1988).

Additional laboratory studies using fungal and bacterial cultures (e.g., Krause *et al.*, 1985; Bailey and Coffey, 1986; Saxena *et al.*, 1987; Liu *et al.*, 1987, 1989) confirmed the ability of some microbes to transform and degrade metolachlor. Chesters *et al.* (1989) listed the metabolites of metolachlor produced by chemical and microbial degradation in various environments, as well as the microorganisms capable of metabolizing metolachlor.

Acclimation of the soil microbial community to metolachlor had a dramatic effect on the rate of biodegradation of ^{14}C ring-labelled metolachlor. The microbial community from a Virginia soil that had received treatments of the commercial formulation Dual® for 5 consecutive years was able to degrade 5 times the amount of metolachlor as an unacclimated microbial community in the same amount of time (Liu *et al.*, 1988). This study contradicted the results of a study by Harvey (1987) in which previous applications of metolachlor for 5 consecutive years to Wisconsin soils failed to enhance metolachlor degradation. Harvey (1987) quantified the amount of metolachlor remaining in sealed 150-mL polyethylene containers after a 12-d incubation at 25°C to be 52% of the initial 4 mg·kg^{-1} application in soils with a previous history of metolachlor application. Liu *et al.* (1988) used a 50 mg·L^{-1} solution of ^{14}C ring-labelled metolachlor continuously perfused through a soil column for 28 d at 24°C–28°C. Labelled CO_2 and volatile metabolites were trapped in appropriate solutions as sterile air was passed through the system. The contradictory results of Harvey (1987) and Liu *et al.* (1988) may have been due to the different techniques used by each investigator.

In a laboratory experiment using silt loam soils from rice fields, metolachlor degradation versus soil moisture potential was studied using two soil

Table 3. Summary of Metolachlor Degradation in Soil/Sediment, Water, and Biota

Soil/Sediment

Photolysis
- $t_{1/2}$ approximately 8 d under ideal conditions in the laboratory; $t_{1/2}$ in the field considerably greater[1]
- volatilization makes lab data difficult to interpret[2]

Oxidation
- no data

Aerobic metabolism
- major degradative pathway [1] [2] [3]
- major metabolites, see U.S. EPA, 1980

Anaerobic metabolism
- little occurs, few data[2]

Volatilization
- relatively nonvolatile, but under certain conditions volatility may be a significant factor in dissipation[1]

Mobility
- little leaching or lateral movement in soils[1]
- adsorption and soil texture dependent; more movement in sandy soils; most leaching occurs in soil columns after heavy precipitation on coarse-textured soils low in organic matter[2]

Adsorption/Desorption
- related to organic matter and clay content but not to silt content[2]
- more adsorption to muck or clay soils[1]
- adsorption decreases with decreasing temperature[2]
  K_d = 1.5–11 on sandy loam soils
  0.71–4.3 on silt loam soils
  11 on silty clay loam soils
  1.8 on a clay soil
  K_d increases with organic matter content[2]
- no sediment adsorption data

Persistence
- depends on temperature and moisture
  $t_{1/2}$ = 13-38 d at 10°C-30°C on clay loam (lab)[4]
  $t_{1/2}$ = 21-110 d at 5°C-30°C on sandy loam (lab)[5]
  $t_{1/2}$ = 14-19 d in sandy to loamy sand soil (lab)[6]
  $t_{1/2}$ = 11-52 d in silt loam (field)[5]
  $t_{1/2}$ = 39-70 d in sandy loam (field)[5]
  $t_{1/2}$ = 26-42 d in sterile and nonsterile sediment[7]

Water

Photolysis
- not a major path of loss
- little occurs in aqueous solution[8]
- slow photolysis; 8% in 30 d[2]

Oxidation, aerobic metabolism, anaerobic metabolism, persistence
- no data

Volatilization
- not a major fate process; relatively nonvolatile [8]
- no "good" data, but may be an important dissipation pathway in the field [2]

Biota

- rapidly absorbed, metabolized, and excreted in the urine and feces of goats, rats, and poultry
- no residues found in eggs, meat, or fat samples of laying chickens
- depuration in rats with $t_{1/2}$ of 28 h
- reactions in rats include dechlorination, O-demethylation, N-dealkylation, and side chain oxidation
- in rats, metabolites were N-(2-ethyl-6-methylphenyl)hydroxyacetamide and N-(2-ethyl-6-methylphenyl)-N-(hydroxyacetyl)-DL-alanine in urine [2]
- absorbed and readily eliminated in fish[2]
- rapidly eliminated from fish, daphnids, and algae[9]

[1] WSSA, 1983
[2] Chesters *et al.*, 1989
[3] Bouchard *et al.*, 1982
[4] Zimdahl and Clark, 1982
[5] Walker and Brown, 1985
[6] Rao *et al.*, 1986
[7] McGahen, 1982
[8] LeBaron *et al.*, 1988
[9] Ellgehausen *et al.*, 1980

moisture regimens: (1) -30 kPa moisture (20% moisture by weight for this soil) over the entire 70-d incubation period; and (2) soils brought to an initial -30 kPa moisture potential and allowed to dry over 70 d (Braverman *et al.*, 1986). The degradation rate of metolachlor was not significantly correlated with declining moisture potentials. The evolution of CO_2 was not correlated with metolachlor degradation during these experiments, but an adequate explanation for this was not given.

Walker and Barnes (1981) wrote a simulation model to predict herbicide persistence in soil. Metolachlor dissipation rates from various U.S. soils observed in the laboratory were compared with those predicted by the model. The model overestimated metolachlor persistence; of 48 predicted soil residue levels, 41 measured values were below those predicted (actual values not given); 16 measured values were more than 30% below the predicted values, and 6 were over 50% less than predicted (Walker and Zimdahl, 1981). Loss through volatilization was considered to be one of the major factors contributing to the observed differences between the model predictions and the measured values.

With a vapour pressure of 7×10^{-3} Pa (1.3×10^{-5} mmHg) at 20°C, metolachlor is relatively nonvolatile. Given the data from laboratory experiments, volatilization loss of metolachlor applied to field soils was estimated to range from 0.6% to 1.4% within the first 24 h (Burkhard, 1977). Metolachlor at 80 $\mu g \cdot g^{-1}$ (wet soil) was calculated to volatilize at a rate of 1.5–4.5 $ng \cdot cm^{-2} \cdot h^{-1}$ at 20°C with an airflow over the soil surface of 30 $L \cdot h^{-1}$ (Burkhard and Guth, 1981). Volatilization rates of 0.03–0.09 $kg \cdot ha^{-1} \cdot d^{-1}$ were reported for three soil types (soil types not given) containing 80 $\mu g \cdot g^{-1}$ metolachlor, 12% moisture at 35°C, 100% relative humidity, and with a 30 $L \cdot h^{-1}$ airflow over the soil surface. Increased volatilization was associated with lower soil organic matter content. Raising the temperature 10°C to 45°C increased the rate of volatilization by a factor of about 3.8, whereas decreasing the temperature to 25°C decreased volatilization by about the same amount. Increasing the flow of air over the soil to 60 $L \cdot h^{-1}$ approximately doubled the loss.

Small volatilization losses (0.1%) of metolachlor from a soil surface after 8 d have been reported (Parochetti, 1978). By contrast, the volatilization loss of metolachlor from a glass surface can approach 50% in several days, and 11%–37% was estimated to volatilize from plant surfaces (straw, tall fescue [*Festuca arundinacea*], and giant foxtail [*Setaria faberi*] left on the soil to simulate a no-till situation) within a few days, depending on the velocity of air passing over the plant surfaces (Parochetti, 1978; Strek and Weber, 1981).

Little chemical hydrolysis of metolachlor occurs. Worthing and Walker (1987) reported that at 20°C, 50% loss of the compound due to hydrolysis was calculated to require more than 200 d over a pH range from 1 to 9. The compound is also stable to decomposition at temperatures up to 300°C.

Loss by photodegradation is considered insignificant for agricultural applications of metolachlor (LeBaron *et al.*, 1988). Aziz (1974) applied ^{14}C-labelled metolachlor to a thin film of soil on glass slides and exposed the slides to sunlight. After 8 d, 50% of the herbicide was photodegraded, but more than 10% had volatilized; temperatures during the experiment reached 50°C–55°C, making the relevance of the results to field situations questionable. Most of the photodegradation products were accounted for by 2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-hydroxy-1-methylethyl)acetamide (Aziz, 1974). Another photoproduct observed was N-chloracetyl-N-(hydroxprop-1-en-2-yl)-2-ethyl-6-methylaniline (Chesters *et al.*, 1989).

As the primary cause of metolachlor dissipation under normal conditions in field soils is biodegradation, environmental factors that favour increased microbial density and activity will decrease the persistence of metolachlor in soil. For instance, Bouchard *et al.* (1982) measured metolachlor degradation at depths of 10–20 cm and 40–50 cm in a silt loam soil in Arkansas. Increased organic matter content of the soil, which favours increased microbial densities and higher adsorption of metolachlor, reduced metolachlor persistence from 455.7 d at the 40- to 50-cm layer (0.5%–0.7% organic matter) to 277.2 d in the 10- to 20-cm layer (0.9%–1.1% organic matter). During soil incubation studies, increased temperature, which favours increased microbial activity, produced metolachlor half-lives of 36–45 d at 37°C, whereas at 15°C half-lives were 182–203 d (Bouchard *et al.*, 1982). Soil moisture, which also influences microbial activity, affects metolachlor persistence,

with greater degradation rates at 80% field capacity moisture (15.8-d half-life) than at 20% moisture (37.6-d half-life) (Zimdahl and Clark, 1982).

Only one report was found that concluded that leaching is a major route of field dissipation for metolachlor (Skipper *et al.*, 1976). This study was conducted in sandy loam soils with 1.6% and 2.3% organic matter. Herbicide persistence was measured using a corn bioassay. Both field sites had relatively high rainfall (22 cm in 4 weeks at one site and 16 cm in 8 weeks at the other) during the study, which may have contributed to the conclusion that leaching caused the observed dissipation. However, consideration was not given to the possible influence of biodegradation; it is possible that biodegradation played a major role in the loss of metolachlor. A summary of the studies of metolachlor persistence that have been conducted in both the field and laboratory is presented in Appendix B.

Rao *et al.* (1986) suggested that metolachlor dissipation rates observed in different studies depend on a combination of the inherent variability in soil due to natural pedogenic processes and the soil and crop management techniques practised during any specific degradation study. Metolachlor half-lives from the surface layer (0–20 cm) of three soils in Georgia ranged from 14 to 19 d. In the same area, half-lives in subsurface soils (i.e., 25–46 cm, 48–63 cm, and 94–107 cm) were approximately twice as long and tended to be more variable. Average half-lives ranged from 27 to 43 d for the subsurface soils (Rao *et al.*, 1986). From lysimeter studies in southern Ontario, Bowman (1988) reported half-lives of 23–28 d in a Plainfield sand soil. Patni *et al.* (1987) measured metolachlor dissipation rates in a field study near Ottawa, Ontario. Under the cool, moist conditions of the growing season, metolachlor degradation followed first-order degradation kinetics. The half-life in the top 0- to 7.5-cm layer of the clay loam soil was 72 d for a preplant incorporation and 39 d for a preemergence application.

Transformation products of metolachlor microbial degradation have been identified by thin-layer chromatographic separation techniques and mass spectrometry. Three metabolites are dechlorinated derivatives. Other metabolites are the result of the replacement of the N-alkyl substituent by a hydroxyl group and hydroxylation of the methyl and ethyl side chains of the aromatic rings (Liu *et al.*, 1988). Dechlorination, dehydrogenation, and hydroxylation of metolachlor by anaerobic communities from lake sediments and the soil fungus *Chaetomium globosum* have been reported by McGahen (1982) and McGahen and Tiedje (1978, 1980).

Water

Little information related to the persistence of metolachlor in the aquatic environment is available. As recently as 1987, the U.S. EPA (1988) stated that the available data were insufficient to assess the environmental fate of metolachlor. However, microcosm studies of the aquatic fate of two structurally related herbicides, alachlor and propachlor, demonstrate a rapid breakdown to numerous metabolites over a 33-d period (Yu *et al.*, 1975).

The soil fungus *Chaetomium globosum* was able to degrade 45% of an aerobic liquid suspension of metolachlor in 144 h (McGahen and Tiedje, 1978). Sterile solutions without fungal mycelia showed no loss of metolachlor. Products of the fungal biodegradation were 2-chloro-N-(2'-ethyl-6'-methylphenyl)acetamide and 2-chloro-N-(2'-ethyl-6'-methylphenyl)-N-(2-hydroxy-1-methylethyl)acetamide. McGahen and Tiedje (1980) also studied anaerobic biodegradation of metolachlor in eutrophic lake sediments. Metolachlor was totally degraded by anaerobic microorganisms within 8 weeks; sterilized controls showed no loss of metolachlor.

LeBaron *et al.* (1988) summarized information on the aquatic fate of metolachlor: the aqueous hydrolysis of metolachlor was slow at a variety of pH levels and temperatures; the half-life at 20°C was calculated to be greater than 200 d at pH 1, 5, 7, and 9, assuming first-order degradation kinetics. Similarly, little aqueous photolysis occurs. When metolachlor was exposed to natural sunlight in aqueous suspension, total photolytic decomposition of only 6% took place over a 1-month period (LeBaron *et al.*, 1988). According to LeBaron *et al.* (1988), metolachlor is hydrolyzed under basic conditions to N-(2-ethyl-6-methylphenyl)-2-hydroxy-N-(2-methoxy-1-methylethyl)acetamide. Under acidic conditions, metolachlor first hydrolyzes to 2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-hydroxy-1-methylethyl)acetamide, which is rapidly converted to 4-(2'methyl-6'-ethylphenyl)-3-methylmorpholinone-5.

No good field data are available on the volatilization of metolachlor from water (Chesters *et al.*, 1989).

RATIONALE

Raw Water for Drinking Water Supply

Guideline

The interim maximum acceptable concentration (IMAC) for metolachlor listed in the Guidelines for Canadian Drinking Water Quality is 50 $\mu g \cdot L^{-1}$ (Health and Welfare Canada, 1989). This IMAC is based on a negligible daily intake (NDI) of 0.005 mg·kg-1·d^{-1} established by a 2-year feeding study with rats. Testicular atrophy, increased kidney and liver weight, decreased spleen weight, and an increased incidence of neoplastic cell changes in the liver noted at higher concentrations were used as effect criteria. This IMAC is currently under review by the Federal–Provincial Subcommittee on Drinking Water of the Federal–Provincial Advisory Committee on Environmental and Occupational Health (G. Wood, 1989, Health and Welfare Canada, pers. com.).

Summary of Existing Guidelines

A recommended health advisory for drinking water for the state of Wisconsin of 25 $\mu g \cdot L^{-1}$ was listed in a paper published by Krill and Sonzogni (1986), but the rationale for this concentration was not given. The U.S. EPA (1987a) calculated a lifetime health advisory concentration of 10 $\mu g \cdot L^{-1}$ for metolachlor in drinking water. This value is based on a 1-year study by Tisdel *et al.* (1983), in which male and female rats were given dietary doses of metolachlor equivalent to 1.5, 15, and 150 $mg \cdot kg^{-1} \cdot d^{-1}$. Treatment-related effects were found for glutamic–oxaloacetic transaminase activity, testicular atrophy with degeneration of the tubular epithelium, and an increased incidence of hepatic eosinophilic foci in both sexes. Based on the data, a no-observed-adverse-effect level (NOAEL) of 1.5 $mg \cdot kg^{-1} \cdot d^{-1}$ was identified. This NOAEL was divided by an uncertainty factor of 100 and converted to a drinking water equivalent of 525 $\mu g \cdot L^{-1}$ by multiplying by an average human body weight of 70 kg and dividing by a daily water consumption of 2 L. Twenty percent of this value (the assumed relative source contribution for drinking water) was divided by an additional uncertainty factor of 10 for possible carcinogenicity to arrive at the lifetime health advisory of 10 $\mu g \cdot L^{-1}$.

The World Health Organization has published a guideline of 5 $\mu g \cdot L^{-1}$ for metolachlor in drinking water (WHO, 1987), but the rationale for this guideline was not provided.

Concentrations in Drinking Water

In Ontario, the OMOE (1987a) in 1985 detected metolachlor in 6 of 31 samples from 15 municipal waterworks (at 0.4-5.1 $\mu g \cdot L^{-1}$) but in none of the treated water samples from these sites. Metolachlor was also found in private wells; 52 of 351 wells showed metolachlor contamination, with a maximum concentration of 1800 $\mu g \cdot L^{-1}$. In 1986, metolachlor was detected in 3 of 37 domestic wells (with a maximum concentration of 3.2 $\mu g \cdot L^{-1}$) but in none of the water samples collected from 5 municipal groundwater supply wells (OMOE 1987b). Metolachlor was found in 40 of 417 (10%) samples collected from 25 municipal surface water waterworks, and in 23 of 150 (15%) treated water samples (maximum concentration of 5.97 $\mu g \cdot L^{-1}$). Metolachlor was found in 16% of the drinking water samples collected from 1981 to 1987 at the Dresden waterworks on the Sydenham River in southwestern Ontario (Frank *et al.*, 1990).

Water Treatment

Adsorption onto granular activated carbon (GAC) is a promising method for removal of metolachlor from contaminated drinking water supplies (U.S. EPA, 1987a). Metolachlor was reported to exhibit the following adsorption capacities at 20°C: 0.173, 0.148, and 0.105 mg metolachlor per milligram GAC at concentrations of 79.8, 10.0, and 1.7 $mg \cdot L^{-1}$, respectively (Whittaker, 1980). Removal of metolachlor from wastewater containing an initial average metolachlor concentration of 16.4 $mg \cdot L^{-1}$ was reported to be 99.5% using GAC columns operated at a hydraulic loading of 0.85 $L \cdot s^{-1} \cdot m^{-2}$ and with a 72-min contact time (Holiday and Hardin, 1981).

The OMOE (1987a, 1987b) reported that the conventional water treatment processes consisting of coagulation, flocculation, filtration, and disinfection were ineffective at removing herbicide residues from water. Powdered activated carbon, used for taste and odour control at doses of 4.4–48.1 $mg \cdot L^{-1}$, depending on the month, was able to reduce the levels of metolachlor in treated water when added to the treatment stream in doses of 40–50 $mg \cdot L^{-1}$ or above. Frank *et al.* (1990) reported

that this amount of carbon removed a mean metolachlor concentration of 2 $\mu g \cdot L^{-1}$ in river water to a level below the detection limit (0.02 $\mu g \cdot L^{-1}$) for the compound.

Freshwater Aquatic Life

Accumulation and Elimination in Aquatic Biota

The expected environmental concentration (EEC) is a measure of the potential exposure of aquatic organisms to a contaminant. Using a worst-case scenario, it is derived by multiplying the maximum recommended application rate (in $kg \cdot ha^{-1}$) to a pond with surface area 0.01 ha and volume 50 000 L by 0.2, which assumes that 20% of the applied herbicide reaches the aquatic environment. This calculation results in an EEC with units of $mg \cdot L^{-1}$. In the case of metolachlor, the EEC is 0.9 $mg \cdot L^{-1}$. Studies concerning the toxicity and bioaccumulative potential of metolachlor can then be compared with the EEC to evaluate the risk to aquatic fauna from application of the compound during normal agricultural operations.

Accumulation studies during which fish have been exposed to concentrations of metolachlor above the EEC have been conducted. Static exposures of bluegills (*Lepomis macrochirus*) to approximately 1.2 $mg \cdot L^{-1}$ of ^{14}C-labelled metolachlor for 70 d resulted in a residual level of 18 $mg \cdot kg^{-1}$ (based on the ^{14}C activity of the tissue) in the edible tissues of the fish. A residue level of 486 $mg \cdot kg^{-1}$ was reported for the nonedible tissues. Depuration for 28 d decreased the residue level in the edible tissues to an equivalent of 12 $mg \cdot kg^{-1}$, and in the nonedible tissues to 13 $mg \cdot kg^{-1}$ (Barrows, 197). Whether the ^{14}C activity was the result of the presence of metolachlor or metolachlor metabolites was not determined.

A flow-through exposure of the bluegill (*L. macrochirus*) to 1 $mg \cdot L^{-1}$ ^{14}C-labelled metolachlor resulted in residues of 28 $mg \cdot kg^{-1}$ in edible tissue and 702 $mg \cdot kg^{-1}$ in nonedible tissue. After 28 d depuration, the activity in the edible tissue decreased to an equivalent of 11.7 $mg \cdot kg^{-1}$ metolachlor (Barrows, 1974). Metolachlor concentrations in nonedible tissues were not reported, and the actual nature of the residues was not defined.

Accumulation studies using concentrations below the EEC have demonstrated apparent bioconcentration factors of 6.5–9.0 for edible portions of catfish (species not given) exposed to 0.08 $mg \cdot L^{-1}$ metolachlor for 30 d (Smith, 1977). The viscera of exposed fish had a 10-fold greater accumulation of metolachlor than the meat (i.e., 55.0–92.4 times the water concentration). After 14 d of depuration, residue concentrations in the edible tissue decreased from 0.72 to 0.03 $mg \cdot kg^{-1}$. The decrease in visceral tissue concentration was from 7.39 to 0.18 $mg \cdot kg^{-1}$ (Smith, 1977).

The water flea (*Daphnia magna*) was reported to accumulate metolachlor to 0.6 $mg \cdot kg^{-1}$ after a 24-h exposure to 0.1 $mg \cdot L^{-1}$. Depuration for 8 h reduced this to 0.30 $mg \cdot kg^{-1}$ (Ellgehausen, 1977). The green alga *Scenedesmus acutus* had a metolachlor concentration of 10.4 $mg \cdot kg^{-1}$ after a 1.5-h exposure to 0.1 $mg \cdot L^{-1}$. A 2-h depuration period reduced the accumulation to 2 $mg \cdot kg^{-1}$ (Ellgehausen, 1977; Ellgehausen *et al.*, 1980).

Using ^{14}C ring-labelled metolachlor, Liu *et al.* (1987, 1989) reported a bioconcentration factor of 11 000 for metolachlor by a mixed bacterial community after 10 d in a chemostat. As no metolachlor was detected by chromatographic analysis, however, they concluded that the radioactivity recovered from the cells represented transformation products. An accumulation of the magnitude seen by Liu *et al.* (1989) seems to be the exception to the generally reported low absorption of metolachlor by microbes (Krause *et al.*, 1985; Saxena *et al.*, 1987). Krause *et al.* (1985), for instance, reported that sorption of metolachlor by an actinomycete from liquid media was less than 1%. Saxena *et al.* (1987) reported about 1% sorption of metolachlor from liquid cultures by two bacterial species and 3%–5% sorption by filamentous microorganisms. A differentiation was not made between adsorption or absorption in either report, and the low levels of metolachlor may have been the result of rapid metabolism of the compound (U.S. EPA, 1987b).

Metolachlor is rapidly metabolized by fish (U.S. EPA, 1987b; Chesters *et al.*, 1989), although confirming studies were not cited. In catfish (*Ictalurus melas*) that had repeatedly been exposed for 4 d to a metolachlor concentration of about

0.01 $mg \cdot L^{-1}$, Ellgehausen *et al.* (1980) measured a depuration half-life of 0.60 d.

Toxicity to Aquatic Organisms

Acute Lethal Toxicity

Only two cold-water species are represented in the vertebrate acute toxicity data base for metolachlor: the fathead minnow (*Pimephales promelas*), with four 96-h LC_{50}s, and the rainbow trout (*Salmo gairdneri*), with two LC_{50}s. The range of LC_{50}s for these two species was 2.0–11.0 $mg \cdot L^{-1}$. The remaining test species are the guppy (*Lebistes reticulata*), bluegill (*Lepomis macrochirus*), channel catfish (*Ictalurus punctatus*), and crucian carp (*Carassius carassius*). The range of LC_{50}s for these test species was 4.9–15 $mg \cdot L^{-1}$. Many of the available toxicity data are proprietary, unpublished information available only through U.S. EPA reviews. Thus, the exact procedures and grade or formulation used were often not available. The available acute toxicity data for vertebrates are presented in Table 4.

Invertebrate toxicity data are available for only two species: *D. magna*, and the midge larva (*Chironomus plumosus*). The 48-h EC_{50} and LC_{50} values for *D. magna* were 23.5 and 25.1 $mg \cdot L^{-1}$, respectively (Vilkas, 1976; Mayer and Ellersieck, 1986). A no-observed-effect level (NOEL) of 5.6 $mg \cdot L^{-1}$ for a 48-h exposure was reported by Vilkas (1976) for *D. magna*. The 48-h EC_{50} for *C. plumosus* was 3.8 $mg \cdot L^{-1}$ for technical-grade metolachlor.

There are currently no acceptable data regarding metolachlor toxicity to freshwater algae or aquatic vascular plants. In their study on bioaccumulation, Ellgehausen *et al.* (1980) determined a no-effect level of 0.1 $mg \cdot L^{-1}$ for *Scenedesmus acutus*; however, details regarding the toxicity measurements were not provided.

Chronic Toxicity and Sublethal Reactions

Chronic toxicity data were reported for the fathead minnow (*P. promelas*) for metolachlor exposures greater than 28 d (Dionne, 1978). These data were reviewed by the U.S. EPA (1987b) and found acceptable in terms of quality. In this study, the effects of technical (97.4%) metolachlor on the reproduction of the fathead minnow (*P. promelas*) were studied. The highest concentration below which no effects were observed (the no-observed-effects concentration, or NOEC) was 780 $\mu g \cdot L^{-1}$.

Guideline

The available vertebrate toxicity data consist of 11 96-h LC_{50} values derived from tests with six species of fish, only one of which was a salmonid. Chronic toxicity data consist of data for the fathead minnow (*P. promelas*) derived from exposures of greater than 4 weeks. Invertebrate toxicity data consist of four 48-h EC_{50}s, one 48-h LC_{50}, and one 48-h NOEL.

After a critical review of the acute toxicity data, most of which were unpublished, the U.S. EPA (1987b) reported that only two 96-h toxicity tests with fish contained sufficient quality assurance information for use in the development of an acute toxicity advisory concentration. These 96-h LC_{50}s were 10 $mg \cdot L^{-1}$ for the bluegill (*L. macrochirus*) and 3.9 $mg \cdot L^{-1}$ for the rainbow trout (*Salmo gairdneri*) (Buccafusco, 1978a, 1978b). The maximum acceptable toxicant concentration (MATC) of 0.78–1.6 $mg \cdot L^{-1}$ for the fathead minnow (*P. promelas*) (Dionne, 1978) was also approved. The toxicity data of Mayer and Ellersieck (1986) were not reviewed by the U.S. EPA (1987b); however, the test procedures used by Mayer and Ellersieck (1986) are U.S. EPA-approved test methods.

The CCME guideline development procedure advocates the use of application factors when sufficient toxicity data are not available (CCREM, 1987, Appendix IX). Application factors are unitless numbers applied to an acute toxicity value to ensure the protection of organisms over a chronic exposure period or to a chronic value when sufficient toxicity or environmental fate data are not available for the compound. Only one chronic toxicity study, the reproduction study with fathead minnow (*P. promelas*) (Dionne, 1978), was found for metolachlor. As the information on the chemical fate of metolachlor in the aquatic environment is limited, an application factor of 0.01 was used (CCREM, 1987). Accordingly, an interim guideline for the protection of freshwater aquatic life of 8 $\mu g \cdot L^{-1}$ was derived. Because of the deficiencies in the metolachlor toxicity data base, this guideline is given interim status. The acute and chronic toxicity data for metolachlor for freshwater aquatic life can be compared with this guideline value in Figure 2. Because of the rapid depuration rate of metolachlor

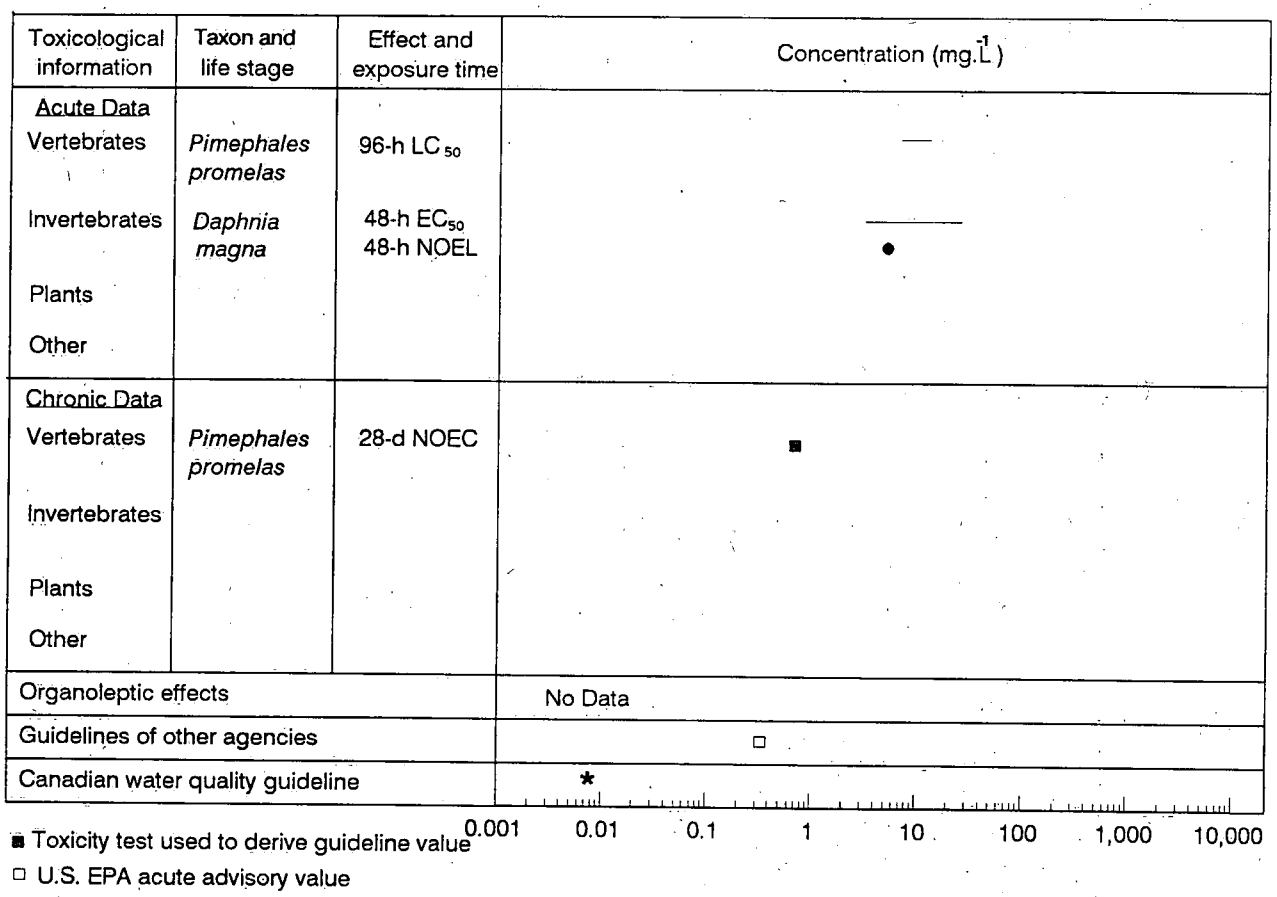

Figure 2. Freshwater aquatic life guideline derivation graph.

Table 4. Summary of Metolachlor Toxicity Data for Aquatic Organisms

Organism	Formulation	Exposure time	Effects*	Comments	Reference
VERTEBRATES					
Fathead minnow (*Pimephales promelas*)	Technical grade 95.40%	96 h	LC_{50} = 8.0 mg·L^{-1} (5.4–12)	Test water pH = 7.4; Hard = 40; 22°C; S; wt = 0.70 g	Mayer and Ellersieck, 1986
	Emulsifiable concentrate 87.00%	96 h	LC_{50} = 8.4 mg·L^{-1} (6.4–11)	Test water pH = 7.4; Hard = 40; 22°C; S; wt = 0.80 g	Mayer and Ellersieck, 1986
	Technical grade 97.4%	Over 4 weeks	MATC between 0.78 and 1.60 mg·L^{-1}	Fish exposed to greater than 1.6 mg·L^{-1} had low survival for 1st- and 2nd-generation fry	Dionne, 1978
		96 h	LC_{50} = 11.0 mg·L^{-1}	S	Dionne, 1978
		96 h	LC_{50} = 9.2 mg·L^{-1}	Flow-through test	Dionne, 1978
Guppy (*Lebistes reticulata*)		96 h	LC_{50} = 8.6 mg·L^{-1}		Sachsse and Ullman, 1974
Bluegill (*Lepomis macrochirus*)		96 h	LC_{50} = 10 mg·L^{-1}		Buccafusco, 1978a
	Technical	96 h	LC_{50} = 15 mg·L^{-1}		WSSA, 1983
Catfish (*Ictalurus punctatus*)		96 h	LC_{50} = 4.9 mg·L^{-1}		Sachsse and Ullman, 1974
Crucian carp (*Carassius carassius*)	Technical	96 h	LC_{50} = 4.9 mg·L^{-1}		Sachsse and Ullman, 1974
Rainbow trout (*Salmo gairdneri*)	Technical	96 h	LC_{50} = 2.0 mg·L^{-1}		WSSA, 1983
		96 h	LC_{50} = 3.9 mg·L^{-1}		Buccafusco, 1978b

Hard = hardness as mg·L^{-1} $CaCO_3$
S = static
*95% confidence limits in parentheses.
wt = weight of fish in grams (reported where available)
NOEL = No-observed-effect level

Table 4. Continued

Organism	Formulation	Exposure time	Effects*	Comments	Reference
INVERTEBRATES					
Cladoceran (*Daphnia magna*) (1st instar)	Technical grade 95.4%	48 h	EC_{50} = 23.5 mg·L^{-1} (18.7–29.5)	Test water pH = 7.2; Hard = 44; 17°C; S	Mayer and Ellersieck, 1986
	Emulsifiable concentrate 87.0%	48 h	EC_{50} = 26.0 mg·L^{-1} (19.4–34.9)	Test water pH = 7.2; Hard = 44; 17°C; S	Mayer and Ellersieck, 1986
Cladoceran (*Daphnia magna*)		48 h	LC_{50} = 25.1 mg·L^{-1} (21.6–29.2) NOEL = 5.6 mg·L^{-1}		Vilkas, 1976
Midge (*Chironomus plumosus*) (3rd instar)	Technical grade 95.4%	48 h	EC_{50} = 3.8 mg·L^{-1} (2.1–10.3)	Test water pH = 6.9; Hard = 40; 22°C; S	Mayer and Ellersieck, 1986
	Emulsifiable concentrate 87.0%	48 h	EC_{50} = 4.4 mg·L^{-1} (3.2–6.1)	Test water pH = 6.9; Hard = 40; 22°C; S	Mayer and Ellersieck, 1986

in fish (U.S. EPA, 1987b; Chesters *et al.*, 1989), this guideline should offer protection from bioconcentration in fish and thus should also protect consumers of fish from ingesting harmful concentrations of metolachlor.

Summary of Existing Guidelines

The U.S. EPA (1987b) divided the lowest acute toxicity value from approved data (3.9 $mg \cdot L^{-1}$ for the rainbow trout) by a safety factor of 11 to calculate an advisory acute value (AAV) of 355 $\mu g \cdot L^{-1}$. They concluded that this value may be a conservative and reasonable concentration to protect aquatic life from acute lethality of metolachlor. A chronic water advisory concentration of 14.2 $\mu g \cdot L^{-1}$ was derived by dividing the AAV by an assumed acute to chronic ratio of 25.

Data Gaps

The aquatic toxicity data base for metolachlor is lacking information on the chronic toxicity of the compound to vertebrates and invertebrates. Information on the adverse effects of metolachlor to phytoplankton and aquatic vascular plants is also lacking. Little information is available on the aquatic fate and persistence of metolachlor; the persistence of the compound in natural waters under field conditions is incompletely known, for instance. No information is available on the volatilization of metolachlor from natural waters, and no field studies were found on the surface transport of metolachlor to water sources.

Agricultural Uses

Livestock Watering

Toxicity to Livestock and Related Biota

Acute Toxicity — A summary of the mammalian toxicity and reproductive effects of metolachlor ingestion is presented in Table 5. Acute toxicity studies indicate that LD_{50}s for metolachlor are in the range 2000–5000 $mg \cdot kg^{-1}$ (body weight) for rats.

Subacute and Chronic Toxicity — White rats given oral doses of Dual® (formulation not given) at 273 $mg \cdot kg^{-1}$ body weight by stomach tube for 15 successive days exhibited ulceration of the buccal mucosa and degradation and necrosis of the visceral epithelium and myocardium. Histopathological examination of lung, liver, heart, and kidney tissues showed widespread congestion and hemorrhage. The organ most severely impacted was the liver, which exhibited centrilobular necrosis (Shihata *et al.*, 1985).

A 180-d feeding study with dogs demonstrated decreased body weight gains in males and females and a failure of the serum alkaline phosphatase enzyme system to decrease with increasing age (Jessup *et al.*, 1979). The NOEL for this study was 100 $mg \cdot kg^{-1}$ (3 $mg \cdot kg^{-1} \cdot d^{-1}$).

Uptake, Metabolism, and Elimination – Although the metabolic pathway for metolachlor is incompletely known (see Table 3), metolachlor appears to be rapidly and completely absorbed from the mammalian gastrointestinal tract and quickly metabolized and excreted. In rats, approximately 70%–90% of single oral doses are excreted as metabolites in the urine and feces within 48 h (Hamböck, 1974a, 1974b, 1974c). Metolachlor was rapidly metabolized in mammals via dechlorination, O-methylation, N-dealkylation, and side chain oxidation; no unaltered metolachlor was detected (Hamböck, 1974a, 1974b).

From excretion studies using rats given oral doses of ^{14}C-labelled metolachlor, a half-life of 28 h was demonstrated. Urine and feces, collected for 48 h after administration of a single oral dose (approximately 31 $mg \cdot kg^{-1}$ body weight), contained 21.5% and 51.4%, respectively, of the dose as metolachlor metabolites. The combined excreta contained 1%, 15%, and 22% of the administered dose as 2-ethyl-6-methylhydroxyacetanilide, 2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-hydroxy-1-methylethyl), and N-(2-ethyl-6-methylphenyl)-N-(hydroxyacetyl)-dl-alanine, respectively. Unaltered metolachlor was not isolated, nor were conjugated forms of metolachlor found (Hamböck, 1974c).

Rats receiving intraperitoneal injections of metolachlor metabolized the herbicide by the hepatic mixed-function oxygenase system to 2,4- and 2,6-disubstituted anilines that were in turn converted to the corresponding nitrosobenzenes (Kimmel *et al.*, 1986). These nitroso compounds have been shown to be highly mutagenic in bacterial assays (Chesters *et al.*, 1989).

An *in vitro* effect of metolachlor on the occurrence of oxidative stress (i.e., decreased concentration of glutathione) in sheep red blood cells was reported at

Table 5. Summary of Mammalian and Avian Health Effects from Metolachlor Ingestion

Animal	Test formulation	Duration	Effects*	Reference
Mouse	NR	18 months (male) 20 months (female)	NOEL = 3000 $mg \cdot kg^{-1}$ (diet)	Industrial Bio-Test Laboratories Inc., 1975
Mouse	NR	2 years	NOEL = 1000 $mg \cdot kg^{-1}$ (diet) (170 $mg \cdot kg^{-1} \cdot d^{-1}$ for males, 224 $mg \cdot kg^{-1} \cdot d^{-1}$ for females) LOEL = 3000 $mg \cdot kg^{-1}$ (diet) (704 $mg \cdot kg^{-1} \cdot d^{-1}$)	Tisdel *et al.*, 1980
Mouse	NR	2 years	NOEL = 1.5 $mg \cdot kg^{-1}$ (body weight) or 30 $mg \cdot kg^{-1}$ (diet); testicular atrophy at 300 $mg \cdot kg^{-1}$ (diet); significant increase in neoplastic liver nodules and proliferative hepatic lesions in females at 3000 $mg \cdot kg^{-1}$ (diet)	Tisdel *et al.*, 1983
Rat	Technical grade	NR	LD_{50} = 2780 $mg \cdot kg^{-1}$ (body weight) (2180-3545)	Bathe, 1973
Rat	Emulsifiable concentrate, 0.72 $kg \cdot L^{-1}$	NR	LD_{50} > 2000, but < 5000 $mg \cdot kg^{-1}$ (body weight)	Affiliated Medical Research Inc., 1974
Rat	NR	2 years	Significant increase in primary neoplasms in females at 3000 $mg \cdot kg^{-1}$ (diet)	Gordon, 1978
Rat	Dual	15 d	Ulceration of buccal mucosa, visceral congestion and hemorrhage, hepatic centrilobular necrosis, 40% mortality at 273 $mg \cdot kg^{-1}$ (body-weight) by gavage	Shihata *et al.*, 1985
Rat	NR (Technical)	10 d (days 6–15 of gestation)	NOEL = 360 $mg \cdot kg^{-1} \cdot d^{-1}$ (body weight) for fetotoxic or developmental effects	Fritz, 1976
Rat	NR	2 generations	NOEL = 300 $mg \cdot kg^{-1}$ (diet) for reproductive effects (14.7 $mg \cdot kg^{-1} \cdot d^{-1}$)	Smith *et al.*, 1981

NR = not reported
NOEL = no-observed-effect level
LOEL = lowest-observed-effect level
* 95% confidence limits in parentheses

Table 5. Continued

Animal	Test formulation	Duration	Effects*	Reference
Rabbit	NR	NR	NOEL = 360 $mg \cdot kg^{-1} \cdot d^{-1}$ (body weight) for fetotoxicity, 36 $mg \cdot kg^{-1} \cdot d^{-1}$ for maternal toxicity	Lightkep *et al.*, 1980
Dog	NR	3 months	$NOEL_a$ = 500 $mg \cdot kg^{-1}$ (body weight) (14–19 $mg \cdot kg^{-1} \cdot d^{-1}$)	Coquet *et al.*, 1974
Dog	NR	6 months	NOEL = 3 $mg \cdot kg^{-1} \cdot d^{-1}$ (body weight) or 100 $mg \cdot kg^{-1}$ (diet)	Jessup *et al.*, 1979
Dog	Technical	7 d	NOEL = 13.7 $mg \cdot kg^{-1}$ (body weight)	Goldenthal *et al.*, 1979
Mallard duck	NR	—	LC_{50} > 2500 $mg \cdot kg^{-1}$	U.S. EPA, 1988
	Technical	8 d	LC_{50} > 10 000 $mg \cdot kg^{-1}$ (diet)	WSSA, 1983
Bobwhite quail	Technical	8 d	LC_{50} > 10 000 $mg \cdot kg^{-1}$ (diet)	WSSA, 1983
Mallard duck and bobwhite quail	NR	112–119 d	Significant reproductive impairment at 10 $mg \cdot kg^{-1}$ (diet)	Chesters *et al.*, 1988

a concentration of 100 $mg \cdot L^{-1}$ (Geiger and Calabrese, 1985). The authors did not speculate as to the actual *in vivo* effect.

Carcinogenicity, Mutagenicity, and Teratogenicity – Evaluations of the genotoxic and mutagenic characteristics of technical-grade metolachlor with and without metabolic activation were negative in the *Salmonella typhimurium* (five strains) assay, the *Saccharomyces cerevisiae* (yeast) assay, and the maize genetic assay (Plewa *et al.*, 1984); details of the evaluation procedures were not given. Commercial-grade metolachlor, however, produced a positive response in one strain of *S. typhimurium* with and without metabolic activation. The only other response produced by the commercial-grade product was in the yeast assay after animal metabolic activation (Plewa *et al.*, 1984). The significance of these positive responses was not discussed by the authors. All other available data indicate no mutagenic potential for metolachlor (U.S. EPA, 1987a).

The U.S. EPA (1987b) tentatively classified metolachlor as a category "C" carcinogen (limited evidence of carcinogenicity in animals). The U.S. EPA also classifies metolachlor as a possible human carcinogen (IRIS, 1989). A 2-year chronic feeding study with rats produced a significantly increased occurrence of primary liver neoplasms in females receiving a dietary metolachlor level of 3000 $mg \cdot kg^{-1}$. Mice fed the same concentration of metolachlor did not demonstrate histological patterns that could be interpreted as carcinogenic. Results of both mouse and rat studies were subsequently confirmed in duplicate studies (U.S. EPA, 1987b; IRIS, 1989).

Metolachlor is not considered to be teratogenic in rats or rabbits or to cause other reproductive effects. Oral doses of 360 $mg \cdot kg^{-1} \cdot d^{-1}$ during gestation did not affect offspring of rats or rabbits, although maternal toxicity was observed at this concentration. Direct effects of oral doses of 1000 $mg \cdot L^{-1}$ metolachlor to rats were not observed during a 2-year reproduction study. The resulting NOEL of 380 $mg \cdot L^{-1}$ for reproductive effects was based on reduced pup weights and decreased food consumption by the females (U.S. EPA, 1987a).

Guideline

No information was found concerning toxicity to livestock consuming metolachlor in their drinking water. Therefore, the derivation of an interim water quality guideline for livestock watering supplies follows the CCREM (1987) procedure of adopting the pesticide guideline for raw drinking water supply as the guideline for livestock watering supplies in the absence of available data.

As the interim guideline for metolachlor in drinking water supplies is 50 $\mu g \cdot L^{-1}$ and is supported by a long-term NOEL derived from rat studies, this value is adopted as an interim guideline for livestock watering supplies.

Summary of Existing Guidelines

No existing guidelines concerning safe concentrations of metolachlor in livestock watering supplies have been found.

Water Supply for Irrigation

Toxicity to Nontarget Plant Species

Laboratory and greenhouse studies have demonstrated that metolachlor adversely affects crop species in concentrations as low as 10^{-7} M (0.028 $mg \cdot L^{-1}$). In these studies, summarized in Table 6, nutrient solutions, moist filter paper, or sand was used as the substrate in which germination and growth of various plant species were examined during exposure to metolachlor. The absence of soil organic matter, specifically the humic matter, may have prevented the reduction of metolachlor activity due to adsorption (Weber *et al.*, 1987). A NOEL of 0.28 $mg \cdot L^{-1}$ for germination of seven crop species was derived by Pillai *et al.* (1979) using moist filter paper as the germination medium.

By contrast, early postemergence spraying of metolachlor on field plots at 1.12 and 4.48 $kg \cdot ha^{-1}$ (3000 and 12 000 $mg \cdot L^{-1}$) did not have a significant effect on the growth of cauliflower (*Brassica oleracea* var. *italica*), cabbage (*B. oleracea* var. *capitata*), or broccoli (*B. oleracea* var. *botrytis*) growing in a loam soil (Sieczka *et al.*, 1986). Other field studies revealed that 2.24 $kg \cdot ha^{-1}$ (9570 $mg \cdot L^{-1}$) had only a slight effect on Chinese cabbage (*Brassica campestris*) growing in a silt loam soil (Grenoble and Ferretti, 1986).

Sandy loam soil plots, which had metolachlor applied at 10 000 mg·L-1·ha^{-1}, were sprinkler irrigated with 1.3 cm of water and planted with grain sorghum (*Sorghum bicolor*). At 10 d postplant, the sorghum seedlings were rated at 98%

Table 6. Summary of Laboratory and Greenhouse Studies of Metolachlor Toxicity to Plants

Metolachlor concentration ($mg \cdot L^{-1}$)	Effect	Reference
2.8	Caused significant increase in loss of ^{32}P from roots of cotton (*Gossypium birsutum*), onion (*Allium cepa*), and cucumber (*Cucumis sativus*) in nutrient solution.	Pillai *et al.*, 1977; Mellis *et al.*, 1982
283 28.3	Inhibited germination of cucumber seeds (*C. sativus*); reduced radicle elongation, fresh weight, and dry weight within 48 h of germination of cucumber seeds; germination in petri dishes with moist paper	Sloan and Camper, 1986
0.0284–28.38	Caused significant inhibition of mevalonic acid incorporation into gibberellic acid precursors in liquid phosphate buffer solution using cell-free extracts of sorghum (*Sorghum bicolor*)	Wilkinson, 1981a
2	Caused 90% decrease of fresh weight of sorghum (*S. bicolor*) grown in sand	Wilkinson, 1981b
0.25	Shoot length decreased above this concentration	Wilkinson, 1981b
0.06	Root length decreased above this concentration	Wilkinson, 1981b
2	Significant adverse effect in shoot growth of peas (*Pisum sativum*) grown in sand	Jordan and Harvey, 1978
28.3	Concentrations above this caused 50% reduction in shoot growth of 4-d-old corn (*Zea mays*) seedlings in nutrient solution	Dixon and Stoller, 1982
283	90% reduction in germination of corn (*Zea mays*), pea (*P. sativum*), sicklepod (*Cassia obtusifolia*), and wheat (*Triticum aestivum*) on moist paper; 100% reduction in germination of oat (*Avena sativa*); 86% reduction in germination of peanut (*Arachis hypogaea*); 89% reduction in germination of lettuce (*Lactuca sativa*) on moist paper	Pillai *et al.*, 1979
0.283	No-observed-effect level for germination of above above species	Pillai *et al.*, 1979
28.4	Caused inhibition of protein synthesis and leucine incorporation into protein in cucumber (*C. sativus*) root tips	Pillai *et al.*, 1979
0.0099	Caused significant inhibition of shoot elongation in yellow nutsedge (*Cyperus esculentus*) sprouts	Cornelius *et al.*, 1985
1-4	Caused inhibition of starch mobilization in the chloroplasts and inhibition of lipid synthesis in sorghum (*S. bicolor*)	Ebert, 1980
0.0028 (in aqueous solution of 0.1% surfactant)	Caused leaf necrosis in soybean (*Glycine max*) at area of application within 96 h	Diner *et al.*, 1977
28.4 (in nutrient solution)	Soybean (*G. max*) roots exposed to metolachlor in nutrient solution for 96 h without apparent harm to plants	Diner *et al.*, 1977

injury due to metolachlor. Additional seeds were replanted in the same soil, and a second 10-d growth period was allowed. At 20 d posttreatment, the second planting seedlings were rated at 70% injury (Banks and Robinson, 1986).

Snap beans and kidney beans (*Phaseolus* spp.) — crops registered for metolachlor application — tolerated 2.8–5.6 $kg \cdot ha^{-1}$ metolachlor regardless of the method of application. These two crop species tolerated 8.4 $kg \cdot ha^{-1}$ metolachlor only when it was preplant incorporated (Higgins and Pruss, 1978). Dual® 720 EC applied to a South African soil at 1.5 $L \cdot ha^{-1}$ (1.08 $kg \cdot ha^{-1}$) did not injure navy beans (*Phaseolus vulgaris*) at 30°C (van Rensburg and van Dyk, 1986). Injury was apparent, however, at 35°C and 40°C. The authors speculated that decreased adsorption at higher temperatures may have occurred, leading to the increased phytotoxicity. Higher dosages of 3–7.5 $L \cdot ha^{-1}$ (2.16–5.4 $kg \cdot ha^{-1}$) also caused injury to the plants (van Rensburg and van Dyk, 1986). Radishes (*Raphanus sativus*) grown in a very organic soil (i.e., muck) were not influenced by metolachlor applications of 1.68 and 3.36 $kg \cdot ha^{-1}$ over 3 successive years (Dusky, 1986).

Guideline

Reports of studies in which metolachlor-contaminated water was used for crop irrigation were not found. However, the phytotoxicity studies summarized in Table 6 demonstrate that significant alterations in plant growth biochemistry may occur at metolachlor concentrations as low as 28 $\mu g \cdot L^{-1}$ in nutrient solutions (Wilkinson, 1981a). Until field studies are conducted using metolachlor-contaminated irrigation water, development of an interim irrigation water quality guideline requires using the laboratory data. An interim guideline of 28 $\mu g \cdot L^{-1}$ is proposed based on the lowest-observed-effects level (LOEL) of 0.0284 $mg \cdot L^{-1}$ for cell-free extracts of sorghum (*Sorghum bicolor*) in phosphate buffer (Wilkinson, 1981a). Because these cells were exposed without added soil material to adsorb or degrade the herbicide, this concentration should be protective of crop species growing under more natural conditions. The data of Diner *et al.* (1977) cannot be used for guideline development, as the 2.8 $\mu g \cdot L^{-1}$ metolachlor solution that produced leaf necrosis in soybeans also contained a surfactant at a concentration of 1000 $mg \cdot L^{-1}$.

Recreational Water Quality and Aesthetics

Organoleptic Effects

Information was not found in the literature related to the ability of metolachlor to impart a taste or odour to water. In addition, information related to the tainting of fish flesh by accumulated metolachlor was not found.

Guideline

At present, there is no evidence to indicate that this water use would be adversely affected by metolachlor residues when this herbicide is used according to label instructions. In addition, water containing metolachlor residues at concentrations that could potentially affect recreational water uses would already be severely impaired for other water uses (i.e., water for the protection of aquatic life). Thus, a water quality guideline has not been determined for recreation and aesthetics.

Industrial Water Supplies

Guideline

There is no indication that metolachlor poses or has the potential to pose a threat to the quality of water used for industry when used according to label instructions. Although of potential concern if found in water supplies, a water quality guideline for metolachlor in industrial water supplies has not been determined.

SUMMARY

After an evaluation of the published information on the pesticide metolachlor, water quality guidelines were derived (Table 7). The background information on metolachlor in terms of uses and production, occurrence in the aquatic environment, and persistence and degradation was reviewed. The rationale employed for the development of the recommended guidelines was summarized.

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Table 7. Recommended water quality guidelines for Metolachlor

Uses	Guideline
Raw water for drinking water supply	50 $\mu g \cdot L^{-1}$ (IMAC)*
Freshwater aquatic life	8 $\mu g \cdot L^{-1}$
Agricultural uses	
Livestock watering	50 $\mu g \cdot L^{-1}$ (interim guideline)
Irrigation	28 $\mu g \cdot L^{-1}$ (interim guideline)
Recreational water quality and aesthetics	No recommended guideline
Industrial water supplies	No recommended guideline

*Existing drinking water guideline, (Health and Welfare Canada, 1989).

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Appendix A

Occurrence of Metolachlor in Surface Water and Groundwater

Table A-1. Occurrence of Metolachlor in Surface Water and Groundwater

Location	Water sample	Concentration ($\mu g \cdot L^{-1}$)	Remarks	Reference
North-central U.S	Rainfall, Apr. through Aug. 1985	24 (maximum)	Concentration considered high relative to other chlorinated pesticides	Baker, 1986; Richards *et al.*, 1987
Rhine River, Germany	River water, Sept. 1985 to Jan. 1986	1.09 (mean) 1.01 (median) 3.18 (maximum) 0.14 (minimum)	34 samples collected, number of samples with contamination not reported	Oehmichen and Haberer, 1986
Southern Ontario	Groundwater (one well)	112 (25 Nov. 1984) (4 Apr. 1985) 7.8 (9 July 1985) 1.1 (13 Aug. 1985) 29 (22 Aug. 1985)	Well contaminated by spills, date of contamination not reported	Frank *et al.*, 1987a
Southern Ontario	Groundwater (one well)	3.4 (15 June 1982)	25 d after back-siphoning into well during filling of herbicide tanks	Frank *et al.*, 1987b
Central Pennsylvania	Groundwater (4 wells out of 82 monitored)	0.1–0.5	"Typical" concentration range	Cohen *et al.*, 1986
Northern Iowa	Groundwater (two springs from limestone aquifer)	0.1–0.5	"Typical" concentration range	Cohen *et al.*, 1986
Wisconsin	Groundwater	55	Maximum value in 126 samples; 17 samples exceeded 25 $\mu g \cdot L^{-1}$	Holden, 1986
United States	Groundwater	0.1–0.4	"Typical" concentration range	Younos and Weigmann, 1988
Southern Ontario	Groundwater	1800 (maximum)	Detected in 106 of 491 private wells; DL = 1 $\mu g \cdot L$-1	OMOE, 1986
Southern Ontario	Treated drinking water	0.20 (maximum)	Samples collected in spring and fall, 1985; detected in 4 of 45 samples; DL = 0.1 $\mu g \cdot L^{-1}$	Agriculture Canada, 1985
Southern Ontario	Groundwater	8.0 (maximum)	Samples collected in spring and fall, 1985; detected in 7 of 44 samples; DL = 0.1 $\mu g \cdot L^{-1}$	Agriculture Canada, 1985
Northeast Kansas	Surface water	1.23 (mean)	Detected in 4 of 7 samples; DL not reported	Arruda *et al.*, 1988
Southern Ontario	Surface water (Grand River)	0.9 ± 0.6 (mean ± SD)	Detected in 4 of 105 samples during period 1981–85; DL < 0.02 $\mu g \cdot L^{-1}$	Frank and Logan, 1988
	Surface water (Saugeen River)	0.7 ± 0.2 (Mean ± SD)	Detected in 2 of 144 samples, during period 1981–85; DL < 0.02 $\mu g \cdot L^{-1}$	
	Surface water (Thames River)	3.6 ± 2.9 (mean ± SD)	Detected in 15 of 205 samples during period 1981–85; DL < 0.02 $\mu g \cdot L^{-1}$	

SD = standard deviation
DL = detection limit

Table A-1. Continued

Location	Water sample	Concentration ($\mu g \cdot L^{-1}$)	Remarks	Reference
Iowa (University of Iowa)	Untreated drinking water	1.8 (maximum)	Detected in 17 of 45 samples; DL = 0.02 $\mu g \cdot L^{-1}$	Wnuk *et al.*, 1987
	Treated drinking water	0.87 (maximum)	Detected in 11 of 48 samples; DL = 0.02 $\mu g \cdot L^{-1}$	
Iowa (Davenport)	Untreated drinking water	0.55 (maximum)	Detected in 1 of 44 samples; DL = 0.02 $\mu g \cdot L^{-1}$	Wnuk *et al.*, 1987
	Treated drinking water	0.23 (maximum)	Detected in 1 of 46 samples; DL = 0.01 $\mu g \cdot L^{-1}$	
Iowa (Clarinda)	Untreated drinking water	0.68 (maximum)	Detected in 11 of 41 samples; DL = 0.02 $\mu g \cdot L^{-1}$	Wnuk *et al.*, 1987
	Treated drinking water	0.82 (maximum)	Detected in 12 of 46 samples; DL = 0.02 $\mu g \cdot L^{-1}$	
Iowa	Treated drinking water (33 municipal water supplies)	2.9 (mean) 0.1–21.0 (range)	Detected in 21 of 33 samples; DL = 0.01 $\mu g \cdot L^{-1}$	Wnuk *et al.*, 1987
	Untreated drinking water	2.8 (mean) 0.1–10.0 (range)	Detected in 11 of 15 samples; DL = 0.01 $\mu g \cdot L^{-1}$	

Appendix B

Summary of Metolachlor Persistence Studies in Soil

Table B-1. Summary of Metolachlor Persistence Studies in Soil

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai)	Soil depth (cm)	Residues (days post-treatment)	Results and comments	Reference
LABORATORY					
Arkansas/Silt loam (1.1% organic matter; pH = 5.2; 9.1% clay; 12% moisture content)	10 $mg \cdot kg^{-1}$ (NR)	10–20 (730 d)	Not detected	Detection limits NR	Bouchard *et al.*, 1982
Arkansas/Silt loam (0.7% organic matter; pH = 5.5; 13.8% clay; 14% moisture content)	6.7 $mg \cdot kg^{-1}$ (NR)	40–500	0.8 $mg \cdot kg^{-1}$ (730 d)		Bouchard *et al.*, 1982
Arkansas/Silt loam (0.9% organic matter; pH = 6.8; 15.8% clay; 12% moisture content)	10 $mg \cdot kg^{-1}$ (NR)	10–20	Not detected (730 d)	Detection limits NR	Bouchard *et al.*, 1982
Arkansas/Silt loam (0.5% organic matter; pH = 7.1; 14.6% clay; 12% moisture content)	6.7 $mg \cdot kg^{-1}$ (NR)	40–50	Not detected (730 d)	Detection limits NR	Bouchard *et al.*, 1982
England/Sandy loam (0.65% organic carbon; pH = 6.4; 70% sand 19% clay)	4.0 $mg \cdot kg^{-1}$ (NR, commercial formulation)	NR	$t_{½}$ = 80.6 d	Temp. = 25°C Moisture = 6% w/w	Walker and Brown, 1985
			$t_{½}$ = 41.8 d	Temp. = 25°C Moisture = 9% w/w	
			$t_{½}$ = 23.9 d	Temp. = 25°C Moisture = 12% w/w	
			$t_{½}$ = 20.9 d	Temp. = 25°C Moisture = 15% w/w	
			$t_{½}$ = 47.4 d	Temp. = 15°C Moisture = 12% w/w	
			$t_{½}$ = 107.8 d	Temp. = 5°C Moisture = 12% w/w	
FIELD					
Colorado/Clay loam (2.5% organic matter; pH = 8.0; 28% clay)	2.2 $kg \cdot ha^{-1}$ (NR)	0–10	1.1 $kg \cdot ha^{-1}$ (17 d)	Half-life of 17 d reported	Zimdahl and Clark, 1982
Colorado/Sandy loam (1.1% organic matter; pH = 7.8; 14% clay)	2.2 $kg \cdot ha^{-1}$ (NR)	0–10	1.1 $kg \cdot ha^{-1}$ (23 d)	Half-life of 23 d reported	
England/Sandy loam (2% organic matter; pH = 6.5; 18% clay, 70% sand)	(NR)	1–10	—	Actual residues NR; figures approximate, interpolated from graph: 75% after 20 d 60% after 35 d 50% after 50 d 42% after 65 d 30% after 90 d	Walker *et al.* 1983

NR = not reported

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai)	Soil depth (cm)	Residues (days post-treatment)	Results and comments	Reference
Arkansas/Silt loam (0.65% organic carbon; pH = 5.2; 9.1% clay)	0.80 $mg \cdot kg^{-1}$ (NR)	0–2.5	0.47 $mg \cdot kg^{-1}$ (7 d) 0.65 $mg \cdot kg^{-1}$ (18 d) 0.44 $mg \cdot kg^{-1}$ (59 d)	Commercial formulation (NR) of metolachlor applied at 300 $L \cdot ha^{-1}$ to provide 0.80 $mg \cdot kg^{-1}$ when incorporated to a depth of 7.5 cm; reported field half-lives: 70 d at 0–2.5 cm 52 d at 2.5–5.0 cm 60 d at 5.0–7.5 cm	Braverman *et al.*, 1986
		2.5–5.0	0.58 $mg \cdot kg^{-1}$ (7 d) 0.53 $mg \cdot kg^{-1}$ (18 d) 0.33 $mg \cdot kg^{-1}$ (59 d)		
		5.0–7.5	0.1 $mg \cdot kg^{-1}$ (7 d) 0.52 $mg \cdot kg^{-1}$ (18 d) 0.36 $mg \cdot kg^{-1}$ (59 d)		
		7.5–10.0	0.61 $mg \cdot kg^{-1}$ (7 d) 0.59 $mg \cdot kg^{-1}$ (18 d) 0.32 $mg \cdot kg^{-1}$ (59 d)		
		10.0–12.5	0.44 $mg \cdot kg^{-1}$ (7 d) 0.52 $mg \cdot kg^{-1}$ (18 d) 0.36 $mg \cdot kg^{-1}$		
England/Sandy loam (0.65% organic matter; pH = 6.4; 70% sand, 19% clay)	2.5 $kg \cdot ha^{-1}$ (100% ai)	0–10	2.0 $kg \cdot ha^{-1}$ (20 d) 1.25 $kg \cdot ha^{-1}$ (40 d) 1.0 $kg \cdot ha^{-1}$ (70 d) 0.75 $kg \cdot ha^{-1}$ (90 d)	Field experiment initiated on 24 April 1982, half-lives and residues remaining interpolated from graphs	Walker and Brown, 1985
			1.23 $kg \cdot ha^{-1}$ (20 d) 1.25 $kg \cdot ha^{-1}$ (40 d) 0.88 $kg \cdot ha^{-1}$ (60 d) 0.63 $kg \cdot ha^{-1}$ (90 d)	Field experiment initiated on 8 June 1982	