Dichlofop-methyl (C₁₆H₁₄Cl₂O₄) is a herbicide used to control annual grass weeds in cereals and broad-leaf crops.

The fate and persistence of dichlofop-methyl and dichlofop in soil may be influenced by major processes such as chemical and biological degradation. In addition, losses in runoff occur because of its ability to adsorb strongly to soil particles. Up to 15% of dichlofop-methyl is hydrolyzed at the time of application and incorporated into the soil, and as much as 85% is hydrolyzed within 24 h of application. Afterwards, the rate of dichlofop-methyl hydrolysis declines (Smith 1977; Martens 1978). Under laboratory conditions, a half-life value for dichlofop-methyl residues of <3 d was reported (Wink and Luley 1988). Degradation of the hydrolysis product, dichlofop, occurs primarily by biological decomposition (Smith 1979b). The half-life of dichlofop ranged from 6 to 38 d in soil under aerobic conditions. Under anaerobic conditions, the half-life was 150 d or longer (Martens 1978). With time, the rate of dichlofop biodegradation levels off, and the residual concentrations are apparently bound to soil organic matter (Martens 1978; Smith 1979b).

The results of several laboratory experiments demonstrate that dichlofop-methyl persistence decreases with higher incubation temperature and increasing soil pH and moisture levels (Wu and Santelmann 1976; Smith 1977; Gaynor 1984; Wink and Luley 1988). Decreased dichlofop-methyl persistence under field conditions is likely related to the proximity of rainfall events to the time of herbicide application (Gaynor 1984).

The low water solubility of dichlofop-methyl (0.8 mg L⁻¹; 20°C), low mobility in soils (log Kₐ = 2.77 mL·g⁻¹), and high log organic carbon-water partition coefficient (log Kₒc = 4.2 mL·g⁻¹) reflect a high adsorption potential. Bound residues, primarily dichlofop-methyl and/or dichlofop, ranging from 37 to 70% of the initially applied dichlofop-methyl, were reported for laboratory and field studies at sampling times up to 5 months after treatment. Typically, the amount of residue in the unextractable form increases with time following dichlofop-methyl application (Smith 1979a, 1979b; Gaynor 1984; Karanth et al. 1984). Smith (1979a) suggested that neither clay or organic material content was associated with the amount of residue present in the bound form, but pH likely had an effect since it determines the chemical state of the resultant acid and subsequent adsorption potential. Unextractable residues of dichlofop are typically bound to soil organic matter, particularly the fulvic acid and humic acid fractions. In addition to soil adsorption, it is also possible that dichlofop-methyl and/or dichlofop become incorporated into the soil microbial biomass (Karanth et al. 1984).

Dichlofop-methyl is considered to be relatively nonvolatile under field conditions because of its low to intermediate vapour pressure (Grover 1983). It was concluded that post-application losses of dichlofop-methyl by volatilization from the crop canopy or from the soil surface was minimal or nonexistent (Smith et al. 1986). Dichlofop-methyl is reported to have a low resistance to decomposition by ultraviolet light (WSSA 1989).

Grover (1983) classified dichlofop-methyl as a herbicide exhibiting little or no leaching potential because of its low water solubility and strong adsorption to organic carbon and soil. Lawrence et al. (1991), using a mesoscale model aquifer system to study the transport and degradation of dichlofop-methyl, found that dichlofop was transported to the 8-cm depth sampler immediately following application. Detection in lower samplers, 22 and 39 cm, was delayed by approximately 12 d. Dichlofop detected at the 54-cm sampler only 4 d after application likely occurred through preferential flow paths. It was advanced that in the absence of these proposed preferential flow paths, the herbicide would not have migrated beyond the 39-cm sampler.

For more information on the use, environmental concentrations, and chemical properties of dichlofop-methyl, see the fact sheet on dichlofop-methyl in Chapter 4 of Canadian Environmental Quality Guidelines.

<table>
<thead>
<tr>
<th>Use</th>
<th>Guideline value (µg·L⁻¹)</th>
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</thead>
<tbody>
<tr>
<td>Irrigation water</td>
<td>0.18</td>
</tr>
<tr>
<td>Livestock water</td>
<td>9</td>
</tr>
</tbody>
</table>

* Interim guideline.
Water Quality Guideline Derivation

The Canadian water quality guidelines for diclofop-methyl for the protection of agricultural water uses were developed on the CCME protocol (CCME 1993b).

Irrigation Water

Field cultivation, environmental chamber, greenhouse, and laboratory petri dish toxicity studies indicate lethal and sublethal effects to seedlings of nontarget plants at application rates and water concentrations as low as 0.036 kg ha⁻¹ and 0.34 µg·L⁻¹ diclofop-methyl, respectively (Shimabukuro et al. 1978; Hoppe 1985). The phytotoxic action of diclofop is reported to be influential in determining the extent of shoot growth inhibition (Hoerauf and Shimabukuro 1979). A reduction in the mitotic index was observed in adventitious root tips of wheat at a diclofop-methyl concentration of 510 µg·L⁻¹. It was suggested that the cell cycle was arrested at a stage preceding mitosis (Morrison et al. 1981). In addition, necrosis of the meristematic and elongation zones of the root tips of germinating corn seedlings was observed at diclofop-methyl concentrations >340 µg·L⁻¹ (Hoppe 1980).

The inhibitory effect of diclofop-methyl on fatty acid biosynthesis at a concentration of 1700 µg·L⁻¹ was documented in studies that followed the incorporation of ¹⁴C-labelled precursors of lipid formation in wheat leaves and in the radicals, leaves, and isolated chloroplasts of corn (Hoppe 1981, 1985; Hoppe and Zacher 1982, 1985). A NOEL of 170.6 µg·L⁻¹ was derived from data on sterol biosynthesis in corn (Hoppe 1981, 1985; Hoppe and Zacher 1982, 1985). A NOEL of 102.36 µg·L⁻¹ was derived based on root growth inhibition (Hoerauf and Shimabukuro 1979). A reduction in the number of nitrogen-fixing root nodules at 225%), increased liver cytochrome P-450 (179%), and increased plasma nitrophenyl acetate esterase (NPAE) (Williamson 1984; Worthing and Walker 1987). Acute oral LD₅₀s for rats range from 563 to 693 mg·kg⁻¹ (Williamson 1984; Worthing and Walker 1987). Acute oral LD₅₀s for birds range from 4400 mg·kg⁻¹ for bobwhite quail (WSSA 1989) to >10 000 mg·kg⁻¹ for Japanese quail (Worthing and Walker 1987). The NOELs of 20 mg·kg⁻¹ (feed) for a 2-year study in rats and 8 mg·kg⁻¹ (feed) for a 15-month study with dogs were reported by Worthing and Walker (1987). A three-generation reproductive study using rats established a NOEL of 30 mg·kg⁻¹ (assumed to be in feed) (WSSA 1989). No details on experimental design or methods were given for the above acute and chronic toxicity studies.

Trapped wild wood mice (Apodemus sylvaticus) and bank voles (Clethrionomys glareolus), fed diclofop-methyl-treated wheat, demonstrated biochemical and histological effects related to the dietary dose. Wood mice fed dietary concentrations of 200 mg·kg⁻¹ for 1-, 2-, and 4-week periods showed increased relative liver weights (173–225%), increased liver cytochrome P-450 (179%), and increased plasma nitrophenyl acetate esterase (NPAE) (135–258%). Higher dietary concentrations of 500 and 1000 mg·kg⁻¹ produced the same effects, as well as increases in hepatocyte size and cell necrosis and loss of cytoplasmic protein in individual hepatocytes. Although the dietary level of 20 mg·kg⁻¹ can be regarded as a NOEL, slight increases in the activity of the enzyme
glutamate oxaloacetate transaminase (GOT) were observed at this concentration (Westlake et al. 1988).

In the voles only increased liver weight (219–265%) and decreased liver NPAE (21–35%) were observed at 200 and 1000 mg·kg⁻¹ over a 2-week period. Histological effects such as increased hypertrophy, inflammation, and necrosis, however, were reported to be more severe for vole livers at 200 mg·kg⁻¹ than for mice livers at the same concentration. The inflammatory response in vole livers was reported to be greater at 200 mg·kg⁻¹ than at 1000 mg·kg⁻¹, while at the higher dose, regenerative replacement of necrotic hepatocytes, greater lipid degeneration, and a decrease in cytoplasmic protein were observed (Westlake et al. 1988). In the same study, Japanese quail were fed diets containing 1000 mg·kg⁻¹ for 2 weeks and 20 mg·kg⁻¹ for 8 weeks. Hepatic cytochrome P-450 was reported to decrease significantly, although the figures were not reported. The authors concluded that diclofop-methyl, when applied at the maximum recommended field rate, does not pose a hazard to small mammals and avian species.

Available data were not sufficient to derive a guideline for livestock water. The existing guideline for drinking water supplies of 9 µg·L⁻¹ (Health Canada 1996), therefore, is recommended as the interim water quality guideline for the protection of livestock water (CCME 1993a).

References


Reference listing:


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