Canadian Water Quality Guidelines for the Protection of Agricultural Water Uses

DICLOFOP-METHYL

Diclofop-methyl ($C_{16}H_{14}Cl_2O_4$) is a herbicide used to control annual grass weeds in cereals and broad-leaf crops.

The fate and persistence of diclofop-methyl and diclofop 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 diclofop-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 diclofop-methyl hydrolysis declines (Smith 1977; Martens 1978). Under laboratory conditions, a half-life value for diclofop-methyl residues of <3 d was reported (Wink and Luley 1988). Degradation of the hydrolysis product, diclofop, occurs primarily by biological decomposition (Smith 1979b). The half-life of diclofop 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 diclofop 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 diclofop-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 diclofop-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 diclofop-methyl ($0.8\ \mathrm{mg \cdot L^{-1}}$; 20°C), low mobility in soils ($\log K_d = 2.77\ \mathrm{mL \cdot g^{-1}}$), and high log organic carbon–water partition coefficient ($\log K_{oc} = 4.2\ \mathrm{mL \cdot g^{-1}}$) reflect a high adsorption potential. Bound residues, primarily diclofop-methyl and/or diclofop, ranging from 37 to 70% of the initially applied diclofop-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 diclofop-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 diclofop 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 diclofop-methyl and/or diclofop become incorporated into the soil microbial biomass (Karanth et al. 1984).

Diclofop-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 diclofop-methyl by volatilization from the crop canopy or from the soil surface was minimal or nonexistent (Smith et al. 1986). Diclofop-methyl is reported to have a low resistance to decomposition by ultraviolet light (WSSA 1989).

Grover (1983) classified diclofop-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 diclofop-methyl, found that diclofop 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. Diclofop 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 diclofop-methyl, see the fact sheet on diclofop-methyl in Chapter 4 of *Canadian Environmental Quality Guidelines*.

Table 1. Water quality guidelines for diclofop-methyl for the protection of agricultural water uses (CCME 1993a).

Use	Guideline value ($\mu g \cdot L^{-1}$)
Irrigation water	0.18
Livestock water	9*

*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 responsible for the inhibition of cell division and elongation at plant meristems (Tomlin 1994). The proximity of diclofop-methyl treatment to the apical and meristematic sites in the leaves of susceptible oat plants is 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 ^{14}C-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 and Zacher 1982), whereas a NOEL of 102.36 µg·L^{1} was derived based on root growth in corn (Hoppe 1980). Hoppe (1985) reported a LOEAR for decreased mean shoot length in corn *(Zea mays)* of 0.036 kg·ha^{-1}. In a greenhouse study, a LOEAR of 1.12 kg·ha^{-1} was observed in the legume red clover *(Trifolium pratense)*. The effects were a 6% decrease in shoot dry weight, a 6% decrease in shoot height, and a 3% decrease in the number of nitrogen-fixing root nodules at 40 d post-treatment (Peters and Zbiba 1979).

The acceptable application rate (AAR) (kg·ha^{-1} a.i.) for tame hay and cereals and other crop species is calculated by dividing the geometric mean of the LOEAR and the NOEAR by an uncertainty factor of 10. The AARs are then divided by the approximate Canadian annual irrigation rate of 10^{7} L·ha^{-1} per year to calculate the SMATC. The most sensitive species of the tame hay and cereals group was corn, with a NOEAR and a LOEAR of 0 and 0.036 kg·ha^{-1} a.i. per year, respectively, resulting in a SMATC of 0.18 µg·L^{-1} (Hoppe 1985). In the “other crop species” group, the most sensitive species was red clover, with a NOEAR and a LOEAR of 0 and 1.12 kg·ha^{-1} a.i. per year, respectively, resulting in a SMATC of 5.6 µg·L^{-1} (Peters Zbiba 1979). The SMATCs can be regarded as guidelines for each crop group. Overall the Canadian water quality guideline for diclofop-methyl in irrigation water for all crops is 0.18 µg·L^{-1} (CCME 1993a).

Livestock Water

Acute oral LD$_{50}$s for rats range from 563 to 693 mg·kg^{-1} (Williamson 1984; Worthing and Walker 1987). Acute oral LD$_{50}$s 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

CCME (Canadian Council of Ministers of the Environment) 1993a. Appendix XII—Canadian water quality guidelines: Updates (April 1993), bromoxynil, dicamba, and diclofop-methyl. In: Canadian water quality guidelines, Canadian Council of Resource and Environment Ministers. 1987. Prepared by the Task Force on Water Quality Guidelines.

———. 1993b. Appendix XV—Protocols for deriving water quality guidelines for the protection of agricultural water uses (October 1993). In: Canadian water quality guidelines, Canadian Council of Resource and Environment Ministers. 1987. Prepared by the Task Force on Water Quality Guidelines. [Updated and reprinted with minor revisions and editorial changes in Canadian environmental quality gudielines, Chapter 5, Canadian Council of Ministers of the Environment, 1999, Winnipeg.]

Gaynor, J.D. 1984. Diclofop-methyl persistence in southwestern Ontario soils and effect of pH on hydrolysis and persistence. Can. J. Soil Sci. 64:283–292.

Grover, R. 1983. Transport of wild oat herbicides in the environment. In: Wild Oat Symposium Proceedings, A.E. Smith, ed.

Health Canada, 1996. Guidelines for Canadian drinking water quality. 6th ed. Prepared by the Federal–Provincial Subcommittee on Drinking Water of the Federal–Provincial Committee on Environmental and Occupational Health.

Hoerauf, R.A., and R.H. Shimabukuro. 1979. The response of resistant and susceptible plants to diclofop-methyl. Weed Res. 19(5):293–300.

Hoppe, H.H. 1980. Changes in membrane permeability, carbohydrate content, lipid content, and lipid composition in root tips from *Zea mays* after treatment with diclofop-methyl. Z. Pflanzenphysiol. 100s:415–426.

———. 1981. Effect of diclofop-methyl on protein nucleic acid and lipid biosynthesis in tips of radicles from *Zea mays*. Z. Pflanzenphysiol. 1025:189–197.

———. 1985. Differential effect of diclofop-methyl on fatty acid biosynthesis in leaves of sensitive and tolerant plant species. Pestic. Biochem. Physiol. 23:297–308.

Hoppe, H.H., and H. Zacher. 1982. Inhibition of fatty acid biosynthesis in tips of radicles from *Zea mays*. Z. Pflanzenphysiol. 1065:287–298.

———. 1985. Inhibition of fatty acid biosynthesis in isolated bean and maize chloroplasts by herbicidal phenoxy-phenoxypropionic acid derivatives and structurally related compounds. Pestic. Biochem. Physiol. 24:298–305.

Karanth, N.G.K., J.P.E. Anderson, and K.H. Domsch. 1984. Degradation of the herbicide diclofop-methyl in soil and influence of pesticide mixtures on its persistence. J. Biosci. 6(6): 829–837.

Lawrence, J.R., M.J. Hendry, R.D. Robarts, and D.E. Caldwell. 1991. Degradation and mobility of diclofop-methyl in a model groundwater system. S7N 0W0. Final report to Pestfund for fiscal year 1990/1991. National Hydrology Research Institute and University of Saskatchewan, Department of Applied Microbiology and Food Science, Saskatoon, SK.

Martens, R. 1978. Degradation of the herbicide (^{14}C)-diclofop-methyl in soil under different conditions. Pestic. Sci. 9: 127–134.

Morrison, I.N., M.G. Owino, and E.H. Stobbe. 1981. Effects of diclofop on growth, mitotic index, and structure of wheat *(Triticum aestivum)* and wild oat *(Avena fatua)* adventitious roots. Weed Sci. 29: 426–432.

Peters, E.J., and M.B. Zbiba. 1979. Effects of herbicides on nitrogen fixation of alfalfa *(Medicago sativa)* and red clover *(Trifolium pratense)*. Weed Sci. 27(1): 18–21.

Shimabukuro, M.A., R.H. Shimabukuro, W.S. Nord, and R.A. Hoerauf. 1978. Physiological effects of methyl 2-[4(2,4-dichloro-phenoxy) phenoxy] propanoate on oat, wild oat, and wheat. Pestic. Biochem. Physiol. 8: 199–207.

Smith, A.E. 1977. Degradation of the herbicide diclofop-methyl in prairie soils. J. Agric. Food Chem. 25: 893–898.

———. 1979a. Extraction of free and bound carboxylic acid residues from field soils treated with the herbicides benzoylprop-ethyl, diclofop-methyl, and flamprop-methyl. J. Agric. Food Chem. 27(2): 428–432.

———. 1979b. Transformation of (^{14}C)-diclofop-methyl in small field plots. J. Agric. Food Chem. 27(6): 1145–1148.

Smith, A.E., R. Grover, A.J. Cessna, S.R. Shewchuk, and J.H. Hunter. 1986. Fate of diclofop-methyl after application to a wheat field. J. Environ. Qual. 15(3): 234–238.

Tomlin, C. (ed.). 1994. The pesticide manual: A world compendium. 10th ed. (Incorporating the Agrochemicals handbook.) British Crop Protection Council and the Royal Society of Chemistry, Thornton Heath, UK.

Westlake, G.E., K.A. Tarrant, and A.R. Hardy. 1988. Biochemical and histological effects of diclofop-methyl in mice and voles under laboratory conditions. Bull. Environ. Contam. Toxicol. 40(1): 153–158.

Williamson, D.A. 1984. A preliminary investigation into the presence of agricultural pesticides in the LaSalle and Assiniboine Rivers, Manitoba, Canada. Water Standards and Studies Report #84-5. Manitoba Department of Environment and Worksplace Safety and Health, Environmental Management Services Branch, Winnipeg.

Wink, O., and U. Luley. 1988. Enantioselective transformation of the herbicides diclofop-methyl and fenoxaprop-ethyl in soil. Pestic. Sci. 22(1): 31–40.

Worthing, C.R., and S.B. Walker (eds.). 1987. The pesticide manual: A world compendium. 8th ed. British Crop Protection Council, Thornton Heath, UK.

WSSA (Weed Science Society of America). 1989. Herbicide handbook. 6th ed. WSSA, Champaign, IL.

Wu, C-H., and P.W. Santelmann. 1976. Phytotoxicity and soil activity of Hoe 23408. Weed Sci. 24(6):601–604.

Reference listing:

Canadian Council of Ministers of the Environment. 1999. Canadian water quality guidelines for the protection of agricultural water uses: Diclofop-methyl. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

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