Canadian Water Quality Guidelines for the Protection of Aquatic Life

CARBOFURAN

Carbofuran ($C_{12}H_{15}NO_3$) is a pesticide with a CAS name and number of 2,3-dihydro-2,2,-dimethyl-7-benzofuranyl methylcarbamate and 1564-66-2, respectively. Carbofuran is a systemic insecticide and nematicide used on vegetables, ornamentals, corn, alfalfa, sorghum, potatoes, sunflowers, and other crops. Tradenames include Furadan, Curaterr, Carbodan, Carbosip, and Chinufur (Tomlin 1994). Carbofuran is a cholinesterase inhibitor with predominantly contact and stomach action. It was among the top ten insecticides sold in Canada in 1985 (Environment Canada/Agriculture Canada 1987). In 1983, 1984, and 1985, the amounts of carbofuran imported into Canada were 997 t, 1736 t, and 1671 t, respectively (Statistics Canada 1986).

Carbofuran may enter the aquatic environment from direct spraying or broadcast of granular formulations, drift deposition of sprayable formulations, and runoff from treated fields. Reported concentrations of carbofuran in Canadian freshwater sources range from 0.03 to 158.5 $mg \cdot L^{-1}$ (Bailey 1985; Krawchuk and Webster 1987).

The persistence of carbofuran is primarily controlled by chemical degradation (Sharom et al. 1980). Hydrolysis may be the most important chemical reaction with half-lives ranging from approximately 0.2 d (pH 9.5) to 1700 d (pH 5.2). This process is base catalyzed and directly influenced by pH. Temperature also has a major influence with a reported 35% increase in hydrolysis rate for each one degree centigrade temperature increase at ambient temperature. Carbofuran degradation in natural ponds (pH about 8.5) showed a maximum carbofuran persistence from 10 to 21 h (Erickson et al. 1977).

Significant photodecomposition of carbofuran within 96 h has been observed in laboratory studies. Volatilization of carbofuran from water in the laboratory was found to be insignificant (Deuel et al. 1979). Koeppe and Lichtenstein (1982) reported that approximately 75% of the radiocarbon found in an aquatic microcosm was contained in the lake sediment, much of it unextractable.

Significant bioconcentration of carbofuran by aquatic organisms has not been found. This is supported by its octanol-water partition coefficient (K_{ow}) of 42.5, which was used to derive a BCF of 10 (Neely et al. 1974). Other BCFs of 2.5, 5, and 6 have also been reported (NRCC 1979). A depuration half-life of 4 d for carbofuran has been reported (NRCC 1979).

Water Quality Guideline Derivation

The Canadian water quality guideline for carbofuran for the protection of freshwater life was developed based on the CCME protocol (CCME 1991).

Freshwater Life

The acute toxicities (96-h LC_{50}) for fish range from 0.088 $mg \cdot L^{-1}$ for bluegill *(Lepomis macrochirus)* (Mayer and Ellersieck 1986) to 11.0 $mg \cdot L^{-1}$ for European carp *(Cyprinus carpio)* (Hejduk and Svobodova 1980). Exposed fish can exhibit the following symptoms: hypoactivity, body paralysis, lateral curvature of the spine (usually with localized hemorrhaging), loss of equilibrium, and opercular and mouth paralysis.

The acute toxicities for invertebrates range from a 48-h LC_{50} of 0.035 $mg \cdot L^{-1}$ for *Daphnia pulex* (Hartman and Martin 1985) to a 24-h LC_{50} of 20 $mg \cdot L^{-1}$ for *Tubifex tubifex* (Dad et al. 1982). Reports concerning the effect of carbofuran on algae and vascular plants demonstrated these organisms to be less sensitive than fish (Kar and Singh 1978, 1979; Hartman and Martin 1985).

Hansen and Parrish (1977) used sheepshead minnows *(Cyprinodon variegatus)* in long-term, partial life-cycle exposures to develop a MATC between 15 and 23 $\mu g \cdot L^{-1}$, based on survival of parental fish, hatching success of eggs, and mortality of developing fry.

Data on sublethal reactions and chronic toxicity have mostly been generated in tropical areas, where carbofuran is used in rice cultivation. Histopathological effects were observed in the liver, kidney, and intestinal tissue of *Anabas testudineus* after exposure to 560 $\mu g \cdot L^{-1}$ for 120 h (Bakthavathsalam et al. 1984). Other enzyme studies have reported significant deviations from control animals with exposure to as little as 31 $\mu g \cdot L^{-1}$ (Verma et al. 1981).

Table 1. Water quality guidelines for carbofuran for the protection of aquatic life (CCME 1989).

Aquatic life	Guideline value ($\mu g \cdot L^{-1}$)
Freshwater	1.8
Marine	NRG*

*No recommended guideline.

The patterns of use of carbofuran, along with its degradation and ability to be metabolized, make long-term, chronic exposure in the aquatic environment much less of a threat than short-term, acute exposure. The available data on chronic, sublethal exposures indicate that the resulting physiological and biochemical disturbances are reversible once carbofuran has dissipated.

The water quality guideline for carbofuran for the protection of freshwater life is 1.8 $\mu g \cdot L^{-1}$. It was derived by multiplying the 48-h LC_{50} (the lowest acute toxicity value derived in an approved manner) of 35 $\mu g \cdot L^{-1}$ for the most sensitive organism to carbofuran, *Daphnia pulex* (Hartman and Martin 1985), by a safety factor of 0.05 (CCME 1989).

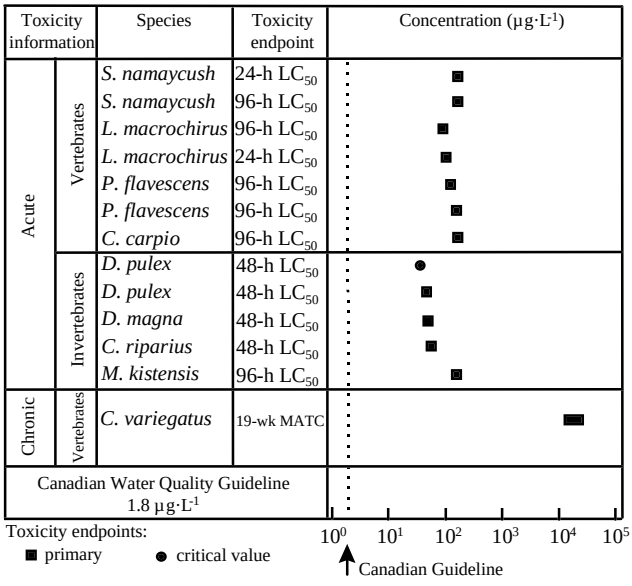

Figure 1. Select freshwater toxicity data for carbofuran.

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