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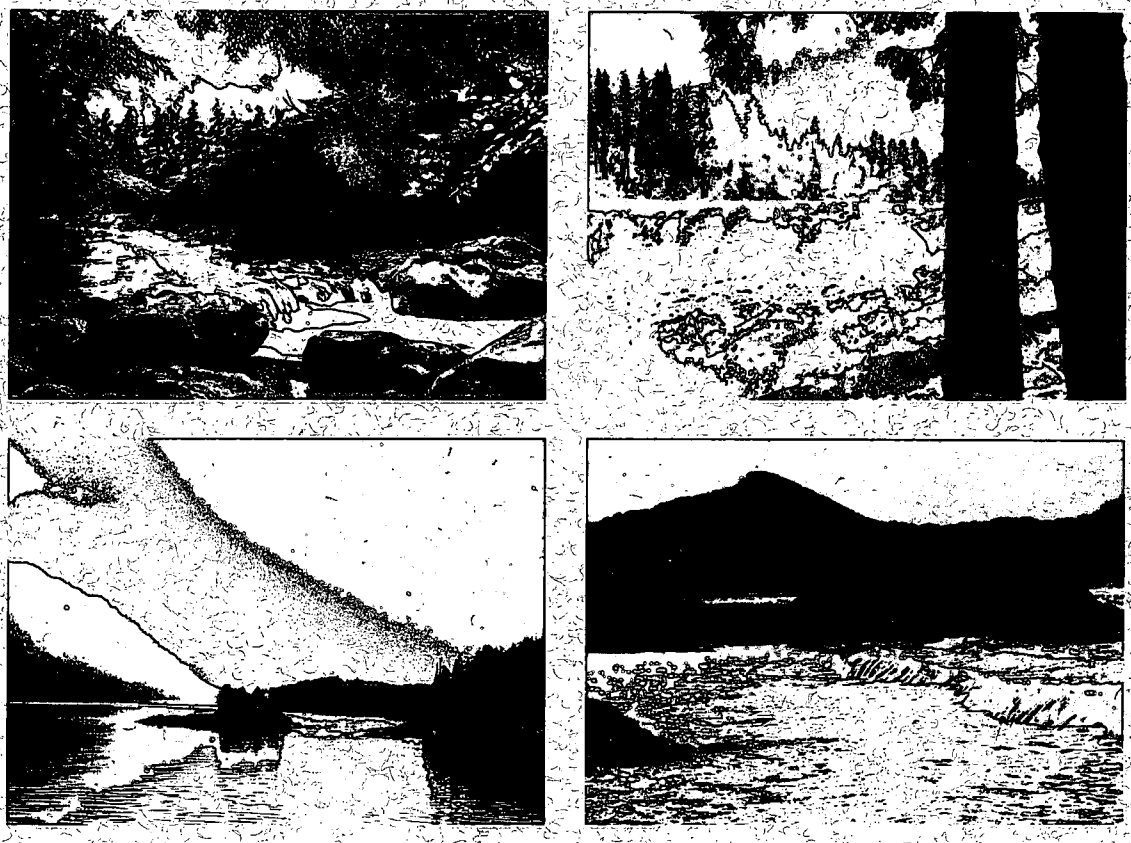

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OTTAWA, ONTARIO, 1990

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Abstract

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

Résumé

On a examiné la documentation relative aux utilisations, au devenir et aux effets du picloram sur l'eau naturelle utilisée comme eau potable non traitée, sur la vie aquatique en 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 les approvisionnements en eau pour l'industrie. Ces renseignements sont résumés dans cette publication. À partir de cette étude, des recommandations sur la qualité de l'eau sont adoptées pour la protection d'utilisations particulières de l'eau.

Canadian Water Quality Guidelines for Picloram

D.M. Trotter, R.A. Kent, and M.P. Wong

SOURCES, OCCURRENCE, AND CHARACTERISTICS

Uses and Production

Picloram, the common name for 4-amino-3,5,6-trichloropyridine-2-carboxylic acid (IUPAC), is a white powder with a chlorine-like odour. The structural formula for picloram is shown in Figure 1. Its Chemical Abstracts Service (CAS) Registry Number is 1918-02-1. The amine and potassium salts of picloram are soluble in water and constitute the active ingredients of several herbicides marketed by the Dow Chemical Company under the trade name Tordon^{R}. At present, three Tordon products are registered for use in Canada: Tordon^{R}22K, Tordon^{R}101, and Tordon^{R}202C (Agriculture Canada 1989). Tordon^{R}22K contains 240 $g \cdot L^{-1}$ picloram as isooctyl esters or as potassium salt in liquid form. It also contains glycol and sorbitol ester-type wetting agents along with alcohol and water. The potassium salt of picloram (2.3%) is also combined with 13.6% boron in Tordon^{R} beads. Picloram organic salts (triisopropanolamine and triethylamine) and the isooctyl ester are also used in combination with other herbicides. Tordon^{R}101 is a mixture of 10.2% picloram and 39.6% 2,4-D both as triisopropylamine salts. This mixture also contains a glycol derivative sequestrant and glycol wetting agent along with alcohol and water. Tordon^{R}202C contains 200 $g \cdot L^{-1}$ 2,4-D and 12 $g \cdot L^{-1}$ picloram. All soil-applied granular formulations of picloram have been discontinued for use in Canada because of concerns regarding contamination of ground water. This includes Tordon^{R}10K, a pellet formulation containing 11.6% picloram. An additional picloram product from the Dow Chemical Company, Tordon^{R}155 (15.1% picloram and 63.4% 2,4,5-T), is no longer in use since the sale of 2,4,5-T has been discontinued in Canada.

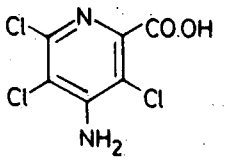

Figure 1. Structural formula for picloram.

Formulated in 1963, picloram is used in Canada as a wide spectrum herbicide for the control of woody and herbaceous broad-leaved plants along rights-of-way. It is also used with spot treatments for the control of "noxious weeds" in pasture and rangeland. Before the cancellation of Tordon^{R}10K, application rates of picloram ranged from 0.1 to 3.3 $kg\text{-}ai \cdot ha^{-1}$ for rights-of-way (ai = active ingredient). The present maximum dosage rate is 2.64 $kg \cdot ha^{-1}$ as Tordon^{R}22K. Application rates for spot treatments vary according to the density of the brush or weeds to be controlled (NRCC 1974).

In Ontario, Tordon^{R}101 is registered for weed and brush control in noncrop locations, industrial sites, and rights-of-way. It cannot be applied to land used for the production of agricultural or horticultural crops, and a permit is required for its purchase and use (Ontario Ministry of Agriculture and Food 1989). Most broadleaf weeds are

sensitive to picloram, including Canada thistle, clover, ragweed, dandelion, goldenrod, burdock, fleabane, and vetch. Most deciduous and coniferous woody plants (except white ash) are also sensitive to picloram (Ontario Ministry of Agriculture and Food 1989).

Imports of picloram-formulated herbicides into Canada for the years 1983, 1984, and 1985 were approximately 384, 749, and 670 metric tonnes, respectively (Statistics Canada 1986).

Physical and Chemical Properties

The physical and chemical characteristics of picloram are presented in Table 1. Although picloram, as the carboxylic acid, has an aqueous solubility of only 430 $mg \cdot L^{-1}$ at 25°C, its potassium salt has a solubility of about 400 000 $mg \cdot L^{-1}$, and the triisopropylamine salt has a solubility of about 800 000 $mg \cdot L^{-1}$ (Mayes and Dill 1984).

Mode of Action

Picloram acts as an auxin type of plant hormone (Foy 1976). It is highly phytotoxic and is easily absorbed and translocated by the leaves and roots of plants (U.S. EPA 1988; Worthing and Walker 1987). Its main mode of action in plants is as a growth regulator. The symptoms of picloram poisoning include epinasty, plugging and browning of xylem vessels, wilting, necrosis, and plant death (Foy 1976). Wilting is apparently due to complex interactions of pathogen-produced hormones, toxins, and toxic enzymes. Picloram has other effects including inhibition of germination and seedling growth, and, possibly, reduction of respiration in mitochondria. Inhibition of oxidative respiration in isolated cucumber mitochondria and of nucleic acid metabolism has been reported as the result of picloram treatment, and the herbicide may also decrease enzyme synthesis and uncouple oxidative phosphorylation (Foy 1976; NRCC 1974). The inhibitory action of picloram may be the result of chelation within the plant tissue. Chelation might interfere with plant respiratory enzyme systems, with carriers such as the cytochrome systems, which contain heavy metals, or with free metal ions in the mitochondria (Foy 1976).

Methods of Analysis

Analysis of picloram is by gas-liquid chromatography or by high-performance liquid chromatography (Worthing and Walker 1987). The NRCC (1974) summarized earlier methods for the analysis for picloram. Gas chromatographic techniques have been used for the analysis of picloram in soil and grass (Bovey et al. 1975). More recently, Wells et al. (1984) used reverse-phase liquid chromatography with UV detection for picloram in soil and water samples. The detection limit was 2 $\mu g \cdot L^{-1}$. For the analysis of picloram residues in surface and ground water, and in soil, sand, and vegetation, a gas chromatograph with an electron capture detector was used (Watson et al. 1989). In this latter investigation, minimum detection limits were 0.5 $\mu g \cdot L^{-1}$ for water, 5 to 6 $\mu g \cdot kg^{-1}$ for soil and sand, and 10 $\mu g \cdot kg^{-1}$ for vegetation. Woodburn et al. (1989) used several chromatographic and mass spectrometry methods for the analysis of picloram and its photolytic decay products in water samples.

Entry into the Environment

The National Research Council of Canada summarized the routes and mechanisms by which picloram can enter various components of the environment (NRCC 1974). Picloram can enter the atmosphere through spray drift during application and as a result of vaporization after application. It may enter the aquatic environment as a result of direct application to surface waters or through surface runoff and leaching from treated soils.

Table 1. Physical and Chemical Characteristics of Picloram

Chemical formula:	$C_6H_3Cl_3N_2O_2$
Molecular weight:	241.48
Physical state:	White powder with chlorine-like odour
Melting point:	Decomposes at 215°C
Sublimation temperature:	190°C at 12 mm Hg
Vapour pressure:	6.16×10^{-17} mm Hg at 35°C 1.07×10^{-6} mm Hg at 45°C
Aqueous solubility:	430 $mg \cdot L^{-1}$ (25°C) as carboxylic acid 400 000 $mg \cdot L^{-1}$ (25°C) as potassium salt 800 000 $mg \cdot L^{-1}$ (?) as amine salt
Solubility in other solvents:	
Acetone	19 800 $mg \cdot L^{-1}$
Ethanol	10 500 $mg \cdot L^{-1}$
Isopropanol	5 500 $mg \cdot L^{-1}$
Acetonitrile	1 600 $mg \cdot L^{-1}$
Diethyl ether	1 200 $mg \cdot L^{-1}$
Methylene chloride	600 $mg \cdot L^{-1}$
Benzene	200 $mg \cdot L^{-1}$
Carbon disulphide	50 $mg \cdot L^{-1}$
Kerosene	10 $mg \cdot L^{-1}$
Dissociation constant (pKa):	3.6
Half-life in soils:	1 to 13 months
Hydrolysis half-life:	18 years (pH 5,7, & 9, 25°C)
Photolysis half-life:	7 d
Elemental analysis:	C, 29.85%; H, 1.25% Cl 44.05%; N, 11.60%; O, 13.25%

From NRCC 1974; Foy 1976; Ghassemi et al. 1981; Windholz et al. 1983; Worthing and Walker 1987; Mayes and Dill 1984; Mullison 1985.

Information on the occurrence of picloram in surface waters and sediments can be found in studies of picloram runoff from treated fields and in special monitoring studies initiated to investigate the movement of picloram to adjacent aquatic environments. A summary of studies concerning picloram in runoff water from treated areas is presented in Appendix A. The solubility of picloram and its various salt formulations allows potentially high concentrations in runoff water if heavy rainfall occurs shortly after application. If light rainfall occurs after application (allowing the picloram to percolate downward) and several days elapse before heavy rainfall (e.g., 30 days), the picloram concentration in runoff water can be reduced by two orders of magnitude (Bovey et al. 1967). Concentrations of picloram in runoff water can be relatively high (3 $mg \cdot L^{-1}$) under certain weather conditions. It has been estimated that 5.5% to 6.3% of the applied picloram can move from the application site (Trichell et al. 1968; Mayeux et al. 1984).

Slope and soil compaction also influence the amount of picloram in runoff. Higher concentrations of picloram in runoff water are generally associated with steeper slopes and occur in the lower half of the slope. There are lower concentrations of picloram in runoff water if the runoff flows over untreated, compacted (fallow) soil (Trichell et al. 1968; Scifres, Hahn et al. 1971).

Another way in which picloram can enter the environment is through spray drift during application. Several incidents of damage to nontarget plants as a result of picloram spray drift have been reported. For instance, the NRCC (1974) reported an incident in which trees 50 m from a treated area were killed by spray drift after picloram application. Ghassemi et al. (1981) also mentioned reports from Minnesota in which drift of picloram from roadside spraying caused injury to nearby corn and soybean fields.

Concentrations in Water, Sediment, and Biota

Several studies outlining the picloram concentrations that have been found in various environmental compartments are summarized in Appendix B. The behaviour of picloram in two east-central Texas watersheds was monitored after single applications of picloram at 0.56 $kg \cdot ha^{-1}$ (Scifres et al. 1977). Picloram was found in the surface waters of one catchment at 170–460 $\mu g \cdot L^{-1}$ 27 d posttreatment. Surface waters of the second catchment contained 80–490 $\mu g \cdot L^{-1}$ picloram after the same time period. After 52 d, only trace amounts (<10 $\mu g \cdot L^{-1}$) of picloram were found in the surface waters of the first watershed. An average of 77 $\mu g \cdot L^{-1}$ picloram was detected in the surface waters of the second watershed. Using the results of picloram monitoring data from both watersheds, it was determined that only 0.05% of the picloram originally applied was detected in runoff water during the first month of the study. Most of the applied picloram remained in the live vegetation and top 15 cm of soil (Scifres et al. 1977).

Dennis et al. (1977) studied runoff from spot-treated (4.5 $kg \cdot ha^{-1}$) land in West Virginia. Runoff resulted in pond water picloram concentrations as high as 437 $\mu g \cdot L^{-1}$ (detection limit of 0.2 $\mu g \cdot L^{-1}$). The concentration declined to approximately 54 $\mu g \cdot L^{-1}$ after 178 d; after 294 d, a mean concentration of 14.2 $\mu g \cdot L^{-1}$ remained in the pond. Picloram was detectable in pond sediments for more than 270 d, but was not detected (detection limit of 5.0 $\mu g \cdot L^{-1}$) in bottom sediments collected from streams. The picloram concentration in wet sediment 2 weeks after application was 243.0 $\mu g \cdot kg^{-1}$. This concentration had been reduced to 15.5 $\mu g \cdot kg^{-1}$ 294 d later. Picloram was detected in water samples collected up

to 5.5 km downstream from the treated sites. Additional information on picloram in runoff water, but with insufficient supplementary data for presentation in Appendix A, includes reports of a concentration of 370 $\mu g \cdot L^{-1}$ at 10 d after soil application of 10.4 $kg \cdot ha^{-1}$ (Davis et al. 1968) and <100 $\mu g \cdot L^{-1}$ at 100 d after application of 1.9 $kg \cdot ha^{-1}$ (Johnsen and Warskow 1968). Both studies were conducted in Arizona watersheds.

Information on the use of picloram pellets and liquid sprays does not indicate differences in the amount of picloram in runoff water from these different formulations. The incorporation of picloram into starch xanthate granules did, however, eliminate the initial large concentration of picloram in the first runoff water compared to the liquid spray application. Concentrations of picloram in subsequent runoff events tended to be higher from the area treated with the xanthate granules than areas treated with the liquid spray. Cumulative loss of picloram from both treatments tends to be similar (NRCC 1974).

A special monitoring program was conducted at the Jimmy Lake Weapons Range (western Saskatchewan) because of large-scale use of picloram at the site. During 12-14 August 1982, a 490-ha area was treated with Tordon^{R}10K pellets (3.3 kg picloram$\cdot ha^{-1}$). Picloram was observed to move into the ground water and travel laterally toward Primrose Lake, 1 km outside the treated area. Concentrations increased in the ground water near Primrose Lake from 0.14 $\mu g \cdot L^{-1}$ in October 1983 to 438.5 $\mu g \cdot L^{-1}$ in October 1984. Surface-water concentrations of picloram in Primrose Lake increased to a maximum of 1.15 $\mu g \cdot L^{-1}$ in October 1984 at a location adjacent to the site with the highest recorded ground-water concentration. Only one sediment sample from Primrose Lake (collected on 20 June 1984) contained detectable levels of picloram (11.97 $\mu g \cdot kg^{-1}$) (Waite et al. 1986; Smith et al. 1988).

Direct injection of picloram into a small semiarid stream in central Arizona demonstrated dissipation through the normal mixing of the stream water in its channel. Picloram injected at 6.26 $mg \cdot L^{-1}$ was detected at a maximum concentration of 2.36 $mg \cdot L^{-1}$ 0.4 km downstream (Johnsen and Warskow 1980).

The extent and duration of stream water contamination by picloram and the loss of the herbicide from a sloped, gravelly loam sand watershed in central Arizona were studied by Davis and Ingebo (1973). Picloram pellets were applied on 1 February 1965 at an average rate of 10.42 $kg \cdot ha^{-1}$. After the application, a rainstorm on 6 and 7 February resulted in a picloram concentration of 370 $\mu g \cdot L^{-1}$ in the first water sample collected on 8 February. The concentration of picloram gradually declined to 31 $\mu g \cdot L^{-1}$ 18 d later. From 15 April to 3 September, picloram concentrations in the runoff water ranged from nondetectable levels to 8 $\mu g \cdot L^{-1}$. There was no picloram detected in a water sample taken on 25 March 1966, 15 months after the initial treatment. From the stream monitoring data, the authors estimated that 3.5% of the applied picloram was lost from the watershed to the stream water.

Field studies of potential picloram contamination of streams crossing electric transmission line rights-of-way (ROW) in British Columbia during aerial herbicide applications were reported by Wilson and Wan (1975). The establishment of 45-m buffer zones on both sides of a creek crossing the ROW prevented detectable concentrations (detection units not given) of picloram from occurring in the creek. In this particular study, the triisopropylamine salt of picloram was sprayed from a helicopter at an altitude of 23-38 m, travelling at a speed of 48 $km \cdot h^{-1}$. The application rate was 2.1 $kg \cdot ha^{-1}$.

Picloram was found in surface and subsurface waters in and adjacent to (i.e., within 100 m) transmission line ROW in Quebec after aerial and ground applications (Varfalvy and Seguin 1987). Samples of water collected during the treatment season had average picloram concentrations ranging from 5.6 to 181 $\mu g \cdot L^{-1}$. The highest concentration reported (1160 $\mu g \cdot L^{-1}$) occurred in a pool located inside the ROW boundary during the first week after aerial spraying of Tordon^{R}10K. A concentration of 190 $\mu g \cdot L^{-1}$ was reported in a stream 30 m outside the treatment area. A lake 12 m outside the ROW boundary was reported to have concentrations of 3.7 $\mu g \cdot L^{-1}$ and 7.6 $\mu g \cdot L^{-1}$ at 4 wk and 8 wk, respectively, after a terrestrial foliage spray treatment with Tordon^{R}101. A maximum picloram concentration of 104 $\mu g \cdot L^{-1}$ in ground water immediately adjacent to the treated ROW was reported 8 wk after treatment. This was not considered unusual given the high water table and the sandy soil of the region (Varfalvy and Seguin 1987). Two other incidents of ground-water contamination (on the order of 1-10 $\mu g \cdot L^{-1}$) after picloram application in Quebec have been reported (Villeneuve et al. 1985).

Ground spraying with 1.12 $kg \cdot ha^{-1}$ picloram of vegetation along roadsides in a granitic upper mountain Montana watershed did not result in detectable residues (i.e., >0.5 $\mu g \cdot L^{-1}$) in an adjacent creek (average distance from road 33.5 m) (Watson et al. 1989).

The National Water Quality Data Base (NAQUADAT) detailed report (Environment Canada 1983) lists picloram among a group of pesticides monitored in 25 selected surface-water sites in western Canada. Picloram was reported to be present at 0.1 $\mu g \cdot L^{-1}$ in March 1985 in the South Saskatchewan River south of Empress, Alberta. Although the period examined was from April 1974 to January 1987, a change in the analytical method for picloram between February and May 1985 reduced the detection limit from 0.2 to 0.05 $\mu g \cdot L^{-1}$. Thus, picloram may have occurred in the river water prior to March 1985.

Reports of the presence of picloram in Canadian surface and ground water are summarized in Appendix B. In addition to these studies, picloram was found in a single Kansas farmstead well out of 103 sampled by Steichen et al. (1988). The well contaminated with picloram had a concentration of 5.6 $\mu g \cdot L^{-1}$ in the initial sample and 3.3 $\mu g \cdot L^{-1}$ when the well was resampled 6 months later (detection limit of 0.40 $\mu g \cdot L^{-1}$). Of 188 wells and 3 rivers sampled in ten North Dakota countries, 6 wells and 2 rivers in five counties were found to contain picloram. Concentrations ranged from <0.1 to 12.8 $\mu g \cdot L^{-1}$ (wells) and from <0.1 to 6 $\mu g \cdot L^{-1}$ (rivers). All areas of contamination were associated with land treated for control of leafy spurge, Euphorbia esula. Picloram had been spilled or misapplied near all but one of the contaminated wells, and the highest concentration of picloram in well water (12.8 $\mu g \cdot L^{-1}$) was apparentlythe result of a spill that occurred during the filling of spray equipment 2 years earlier (Lym and Messersmith 1988).

Ten fish sampled in 1983 and 1984 from Primrose Lake in Saskatchewan did not contain detectable concentrations (detection limit 5 $\mu g \cdot kg^{-1}$) of picloram in the dorsal muscle tissue of walleye, Stizostedion vitreum (1983), or whitefish, Coreogonus clupeaformis (1984) (Waite et al. 1986).

Environmental Fate, Persistence, and Degradation

Soil

Picloram-formulated herbicides may be applied by ground or aerial application equipment. Foliage sprays are usually applied during active growing periods. Sprays may also be applied to the bark of trees. Granular formulations may be applied over the roots of plants to be controlled during the

normal growing season and when rainfall can be expected soon after treatment (Thomson 1979).

Picloram is rapidly absorbed by plant roots and to a lesser extent by the foliage. Unabsorbed picloram may move downward or laterally through the soil because of its solubility and low adsorption on some soils. The major factor controlling the extent of adsorption is soil organic matter content. Grover (1977) found an inverse relationship between adsorption and mobility in five Canadian prairie soils. Adsorption was significantly related to the soil organic matter content.

Picloram leaches to the greatest extent in sandy, light-textured soils with low organic matter. Little picloram moved below 45 cm 9 months after application of up to 4.48 $kg \cdot ha^{-1}$ to a silty clay and silt loam soil in Ohio (Herr et al. 1966). In a sandy silt loam, the herbicide moved through the top 60 cm of soil, and the greatest residue concentrations were found in the deeper soil layers after 9 months. The herbicide persisted longer in the heavier-textured soils with the highest organic matter content. Similarly, more downward movement of picloram was observed in sandy loam soil (organic matter content 1.8%) than in silty clay loam soils (average organic matter content about 4%) in Nebraska (Scifres, Hahn, et al. 1969).

In any soil, the adsorption and mobility of picloram is influenced by average pore-water velocity, bulk density, and soil aggregate size (Davidson and Chang 1972). Besides adsorption, the resistance to leaching of picloram in some soils may be the result of retention of the herbicide in soil micropores (Rao et al. 1974), or diffusion from conducting pores in the soil to adjacent micropores (Ping et al. 1975).

The formulation of picloram may also affect its movement. The potassium salt is more mobile than the triisopropanolamine salt in soil columns (Ghassemi et al. 1981). Sirons et al. (1977) found that the triisopropanolamine salt was highly mobile vertically. The rate of picloram application and rainfall amount also has a significant impact on picloram movement even in soils in which it is strongly adsorbed (Grover 1967; Keys and Friesen 1968; Scifres et al. 1971; Hunter and Stobbe 1972).

The persistence and movement of picloram in clay soils and vegetation, and its occurrence in subsurface water after application to a watershed and to the soil surface above a lysimeter in Texas was monitored by Bovey et al. (1975). Four biannual applications of 2.24 $kg \cdot ha^{-1}$ of a 1:1 mixture of the triethylamine salts of 2,4,5-T and picloram were initiated on 4 May 1970, followed by a single application of 1.12 $kg \cdot ha^{-1}$ of the same mixture. Subsurface water flow was about 1.5-3 m below the soil surface. The maximum concentration of picloram in the soil was 162 $\mu g \cdot kg^{-1}$ in the 0-15 cm soil layer on the last day of application (8 October 1971) of the 2.24-$kg \cdot ha^{-1}$ treatment. (The low concentration in the soil was attributed to spray interception by the heavy grass cover.) After 191 d, this concentration had been reduced to below detection limits (detection limits not given). In the grass growing on the study area, the maximum concentration of picloram measured was 70 265 $\mu g \cdot kg^{-1}$ after the 8 October 1971 application, but, again, this concentration was reduced to below detection limits after 191 d. Thus there was no apparent tendency for picloram to accumulate in the soil or vegetation even after repeated applications. Subsurface water samples usually contained no detectable picloram even after the watershed had received five applications of the relatively high treatment rate; trace amounts ($<0.1 \mu g \cdot L^{-1}$) of picloram were detected in only a few samples from observation wells in treated and untreated areas of the watershed. A maximum or 4 $\mu g \cdot L^{-1}$ picloram was found in the water collected by the lysimeter.

The authors noted that the low levels of picloram in the soil profile, especially in the lower soil horizons, indicated the low amount of herbicide available for leaching to the subsoil and contamination of the subsurface water.

Soils with high organic matter content may adsorb and retain considerable quantities of picloram, which can injure crops years after application. Adsorption generally increases with decreasing pH and is much lower in neutral and alkaline soils. Clay soils exhibit very strong adsorption of picloram because of the presence of aluminum and iron oxides (Norris 1970; Grover 1971; Biggar and Cheung 1973; Davis and Ingebo 1973; Farmer and Aochi 1974). Adsorption and binding of picloram to organic matter forming unextractable residues also appear to increase with time (Evans and Norris 1986). In spite of this, picloram usually does not persist or accumulate in the soil even after repeated applications. Picloram applied at rates of up to 350 $g \cdot ha^{-1}$ in alternate years for 7 years did not accumulate in the top 50 cm of soil (Sirons et al. 1977); 36 months after spraying, picloram was not detected. After a single application of approximately 17.4-20.8 $kg \cdot ha^{-1}$ to clumps of bushes in Arizona, Davis and Ingebo (1973) found 1.0 $\mu g \cdot kg^{-1}$ picloram in the top 15 cm of soil 6.6 years after treatment. After application of 0.28 $kg \cdot ha^{-1}$ picloram to ryegrass on a sandy loam soil in the United Kingdom, the herbicide dissipated with a half-life in the soil of less than 2 wk (Fryer et al. 1979). An application of 1.68 $kg \cdot ha^{-1}$ dissipated with a half-life of slightly longer than 2 wk. The latter authors reported that approximately 1 year after each picloram application between 2% and 6% of the picloram applied was recoverable. Further, no accumulation took place; however, after an initial rapid dissipation, there was only a slow disappearance of the remaining residue. In a montmorillonitic clay soil, picloram applications of 23.3-1890 $g \cdot ha^{-1}$ had not moved below the surface 30 cm of soil 28.4 months after application in Australia (Marley 1980). With the lower application rates of 23.3 and 70 $g \cdot ha^{-1}$, less than 10% of the applied picloram was present in the soil 7.4 months after application.

Microbial degradation is the principle method by which picloram is broken down in the soil (Mullison 1985). Chemical routes of degradation in soils seem relatively insignificant (Ghassemi et al. 1981). Although microbial degradation increases with favourable conditions for microbial growth, the overall amount of picloram decomposed to CO_2 is relatively small. This suggests that microbial degradation does not involve cleavage of the pyridine ring. Picloram is apparently not used as a sole carbon source for microbes, and any microbial degradation results from the co-metabolic activity with other microbial carbon substrates (Grover 1967; Youngson et al. 1967; Naik et al. 1972; Foy 1976). The degradation of picloram in soil is inversely related to the picloram concentration; as picloram concentrations increase, degradation decreases (Mullison 1985; Herr et al. 1966). As biological degradation can be the major route of dissipation in soil, degradation rates increase in warm, moist soils high in organic matter, which have enhanced microbial activity, while in cool and dry soils, picloram will have a longer persistence (U.S. Department of Agriculture 1984; Davis and Ingebo 1973; Hamaker et al. 1967).

Degradation rate kinetics for picloram in soil have been reported to be one-half to first order under practical application rates (i.e., 0.1-3.3 $kg \cdot ha^{-1}$) (Davis and Ingebo 1973). Calculated half-order rates for picloram degradation in 18 U.S. states and 2 Canadian provinces ranged from 2.9 to 7.4 $g \cdot ha^{-1}$ per month and were correlated with temperature (NRCC 1974). Half-order rate constants for Alberta and Saskatchewan were 5.3 and 2.9 $g \cdot ha^{-1}$ per month, respectively (NRCC 1974; Hamaker et al. 1967). Other degradation rates

were reported to be 4% in 15 d by plant-root microorganisms (Meikle et al. 1966), and 0.24% to 1.21% over 63 d by different types of bacteria and fungi exposed to 1 $mg \cdot L^{-1}$ (Youngson et al. 1967).

In grass, Getzendaner et al. (1969) found that the picloram residue level decreases very rapidly. An initial application of 1.12 $kg \cdot ha^{-1}$ resulted in residues on the grass of approximately 200 $mg \cdot kg^{-1}$ at the time of application. The average residue level in the grass was 150 $mg \cdot kg^{-1}$ at the same time, which decreased to 50 $mg \cdot kg^{-1}$ in 2 wk. After 1 year, the grass contained little detectable residue (maximum of 12 $mg \cdot kg^{-1}$). In a similar investigation (Scifres, Hahn, and Merkle 1971), about 25 $mg \cdot kg^{-1}$ of picloram was detected on grass immediately after application of 0.28 $kg \cdot ha^{-1}$ in Texas. The picloram rapidly dissipated, and usually less than 1 $mg \cdot kg^{-1}$ was detected in grass tissue 30-60 d after treatment. The level of picloram residue in grass tissue was reduced by 99% 72 d after the application. In this study, detectable picloram had also been reduced 93% in herbaceous, broadleaf species 30 d after application.

Photodecomposition, apparently by pyridine ring cleavage, is a significant pathway for picloram degradation on plant or soil surfaces. Approximately 20% of a 4.8-$g \cdot L^{-1}$ solution of picloram, as the acid, was decomposed after 48-h exposure to UV light (253.7 nm) under laboratory conditions (Hall et al. 1968). However, since the emission spectrum of natural sunlight is approximately 290-750 nm, the environmental relevance of this data is questionable. In other studies, the isooctyl ester of picloram was degraded more rapidly (96% decomposition) than the potassium salt (26% decomposition) after 72-h exposure to UV light (300-380 nm) in open petri dishes containing wet and dry soil under laboratory conditions (Bovey et al. 1970). The sodium salt of picloram in aqueous solution (502 $mg \cdot L^{-1}$) exhibited 30.7% and 60.5% photolytic decomposition after 25 and 34 h, respectively, when irradiated in cylindrical quartz cells by UV light (300-380 nm) at 30°C (Mosier and Guenzi 1973). However, photodecomposition of picloram is slower and more variable in natural sunlight than under UV irradiation in the laboratory (Bovey et al. 1970; Norris and Morre 1970; Bovey and Scifres 1971; Mosier and Guenzi 1973). When spread on a glass surface, 60% of the picloram was degraded by short-wavelength UV light (155 $\mu W \cdot cm^{-2}$) within 48 h, but only 35% was degraded in the same time by natural sunlight (Merkle et al. 1967). One-week exposure of picloram on a glass surface produced 90% degradation by the same UV light (wavelength not given), but only 65% degradation by natural sunlight. A 44.7% loss of picloram, as Tordon^{R}22K, sprayed at 0.28 $kg \cdot ha^{-1}$ on old field vegetation was attributed to photodegradation during the first week after application (Watson et al. 1989).

Volatilization is not expected to be a major mechanism of loss of picloram from soil due to the low vapour pressure of picloram and its various formulations. This is also indicated by laboratory studies where <5% of the applied picloram (as the potassium salt) was lost from open petri dishes maintained at 55°C-60°C over 1 wk (NRCC 1974). Anaerobic nonbiological chemical degradation of picloram does not appear to occur (Hance 1967, 1969).

The general structure of picloram, a pyridine-2-carboxylic acid, is known to function as a chelating agent for metal ions. Strong interactions occur with Fe(II) and Ni(II) under conditions similar to those in soils or aquifers. This prompted Michaud and Hoggard (1988) to speculate that picloram complexation with Fe(II) represented a possible removal mechanism for picloram in ground water.

Water and Sediment

Picloram mobility in sediment has not been studied, but it has been reported to be not strongly adsorbed to dilute solutions of soil organic matter and natural sediments (Muir 1990). Laboratory studies indicate that photolysis is the primary mechanism for picloram degradation in water (Hall et al. 1968; Haas et al. 1971; Mosier and Guenzi 1973; Ghassemi et al. 1981). Photolytic half-lives varying from 5 to 60 d are reported for picloram in 2.5-cm and 3.6-cm deep containers, respectively. Circulating solutions as deep as 3.64 m and containing up to 100 $mg \cdot L^{-1}$ picloram followed pseudo-first-order degradation kinetics under natural sunlight (Hedlund and Youngson 1972). In areas of abundant sunlight, picloram decomposed rapidly in distilled water with a half-life of 6-8 d. With the exception of the highest picloram concentrations (e.g., 2500 $mg \cdot L^{-1}$), a 30-d exposure yielded 90% picloram decomposition. The rate of picloram photodecomposition in water is proportional to the light intensity and depth of solution, but is independent of the initial concentration (Hedlund and Youngson 1972).

The photolysis of picloram involves an ionic mechanism resulting in chloride ion production. Photolysis rates increase with the ionic strength of the solution. Free radicals of oxygen also contribute to the decomposition process. These mechanisms, however, produce different decomposition products with the free radical process probably producing oxidation products. The products of the ionic mechanism are not known (Mosier and Guenzi 1973).

Field experiments in semiarid central Arizona showed photolysis was responsible for the decomposition of 57% of the picloram in glass jars during an 8.8-h exposure to natural sunlight (Johnsen and Warskow 1980). A later study investigated the effect of altitude on picloram disappearance in sunlight (Johnsen and Martin 1983). Losses of picloram in water exposed to sunlight ranged from 50% to 80% after exposure to 14 h of sunlight, and to 95% or more after 56 h of sunlight in southern Arizona (altitudes of 700-2800 m). Finally, Woodburn et al. (1989) studied the photolysis of ring-labelled picloram at 25°C in sterile, buffered (pH 7) water and in a water sample taken from a forest ecosystem. Water samples containing picloram were held in 750-mL quartz flasks and were irradiated by eight phosphor-coated mercury vapour arc lamps (290-320 nm) for 24 hours, an exposure the authors claim to be equivalent to a midsummer sunlight day at 40°N latitude. The half-lives for these conditions, calculated using first-order kinetics, were 2.7 d for the sterile water and 2.5 d for the natural water. The two major photoproducts were low molecular weight organic acids, indicating that in aqueous solutions picloram undergoes photolytic dechlorination and cleavage of the pyridine ring.

Hydrolysis of picloram in water is negligible as it was reported to be stable in ground water at 10°C and 25°C for up to 15 months (Weidner 1974) and in darkened controls maintained during photolysis experiments. Similarly, picloram is unlikely to volatilize from water at neutral pH because it exists as an anion (Muir 1990). There was no volatilization of picloram from stream water held in open beakers (Johnsen and Warskow 1980).

RATIONALE

Raw Water for Drinking Water Supply

Guideline

The interim maximum acceptable concentration (IMAC) for picloram in drinking water is 190 $\mu g \cdot L^{-1}$ (Health and Welfare Canada 1987). This value is based on a negligible daily intake of 0.02 $mg \cdot kg^{-1} \cdot d^{-1}$ established by a 2-year feeding study with rats using increased

mortality, organ weight changes, andreduced activity as effect criteria. This IMAC is currently under review by the Federal-Provincial Subcommittee on Drinking Water of Health and Welfare Canada (G. Wood, 1988, Health and Welfare Canada, pers. comm.)

Concentrations

Canadian data on the concentrations of picloram in raw and treated drinking water can be found in Appendix B. Picloram has not been found in treated drinking water. In one area of New Brunswick, almost 50% (5 of 12) of the ground-water samples collected were contaminated (Franklin 1985). Between 1969 and 1978, Frank et al. (1979) investigated 237 farm wells in Ontario where herbicide contamination was known or suspected. Picloram was found in 6 wells in concentrations from 0.1 to 100 $\mu g \cdot L^{-1}$. The authors noted that the herbicide gained entry to the wells through spray drift, during storm runoff, or through subterranean intrusion. In a sand point well 5 m deep, about 1 L of a 1% solution of picloram was backsiphoned into the well. After 706 days, picloram was still present in this well (at 0.08 $\mu g \cdot L^{-1}$) even after decontamination efforts. Another well contaminated with picloram was abandoned 3 years after the initial contamination. The authors concluded that picloram was a particularly persistent well contaminant. These wells, however, were contaminated because of the misuse of the herbicide around the wells.

Removal by Water Treatment Operations

No information was found on the treatment technologies that might be capable of removing picloram from contaminated water (U.S. EPA 1987).

Freshwater Aquatic Life

Bioaccumulation

The solubility of picloram and its associated salts, as well as the other chemical properties of these compounds, do not indicate significant uptake or accumulation by lipid-containing tissues of aquatic organisms. Available bioconcentration factors for picloram in aquatic organisms are usually 1 or less (Hardy 1966; Youngson and Meikle 1972). These values indicate that, at equilibrium, picloram is excreted from the organism at the same rate of uptake and/or picloram is not easily taken up through aquatic food chains (i.e., no biomagnification) (Lynn 1965; Hardy 1966).

Toxicity to Aquatic Organisms

Acute Lethal Toxicity

A summary of aquatic toxicity data for picloram is presented in Appendices C and D. There are obvious differences between the 24-h LC_{50} data of Lynn (1965) and Kenaga (1969) for the same fish species exposed to Tordon^{R}22K. The reason for the much larger 24-h LC_{50} values reported by Lynn (1965) is due to the reporting of Kenaga's (1969) data as acid equivalents as opposed to the concentration of the formulation as reported by Lynn (1965). The Tordon^{R}22K formulation used by Lynn (1965) represented only 21.5% acid equivalents of picloram. When this is taken into account, the data of Lynn (1965) become similar to that of Kenaga (1969). The data of Weimer et al. (1967) were not presented as these tests used the Tordon^{R}101 mixture of picloram and 2,4-D. These data were originally reported by Lynn (1965).

A compilation of previously published and unpublished data (Mayes and Oliver 1985) presented the toxicity of the various formulated products of picloram on the basis of picloram acid equivalents (ae). The compiled data indicate that the isooctyl ester of picloram is the most toxic formulation to rainbow trout, Salmo gairdneri (96-h LC_{50} = 4.0 $mg \cdot L^{-1}$; data from Johnson and Finley 1980). For the goldfish, Carassius auratus, the toxicity of the carboxylic acid and the isooctyl ester were

similar (14-32 µg·L^{-1} ae and 10.4 µg·L^{-1}ae as 96-h LC_{50}s, respectively) (Kenaga 1969). The increased toxicity of the picloram ester formulation to S. gairdneri may have been due to the presence of a more toxic impurity, 2-(3,4,5,6-tetrachloro-2-pyridyl)-guanidine, in the formulation (Sargent et al. 1971). Further information regarding the toxicity of this contaminant was not found. Salmonids and the channel catfish, Ictalurus punctatus, are generally the fish species most sensitive to the toxic effects of picloram, regardless of the formulation tested. An analysis of the 96-h LC_{50} data presented by Mayer and Ellersieck (1986) shows the following mean 96-h LC_{50} values for salmonid species: lake trout, Salvelinus namaycush, 3.9 mg·L^{-1}; cutthroat trout, Salmo clarki, 5.0 mg·L^{-1}; and rainbow trout, S. gairdneri, 8.8 mg·L^{-1}. The average 96-h LC_{50} for the channel catfish, I. punctatus, was 10 mg·L^{-1}. By contrast, the mean 96-h LC_{50} for the bluegill, Lepomis macrochirus, was 23.3 mg·L^{-1}.

There is much less information on the acute toxicity of picloram to invertebrates. Twenty-four-hour LC_{50}s range from 20 mg·L^{-1} for the amphipod Gammarus pseudolimnaeus to 140 mg·L^{-1} for the stonefly nymph, Pteronarcys californica (Mayer and Ellersieck 1986). Both values are for exposure to the acid form of picloram as >90% active ingredient. Forty-eight-hour LC_{50}s for picloram (>90% ai) as the acid range from 50.7 to 76.0 mg·L^{-1} for first instar Daphnia magna (Mayes and Dill 1984; Mayer and Ellersieck 1986). Although limited data are available, the amphipod G. pseudolimnaeus, appears to be the most sensitive invertebrate; 96-h exposures gave an LC_{50} of 16.5 mg·L^{-1} picloram as the acid (Mayer and Ellersieck 1986). The least sensitive invertebrate appears to be the stonefly nymph, P. californica, with a 96-h LC_{50} of 48 mg·L^{-1} picloram as the acid (Johnson and Finley 1980). The LC_{50}s of formulated products containing lower percentages of picloram as the potassium salt produce higher median lethal values.

Acute algal toxicity data are scarce, with only one 24-h EC_{50} of 115 mg·L^{-1} for Selenastrum capricornutum, based on oxygen evolution, which was published as part of a screening methodology (Turbak et al. 1986). Growth and development (assessed using cell counts and changes in optical density) of various species of freshwater and marine algae (species not given), including the rate of carbon fixation, were not affected by doses of picloram of up to 240 mg·L^{-1} (Elder et al. 1970). Elder et al. (1970) concluded that normal use of picloram-containing herbicides did not appear to pose a hazard to algal populations from terrestrial runoff or indirect contamination. The algal toxicity data provided by Walsh (1972) are not presented in Appendix C because the tests used a mixture of picloram and 2,4-D.

Kratky and Warren (1971) reported a <50% inhibition of Chlorella cell growth in nutrient solution (actual inhibition not given) after an 18- to 36-h exposure to picloram concentrations of 1 or 10 mg·L^{-1}. A paper disc agar diffusion method for algal sensitivity to herbicides demonstrated that 1000 mg·L^{-1} picloram did not inhibit the growth of Chlorella-seeded agar plates outside the diameter of the paper disc containing the picloram (Thomas et al. 1973).

Chronic Toxicity and Sublethal Reactions

An 8-d flow-through toxicity test using 90-d-old rainbow trout, S. gairdneri, produced a 192-h LC_{50} of 14 mg·L^{-1} and a no-observed-adverse-effect level (NOAEL) of 6.9 mg·L^{-1}. Toxicity tests using the embryo and larval stages of the same species over approximately 70 d produced an NOAEL of 0.55 mg·L^{-1} and a maximum acceptable toxicant concentration (MATC) of 0.70 mg·L^{-1} (Mayes et al. 1987).

Woodward (1979) simulated the effects of picloram pulsed exposure on an early life stage (3-d posthatch) of the cutthroat trout, S. clarki. Technical grade picloram (90% ai) was slowly me-

tered into continuous flow test tanks to permit the gradual increase of picloram to a predetermined concentration at the end of 48 h. Picloram input was then stopped and the concentration allowed to drop for 5 d prior to the second input of picloram. The concentration in each successive exposure was reduced by 50% to simulate the decreased presence of picloram in runoff water with time. Five testing regimens representing initial picloram concentrations of 7.90, 3.20, 1.60, 0.790, and 0.290 $mg \cdot L^{-1}$ were used. Each regimen was tested against the same group of fish 4 times. Although picloram exposure was terminated on day 24, observations were continued until day 60 for latent effects on survival and growth. The lowest concentration in an exposure regimen that adversely affected the test fish was 0.790-$mg \cdot L^{-1}$ (first exposure), which was lowered to a fourth exposure of 0.076 $mg \cdot L^{-1}$. Although fry survival in the 0.790-$mg \cdot L^{-1}$ regimen was not significantly different from the controls (92% versus 84%, respectively), fry growth to 60 d was significantly retarded (26% decrease in weight). Differences among the alevins and fry and controls in the 0.290-$mg \cdot L^{-1}$ exposure regimen did not exist in terms of development, growth, and survival (Woodward 1979). This type of toxicity assessment (i.e., pulsed exposure to toxic substances) is promoted for the establishment of water quality criteria by Holdway and Dixon (1986a).

Invertebrate chronic toxicity data consist of one MATC (14.6 $mg \cdot L^{-1}$) derived using Daphnia magna exposed to picloram as the acid (93.8% ai) for 21 d (Gersich et al. 1985).

Algal chronic toxicity data are represented by 10- to 14-d exposures of Chlorella to picloram as the acid and as the decarboxylated derivative measured in a microplate assay system (Appendix D). This system produced EC_{50}s of >160 $mg \cdot L^{-1}$ (Baarschers et al. 1988). The decarboxylated picloram was more toxic, with EC_{50}s of 8 and 49 $mg \cdot L^{-1}$. The criterion used in these assays was reduction in cell numbers. An EC_{50} of 44.8 $mg \cdot L^{-1}$ Tordon^{R}22K, based on reductions in cell biomass, resulted from a 2- to 3-wk exposure using Selenastrum capricornutum (Turbak et al. 1986).

According to the NRCC (1974), the only existing aquatic macrophyte study was performed by the Dow Chemical Co. This study included a 400-h exposure of five species (numbers of each species not given) to 10 $mg \cdot L^{-1}$ picloram under greenhouse conditions (21°C-27°C). The only species that exhibited a negative response was a small shoreline herbaceous plant, Lysimachia nummularia, which incurred significant (50%) injury. Details on what constituted injury and how it was measured were not provided. No obvious damage to aquatic macrophyte beds adjacent to a surface-water sampling site, where 1.15 $\mu g \cdot L^{-1}$ of picloram was detected, was reported in a field study of picloram off-target contamination (Waite et al. 1986).

Guideline

The vertebrate aquatic toxicity data base for picloram consists of data for 17 fish species, including 240 acute toxicity tests (i.e., exposures of 96 h or less) and 6 chronic studies. Of the 240 acute toxicity tests, 144 or 60% were conducted using six North American freshwater salmonid species. The remainder of the acute tests used eight North American warm-water species and three freshwater tropical species commonly used in toxicity testing. Of the six chronic studies, four were conducted with salmonid species and two with tropical species. One of the four salmonid chronic studies used early life stages. A separate study, not included in the above categories, used intermittent exposure with early life stages over a period of 192 h.

The invertebrate toxicity data base consists of 18 acute toxicity tests representing six different species from six different families. One 21-d chron- test was conducted with Daphnia magna.

The aquatic plant toxicity data base includes three common green algae: Selenastrum capricornutum and two species of Chlorella. Tests conducted with S. capricornutum consisted of both acute (24 h) and chronic exposures (14-21 d). Tests using the Chlorella species were chronic exposures (10-14 d).

The toxicity data contained in this review were of sufficient quality and quantity to derive a Canadian water quality guideline for the protection of freshwater aquatic life from picloram. Toxicity data reported for lake trout, Salvelinus namaycush, show that exposure to 35 $\mu g \cdot L^{-1}$ technical grade picloram from 10-d prehatch to 60-d posthatch was capable of reducing fry survival and significantly inhibiting fry growth (Woodward 1976). Mayes et al. (1987) were unable to interpret these data because of insufficient picloram test concentration measurements. Only the highest concentration of the test series appears to have been measured. Thus, the latter authors disregarded the 35-$\mu g \cdot L^{-1}$ effect datum because of insufficient analytical support.

The no-observed-effect concentration of 0.29 $mg \cdot L^{-1}$ (Woodward 1979) was derived from early life stage (3-d posthatch) exposure to pulsed or variable concentrations of picloram. The criteria for the no-observed effect were survival and growth. The next highest concentration used by Woodward (1979), 0.79 $mg \cdot L^{-1}$, produced significant growth retardation. Similarly, the lowest concentration that produced an effect in the Mayes et al. (1987) study was 0.88 $mg \cdot L^{-1}$. These two studies appear to be in close agreement as to the concentration causing adverse effects on growth in the early life stage of a sensitive fish species. The no-observed effect concentrations from these studies are somewhat further apart at 0.29 $mg.L^{-1}$ for the Woodward (1979) study and 0.550 $mg \cdot L^{-1}$ for the Mayes et al. (1987) study.

The value of 0.29 $mg \cdot L^{-1}$ (290 $\mu g \cdot L^{-1}$) (Woodward 1979) is the lowest no-observed-effect value in the scientific literature that had sufficient quality control/quality assurance support. With the exception of Woodward's (1976) report of effects at 35 $\mu g \cdot L^{-1}$, the value of 290 $\mu g \cdot L^{-1}$ is below all reported concentrations causing an effect in fish, invertebrates, and algae. It is also less than the MATC of 14 600 $\mu g \cdot L^{-1}$ based on chronic exposures with Daphnia magna (Gersich et al. 1985) and the MATC of 700 $\mu g \cdot L^{-1}$ based on chronic exposures of rainbow trout, Salmo gairdneri, early life stages (Mayes et al. 1987).

Of equal importance is the fact that the no-observed-effect value of 290 $\mu g \cdot L^{-1}$ was determined using an early life stage exposed to pulsed or variable concentrations of picloram. Early life stages (i.e., embryo-larva) are reported to be the period of vertebrate aquatic life most sensitive to toxicant exposure (Woltering 1984). Pulsed exposure has been shown to be significantly more detrimental than continuous exposure for another pesticide (methoxychlor) (Holdway and Dixon 1985, 1986b).

The available aquatic toxicity data do not meet the requirements for a Canadian water quality guideline (CCREM 1987, Appendix IX). Additional invertebrate toxicity data, in particular, chronic tests with nonlethal endpoints employing sensitive planktonic species, are necessary to support a full guideline. In addition, the algal and aquatic vascular plant data base is also in need of studies on the effects of picloram. For this reason an interim guideline for picloram was developed.

The derivation of an interim guideline for freshwater aquatic life is based on the no-observed-effect concentration of 290 $\mu g \cdot L^{-1}$ (Woodward 1979). Additional supporting data on pulsed exposures to early life stages of other North American fish or invertebrates

were not found. In accordance with the CCREM (1987) guideline development procedure, the NOEL value of 290 $\mu g \cdot L^{-1}$ is reduced by an order of magnitude to a guideline of 29 $\mu g \cdot L^{-1}$ for an additional margin of safety.

Agricultural Uses

Livestock Watering

Toxicity to Livestock and Related Biota

Acute Toxicity

The available data show that picloram is not very toxic to birds and mammals. Avian toxicity studies conducted by the Dow Chemical Co. and cited by NRCC (1974) and Ghassemi et al. (1981) are presented in Table 2. Mammalian acute dietary LD_{50}s range from 2000 to 8200 $mg \cdot kg^{-1}$ for rabbits and rats, respectively. This range also includes mice (2000–4000 $mg \cdot kg^{-1}$) and guinea pigs (3000 $mg \cdot kg^{-1}$). Sheep did not show acute adverse effects after ingesting up to 4650 $mg \cdot kg^{-1}$ of the potassium salt of picloram (25% ai) in their feed (Lynn 1965).

Sublethal and Chronic Toxicity

Long-term ingestion of picloram by rats of dietary concentrations as high as 1000 $mg \cdot kg^{-1}$ did not result in adverse effects after 90 d. Dietary concentrations of 3000 $mg \cdot kg^{-1}$ produced increased liver weight in female rats. Slight to moderate pathological changes in rat liver and kidney tissues were caused by a dietary level of 10 000 $mg \cdot kg^{-1}$ (McCollister and Leng 1969).

Two-year studies with beagle dogs and rats ingesting 150 $mg \cdot kg^{-1}$ did not produce morphological, pathological, or physiological effects (McCollister and Leng 1969). Studies by Dow Chemical Co. (1983) showed a no-observed-adverse-effect level (NOAEL) of 7 $mg \cdot kg^{-1} \cdot d^{-1}$ (body weight) for beagle dogs ingesting picloram for 6 months. The NOAEL is based on increased liver weight. Another NOAEL of 50 $mg \cdot kg^{-1} \cdot d^{-1}$ for CDF Fischer 344 rats was based on liver swelling after 13 weeks. Male and female Fischer 344 rats administered daily oral doses of 200 $mg \cdot kg^{-1}$ body weight for 1 year did not exhibit significant changes in body weight, food consumption, clinical chemistry, or hematological properties. The only treatment effects generally manifested were an increase in the liver-to-body weight ratio and slight hypertrophy and pallor of the centrilobular hepatocytes. The NOAEL for the 1-year study was 20 $mg \cdot kg^{-1} \cdot d^{-1}$ for both male and female rats (Gorzinski et al. 1987).

Short-term NOAELs are 1 to 2 orders of magnitude greater than the long-term NOAELs. A NOAEL of 200 $mg \cdot kg^{-1} \cdot d^{-1}$ was based on the absence of reduced food intake by beagle dogs ingesting picloram for 7–10 d (U.S. EPA 1987).

Dietary concentrations from 100 to 10 000 $mg \cdot kg^{-1}$, increased over a 1-year period, were fed to Japanese quail, Coturnix coturnix, without effect. Calculated LD_{50}s for bobwhite quail, Colinus virginianus, were 23 366 and 10 000 $mg \cdot kg^{-1}$ feed for adults and 5- to 7-d old chicks, respectively.

Uptake, Metabolism, and Elimination

Picloram is easily absorbed from the gastrointestinal tract of mammals (Nolan et al. 1980; Dow Chemical Co. 1983). Feeding studies demonstrate that almost all of the ingested picloram is excreted in the urine (Redemann 1963, 1964; Fisher et al. 1965; McCollister and Leng 1969). Accumulation of low levels of picloram in animal tissues (i.e., 0.5 $mg \cdot kg^{-1}$) occurs at dietary picloram concentrations of 100–200 $mg \cdot kg^{-1}$ (Leasure and Getzander 1964). Picloram is not metabolized significantly by mammals (Redemann 1964; Nolan et al. 1980; Dow Chemical Co. 1983).

Table 2. Picloram Toxicity to Avian Species - Dietary Studies[1]

Species	Acid[2] equivalent ($mg \cdot kg^{-1}$ feed)	Observations
Mallard ducklings (*Anas platyrhynchos*)	500 - 10 000	0% mortality in 5-d feeding, 8-d observation.
	385 200	LC_{50} for 5-d feeding, 8-d observation.
Japaneses quail (*Coturnix coturnix japonica*)	100	14-d exposure, reproduction study. No effect on plumage, feathering, egg production, fertility, hatchability, mortality, or weight.
	1000	14-d exposure, reproduction study. No effect on egg production, body weight or adult mortality. Egg fertility reduced 55%, egg hatchability reduced first week, but not second week of treatment. Hatchability and fertility normal first week after treatment.
	100 - 10 000	Dosage increased over a period of nearly one year. No increased mortality, no decrease in consumption or body weight; no impaired reproductive effect compared to controls.
(5- to 7-d-old chicks)	100; 500; 1000	Reproductive, three-generation study. F_0 generation fed 20 weeks, F_1 generation fed 12 weeks, F_2 generation fed 8 weeks. No statistically significant difference between controls and treatments as measured by food consumption, egg production, fertility, and hatchability, survival and body weight gain. No adverse symptoms noted when medicated diets were withdrawn.

[1]From NRCC (1974).
[2]4-amino-3,5,6-trichloropicolinic acid used in all tests, except where noted.
[3]Isooctyl ester.

Table 2. Continued

Species	Acid[2] equivalent ($mg \cdot kg^{-1}$ feed)	Observations
Bobwhite quail (*Colinus virginianus*)	23 366	LC_{50} for 5-d feeding, 8-d observation.
Bobwhite quail (*Colinus virginianus*) (5- to 7-d-old chicks)	10 000	LC_{50} for 5-d feeding, 8-d observation.
	17 075[3]	LC_{50} for 5-d feeding, 8-d observation.

Three dairy cows receiving dietary levels of 10, 30, and 100 $mg \cdot kg^{-1}$ of picloram in their feed did not have detectable residues of picloram (<0.05 $mg \cdot L^{-1}$) in their milk after 13 d on the diet. Dietary levels of 300 and 1000 $mg \cdot kg^{-1}$ (the latter rate equivalent to 18 $mg \cdot kg^{-1} \cdot d^{-1}$) resulted in mean milk residues of approximately 0.05 and 0.19 $mg \cdot L^{-1}$, respectively, after the same time period. The 0.19-$mg \cdot L^{-1}$ residue level decreased to below detection limits 2-3 d after picloram ingestion ceased (Kutschinski 1969). Beef cattle receiving picloram levels of 200 and 1600 $mg \cdot kg^{-1}$ feed for 3 d exhibited picloram concentrations in the blood of 0.18 and 1.18 $mg \cdot L^{-1}$, respectively. Tissue residues were proportional to dietary levels with 0.32, 1.61, 18.0, and 0.45 $mg \cdot kg^{-1}$ in muscle, liver, kidney, and peritoneal fat, respectively, from the 1600-$mg \cdot kg^{-1}$ feed treatment level. These residues decreased rapidly 3 d after cessation of picloram ingestion (Kutschinski and Riley 1969).

Mutagenicity, Teratogenicity, and Carcinogenicity

The results of several microbial mutagenicity assays indicated that picloram is not mutagenic with or without metabolic activation (Andersen et al. 1972; Torracca et al. 1976; Carere et al. 1978). There appears to be only one report (Ercegovich and Rashid 1977) that considered picloram a weak microbial mutagen. The absence of cytological changes in bone marrow cells in laboratory rats supports the suggestion of the nonmutagenic nature of picloram (Mensik et al. 1976).

A three-generation (two litters per generation) fertility, reproduction, lactation, and teratology study concluded that 75 $mg \cdot kg^{-1} \cdot d^{-1}$ (body weight) Tordon^{R} (95% ai) was not teratogenic in rats. Picloram reduced fertility at 75 $mg \cdot kg^{-1} \cdot d^{-1}$, with a NOAEL for this effect at 25 $mg \cdot kg^{-1} \cdot d^{-1}$. Effects on lactation or other reproductive responses were not observed (McCollister et al. 1967). A teratogenic study with 500-, 750-, and 1000-$mg \cdot kg^{-1} \cdot d^{-1}$ treatments on days 6 to 15 of gestation found evidence of retarded fetal growth, but this did not occur in a dose-related manner (Thompson et al. 1972). Oral ingestion of picloram at 400 $mg \cdot kg^{-1} \cdot d^{-1}$ as the potassium salt by pregnant New Zealand white rabbits failed to produce embryotoxic or teratogenic effects (Mullison 1985).

An initial study of the carcinogenicity of picloram to rats and mice (NCI 1978) indicated that picloram induced benign liver tumors in rats. Subsequent review of the study by the National Toxicology Program questioned the findings (U.S. EPA 1987). Retesting by the Dow Chemical Co. (1986) established the absence of an oncogenic effect. The original NCI (1978) mouse study also failed to find treatment-related carcinogenic responses.

Guideline

Derivation of a recommended guideline for picloram in livestock watering supplies presumes the protection of the most sensitive species (CCREM 1987). Long-term picloram ingestion studies that used typical livestock species were not found. Under these circumstances, the derivation of a guideline for livestock watering supplies necessitated the implementation of the CCREM (1987) procedure to use the guideline for pesticides in raw water for drinking water supply as the guideline for livestock watering. This procedure is used "as a means of providing a margin of safety for livestock and preventing unacceptable residues in animal products" (CCREM 1987). As an interim guideline for picloram in raw water for drinking water supply is available (190 $\mu g \cdot L^{-1}$), this value is adopted as the interim guideline for livestock watering.

Irrigation

Toxicity to Nontarget Plant Species

The toxicity of picloram to plants is enhanced by its mobility and resistance to plant metabolic degradation. Picloram is easily absorbed by roots or foliage and readily transported by means of phloem throughout the plant, eventually accumulating at the growing regions (Foy 1976).

A number of important crop species are highly sensitive to picloram (Davis and Ingebo 1973). Concentrations of picloram reported in runoff water are, under certain circumstances, sufficient to injure the growth of sensitive plants (e.g., black valentine beans) for as long as 4 months after application (Trichell et al. 1968). Investigations of the effect of low concentrations of picloram on crop species as would be found in runoff water downstream from a picloram-treated watershed, have been conducted (Baur et al. 1970). Significant reductions in soybean dry weight were found at soil concentrations of 1.0 $\mu g \cdot kg^{-1}$. A concentration of 0.25 $\mu g \cdot kg^{-1}$ in the soil produced observable damage to sunflowers (Baur et al. 1970). It has been concluded that picloram residues of 10 $\mu g \cdot L^{-1}$ or greater could significantly affect the growth of some crop seedlings (Bovey and Scifres 1971).

Guideline

The main use of picloram in Canada appears to be for brush control along utility and transport rights-of-way. Use along irrigation canals or on fields in irrigated areas appears limited. Given the extreme sensitivity of some crops to picloram, however, the value of 10 $\mu g \cdot L^{-1}$, as suggested by Bovey and Scifres (1971) to be the lower limit for toxic effects, may be too high. Reports exist that 1-4 $\mu g \cdot L^{-1}$ picloram in irrigation water injured tomato and field bean crops near Kimball, Nebraska (Mullison 1985). Concentrations of 0.05 and 0.4 $\mu g \cdot L^{-1}$ were also reported to have injured plants in West Virginia (Mullison 1985). Frank et al. (1979) noted that 0.08 $\mu g \cdot L^{-1}$ affected tobacco seedlings in southern Ontario. Until these reports are confirmed and supported by other no-observed-effect data, a guideline for picloram in irrigation water cannot be derived.

Recreational Water Quality and Aesthetics

Organoleptic Effects

Information related to the concentration of picloram in water that causes a taste or odour was not found in the published literature. The low volatility of picloram and its formulations makes it unlikely that small concentrations (i.e., 10 $\mu g \cdot L^{-1}$) could cause water to have an odour.

Guideline

At present, there is no evidence to indicate that recreational water quality and aesthetics would be adversely affected by picloram residues when this herbicide is used according to label instructions. Thus, a water quality guideline has not been determined for recreation and aesthetics.

Industrial Water Supplies

Guideline

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

SUMMARY

Following an extensive evaluation of the published literature on the herbicide picloram, Canadian water quality

Table 3. Recommended Water Quality Guidelines for Picloram

Uses	Recommended guidelines
Raw water for drinking water supply	190 $\mu g \cdot L^{-1}$ (IMAC)*
Freshwater aquatic life	29 $\mu g \cdot L^{-1}$ (interim guideline)
Agricultural uses	
Livestock watering	190 $\mu g \cdot L^{-1}$ (interim guideline)
Irrigation	No recommended guideline
Recreational water quality and aesthetics	No recommended guideline
Industrial water supplies	No recommended guideline

*Existing interim drinking water guideline (Health and Welfare Canada 1987)

guidelines were derived (Table 3). The background information on picloram, 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 A-1. Continued

Plot description (soil type, crop)	Formulation (% ai)	Application rate	Method of application	Residues in runoff ($\mu g \cdot L^{-1}$) (days posttreatment)		Reference
College Station, Texas; clay loam soil, pH = 7.5; organic matter = 1.5%-2.0% sod consisted of bermudagrass, silver beardgrass, and three-awn	NR (K-salt)	1.12 $kg \cdot ha^{-1}$	spray	2170 27	(1) (120)	Trichell et al. 1968
College Station, Texas; clay loam soil, pH = 7.5; organic matter = 1.5%-2.0% sod consisted of bermudagrass, silver beardgrass, and three-awn; plowed to depth of 1.6-2.4 cm	NR (K-salt)	1.12 $kg \cdot ha^{-1}$	spray	650 15	(1) (120)	Trichell et al. 1968
Power line right-of-way; southern Ontario; podsolized soils with 0-10 cm layer of organic matter over 3-cm ash grey horizon; pH = 3.5-6.0; 25 x 25 m plot	Tordon 101 (10.2)	9.35 $kg \cdot ha^{-1}$	spray	38 28 26	(0) (12) (18)	Suffling et al. 1974
4-ha plot at the Coweeta Hydrological Lab, western North Carolina; soils mainly stony loams, coarse-loamy, mixed, mesic Umbric Dystrochrepts; forest stand was mixture of low quality hardwoods	pellets (10)	5.0 $kg \cdot ha^{-1}$	broadcast	3	(27)	Neary et al. 1985
7.6 x 23 m plots near Carlos, Texas; soil was fine sandy loam; predominant vegetation consisted of yaupon, post oak and blackjack oak	NR	1.12 $kg \cdot ha^{-1}$ (29 April 1969)	spray	26.2 78.9 89.7 88.5 32.3	(2) (4) (6) (8) (9)	Baur, Bovey, and Merkle et al. 1972
		1.12 $kg \cdot ha^{-1}$ (20 May 1969)	spray	10.0 13.8	(13) (15)	
		1.12 $kg \cdot ha^{-1}$ (17 June 1969)	spray	1.0 1.4 2.4 < 1.0 1.9	(30) (67) (71) (88) (124)	

Table A-1. Continued

Plot description (soil type, crop)	Formulation (% ai)	Application rate	Method of application	Residues in runoff ($\mu g \cdot L^{-1}$) (days posttreatment)		Reference
				Surface Water		
Jimmy Lake Weapons Range, Saskatchewan; boreal forest; soil was fine sand mixed with gravel and rock; ground water approximately 15 cm below surface	Tordon 10K pellets (NR)	3.38 $kg \cdot ha^{-1}$ (as active ingredient) on 490 ha	broadcast	0.26	(390)	Waite et al. 1986; Smith et al. 1988
				0.03	(660)	
				1.15	(780)	
				0.39	(1050)	
				Ground water		
				0.14	(390)	
				12.6	(660)	
				438.5	(780)	
				88.3	(1050)	
113-ha pinyon-juniper watershed in central Arizona (Cocoino National Forest); soil was very stony clay, a fine, montmorillonic, mesic Typic Chromusterts	NR	2.8 kg $ae \cdot ha^{-1}$	spray	320	(157)	Johnsen 1980
				260	(158)	
				235	(159)	
				180	(161)	
				200	(178)	
				160	(181)	
				200	(185–186)	
				175	(187–196)	
				98	(202)	
				135	(203)	
				130	(204)	
				94	(205)	
				135	(207)	
				14	(350)	
				10	(356)	
				16	(369)	
				8	(391–402)	
				18	(451)	
				12	(452)	
				16	(455)	
				10	(557–561)	
				11	(564)	
				10	(566)	
				7	(568)	
				2	(745)	
				3	(747)	
				7	(915)	
				< 0.4	(1087)	
				< 0.4	(1089)	

Table A-1. Continued

Plot description (soil type, crop)	Formulation (% ai)	Application rate	Method of application	Residues in runoff ($\mu g \cdot L^{-1}$) (days posttreatment)		Reference
2–2.5–ha watershed in southern Oregon (Boyer Ranch); soils were heavy, dark clay, slightly acidic with 3%–4% organic matter	NR	1.2 $kg \cdot ha^{-1}$ (as picloram)	spray	110	(105)	Norris et al. 1982
				43	(126)	
				64	(130)	
				39	(138)	
				<1	(162–197)	
				12	(202)	
				1	(209)	
				<1	(222)	
5–ha watershed in southern Oregon (Ronk Ranch); soil as above	NR	1.68 $kg \cdot ha^{-1}$	spray	57	(105)	Norris et al. 1982
				71	(127)	
				49	(132)	
				7	(141)	
				6	(147)	
				19	(153)	
				10	(161–165)	
				4	(171)	
				2	(178)	
				3	(186)	
				26	(189)	
				2	(196)	
				<1	(199)	
				<1	(208)	
1.5–ha "downstream" portion of 4.1–ha watershed in Tuskegee National Forest, Alabama; loamy sand soils generally underlain by sandy clay loam; soil pH 4.5–5.5; organic matter <1%; main forest overstory and understory destroyed by fire in 1978; extensive Kudzu growth	pellets (10)	56 $kg \cdot ha^{-1}$	broadcast from air	241	(14)	Michael et al. 1989
				<2	(140)	

Appendix B

Environmental Concentrations of Picloram Residues in Canadian Surface Water and Ground Water

Table B-1. Environmental Concentrations of Picloram Residues in Canadian Surface Water and Ground Water

Location, years, and conditions	Matrix	Concentration (maximum)	Samples with pesticide/N	Reference
Rural wells in southern Ontario	Ground water	32 $\mu g \cdot L^{-1}$	1/5	Frank et. al. 1987
Canadian Forces Base Gagetown, New Brunswick	Ground water	17.1 $\mu g \cdot L^{-1}$	5/12	Franklin 1985
15 Alberta municipalities using surface-water supplies	Treated drinking water	N/A	0/284 (detection limit = 0.01 - 0.3 $\mu g \cdot L^{-1}$)	Hiebsch 1988
13 Alberta municipalities using ground-water supplies	Treated drinking water	N/A	0/26 (detection limit = 0.01 - 0.3 $\mu g \cdot L^{-1}$)	Hiebsch 1988
Metropolitan Toronto, Ont. 1971 - 1982	Raw drinking water	N/A	0/3 (detection limit NR)	Hiebsch 1988
	Treated drinking water	N/A	0/3 (detection limit NR)	Hiebsch 1988
Amherstburg, Ont. (treatment plant)	Raw drinking water	N/A	0/1 (detection limit NR)	Hiebsch 1988
	Treated drinking water	N/A	0/1 (detection limit NR)	Hiebsch 1988
Mitchell's Bay, Ont. (treatment plant)	Raw drinking water	N/A	0/1 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	
	Treated drinking water	N/A	0/1 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	
Stoney Point, Ont. (treatment plant)	Raw drinking water	N/A	0/2 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	Hiebsch 1988
	Treated drinking water	N/A	0/2 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	

N = no. of samples
N/A = not applicable
NR = not reported

Table B-1. Continued

Location, years, and conditions	Matrix	Concentration (maximum)	Samples with pesticide/N	Reference
Wallaceburg, Ont. (treatment plant)	Raw drinking water	N/A	0/2 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	Hiebsch 1988
	Treated drinking water	N/A	0/2 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	
Walpole Island, Ont. (treatment plant)	Raw drinking water	N/A	0/1 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	Hiebsch 1988
	Treated drinking water	N/A	0/1 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	
Windsor, Ont. (treatment plant)	Raw drinking water	N/A	0/1 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	Hiebsch 1988
	Treated drinking water	N/A	0/1 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	
Harrow, Ont. (treatment plant)	Raw drinking water	N/A	0/1 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	Hiebsch 1988
	Treated drinking water	N/A	0/1 (detection limit = 0.1 $\mu g \cdot L^{-1}$)	

Appendix C

Summary of Picloram Acute Toxicity Data for Aquatic Organisms

Table C-1. Summary of Picloram Acute Toxicity Data for Aquatic Organisms[1]

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
VERTEBRATES					
Brook trout (*Salvelinus fontinalis*)	Tordon 22K (K salt)	24 h	LC_{50} = 91 mg·L^{-1} ae	10°C	Kenaga 1969
		96 h	LC_{50} = 91 mg·L^{-1} ae	10°C	
		96 h	No mortality at 69 mg·L^{-1} ae	10°C	
		96 h	LC_{50} = 420 mg·L^{-1}	10°C	Lynn 1965
Brown trout (*Salmo trutta*)	Tordon 22K (K salt)	24 h	LC_{50} = 52 mg·L^{-1} ae	10°C	Kenaga 1969
		96 h	LC_{50} = 52 mg·L^{-1} ae	10°C	
		96 h	No mortality at 22 mg·L^{-1}		
		96 h	LC_{50} = 240 mg·L^{-1}	10°C	Lynn 1965
Coho salmon (*Oncorhynchus kisutch*)	Picloram as acid	24 h	LC_{50} = 29.0 mg·L^{-1} ae	17°C	Kenaga 1969
		48 h	LC_{50} = 25.0 mg·L^{-1} ae	17°C	
		96 h	LC_{50} = 21.0 mg·L^{-1} ae	17°C	
		24 h	LC_{50} = 25.0 mg·L^{-1} ae		
		48 h	100% mortality at 25 mg·L^{-1} ae		
		24 h	35% mortality at 24 mg·L^{-1} ae	17°C	
		48 h	90% mortality at 24 mg·L^{-1} ae		
		24 h	30% mortality at 21 mg·L^{-1} ae	17°C	
		48 h	45% mortality at 21 mg·L^{-1} ae		
Fathead minnow (*Pimephales promelas*)	Tordon 22K (K salt)	24 h	LC_{50} = 52 mg·L^{-1} ae	10°C	Kenaga 1969
		48 h	LC_{50} = 32 mg·L^{-1} ae	10°C	
		72 h	LC_{50} = 32 mg·L^{-1} ae	10°C	
		96 h	LC_{50} = 29 mg·L^{-1} ae	10°C	
		96 h	No mortality at 22 mg·L^{-1}	10°C	
		96 h	LC_{50} = 135 mg·L^{-1}	10°C	Lynn 1965
Rainbow trout (*Salmo gairdneri*)	Picloram as acid	24 h	LC_{50} = 34 mg·L^{-1} ae	13°C	Kenaga 1969
		48 h	LC_{50} = 25 mg·L^{-1} ae	13°C	
		96 h	LC_{50} = 24 mg·L^{-1} ae	13°C	

[1]Based on NRCC 1974 with additional data.
[2]95% confidence limits in parentheses.
Note: TIPA = triisopropanolamine
TEA = triethylamine
Hard = test water hardness as mg·L^{-1} $CaCO_3$
MATC = maximum acceptable toxicant concentration

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Rainbow trout (cont'd)	Picloram as TIPA salt	24 h	LC_{50} = 279 mg·L^{-1} ae	16°C	
		48 h	LC_{50} = 210 mg·L^{-1} ae	16°C	
		72 h	LC_{50} = 210 mg·L^{-1} ae	16°C	
		96 h	LC_{50} = 210 mg·L^{-1} ae	16°C	
	Picloram as TEA salt	24 h	LC_{50} = 43.4 mg·L^{-1} ae	16°C	
		48 h	LC_{50} = 30.2 mg·L^{-1} ae	16°C	
		72 h	LC_{50} = 29.2 mg·L^{-1} ae	16°C	
		96 h	LC_{50} = 29.2 mg·L^{-1} ae	16°C	
	Tordon 22K (K salt)	24 h	LC_{50} = 50 mg·L^{-1} ae	10°C	
		96 h	LC_{50} = 58 mg·L^{-1} ae	10°C	
		96 h	No mortality at 22 mg·L^{-1} ae	10°C	
	Picloram as isooctyl ester	24 h	LC_{50} = 9.6 mg·L^{-1} ae	16°C	
		48 h	LC_{50} = 5.1 mg·L^{-1} ae	16°C	
		72 h	LC_{50} = 3.3 mg·L^{-1} ae	16°C	
		96 h	LC_{50} = 3.1 mg·L^{-1} ae	16°C	
	Tordon 22 K (K salt)	96 h	LC_{50} = 230 mg·L^{-1}	10°C	Lynn 1965
	Picloram as acid	24 h	LC_{50} = 2.5 mg·L^{-1}		FWPCA 1968
Green sunfish (Lepomis cyanellus)	Tordon 22 K (K salt)	24 h	LC_{50} = 91 mg·L^{-1} ae	10°C	Kenaga 1969
		96 h	LC_{50} = 91 mg·L^{-1} ae	10°C	
		96 h	No mortality at 39 mg·L^{-1} ae	10°C	
	Tordon 22 K (K salt)	96 h	LC_{50} = 420 mg·L^{-1}	10°C	Lynn 1965
Largemouth bass (Micropterus salmoides)	Picloram as acid	24 h	LC_{50} = 19.7 mg·L^{-1} ae	24°C	Kenaga 1969
		48 h	LC_{50} = 13.1 mg·L^{-1} ae	24°C	
Black bullhead (Ictalurus melas)	Tordon 22K (K salt)	24 h	LC_{50} = 91 mg·L^{-1} ae	10°C	Kenaga 1969
		96 h	LC_{50} = 91 mg·L^{-1} ae	10°C	
		96 h	No mortality at 69 mg·L^{-1} ae	10°C	
Bluegill (Lepomis macrochirus)	Picloram as acid	24 h	LC_{50} = 26.5 mg·L^{-1} ae	17°C	Kenaga 1969
		48 h	LC_{50} = 22.5 mg·L^{-1} ae	17°C	
		72 h	LC_{50} = 21.0 mg·L^{-1} ae	17°C	
		96 h	LC_{50} = 21.0 mg·L^{-1} ae	17°C	
	Tordon 22K (K salt)	24 h	LC_{50} = 8.2 mg·L^{-1} ae	18°C	
		48 h	LC_{50} = 7.3 mg·L^{-1} ae	18°C	
		96 h	LC_{50} = 5.4 mg·L^{-1} ae	18°C	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Channel catfish (*Ictalurus punctatus*)	Picloram as TEA salt	24 h	LC_{50} = 70.5 mg·L^{-1} ae	27°C	Kenaga 1969
		48 h	LC_{50} = 64.9 mg·L^{-1} ae	27°C	
		72 h	LC_{50} = 52.9 mg·L^{-1} ae	27°C	
		96 h	LC_{50} = 52.9 mg·L^{-1} ae	27°C	
	Picloram as isooctyl ester	24 h	LC_{50} = 2.2 mg·L^{-1} ae	18°C	
		96 h	LC_{50} = 0.9 mg·L^{-1} ae	18°C	
		24 h	LC_{50} = 16.4 mg·L^{-1} ae	27°C	
		48 h	LC_{50} = 15.5 mg·L^{-1} ae	27°C	
		72 h	LC_{50} = 8.9 mg·L^{-1} ae	27°C	
		96 h	LC_{50} = 8.9 mg·L^{-1} ae	27°C	
Goldfish (*Carassius auratus*)	Picloram as acid	24 h	LC_{50} = 27–36 mg·L^{-1} ae	24°C	Kenaga 1969
		48 h	LC_{50} = 21–32 mg·L^{-1} ae	24°C	
		96 h	LC_{50} = 14–32 mg·L^{-1} ae	24°C	
	Picloram as isooctyl ester	48 h	LC_{50} = 27.0 mg·L^{-1} ae	27°C	
		72 h	LC_{50} = 13.5 mg·L^{-1} ae	27°C	
		96 h	LC_{50} = 10.4 mg·L^{-1} ae	27°C	
	Picloram as TEA salt	24 h	LC_{50} = 90.6 mg·L^{-1} ae	27°C	
		48 h	LC_{50} = 61.3 mg·L^{-1} ae	27°C	
		72 h	LC_{50} = 45.5 mg·L^{-1} ae	27°C	
		96 h	LC_{50} = 43.7 mg·L^{-1} ae	27°C	
Emerald shiner (*Notropis atherinoides*)	Tordon 22K (K salt)	24 h	LC_{50} = 34.1 mg·L^{-1} ae	21–26°C	Kenaga 1969
		48 h	LC_{50} = 34.1 mg·L^{-1} ae	21–26°C	
		96 h	LC_{50} = 30.3 mg·L^{-1} ae	21–26°C	
Harlequin fish (*Rasbora heteromorpha*)	Picloram as K salt	24 h	LC_{50} = 34 mg·L^{-1} ae		Alabaster 1969
Cutthroat trout (*Salmo clarki*)	Picloram as acid Tech. grade	96 h	LC_{50} = 6.5 mg·L^{-1} (5.55 to 7.61)	Static test; Hard = 44	Woodward 1976; Mayer and Ellersieck 1986
	(90% ai)	24 h	LC_{50} = 8.3 mg·L^{-1} (5.5 to 12.6)	5°C; pH = 7.4 as above	
	as above	96 h	LC_{50} = 5.0 mg·L^{-1} (4.36 to 5.73)	as above, except 10°C	
		24 h	LC_{50} = 5.5 mg·L^{-1} (5.0 to 6.0)	as above	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Cutthroat trout (cont'd)	as above	96 h	LC_{50} = 4.1 mg·L^{-1} (3.38 to 4.97)	as above, except 15°C	
		24 h	LC_{50} = 4.8 mg·L^{-1} (3.7 to 6.2)	as above	
	as above	96 h	LC_{50} = 8.6 mg·L^{-1} (7.60 to 9.73)	Static test; Hard = 44	
		24 h	LC_{50} = 12.5 mg·L^{-1} (9.0 to 17.4)	10°C; pH = 6.5 as above	
	as above	96 h	LC_{50} = 4.70 mg·L^{-1} (3.94 to 5.60)	as above, except pH = 7.5	
		24 h	LC_{50} = 6.0 mg·L^{-1} (4.3 to 5.8)	as above	
	as above	96 h	LC_{50} = 4.15 mg·L^{-1} (3.38 to 5.10)	as above, except pH = 8.5	
		24 h	LC_{50} = 4.7 mg·L^{-1} (3.9 to 5.6)	as above	
	as above	96 h	LC_{50} = 3.7 mg·L^{-1} (2.89 to 4.74)	Static test; 10°C; pH = 7.8	
		24 h	LC_{50} = 3.7 mg·L^{-1} (2.9 to 4.7)	Hard = 44 as above	
	as above	96 h	LC_{50} = 3.45 mg·L^{-1} (2.97 to 4.00)	as above, except Hard = 170	
		24 h	LC_{50} = 4.4 mg·L^{-1} (4.0 to 4.9)	as above	
	as above	96 h	LC_{50} = 3.45 mg·L^{-1} (2.87 to 4.00)	as above, except Hard = 300	
	as above, except 99%	96 h	LC_{50} = 5.8 mg·L^{-1} (5.0 to 6.7)	Static test; 10°C; pH = 7.4	Mayer and Ellersieck 1986
	as above	96 h	LC_{50} = 4.8 mg·L^{-1} (3.7 to 6.1)	as above, except test solution aged for 7 d	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Cutthroat trout (cont'd)	as above	24 h	LC_{50} = 5.3 mg·L^{-1} (4.1 to 6.8)	as above	
	as above	96 h	LC_{50} = 4.8 mg·L^{-1} (4.1 to 5.6)	as above, except test solution aged for 14 d	
	as above	24 h	LC_{50} = 6.0 mg·L^{-1} (5.4 to 6.7)	as above	
	as above	96 h	LC_{50} = 7.8 mg·L^{-1} (6.2 to 9.8)	as above, except test solution aged for 21 d	
	as above	96 h	LC_{50} = 5.9 mg·L^{-1} (4.8 to 7.3)	as above, except test solution aged for 28 d	
	as above	96 h	LC_{50} = 1.5 mg·L^{-1} (1.2 to 1.8)	Flow-through test; Hard = 162; 10°C; pH = 7.4	
	as above	96 h	LC_{50} = 5.4 mg·L^{-1}	Static test; Hard = 44; 10°C; pH = 7.4	
	as above	24 h	LC_{50} = 6.7 mg·L^{-1} (5.6 to 8.0)	as above	
	as above	24 h	LC_{50} = 3.4 mg·L^{-1} (2.5 to 4.4)	Static test; Hard = 162; 10°C; pH = 7.4	
	as above	96 h	LC_{50} = 5.8 mg·L^{-1} (4.6 to 7.4)	as above	
	as above	24 h	LC_{50} = 4.4 mg·L^{-1} (3.6 to 5.4)	as above	
	as above	96 h	LC_{50} = 4.7 mg·L^{-1} (3.8 to 5.8)	as above, except Hard = 44	
	as above	24 h	LC_{50} = 4.7 mg·L^{-1} (3.8 to 5.8)	as above, except 12°C	
Lake trout (Salvelinus namaycush)	Picloram as acid tech. grade (90% ai)	96 h	LC_{50} = 3.60 mg·L^{-1} (2.98 to 4.35)	Static test; Hard = 44 pH = 7.2, 5°C	Meyer and Ellersieck 1986
		24 h	LC_{50} = 4.6 mg·L^{-1} (3.0 to 7.2)	as above	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Lake trout (cont'd)	as above	96 h	LC_{50} = 4.25 $mg \cdot L^{-1}$ (4.00 to 4.51)	as above, except 10°C	
		24 h	LC_{50} = 4.3 $mg \cdot L^{-1}$ (3.9 to 4.8)	as above	
	as above	96 h	LC_{50} = 2.35 $mg \cdot L^{-1}$ (1.66 to 3.34)	as above, except 15°C	
		24 h	LC_{50} = 2.7 $mg \cdot L^{-1}$ (1.9 to 3.6)	as above	
	as above	96 h	LC_{50} = 4.95 $mg \cdot L^{-1}$ (4.18 to 5.87)	Static test; Hard = 44	
		24 h	LC_{50} = 5.0 $mg \cdot L^{-1}$ (4.2 - 5.9)	as above	
	as above	96 h	LC_{50} = 2.70 $mg \cdot L^{-1}$ (1.82 to 4.00)	as above, except pH = 7.5	
		24 h	LC_{50} = 3.0 $mg \cdot L^{-1}$ (2.2 - 4.1)	as above	
	as above	96 h	LC_{50} = 2.70 $mg \cdot L^{-1}$ (1.82 to 4.00)	as above, except pH = 7.5	
		24 h	LC_{50} = 3.0 $mg \cdot L^{-1}$ (2.2 - 4.1)	as above	
	as above	96 h	LC_{50} = 2.05 $mg \cdot L^{-1}$ (1.55 to 2.71)	as above, except pH = 8.5	
		24 h	LC_{50} = 2.1 $mg \cdot L^{-1}$ (1.6 to 2.7)	as above	
	as above	96 h	LC_{50} = 2.15 $mg \cdot L^{-1}$ (1.6 to 2.9)	Static test; 10°C; pH = 7.8; Hard = 44	
		24 h	LC_{50} = 2.2 $mg \cdot L^{-1}$ (1.6 to 2.9)	as above	
	as above	96 h	LC_{50} = 1.55 $mg \cdot L^{-1}$ (1.18 to 2.03)	as above, except Hard = 160	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Lake trout (cont'd)		24 h	LC_{50} = 1.8 mg·L^{-1} (1.4 to 2.2)	as above	
	as above	96 h	LC_{50} = 2.10 mg·L^{-1} (1.55 to 2.84)	as above, except Hard = 300	
		24 h	LC_{50} = 2.4 mg·L^{-1} (1.9 to 3.1)	as above	
	as above except 99%	96 h	LC_{50} = 3.5 mg·L^{-1} (2.5 to 5.0)	Static test; Hard 44; 10°C; pH = 7.4	
		24 h	LC_{50} = 3.5 mg·L^{-1} (2.5 to 5.0)	as above	
	as above	96 h	LC_{50} = 1.9 mg·L^{-1} (1.6 to 2.1)	Flow-through test; Hard = 162; 10°C; pH = 7.4	
Lake trout (swimup fry)	as above	96 h	LC_{50} = 2.9 mg·L^{-1} (2.4 to 3.4)	as above, except static test	Mayer and Ellersieck 1986
	as above	24 h	LC_{50} = 3.7 mg·L^{-1} (3.3 to 4.2)	as above	
(yolk-sac fry)	as above	96 h	LC_{50} = 16.8 mg·L^{-1} (11.4 to 24.6)	as above	
		24 h	LC_{50} = 16.8 mg·L^{-1} (11.4 to 24.6)	as above	
Rainbow trout (Salmo gairdneri)	as above	96 h	LC_{50} = 4.0 mg·L^{-1} (2.8 to 5.9)	Static test; Hard = 44; 13°C; pH = 7.1	Mayer and Ellersieck 1986
	as above	24 h	LC_{50} = 5.2 mg·L^{-1} (2.8 to 9.5)	as above	
	as above	96 h	LC_{50} = 3.1 mg·L^{-1} (1.7 to 5.6)	as above, except Hard = 272	
	as above	24 h	LC_{50} = 3.1 mg·L^{-1} (1.7 to 5.6)	as above	
	Liquid 24.9% ai	96 h	LC_{50} = 12 mg·L^{-1} (9.8 to 14.7)	Static test; Hard = 40; 12°C; pH = 7.4	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Rainbow trout (cont'd)		24 h	LC_{50} = 12 mg·L^{-1} (9.8 to 14.7)	as above	
	Technical grade 99% ai	96 h	LC_{50} = 10 mg·L^{-1} (7.2 to 13.9)	as above, except 7°C	
	as above	24 h	LC_{50} = 11 mg·L^{-1} (7.6 to 15.8)	as above	
	as above	24 h	LC_{50} = 13.5 mg·L^{-1} (11.1 to 16.5)	as above, except 12°C	
	as above	96 h	LC_{50} = 12 mg·L^{-1} (9.0 to 16.1)	as above, except 17°C	
	as above	96 h	LC_{50} = 13 mg·L^{-1} (10.7 to 15.8)	Static test; Hard = 40; 12°C; pH = 6.5	
	as above	24 h	LC_{50} = 13 mg·L^{-1} (10.7 to 15.8)	as above	
	as above	96 h	LC_{50} = 6.0 mg·L^{-1} (4.3 to 8.3)	as above, except pH = 7.5	
	as above	24 h	LC_{50} = 6.8 mg·L^{-1} (4.9 to 9.4)	as above	
	as above	96 h	LC_{50} = 5.8 mg·L^{-1} (4.3 to 7.8)	as above, except pH = 8.5	
	as above	24 h	LC_{50} = 6.0 mg·L^{-1} (4.1 to 8.7)	as above	
	as above	96 h	LC_{50} = 14 mg·L^{-1} (10.5 to 18.5)	as above, except pH = 7.4	
	as above	24 h	LC_{50} = 16 mg·L^{-1} (10.7 to 23.9)	as above	
	as above	96 h	LC_{50} = 8.0 mg·L^{-1} (6.1 to 10.4)	Static test; Hard = 320; 12°C; pH = 8.0	
	as above	24 h	LC_{50} = 8.7 mg·L^{-1} (6.5 to 11.5)	as above	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Rainbow trout (cont'd) (fingerling)	as above	95 h	LC_{50} = 11.0 mg·L^{-1} (7.4 to 16.4)	Static test; Hard = 40; 12°C; pH = 7.4	
	as above	24 h	LC_{50} = 12.0 mg·L^{-1} (8.7 to 16.6)	as above	
(swimup fry)	as above	96 h	LC_{50} = 8.0 mg·L^{-1} (6.5 to 9.9)	as above, except 12°C	
	as above	24 h	LC_{50} = 17.0 mg·L^{-1} (11.5 to 25)	as above	
(yolk-sac fry)	as above	96 h	LC_{50} = 8.0 mg·L^{-1} (6.0 to 10.5)	as above	
	as above	24 h	LC_{50} = 17.0 mg·L^{-1} (13.3 to 21.6)	as above, except pH = 7.5	
Fathead minnow (Pimephales promelas)	Picloram as acid	96 h	LC_{50} = 55.3 mg·L^{-1} (47.4 to 64.6)	pH = 7.2–8.0; 17.0–17.4°C	Mayes and Dill 1984
	K salt	96 h	LC_{50} = 201 mg·L^{-1} (161 to 288)	pH = 7.5–8.0; 17.0–17.4°C	
	TIPA salt	96 h	LC_{50} = 150 mg·L^{-1} (132 to 176)	pH = 7.3–7.9; 17.5–18.0°C	
Rainbow trout (Salmo gairdneri)	Picloram as acid	96 h	LC_{50} = 19.3 mg·L^{-1} (16.5 to 21.8)	pH = 7.5–8.1; 11.7–12.5°C	Mayes and Dill 1984
	K salt	96 h	LC_{50} = 48 mg·L^{-1} (42 to 54)	pH = 7.5–8.1; 12–13°C	
	TIPA salt	96 h	LC_{50} = 51 mg·L^{-1} (43 to 61)	pH = 6.9–7.6; 12–12.5°C	
Bluegill (Lepomis macrochirus)	Picloram as acid	96 h	LC_{50} = 44.5 mg·L^{-1} (33.9 to 88.2)	pH = 7.4–8.1; 17–17.4°C	Mayes and Dill 1984
	K salt	96 h	LC_{50} = 137 mg·L^{-1} (114 to 166)	pH = 7.7–8.0; 16.5–17.7°C	
	TIPA salt	96 h	LC_{50} = 109 mg·L^{-1} (92 to 132)	pH = 7.7–8.0; 17–17.4°C	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Rainbow trout (Salmo gairdneri) (90 d old)	Picloram (93.8% ai)	96 h	LC_{50} = 15.6 mg·L^{-1} (14.3 to 17.0)	Flow-through test at 3 L·h^{-1}; pH = 7.8–8.5; Hard = 73–83	Mayes et al. 1987
		192 h	LC_{50} = 14.0 mg·L^{-1} (12.5 to 15.8)	NOEL = 6.9 mg·L^{-1}	
Zebrafish (Brachydanio rerio)	Tordon 22K (240 g·L^{-1} as K salt)	96 h	LC_{50} = 35.5 mg·L^{-1}	Flow-through test with average 90% replacement in 8.4 h; 25°C; Hard = 350–375; pH = 8.0–8.3	Fogels and Sprague 1977
Flagfish (Jordanella floridae)	Tordon 22K (240 g·L^{-1} as K salt)	96 h	LC_{50} = 26.1 mg·L^{-1}	Flow-through test with average 90% replacement in 8.6 h; 25°C; Hard = 350–375; pH = 8.0–8.3	Fogels and Sprague 1977
Rainbow trout (Salmo gairdneri)	Tordon 22K (240 g·L^{-1} as K salt)	48 h	LC_{50} = 31.0 mg·L^{-1}	Flow-through test with average 90% replacement 5.6 h; 15°C; Hard = 350–375; pH = 8.0–8.3	Fogels and Sprague 1977
	as above	96 h	LC_{50} = 26.0 mg·L^{-1}	Flow-through test with average 90% replacement 5.6 h; 15°C; Hard = 350–375; pH = 8.0–8.3	
Lake trout (Salvelinus namaycush)	Picloram as acid (90–100% ai)	96 h	LC_{50} = 4.3 mg·L^{-1} (4.0 to 4.5)	Static test; 10°C	Mayer and Ellersieck 1986
		24 h	LC_{50} = 4.3 mg·L^{-1} (3.9 to 4.8)	as above	
Cutthroat trout (Salmo clarki)	as above	96 h	LC_{50} = 4.8 mg·L^{-1} (3.8 to 6.2)	Static test; 12°C; Hard = 44	Mayer and Ellersieck 1986
		24 h	LC_{50} = 5.5 mg·L^{-1} (4.5 to 6.6)	as above	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Channel catfish (Ictalurus punctatus)	as above	96 h	LC_{50} = 6.3 mg·L^{-1} (3.6 to 11.1)	Static test; 18°C	Mayer and Ellersieck 1986.
	as above	96 h	LC_{50} = 15.5 mg·L^{-1} (11.4 to 20.9)	Static test; 22°C Hard = 40; pH = 7.4	
	as above	96 h	LC_{50} = 1.4 mg·L^{-1} (0.7 to 2.5)	Static test; Hard = 44; 18°C; pH = 7.1	
	as above	24 h	LC_{50} = 3.2 mg·L^{-1} (2.5 to 4.1)	as above	
	as above	96 h	LC_{50} = 13 mg·L^{-1} (10.3 to 16.4)	Static test; Hard = 40; 22°C; pH = 7.4	
	as above	24 h	LC_{50} = 14 mg·L^{-1} (10.8 to 18.2)	as above	
	as above	96 h	LC_{50} = 22 mg·L^{-1} (17.0 to 28.0)	as above	
	as above	24 h	LC_{50} = 24 mg·L^{-1} (20.0 to 29.0)	as above	
(swimup fry)	as above	96 h	LC_{50} = 6.8 mg·L^{-1} (3.5 to 13.0)	as above	
(yolk-sac fry)	as above	96 h	LC_{50} = 5.8 mg·L^{-1} (4.6 to 7.2)	as above	
Bluegill (Lepomis macrochirus)	as above	96 h	LC_{50} = 23.0 mg·L^{-1} (17.8 to 29.9)	Static test; 22°C Hard = 40; pH = 7.4	Mayer and Ellersieck 1986.
	K salt	24 h	LC_{50} = 39 mg·L^{-1} (31.0 to 48.0)	Static test; 18°C; Hard = 44; pH = 7.1	
	Technical grade (99% ai)	96 h	LC_{50} = 33 mg·L^{-1} (22 to 49)	Static test; Hard = 40; 12°C; pH = 7.4	
	as above	96 h	LC_{50} = 31 mg·L^{-1} (22 to 43)	as above, except 17°C	
	as above	96 h	LC_{50} = 23 mg·L^{-1} (18 to 30)	as above, except 22°C	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Bluegill (cont'd)	as above	24 h	LC_{50} = 90 mg·L^{-1} (57.0 to 141)	as above	
	as above	96 h	LC_{50} = 20 mg·L^{-1} (14 to 27)	as above, except pH = 6.5	
	as above	24 h	LC_{50} = 92 mg·L^{-1} (58.0 to 146)	as above	
	as above	96 h	LC_{50} = 18 mg·L^{-1} (14 to 25)	Static test; Hard = 40; pH = 7.5	
	as above	24 h	LC_{50} = 30 mg·L^{-1} (23 to 39)	as above	
	as above	96 h	LC_{50} = 19 mg·L^{-1} (13 to 28)	as above, except pH = 8.5	
	as above	24 h	LC_{50} = 32 mg·L^{-1} (19 to 53)	as above	
	as above (99% ai)	96 h	LC_{50} = 26 mg·L^{-1} (21 to 32)	as above, except pH = 7.4	
	as above	24 h	LC_{50} = 68 mg·L^{-1} (52 to 88)	as above	
	as above	96 h	LC_{50} = 13.5 mg·L^{-1} (10.4 to 17.0)	as above, except Hard = 320	
	as above	24 h	LC_{50} = 43 mg·L^{-1} (33 to 56)	as above	
Rainbow trout (Salmo gairdneri)	as above	96 h	LC_{50} = 12.5 mg·L^{-1} (9.5 to 16.5)	Static test; 12°C; Hard = 40; pH = 7.4	Mayer and Elliersieck 1986
	Isooctyl ester (90% ai)	96 h	LC_{50} = 4.0 mg·L^{-1} (2.8 to 5.9)	Static test; 12°C	Johnson and Finley 1980
Channel catfish (Ictalurus punctatus)	as above	96 h	LC_{50} = 1.4 mg·L^{-1} (0.7 to 2.5)	Static test; 18°C; Hard = 44; pH = 7.1	Johnson and Finley 1980
Cutthroat trout (Salmo clarki)	K salt (24.9% ai)	96 h	LC_{50} = 1.5 mg·L^{-1} (0.8 to 3.0)	Static test; 10°C	Johnson and Finley 1980

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Bluegill (Lepomis macrochirus)	as above	96 h	LC_{50} = 26.8 mg·L^{-1} (22.9 to 31.3)	Static test; 18°C Hard = 44; pH = 7.1	Johnson and Finley 1980
	Picloram as acid (90–100 ae)	96 h	LC_{50} = 23.0 mg·L^{-1} (17.8 to 29.9)	22°C ± 1°C; pH = 7.2–7.5; Hard = 40–50	
Cutthroat trout (Salmo clarki) (3 d posthatch)	Picloram as acid (90% ai)	4 periods of 48 h each	Increased fry mortality at GT >1.3 mg·L^{-1}; reduced fry growth at >0.610 mg·L^{-1}; no adverse effect below 0.290 mg·L^{-1}	Flow-through test; picloram slowly added to test water over 48 h then stopped for 5 d, prior to readdition of picloram at 50% of previous concentration	Woodward 1979
Bluegill (Lepomis macrochirus)	Picloram as acid (93.8% ae)	96 h	LC_{50} = 21.9 mg·L^{-1} (18.0 to 27.5)	22°C ± 1°C; pH = 7.4–8.1; Hard = 103; static test	Mayes and Oliver 1985
	Picloram as acid (91.9% ae)	96 h	LC_{50} = 32.9 mg·L^{-1} (23.7 to 58.2)	22°C ± 1°C; pH = 7.9; Hard = 100; static test	
	Picloram as acid (92.7% ae)	96 h	LC_{50} = 19.4 mg·L^{-1} (18.0 to 21.0)	22°C ± 1°C; pH = 7.9; Hard = 100; static test	
	Picloram as acid (92.9% ae)	96 h	LC_{50} = 14.5 mg·L^{-1} (13.7 to 15.3)	24°C ± 1°C; pH = 7.2; Hard = 40–50; static test	
Rainbow trout (Salmo gairdneri)	as above	96 h	LC_{50} = 5.5 mg·L^{-1} (5.2 to 5.8)	13°C ± 1°C; pH = 7.2; Hard = 40–50; static test	Mayes and Oliver 1985
Bluegill (Lepomis macrochirus)	Picloram (K salt: 91% ae)	96 h	LC_{50} = 24 mg·L^{-1}	26°C ± 1°C; pH = 7.2–7.5; Hard = 40–50; static test	Mayes and Oliver 1985
Channel catfish (Ictalurus punctatus)	Picloram (K salt: 91% ae)	96 h	LC_{50} = 14 mg·L^{-1}	26°C ± 1°C; pH = 7.2–7.5; Hard = 40–50; static test	Mayes and Oliver 1985
Rainbow trout (Salmo gairdneri)	K salt (91% ae)	96 h	LC_{50} = 13 mg·L^{-1}	15°C ± 1°C; pH = 7.2–7.5; Hard = 40–50; static test	Mayes and Oliver 1985
Bluegill (Lepomis macrochirus)	TIPA salt (36.9% ae)	96 h	LC_{50} = 80 mg·L^{-1} (74.3 to 85.5)	17°C ± 1°C; pH = 7.2–8.0; Hard = 78; static test	Mayes and Oliver 1985
	TIPA salt (36.9% ae)	96 h	LC_{50} = 79.3 mg·L^{-1} (74.3 to 85.0)	17°C ± 1°C; pH = 7.6–7.8; Hard = 78; static test	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Bluegill (cont'd)	TIPA salt (36.9% ae)	96 h	LC_{50} = 79.3 mg·L^{-1} (79.5 to 95.6)	17°C ± 1°C; pH = 7.6–7.9; Hard = 78; static test	
Fathead minnow (Pimephales promelas)	TIPA salt (36.9% ae)	96 h	LC_{50} = 81.5 mg·L^{-1} (63.2 to 102)	15°C ± 1°C; pH = 7.6–7.9; Hard = 75; static test	Mayes and Oliver 1985
Rainbow trout (Salmo gairdneri)	TIPA salt (98–99% ae)	96 h	LC_{50} = 310 mg·L^{-1} (222 to 518)	12°C ± 1°C; pH = 7.2–7.5; Hard = 40–50; static test	Mayes and Oliver 1985
	TIPA salt (36.9% ae)	96 h	LC_{50} = 21.6 mg·L^{-1} (17.6 to 24.7)	26°C ± 1°C; pH = 7.6–7.8; Hard = 78; static test	
	TIPA salt (36.9% ae)	96 h	LC_{50} = 21.8 mg·L^{-1} (19.3 to 25.4)	15°C ± 1°C; pH = 7.6–7.9; Hard = 78; static test	
Channel catfish (Ictalurus punctatus)	TEA salt (35.3% ae)	96 h	LC_{50} = 74.8 mg·L^{-1} (56 to 100)	26°C ± 1°C; pH = 7.2–7.5; Hard = 40–50; static test	Mayes and Oliver 1985
Rainbow trout (Salmo gairdneri)	TEA salt (35.5% ae)	96 h	LC_{50} = 41.4 mg·L^{-1} (32 to 56)	15°C ± 1°C; pH = 7.2–7.5; Hard = 40–50; static test	Mayes and Oliver 1985
Channel catfish (Ictalurus punctatus)	Isooctyl ester (63.4% ae)	96 h	LC_{50} = 16.5 mg·L^{-1} (13.5 to 19.6)	26°C ± 1°C; pH = 7.2–7.5; Hard = 40–50; static test	Mayes and Oliver 1985
INVERTEBRATES					
Cladoceran (Daphnia magna) (first instar)	Picloram as acid (93.8% ai)	48 h	LC_{50} = 50.7 mg·L^{-1} (44.7 to 57.6)	Static test; 20°C ± 1°C; pH = 7.8–7.9	Mayes and Dill 1984
	K salt (43.5% ai)	48 h	LC_{50} = 212 mg·L^{-1} (180 to 253)	as above	
	TIPA salt (65.2% ai)	48 h	LC_{50} = 125 mg·L^{-1} (111 to 141)	as above	
Stonefly (Pteronarcella bodia) (Nymph)	Picloram as acid (90–100% ai)	96 h	LC_{50} = >10.0 mg·L^{-1}	Static test; 10°C	Johnson and Finley 1980
Stonefly (Pteronarcys californica) (Nymph)	as above	96 h	LC_{50} = 48 mg·L^{-1} (37 to 62)	Static test; 15°C; pH = 7.1; Hard = 44	
	as above	24 h	LC_{50} = 140 mg·L^{-1} (110 to 180)	as above	Mayer and Ellersieck 1986

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Amphipod (Gammarus fasciatus)	as above	96 h	LC_{50} = 27 mg·L^{-1} (20 to 37)	Static test; 21°C; Hard = 44; pH = 7.1	Mayer and Ellersieck 1986
		24 h	LC_{50} = 50 mg·L^{-1} (35 to 71)	as above	
Amphipod (Gammarus pseudolimnaeus)	as above	96 h	LC_{50} = 16.5 mg·L^{-1} (10.8 to 25.3)	Static test; 17°C; pH = 7.0	Mayer and Ellersieck 1986
	as above	24 h	LC_{50} = 20 mg·L^{-1} (15 to 26)	as above	
Cladoceran (Daphnia magna) (first instar)	as above	48 h	EC_{50} = 76 mg·L^{-1} (59 to 97)	Static test; 17°C Hard = 44; pH = 7.4	Mayer and Ellersieck 1986
Amphipod (Gammarus lacustris)	Picloram as acid (90%–100% ai)	96 h	LC_{50} = 27 mg·L^{-1} (20 to 37)		Sanders 1969
Stonefly (Pteronarcys californica) (Nymph)	Picloram as acid (90%–100% ai)	96 h	LC_{50} = 48 mg·L^{-1} (37 to 62)		Sanders and Cope 1968
Cladoceran (Daphnia magna)	Picloram as acid (93.8% ai)	48 h	LC_{50} = 68.3 mg·L^{-1} (63.0 to 75.0)	20°C ± 1°C; pH = 7.2–8.1; Hard = 156	Gersich et al. 1985
Oyster (Crassostrea virginica)	K salt (21.5% ae)	96 h	LC_{50} = >18, <32 mg·L^{-1}	20°C ± C; pH = 8.0 ± 0.5; Salinity = 23 g·L^{-1}; Static test	Mayes and Oliver 1985
	K salt (10% ae)	96 h	LC_{50} = >1000 mg·L^{-1}	20°C ± 1°C; pH = 8.0 ± 0.5; Salinity = 23 g·L^{-1}; Static test	
Pink shrimp (Penaeus duorarum)	K salt (21.5% ae)	96 h	LC_{50} = 125 mg·L^{-1} (114 to 138) Static test	15°C ± 1°C; pH = 8.0 ± 0.5; Salinity = 28 g·L^{-1}	Mayes and Oliver 1985
	K salt (10% ae)	96 h	LC_{50} = 1230 mg·L^{-1} (702 to 2140)	15°C ± 1°C; pH = 8.0 ± 0.5; Salinity = 28 g·L^{-1}	

Table C-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Fiddler crab (*Uca pugilator*)	K salt (21.5% ae)	96 h	LC_{50} = >1000 mg·L^{-1}	8°C ± 0.5°C; pH = 8.0 ± 0.5; Salinity = 20 g·L^{-1}; Static test	Mayes and Oliver 1985.
	K salt (10% ae)	96 h	LC_{50} = >1000 mg·L^{-1}	8°C ± 0.5°C; pH = 8.0 ± 0.5; Salinity = 20 g·L^{-1}; Static test	
ALGAE					
Green alga (*Selenastrum capricornutum*)	Tordon 22K	24 h	EC_{50} = 115 mg·L^{-1} (86.1 to 153)	EC_{50} = 50% decrease in oxygen evolution in 24 h; 24°C	Turbak et al. 1986

Appendix D

Summary of Picloram Chronic Toxicity Data for Aquatic Organisms

Table D-1. Summary of Picloram Chronic Toxicity Data for Aquatic Organisms[1]

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Lake trout (*Salvelinus namaycush*)	Technical grade picloram as acid (90% ai)	70 d (10-d prehatch; 60 d posthatch)	35 $\mu g \cdot L^{-1}$ reduced fry survival and significantly inhibited growth	35 $\mu g \cdot L^{-1}$ concentration not measured in test container; extra-plated from higher concentration in test series	Woodward 1976
Rainbow trout (*salmo gairdneri*) (10-d prehatch embryos)	Picloram (93.8% ai)	70 d (60-d post day-to-mean hatch)	Larval survival significantly reduced at 2.02 $mg \cdot L^{-1}$; length and weight significantly reduced at 0.88 $mg \cdot L^{-1}$; MATC estimated to be 0.70 $mg \cdot L^{-1}$; NOEL = 0.55 $mg \cdot L^{-1}$	11 ± 1°C; pH = 7.4-8.4; Flow-through test at 3 $L \cdot h^{-1}$	Mayes et al. 1987
Zebrafish (*Brachydanio rerio*)	Tordon 22K (240 $g \cdot L^{-1}$ as K salt)	10 d	Threshold LC_{50} = 35.5 $mg \cdot L^{-1}$ (32.7 to 38.5)	Flow-through test with average 90% replacement in 8.4 h; 25°C; Hard = 350-375; pH = 8.0-8.3; no definite acute threshold; no mortality	Fogels and Sprague 1977
Flagfish (*Jordanella floridae*)	Tordon 22K (240 $g \cdot L^{-1}$ as K salt)	10 d	LC_{50} = 12.3 $mg \cdot L^{-1}$ (9.84 to 15.4)	Flow-through test with average 90% replacement in 8.6 h; 25°C; Hard = 350-375; pH = 8.0-8.3	Fogels and Sprague 1977
Rainbow trout (*Salmo gairdneri*)	Tordon 22K (240 $g \cdot L^{-1}$ K salt)	10 d	LC_{50} = 22.2 $mg \cdot L^{-1}$	Flow-through test with average 90% replacement in 5.6 h; 15°C; Hard = 350-375; pH = 8.0-8.3	Fogels and Sprague 1977
Cladocera (*Daphnia magna*)	Picloram as acid (93.8% ai)	21 d	MATC = 14.6 $mg \cdot L^{-1}$	20°C ± 1°C; pH = 7.2-8.1; Hard = 156	Gersich et al. 1985

[1]Based on NRCC 1974 with additional data.
[2]95% confidence limits in parantheses.
Note: MATC = maximum acceptable toxicant concentration
Hard = test water hardness as $mg \cdot L^{-1}$ $CaCO_3$

Table D-1. Continued

Organism	Chemical or formulation	Exposure time	Effects[2]	Comments	Reference
Green alga (*Selenastrum capricornutum*)	Tordon 22K	2-3 weeks	EC_{50} = 44.8 mg·L^{-1} (36.4 to 54.2)	EC_{50} = 50% decrease in cell biomass	Turbak et al. 1986
Green alga (*Chlorella pyrenoidosa*)	Picloram as acid	10-14 d	EC_{50} = >160 mg·L^{-1}	Measured by microplate assay; EC_{50} = 50% reduction in cell counts	Barschers et al. 1988
	Decarboxy picloram	10-14 d	EC_{50} = 8 mg·L^{-1}		
Green alga (*Chlorella vulgaris*)	Picloram as acid	10-14 d	EC_{50} = >160 mg·L^{-1}	Measured by microplate assay; EC_{50} = 50% reduction in cell counts	Barschers et al. 1988
	Decarboxy picloram	10-14 d	EC_{50} = 49 mg·L^{-1}		