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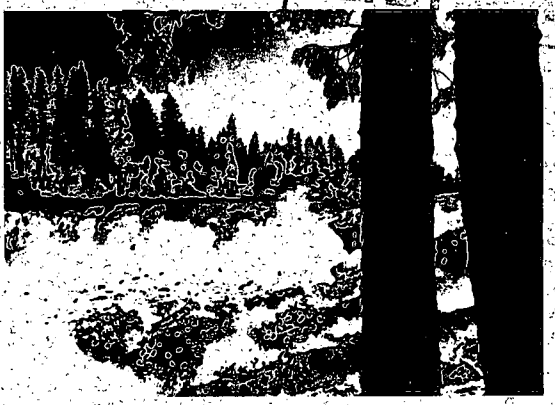

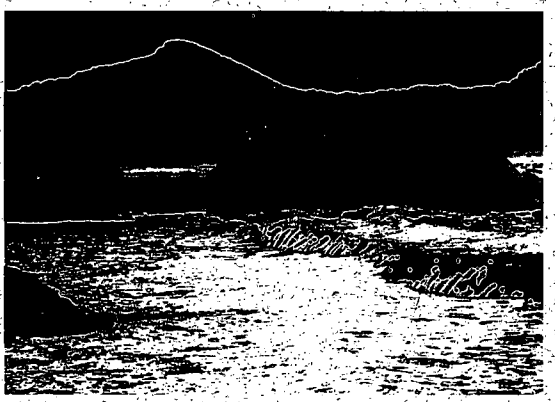

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Abstract

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

Résumé

On a examiné la documentation relative aux utilisations, au devenir et aux effets du triallate 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 lignes directrices sur la qualité de l'eau sont recommandées pour la protection d'utilisations particulières de l'eau.

Canadian Water Quality Guidelines for Triallate

R.A. Kent, M. Taché, P.-Y. Caux, S. De Silva and K. Lemky

INTRODUCTION AND BACKGROUND

Production and Uses

Triallate is the common name for the agricultural herbicide with the Chemical Abstracts Service (CAS) and IUPAC name N,N-diisopropyl-thiocarbamate 2,3,3-trichloroallyl. It is an amber oil with a molecular formula of $C_{10}H_{16}Cl_3NOS$ and a molecular weight of 304.7. The CAS registry number for triallate is 2303-17-5. It is also known as bis(1-methylethyl)-carbamothioic acid or S-(2,3,3-trichloro-2-propenyl) ester. Triallate was introduced into Canada in the early 1960s by Monsanto and is currently marketed under the trade names Avadex BW and Fortress (Agriculture Canada 1990). Triallate is not manufactured in Canada. Canadian import data for triallate are presented in Table 1. At present, three Avadex BW products, consisting of 400 and 480 g•L^{-1} active ingredient (ai) emulsifiable concentrates and a 10% ai granular formulation, are registered in Canada. Avadex granules have recently (September 1990) been registered for a fall surface treatment. (P. Marshall, 1991 Monsanto Canada, Ottawa, pers. com.). In this application, herbicide is spread in the fall just prior to freeze-up, and incorporation is delayed until spring. This treatment is intended for prairie soils that are erosion prone; the removal of a fall tillage operation can drastically decrease erosion vulnerability. A fourth product (Fortress) contains a 4% trifluralin, 10% triallate granular mixture. Triallate is a very popular preemergence herbicide highly effective in controlling certain monocots, particularly wild oats. It is recommended for control of wild oats in barley, durum wheat, spring wheat, and dry peas (Worthing and Walker 1987). It is also recommended for use on canola, flax, sugar beets, and mustard (Agriculture Canada 1982).

Preplant treatments require that triallate be sprayed on the soil surface and worked into the top 5–8 cm of soil with a disk or cultivator. Postplant treatment of cereals requires that triallate be sprayed on the soil surface and worked into the soil by harrowing (the crop must be seeded deep enough to prevent disturbance by harrowing). In both pre- and postplant treatments, triallate should be worked into the soil within 2 h after spraying (OMAF 1989). Normal applications range from 1.12 to 1.68 kg ai•ha^{-1} (Worthing and Walker 1987).

Table 1. Statistics Canada Import Data for Triallate

	1983	1984	1985	1986	1987
Triallate formulated herbicides (tonnes)	19 185	23 980	16 607	11 862	7009
Triallate technical (tonnes)	2 672	3 000	1 560	972	562

Source: Statistics Canada (1986, 1988).
Note: The quantities refer to the mass of the product (i.e., not the active ingredient) and likely include solvents and additives (e.g., surfactants, etc.). Secondary pesticide active ingredients may also be included.

Physical and Chemical Characteristics

The physical and chemical characteristics of triallate are presented in Table 2. The water solubility is reported to be 4 mg•L^{-1}. The structural formula for triallate is presented in Figure 1.

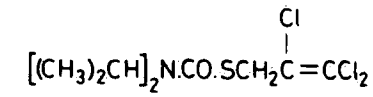

Figure 1. Structural formula for triallate.

Mode of Action

The major phytotoxic effect of triallate is inhibition of cell elongation or expansion. The effect is more pronounced on the stem and leaf meristematic tissue than on root tissue (Banting 1967, 1970; Thiele and Zimdahl 1976). In wild oats (*Avena fatua* L.), 63% mitotic inhibition occurred in stem and leaf meristematic tissues during a 3-d germination period when the plants were exposed to vapour from a 249.85-mg•L^{-1} triallate solution (Banting 1970). Inhibitory effects on elongation were observed at concentrations that did not affect mitosis. Thus inhibition of mitosis appears to be a secondary effect (Banting 1970). The herbicidal action of triallate apparently depends on the diffusion of the vapour phase into the coleoptile, resulting in the suppression of development of the first leaf and interference

Table 2. Physical and Chemical Characteristics of Triallate

Chemical formula	$C_{10}H_{16}Cl_3NOS$[1]
Molecular weight	304.7[1]
Physical state	Amber oil°C[2] (°C not given)
Henry's law constant	1.02 Pa $m^3 mol^{-1}$[3]
Melting point	29–30°C[1]
Boiling point	148–149°C[1]
Vapour pressure	13.3 mPa at 20°C[4] 20.2 mPa at 23°C[4] 16.0 mPa at 25°C[2] 27.5 mPa at 25°C[4] 27.6 mPa at 25°C[1] 44.6 mPa at 30°C[4] 70.4 mPa at 35°C[4] 131.5 mPa at 40°C[4] 266.9 mPa at 45°C[4]
Log octanol/water partition coefficient (K_{ow})	4.6[5]
Log sediment/water distribution coefficient (K_{oc})	3.3[6] 3.45–3.53[7]
Solubility: Water	4 $mg L^{-1}$ at 25°C[1][2]
Half-life in topsoil*	3–88 d[8,9]
Bioconcentration factor	150[5]

Note: Half-life is strongly dependent on soil humidity i.e., 3 days in wet soil (greenhouse)[8], up to 70 d (greenhouse)[8], and 88 d (field study, Regina, Saskatchewan)[9] in dry soil.

[1]U.S. EPA 1983.
[2]Worthing and Walker 1987.
[3]Suntio *et al.* 1988.
[4]Grover *et al.* 1978.
[5]Estimated from Chiou *et al.* 1977.
[6]Kenaga 1980.
[7]Singh *et al.* 1990.
[8]Hance, Holroyd, and McKone 1973.
[9]Grover, Smith, *et al.* 1988.

in the maturation of the cells of the coleoptile (Miller and Nalewaja 1976). Thiocarbamates are known to interfere with lipid formation, resulting in decreased epicuticular wax formation and thinner cuticula, thus increasing leaf wettability and plant susceptibility to foliage–applied herbicides (Hess 1989). These symptoms and the production of necrotic lesions have also been observed by Billet and Ashford (1978). The effects of triallate on the elongation of shoot cells and disruption of wax formation appear to have a common cause in the inhibition of fatty acid synthesis, which reduces cuticular wax formation by inhibiting fatty acid elongation (Bolton and Harwood 1976). Thiocarbamates, for example, EPTC, have been shown to inhibit gibberellic acid synthesis, which eventually affects cell elongation (Wilkinson and Ashley 1979). Triallate, having a similar structure, is expected to act similarly.

Methods of Analysis

McKone and Hance (1967) described an extraction and gas chromatographic (GC) analysis of triallate in soil and vegetable matter that had a detection limit of 0.05 $mg \cdot kg^{-1}$. The extractant used was a mixture of 2,2,4-trimethylpentane and isopropyl alcohol. Vegetable matter extracts required further cleanup techniques (i.e., thin layer or column chromatographic separation), which were not necessary for the soil extracts.

Several authors (Beestman and Deming 1976; Anderson and Domsch 1980a, 1980b; Anderson 1981) used a mixture of benzene and isopropanol (2:1, v/v) to extract triallate from soil. Benzene alone was used to extract triallate from water. Detection limits were not reported by these authors.

A second group of researchers (Smith 1970, 1979; Jury *et al.* 1980; Smith and Hayden 1982a, 1982b; Smith and Milward 1985) extracted triallate from soil samples using 30% aqueous acetonitrile containing 2.5%–3.0% glacial acetic acid. The extract was subsequently partitioned into n-hexane prior to GC analysis. Detection limits were not reported by these researchers.

Extraction of triallate from water was described by Muir and Grift (1987). Adjustment of pH to 2.0 with HCl was followed by extraction with dichloromethane. Final water removal was accomplished by passing the extract through an anhydrous sodium sulphate column. Florisil column cleanup was followed by ethyl acetate/hexane elution. The triallate detection limit for the GC/MS method was 3 $ng \cdot L^{-1}$. Triallate was extracted from surface water samples, sediment, fish, and macrophytes using diethyl ether and analyzed by GC with electron capture detector (ECD) (Therrien-Richards and Williamson 1987). They reported an analytical detection limit of 0.10 $\mu g \cdot L^{-1}$ for water samples and 2.7 $ng \cdot g^{-1}$ for sediment, fish, and macrophytes. Environment Canada's National Water Quality Laboratory uses a gas-liquid chromatography method with ECD for the analysis of triallate in surface waters. A detection limit of 0.01 $\mu g \cdot L^{-1}$ was reported for river water samples (Environment Canada 1984).

Entry into the Environment

Triallate has the potential to leave the site of application and enter the nontarget environment by direct volatilization and subsequent atmospheric transport mechanisms, surface water runoff, and soil adsorption.

Concentrations in Atmosphere

A soil-applied herbicide such as triallate with a relatively high vapour pressure has a great potential for evaporation or volatilization (Grover 1983). Atmospheric concentrations as high as 198 $ng \cdot m^{-3}$ have been recorded in Regina and Melfort, Saskatchewan, where triallate is extensively used in the surrounding area (Grover, Kerr, *et al.* 1988). The seasonal occurrence of triallate in the air generally follows the seasonal use patterns for this herbicide. Soil moisture conditions and rainfall events, however, greatly influence the occurrence and concentration of triallate in the air (Grover, Kerr, *et al.* 1988). Reported maximum concentrations of triallate (200 $ng \cdot m^{-3}$) in Saskatchewan occurred during the spray season of May 1978 when the soil was relatively wet (Grover 1983). During the summer, when the soil was dry, or following freezing of the soil in the fall, airborne residues of triallate were less than 10 $ng \cdot m^{-3}$.

In the field, vapour losses are influenced by the nature of the target, atmospheric turbulence, and soil moisture (Grover 1983). Volatilization losses of triallate are increased when it is applied as an emulsifiable concentrate as opposed to the granular formulation (Hance, Holroyd, and McKone 1973; Smith and Hayden 1981), when the soil is moist (Beestman and Deming 1976; Hance, Holroyd, and McKone 1973; Smith 1983), and when the compound is not soil incorporated (Worthing and Walker 1987).

Concentrations in Water, Sediment, and Biota

A summary of triallate concentrations in Canadian surface water and biota is presented in Appendix A. Snowmelt runoff from fields treated the previous fall may be a significant factor in the presence of triallate in surface waters on the Canadian prairies. Support for this comes from the positive linear correlation ($r^2 = 0.713$) between the flow rates of the La Salle River (southern Manitoba) in the spring and the observed concentrations of triallate. When the river flow increased in June, the same correlation could not be found (Williamson 1984). In southern Saskatchewan, triallate entry into surface waters in spring runoff was also observed by Waite *et al.* (1986). They reported 0.47 and 0.64 $\mu g \cdot L^{-1}$ on March 27 and 28, 1984, respectively, in the runoff from 648 ha in the South Saskatchewan River basin. In 1985 and in 1987, on an agricultural watershed north of Regina, Waite *et al.* (1990) recorded maximum levels of 0.62 $\mu g \cdot L^{-1}$ and 0.98 $\mu g \cdot L^{-1}$, respectively. The presence of low triallate concentrations in rivers during spring and fall application periods suggests that transport and deposition of triallate vapours and triallate adsorbed to dust particles may be the cause of low concentrations of triallate in surface waters not contaminated by surface runoff (Muir and Grift 1987). This is further supported by a study in a Saskatchewan watershed by Grover, Kerr, *et al.* (1988), which showed that aerial transport is a significant path of herbicide input to surface waters.

In a shallow groundwater study, in the Outlook Irrigation District, Saskatchewan, Maathuis *et al.* (1988) recorded triallate concentrations in piezometers with ranges between 0.13 and 0.39 $\mu g \cdot L^{-1}$ and between 0.13 and 0.15 $\mu g \cdot L^{-1}$. These high concentrations of triallate could not be explained because triallate had not been applied in the region in the past few years. During a monitoring survey for triallate in the La Salle River in August–December 1984, triallate was not detected in the water column in an area where it was heavily used. The limit of detection was 0.10 $\mu g \cdot L^{-1}$. Triallate, however, was found in the river sediments at concentrations ranging from 16.9 to 119 $\mu g \cdot kg^{-1}$ (Therrien-Richards and Williamson 1987). Triallate is strongly adsorbed to soil particles. As a result, another major transport pathway from treated fields is by soil erosion via surface runoff and atmospheric suspension. Reports of triallate concentrations in edge-of-field runoff are relatively few. Triallate concentrations in runoff water would be expected to be reduced by soil incorporation through a reduction in the amount available for runoff loss. Triallate concentrations in rivers such as the Ochre, Turtle, La Salle, and Assiniboine in Manitoba, which drain areas where the herbicide is used, ranged from 3 $ng \cdot L^{-1}$ to 150 $ng \cdot L^{-1}$ (Muir and Grift 1987; Williamson 1984). Triallate concentrations ranging from 1.58 to 6.77 $\mu g \cdot L^{-1}$ were detected in spring runoff and snowmelt in Saskatchewan (Grover, Kerr, *et al.* 1988). In a long-term field experiment in Saskatchewan, triallate concentrations in irrigation tailwaters were reported to be 1.8 $\mu g \cdot L^{-1}$. The concentration of triallate in the drainage canal, which carried all tailwaters and return irrigation flows from the basin, however, was <0.1 $\mu g \cdot L^{-1}$ following the first irrigation event after triallate application (Cessna and Grover 1982).

Small forage fish collected from the La Salle River, Manitoba, were found to contain triallate.

Sufficient numbers of individual species (whole body samples) were composited to produce 100-g samples. Maximum triallate concentrations in brown bullhead (*Ictalurus nebulosus*), brook stickleback (*Culaea inconstans*), and the central mudminnow (*Umbra limi*) were reported to be 4.2, 3.3, and 9.2 $\mu g \cdot kg^{-1}$, respectively (Therrien-Richards and Williamson 1987). These data, together with the lack of detectable residues in aquatic macrophytes (*Myriophyllum* sp.) in the La Salle River, further support the rapid selective partitioning to sediment phases and subsequent incorporation into sediment-associated biota.

The U.S. national water quality monitoring data base, STORET, did not contain monitoring data for triallate (U.S. EPA 1983).

Environmental Fate, Persistence, and Degradation

Soil

Processes such as adsorption, leaching, chemical and biological degradation, volatilization, and photodecomposition (influenced by environmental conditions including soil temperature, moisture, and composition) affect the rate of triallate loss from soils (Smith 1970). Of these factors, soil adsorption, microbial degradation, and volatilization appear to be the most important to triallate dissipation (Smith 1970; Anderson 1981; Grover, Smith *et al.* 1988), with adsorption affecting the amount of triallate available in the soil solution for degradation and volatilization.

Persistence in Soil

Reported values of triallate persistence in soil are quite variable depending on the environmental conditions (see Appendix B). The 6-month carry-over of triallate residues from spring and fall applications for various locations in Saskatchewan is reported to range from 3% to 75% of the initial application (Smith 1970, 1971, 1975, 1979; Smith and Hayden 1976, 1982a, 1982b; Cessna *et al.* 1988; Grover, Smith, *et al.* 1988). The upper values of this range generally correspond to fall–spring carry-over rates, while the lower values typically represent spring–fall carry-over.

Half-life values for triallate persistence, obtained from various laboratory, greenhouse, and field studies, ranged from 3 to 88 d (Banting 1967; Smith 1969; Hance, Holroyd, and McKone 1973; Anderson 1981; Grover, Smith, *et al.* 1988). The lower portion of this range represents surface applications without incorporation on wet soil. Thorough incorporation of the herbicide into the soil typically produces half-lives in the upper portion of this range indicating the importance of volatilization to triallate dissipation.

In a soil at Oxford, England, to which triallate was applied and soil incorporated in the spring at a rate of 1.68 $kg \cdot ha^{-1}$, residues could not be detected at the end of the growing season. However, the domestic oat (*Avena sativa*) bioassay method that was used to detect triallate presence (detection limit of about 0.1 $mg \cdot kg^{-1}$) was concluded to be too imprecise and insensitive for persistence studies (Fryer and Kirkland 1970). Studies at the same location found little evidence for the accumulation of triallate in soils even after repeated applications and at rates above normal; plots treated twice annually for 6 years at 3.3 $kg \cdot ha^{-1}$ were found to contain 5.50 $kg \cdot ha^{-1}$ after the final application, but only 0.62 $kg \cdot ha^{-1}$ a year later and 0.09 $kg \cdot ha^{-1}$ (2% of initial value of 5.5 $kg \cdot ha^{-1}$) 3.5 years later (Fryer and Kirkland 1970; Fryer, Smith, and Hance 1980).

In Saskatchewan, climatic conditions are typically represented by long, cold winters and hot, dry summers. Triallate residues recovered from the top 5 cm of field plots in May 1972 represented 54% (for a sandy loam soil), 75% (for a heavy clay soil), and 75% (for a silty clay soil) of the initial 1.7-$kg \cdot ha^{-1}$ treatment applied the previous October. Comparable values for May 1973 were 37% (sandy loam), 23% (heavy clay), and 43% (silty clay) from an October 1972 application. Actual residue concentrations were not reported (Smith 1975).

Residues recovered in October 1972 following a May 1972 application of 1.7 $kg \cdot ha^{-1}$ were 14% (sandy loam), 18% (heavy clay), and 35% (silty clay). Comparable values for October 1973 were 10% (sandy loam), 11% (heavy clay), and 3% (silty clay) of a May 1973 application. Actual residue concentrations were not reported (Smith and Hayden 1976). Comparisons of percent triallate soil residues between fall–spring carry-overs and spring–fall carry-overs indicate that these were generally higher on the silty clay. Further, the fall–spring carry-over exhibits greater residue recoveries. Smith (1975) attributes this finding to triallate volatilization and biological degradation mechanisms being more significant over the spring–fall period.

The soils of the subarctic interior of Alaska are frozen for 6 months or longer each year; greater persistence is expected as a result of these colder conditions. In this region, average triallate carry-over of 54%, 36%, and 14% was reported after 1, 2, and 3 years (Conn and Cameron 1988). The average annual carry-over did not vary greatly, despite different initial spring application rates (0.7, 1.4, or 2.8 kg•ha-1).

Although the application rate of triallate is reported to have no effect on persistence (Conn and Cameron 1988), Banting (1967) found an increase in application rate from 0.56 to 1.12 $kg \cdot ha^{-1}$ corresponded to a half-life increase from 49 to 66 d in a laboratory study. In another laboratory study, persistence was very similar for triallate soil application rates ranging from 0.25 to 1.0 $mg \cdot kg^{-1}$. The absolute amounts of triallate dissipating from the soil were greater as the application rate increased from 5.0 to 50.0 $mg \cdot kg^{-1}$ (Anderson and Domsch 1980b).

There appears to be a relationship between the organic matter content of a soil and triallate persistence, however, the nature of this relationship is not clear. Various investigators report the following: (1) an increase in persistence with increasing soil organic matter from approximately 3.2% to 11.7% (Smith 1975, 1979); (2) no clear trend in triallate persistence over a range of 3.2%–10.6% organic matter (Smith 1971); (3) decreasing persistence with an increase in organic matter from 4.2% to 6.5% (Smith and Fitzpatrick 1970); and (4) little difference in the persistence of triallate among different soil types (Smith 1969). Persistence was reported to be greater in organic soils than in light-textured soils (Smith 1983).

The discrepancy in triallate persistence data in relation to soil organic matter may be due to variations in soil moisture and temperature (Smith and Hayden 1982a). Increased soil moisture and temperature result in a decrease in persistence (Smith 1970; Hance, Holroyd, and McKone 1973; Smith and Hayden 1976, 1982a, 1982b; Anderson 1981; Conn and Cameron 1988; Grover, Smith, *et al.* 1988), probably due to increased volatilization and/or biodegradation. Increased soil aeration, soil moisture content, and temperature also contribute to reducing the persistence of carbamate herbicides as a group by providing conditions conducive to increased microbial activity (Kaufman 1967). A decrease in triallate persistence is associated with both an increase in the biomass of soil microorganisms (Anderson 1981, 1984) and amendment of soils with glucose or a carbohydrate mixture (Anderson 1984). Triallate persistence in soil may also be due in part to its adsorption onto microbial cell walls (Cullimore and Smith 1972). Under controlled laboratory conditions, triallate adsorption on different adsorbents showed that triallate has a greater affinity for organic adsorbents (peat moss, straw wheat) than for inorganic adsorbents (clay). Triallate bound to montmorillonite is more easily desorbed with water than from peat moss, suggesting that weak physical forces (Van der Waals) are involved in the compound's binding to montmorillonite (Grover 1974). Leaching of triallate is shown to be higher in soils with high clay and low organic content than in soils with low clay and high organic content (Smith 1969).

Although the soil persistence of a number of herbicides may be affected when used in combination with other chemicals (Hurle and Walker 1980), several studies have shown that herbicidal combinations with triallate have little or no effect on the persistence of the compound. Anderson and Domsch (1980a) found triallate persistence to be reduced by the addition of chlorpyrifos to soils, but various combinations of other pesticides did not affect carry-over. As well, the addition of trifluralin (Smith 1979) and chloramben (Smith and Hayden 1982b) to soils had little effect on the persistence of triallate.

The availability of triallate in soil to various dissipation and degradation mechanisms also affects its persistence. The formation of bound or unextractable soil residues is an important process controlling the availability of triallate (Anderson 1981), but little information is available describing the formation or structure of these bound residues. In view of the information related to decreased dissipation of diallate with increased adsorption, triallate appears to be unavailable for short-term (i.e., hours) phytotoxic or biodegradation reactions when adsorption and bound residue formation are prominent processes. Over longer terms (i.e., months), however, these bound residues are apparently susceptible to biodegradation (Anderson and Domsch 1980b).

Triallate persistence in soil is greatly influenced by the formulation with which it is applied. Granular formulations of triallate are reported to be more persistent than the emulsifiable concentrates be-

cause of their slower release into the environment and their incorporation in soils (Hance, Holroyd, and McKone 1973; Smith and Hayden 1981; Qureshi 1987).

Dissipation

Three distinct phases of triallate dissipation in Canadian soils are described by Grover, Smith, *et al.* (1988) as (1) an initial rapid phase with volatilization as the major means of dissipation after application and incorporation, followed by (2) a slow and continuous dissipation over the entire growing season with volatilization and microbial degradation as the major pathways of dissipation, and (3) little or no dissipation in winter. Initial rapid volatilization losses, followed by slow dissipation, is congruent with field and laboratory investigations of triallate (Smith 1970, 1971; Anderson and Domsch 1980b; Jury *et al.* 1980; Cessna *et al.* 1988).

The reported rate kinetics values for triallate are quite variable. First-order kinetics were described for triallate soil dissipation by Banting (1967) and Smith and Milward (1985). Banting (1967), however, found a lag period in triallate dissipation of 28 and 45 d, which depended on the application rate between the time of triallate application and the onset of breakdown. The influence this lag period may have on the half-life range of 3–88 d generally attributed to triallate was not discussed.

The gross dissipation of triallate for the entire growing season, although described earlier by Grover, Smith, *et al.* (1988) as occurring in two distinct phases, was reported to follow first-order kinetics. Because triallate is lost from soil by three different routes (i.e., volatilization, biodegradation, and bound residue formation), a rate of loss between first- and second-order kinetics is considered nonetheless to be more representative than first-order kinetics (Anderson and Domsch 1980b).

Volatilization—Volatilization of triallate is considered to be the initial dominant route of soil dissipation from treated areas (Smith 1979, 1983; Grover 1983; Grover, Kerr, *et al.* 1988, Grover, Smith, *et al.* 1988). Since triallate is a very volatile substance, it must be incorporated into the soil shortly after application (Smith 1969, 1970; Cullimore and Smith 1972). Volatilization of triallate from deep incorporations is less than that from shallower incorporations (Smith 1983). In areas where triallate is used extensively, airborne residues (measured by using an air sampling train [tube, chamber, flow meter, and pump] with polyurethane foam as the adsorbent material) can be detected throughout the growing season (Grover 1983). However, over long periods and after soil incorporation, volatilization losses are considerably less than those due to biodegradation and bound residue formation (Anderson 1981, 1984; Anderson and Domsch 1980b). Extensive adsorption has been reported to substantially reduce losses due to volatilization (Smith 1970). Volatilization of preemergence, soil-incorporated herbicides is a function of vapour pressure, but under field conditions, loss due to volatilization is governed by (1) the rate of herbicide desorption from soil (adsorption/ desorption potential), (2) movement to the soil surface (diffusion and mass flow potential), (3) the rate of volatilization at the soil surface (vaporization potential), and (4) the rate of vapour movement away from the surface (atmospheric turbulence potential) (Jury *et al.* 1980; Grover 1983). In addition to soil-adsorbed and solution-phase triallate, the gaseous phase of the herbicide can also move to the soil surface by diffusion (Jury *et al.* 1980).

Under field conditions, maximum triallate vapour concentrations were typically found during peak application periods in May when soil moisture conditions were relatively high. During relatively dry springs, airborne residues were lower than those measured following summer rainfall events (Grover 1983; Cessna *et al.* 1988; Grover, Kerr, *et al.* 1988; Grover, Smith, *et al.* 1988). Although soil water was reported to have little influence on volatilization rates in closed systems without air exchange (Anderson 1981), several other investigators have reported increased triallate volatilization with increased soil moisture (Hance, Holroyd, and McKone 1973; Miller and Nalewaja 1976; Smith and Hayden 1982a; Grover 1983; Smith 1983; Cessna *et al.* 1988). For instance, appreciable volatilization losses were not found from dry soils kept in the laboratory at 50°C for 28 d (Smith 1970). Triallate volatilization losses were suggested to be minimal during summer months on the Canadian prairies where the top 5 cm of soil are often dry even though soil temperatures of 50°C and higher have been recorded.

Water is thought to displace triallate from soil adsorption sites as soil moisture levels increase beyond that necessary to produce a monolayer around the soil particles (Hance, Holroyd, and McKone 1973; Miller and Nalewaja 1976; Menzer and Nelson 1980). Triallate in the liquid phase moves upward

primarily by convection when evaporation occurs at the soil surface (Jury *et al.* 1980; Grover, Smith, *et al.* 1988). The mass flow of thiocarbamates to the soil surface has been referred to as the "wick effect" (Menzer and Nelson 1980), which is the capillary action of water flowing upward against gravity. Both a gas phase and a liquid phase (by convection) are contributing to the upward movement of water as evaporation from the surface occurs. Convection is the mechanism whereby triallate is resupplied at the surface soil layer as it is lost by diffusion to the air (Jury *et al.* 1980).

Volatilization of triallate from soils decreased with increasing organic matter content (Beestman and Deming 1976; Miller and Nalewaja 1976), which may reflect a higher adsorption in these soils (Hance, Holroyd, and McKone 1973). Similar triallate volatilization losses from two soils of different organic matter contents (1.24% and 5.1%), however, have also been reported under laboratory conditions (Jury *et al.* 1980). The higher adsorptive capacity of the more organic soil was thought to be offset by its lower bulk density and higher porosity, which resulted in a higher triallate diffusion coefficient (Jury *et al.* 1980).

Both the formulation and application rate affect triallate volatilization, with the volatilization rate decreasing from the emulsifiable concentrate to the unformulated technical grade triallate to the granular formulation (Hance, Holroyd, and McKone 1973; Miller and Nalewaja 1976; Smith and Hayden 1981). Volatilization increases with increasing application rate (Hance, Holroyd, and McKone 1973; Anderson and Domsch 1980b).

Under conditions favouring triallate volatilization from soils, maximum rates of loss are typically reached soon after application, followed by a rapid decrease, which is likely associated with a quick loss of the herbicide near the soil surface (Jury *et al.* 1980).

A volatilization loss equal to 17.6% of the amount of triallate applied was reported for a single growing season in southern Saskatchewan. Approximately 50% of the volatilization loss occurred during the first 4–5 d following application, with the subsequent vapour flux from the soil decreasing with time over the growing season (Grover, Smith, *et al.* 1988).

Jury *et al.* (1990) recently evaluated the volatilization of organic chemicals residing below the soil surface. Their model was designed as a screening tool to assess the volatilization potential of compounds under standard soil and environmental conditions. They found the soil cover thickness required to restrict volatilization to less than 0.7% of the triallate mass incorporated in soil was 3.6 cm for a sandy soil and 1.5 cm for a clay soil.

The vapour flux of triallate from a glass surface was successfully predicted using a mathematical model based on triallate vapour pressure and molecular weight (Grover *et al.* 1978). The average volatilization rate from glass plates was 5.71 $\mu g \cdot cm^{-2} \cdot h^{-1}$ at 25°C during a 4- to 6-h period. This value may be equalled or exceeded under field conditions when adsorptive processes are not operating in moist soils and air exchange rates are high. Since in practice triallate is incorporated into the soil, however, it is difficult to assess the exact relationship between volatilization rates from the nonadsorbing surfaces in this study to those expected in the field where adsorption is important (Grover *et al.* 1978). Another field study in Saskatchewan demonstrated a maximum volatilization rate of 0.04 $\mu g \cdot cm^{-2} \cdot h^{-1}$ during the 4–6 h following application of 1.5 $kg \cdot ha^{-1}$ triallate as an emulsifiable concentrate to a heavy clay soil (air temperature of 14.4°C) (Grover, Smith, *et al.* 1988).

Microbial Degradation—While volatilization is initially important, the breakdown of triallate by soil microorganisms is the most important factor affecting the dissipation of the herbicide from agricultural soils in the long term (Smith 1969, 1970; Anderson and Domsch 1980a, 1980b; Smith and Hayden 1981, 1982a; Anderson 1984; Smith and Milward 1985). This is particularly true when triallate is incorporated into the soil (Banting 1967; Kaufman 1967).

Most temperate agricultural soils contain microorganisms and/or systems of cell-free enzymes that can degrade triallate (Anderson and Domsch 1980b). The overall rate of metabolism of herbicides in soils is a function of (1) the amount of herbicide in the soil and its distribution, (2) the amount of the enzymatic material in the soil (both within and outside the microbial cells) and its distribution, and (3) the activity level of the enzymatic degradation systems. The herbicide bioavailability is influenced by variables such as soil moisture, temperature, aeration, pH, nutrient status, and organic content. Rates of triallate metabolism are expected to change

temporarily with changes in these factors. Thus, a linear relationship between microbial biomass and triallate degradation has not always been supported by the available data (Anderson 1981, 1984). Also, as the total amount of triallate in the soil decreases with time, the availability of the herbicide to the degradation systems is reduced and the rate of degradation declines.

For most herbicides, the pool of enzymatic material that accounts for the biodegradation potential usually requires no induction period for the initiation of biodegradation (Anderson and Domsch 1976). An exception is triallate; Banting (1967) reported a lag period for the initiation of triallate biodegradation.

Very little information is available concerning the metabolic pathways and metabolites of triallate degradation in soil. In a series of laboratory investigations, the major products of triallate degradation were reported to be CO_2 and soil-bound residues, the formation of which was related to the water content of the soil (Anderson and Domsch 1980a). Almost without exception, the quantity of the unextractable residues was initially greater than CO_2 production. Over longer periods of time, CO_2 production was found to increase relative to the unextractable residues as would be expected as the residues were biodegraded. In addition, degradation products also included traces of water- and benzene-soluble metabolites (Anderson and Domsch 1980b; Anderson 1981).

Climatic factors have been reported to strongly affect the degradation of triallate in soils (Heinonen-Tanski *et al.* 1985), with warm soil temperatures being more conducive to the breakdown process than cold soil temperatures (Smith 1970; Conn and Cameron 1988). Increasing soil moisture also appears to increase triallate breakdown. Soil moisture not only acts as a solvent making herbicides available for degradation, but also influences microbial biomass in the soil (Anderson 1981, 1984). Degradation appears to be retarded as soil moisture falls below field capacity (McKercher and Thangudu 1982); moisture levels in excess of the wilting point are considered to be required for effective microbial degradation (Smith 1970, 1971). During the summer months, soils of the Canadian prairies typically have moisture levels well below field capacity, and thus microbial activity and, consequently, triallate degradation are expected to be low (Smith 1969). In flooded soils, persistence of triallate suggests that anaerobic conditions are unfavourable for microbial degradation (McKercher and Thangudu 1982).

A review of the microbial breakdown of the general category of thiocarbamates failed to provide information concerning triallate degradation, but suggested the possible metabolic processes affecting this family of herbicides (Kaufman 1967). The possible sites of metabolic attack on the thiocarbamate molecule are the alkyl groups, the amide linkage, or the ester linkage. The initial site of attack is determined by the nature of the alkyl groups attached to the amide linkage; in the presence of relatively small alkyl groups at the ester linkage, the thiocarbamate molecule is likely to be hydrolyzed at the ester linkage. Triallate in aqueous solution, however, has been found to be resistent to hydrolysis over a pH range of 4–8. Only a maximum of 15% of the herbicide was degraded in this manner over 24 weeks (Smith 1969).

Mobility and Leaching—Adsorption of triallate to soil clay and organic matter combined with the low water solubility of the herbicide are considered important factors contributing to the low leachability of triallate in soils. The mobility or leaching of triallate in field soils can be expected to be minimal due to its strong adsorption to soils (Smith 1971; Grover, Banting, and Morse 1979; Grover 1983). This is supported by observations of negligible triallate residue movement beyond the depth of soil incorporation (Fryer and Kirkland 1970; Smith 1970, 1971, 1975; Fryer, Smith, and Hance 1980; Smith and Hayden 1982a, 1982b). Approximately 96% of the applied granular triallate remained in the upper 0–1 cm of laboratory soil columns after 15.2 cm of simulated rainfall was applied at 2.5 $cm \cdot h^{-1}$ (Beestman and Deming 1976). The addition of an emulsifier to the granules enhanced triallate movement through the soil; four times more triallate was moved beyond 1 cm, but 95% of it was concentrated in the upper 3 cm of the soil. In a similar experiment, only 5%–13% of the triallate applied to two soil types (Regina heavy clay and Weyburn loam) was eluted from columns with 23 cm of simulated rainfall (Smith 1969). Since the annual summer rainfall on the Canadian prairies is normally less than 25.4 cm, it is thought that excessive leaching of triallate in the field is unlikely (Smith 1969).

The extent of adsorption of a substance to various soils is often described by the Freundlich equation, $X/M = kC^n$, where X is the mass of

adsorbed solute, M is the adsorbent (sediment or soil) mass, C is the equilibrium concentration, and k (the adsorption coefficient) and n are estimated from the linear regression of log X/M vs. log C (Grover, Banting, and Morse 1979). X/M has units of soil-adsorbed concentration ($\mu g \cdot g^{-1}$) (B.T. Bowman, 1990, Agriculture Canada, London, Ont., pers. com.). The adsorption coefficients for triallate on various soils from England and Saskatchewan have been reported to range from 23 to 150 $\mu g^{(1-n)} \cdot mL^{-n} \cdot g^{-1}$ (concentration range at equilibrium 4–30 $\mu g \cdot L^{-1}$ soil and 0.03–0.9 $\mu g \cdot mL^{-1}$ solution, n ranging from 0.96 to 0.98) (Hance, Holroyd, and McKone 1973; Grover, Banting, and Morse 1979). Triallate is strongly adsorbed to soil colloids, and this may be the most important factor regulating its availability in soil. Between 79%–96% of the original amount of triallate in aqueous solutions, ranging in concentrations from 0.5 to 3 $mg \cdot L^{-1}$, was adsorbed by several Saskatchewan soils. As well, the soil solution concentrations of triallate at equilibrium were well below its solubility in water (Grover, Banting, and Morse 1979).

The structure of triallate (Fig. 1) supports the suggestion that adsorption will be by nonionic interactions (Grover, Banting, and Morse 1979). Thus, pH has little effect on adsorption of triallate to soils (Grover 1974). A report of triallate adsorption increasing with decreasing pH was attributed to the strong inverse relationship between organic matter content and pH of soils (Grover, Banting and Morse 1979).

Triallate is strongly adsorbed on hydrophobic, organic adsorbents, such as activated charcoal, peat moss, and cellulose, and is negligibly desorbed by water. Wheat straw, which is a mixture of cellulose, hemi-cellulose, lignin, and proteins, also exhibits strong adsorption of triallate coupled with minimal desorption by water (Grover 1974). Triallate mobility in soils, in contrast to volatization, is not substantially affected by emulsifying agents used in some triallate formulations.

Organic matter content appears to be one of the most important factors governing the adsorption of triallate in soils. A positive relationship between soil organic matter and adsorption of triallate has been found by various investigators (Smith 1970; Hance, Holroyd, and McKone 1973; Beestman and Deming 1976; Jury *et al.* 1980). Organic matter content is highly correlated ($r = 0.97$) with the triallate adsorption coefficients for several Saskatchewan soils and is considered to be the most important factor affecting the behaviour of triallate in these soils (Grover, Banting, and Morse 1979).

Khan (1973) studied the nature of a triallate-montmorillonite complex and showed that triallate adsorption onto clay is by complexation of the triallate carbonyl group to the exchangeable cations on the clay. The triallate-montmorillonite complex was stable even on heating to 50°C under dry conditions, but when shaken with distilled water, it was completely displaced from the clay (Khan 1973). The affinity of triallate for clay explains its higher persistence in clay-enriched soils at field capacity moisture levels (Smith and Fitzpatrick 1970).

Photodecomposition—The dissipation of triallate from soil occurring as a result of photodecomposition does not appear important (WSSA 1983). The ultraviolet absorption spectrum of triallate does not indicate absorption at wavelengths greater than 280 nm. Since the spectrum of solar radiation at the earth's surface has a minimum wavelength of about 290 nm, photodecomposition is not expected to be a determining component in the dissipation of triallate from the soil (Beestman and Deming 1976). Minimal losses of triallate from photodecomposition were reported by Grover, Banting, and Morse (1979).

Water and Sediment

Compared to soil studies, information related to the fate and persistence of triallate in the aquatic environment is scarce. Although triallate might react with available free radicals and be subjected to photochemical reactions, specific data supporting this hypothesis were not found (U.S. EPA 1983). Based on the previously discussed work of Smith (1969), who found low (10%–15%, pH 4–8) hydrolyzation values, this mode of action for triallate dissipation is not expected to be a significant degradation factor in the aquatic environment.

Studies of triallate biodegradation in water or sediments were not found. Retention of triallate in flooded soils suggests that anaerobic conditions in sediments are not favourable for microbial degradation (McKercher and Thangudu 1982).

The measured half-life of triallate in aquatic systems is available from only one study. Monsanto Company (1987) measured the half-life of triallate in water to range between 3 and 15 days under various

laboratory conditions. A major portion of the loss, however, was due to the volatilization. More details of this study were not provided (P. Marshall, 1991, Monsanto Canada, Ottawa, pers. com.). A Henry's law constant has been estimated by Suntio *et al.* (1988) at 1.02 Pa $m^3 \cdot mol^{-1}$. Volatilization from water may or may not be significant depending on the rates of competitive processes (Suntio *et al.* 1988). The half-life of triallate in water due to volatilization has been estimated to be "several days" (U.S. EPA 1983). This estimate was based on the known vapour pressure and water solubility of triallate and data for the volatilization from water of the closely related herbicide diallate. Muir (in press) has predicted that triallate will volatilize rapidly from shallow waters based on its high transfer coefficient and has estimated that the half-life for volatilization from water of 1-m depth (20°C) would be 8 d.

The strong adsorption of triallate from aqueous solution onto soil particles (95% to a Regina heavy clay and Weyburn loam) (Smith and Fitzpatrick 1970) indicates that adsorption onto particulate material in the aquatic environment is a major fate process. The sediment detections reported by Therrien-Richards and Williamson (1987) in the La Salle River in Manitoba (16.9–119 $ng \cdot g^{-1}$, Appendix A) support this assumption.

RATIONALE

Raw Water for Drinking Water Supply

Guideline

The Federal–Provincial Subcommittee on Drinking Water has recommended a maximum acceptable concentration of 230 $\mu g \cdot L^{-1}$ as the Canadian drinking water quality guideline for triallate (Health and Welfare Canada 1989).

Concentrations in Drinking Water

Published measurements of triallate in treated (municipal and private) water in Canada were not found (Hiebsch 1988).

Freshwater Aquatic Life

Bioaccumulation

Published studies on the experimental bioaccumulation of triallate in aquatic animals were not found in the scientific literature. However, several unpublished studies provide preliminary bioaccumulation data. Monsanto Company (1982) found that the daily bioconcentration factors during the exposure phase ranged from 210 to 574 for channel catfish *(Ictalurus punctatus)*, and from 282 to 778 for bluegill sunfish (*Lepomis macrochirus)*. In both cases, rapid elimination occurred within 2 weeks during the depuration period. Environment Canada (1990) has a preliminary report on the bioconcentration potential of triallate in rainbow trout (*Oncorhynchus mykiss*). In this study, trout were exposed to triallate at a mean measured concentration of 0.14 $\mu g \cdot L^{-1}$ in a continuous flow system; steady state body burdens of 0.069 $\mu g \cdot L^{-1}$ were achieved after 3 d of exposure; and BCFs of 789 to 838 were generated by the static model (mean fish concentration divided by mean water concentration). An estimated bioconcentration factor of 150 was published by Kenaga (1980) based on equations developed by Kenaga and Goring (1980). Using the equations published by Chiou *et al.* (1977), a log octanol/water partition coefficient of 4.6 can be calculated. This value would seem to suggest a higher bioconcentration factor than 150. Triallate, however, is known to be easily metabolized and excreted by terrestrial animals (Khokhol'kova and Pestova 1969; Zhavoronkov, Polyakova, and Verkhovskii 1972; Marsden and Casida 1982). The same would be expected of aquatic animals, thus limiting an organism's ability to retain (i.e., bioaccumulate) triallate.

Although triallate could not be detected in the water of the La Salle River, Manitoba, with a detection limit of 0.10 $\mu g \cdot L^{-1}$, it was detected in three species of forage fish (brown bullhead, brook stickleback, and central mudminnow). The tissue concentrations ranged from 3.3 to 9.2 $ng \cdot g^{-1}$, with a detection limit of 2.7 $ng \cdot g^{-1}$ (Therrien-Richards and Williamson 1987). If the detection limit for triallate in water (0.10 $\mu g \cdot L^{-1}$) is used with the maximum tissue concentration reported (9.2 $ng \cdot g^{-1}$), a bioaccumulation factor of 92 results. Water concentrations below 0.10 $\mu g \cdot L^{-1}$ would produce higher bioaccumulation factors, which could be similar to the value of 150 as predicted by Kenaga (1980).

At four sampling locations in the La Salle River, Therrrien-Richards and Williamson (1987) found no bioaccumulation of triallate in an aquatic macrophyte *Myriophyllum* sp. (detection limit 2.7 $ng \cdot g^{-1}$).

Toxicity to Aquatic Organisms

Acute Lethal Toxicity

Vertebrate acute toxicity data for technical triallate (95.3% ai) consists of 24-h LC_{50}s of 1300 $\mu g \cdot L^{-1}$ for rainbow trout (*Oncorhynchus mykiss*) and 2500 $\mu g \cdot L^{-1}$ for channel catfish (*Ictalurus punctatus*). The 96-h LC_{50}s are 620 and 1700 $\mu g \cdot L^{-1}$ for the respective species. Tests conducted with the formulated emulsifiable concentrate (46.3% ai) produced 24-h LC_{50}s of 1300 and 1800 $\mu g \cdot L^{-1}$ for rainbow trout and channel catfish, respectively. The 96-h LC_{50}s are 1000 and 1100 $\mu g \cdot L^{-1}$ for the respective species (Mayer and Ellersieck 1986).

Invertebrate aquatic organisms were considerably more sensitive to triallate. Invertebrate acute toxicity testing using technical triallate produced 48-h LC_{50}s of 80 $\mu g \cdot L^{-1}$ for first instar *Daphnia magna* (Mayer and Ellersieck 1986) and a 48-h EC_{50} of 2300 $\mu g \cdot L^{-1}$ for fourth instar *Chironomus riparius* (Buhl and Faerber 1989). A 96-h test using third instar *Chironomus plumosus* produced an LC_{50} of 490 $\mu g \cdot L^{-1}$ (Mayer and Ellersieck 1986).

Acute toxicity of the emulsifiable concentrate formulation ranged from a 48-h LC_{50} of 57 $\mu g \cdot L^{-1}$ for first instar *D. magna* (Mayer and Ellersieck 1986) to an EC_{50} of 1230 $\mu g \cdot L^{-1}$ for *C. riparius* (Buhl and Faerber 1989). A summary of the limited acute toxicity data of triallate to aquatic vertebrates and invertebrates is presented in Appendix C. A solvent carrier was not used in the development of toxicity data by Mayer and Ellersieck (1986). An acetone solvent carrier was used in one of the controls to simulate the formulation additive in the tests conducted by Buhl and Faerber (1989). They found that immobilization and mortality in the untreated control and solvent control did not exceed 10% in any of the tests.

Chronic Toxicity and Sublethal Reactions

Fathead minnow (*Pimephales promelas*) larvae used in 7-d survival and growth tests with triallate demonstrated a sharp dose–response relationship. Mortality was not observed at 202 $\mu g \cdot L^{-1}$, but 100% mortality occurred at 531 $\mu g \cdot L^{-1}$. A 7-d LC_{50} of 330 $\mu g \cdot L^{-1}$ was estimated from the data. Fathead minnow growth (based on the dry weight of fry) was reduced (33%) at 202 $\mu g \cdot L^{-1}$ (lowest-observed-effect concentration), but not at 125 $\mu g \cdot L^{-1}$ (no-observed-effect concentration) producing an estimated MATC (maximum acceptable toxic concentration) of 160 $\mu g \cdot L^{-1}$ (Environment Canada 1989).

As with acute lethality data, the limited chronic data also indicate the greater sensitivity of aquatic invertebrates when compared to vertebrates. Standard 7-d survival and reproduction bioassays conducted using *Ceriodaphnia dubia* produced a more gradual dose–response relationship with mortalities observed over almost the entire exposure range (0.35–531 $\mu g \cdot L^{-1}$). The 7-d LC_{50} was 12 $\mu g \cdot L^{-1}$. Reproduction (measured as the daily production of young) was reduced (by 59%) at a concentration of 2.4 $\mu g \cdot L^{-1}$ (the lowest-observed-effect concentration), but not at 1.3 $\mu g \cdot L^{-1}$. The resulting estimated MATC was calculated to be 1.8 $\mu g \cdot L^{-1}$ (Environment Canada 1989).

Aquatic Plants

Information related to the acute toxicity of triallate to aquatic plants is also scarce. An algal bioassay of 18–36 h duration resulted in less than 50% inhibition of chlorophyll production in *Chlorella pyrenoidosa* when tested with 1000- and 10 000-$\mu g \cdot L^{-1}$ triallate concentrations (Kratky and Warren 1971). More specific data were not generated by these authors. The 10 000-$\mu g \cdot L^{-1}$ triallate solution was produced with either an acetone or methanol solvent carrier; the report did not specify which solvent was used.

Algal bioassays of 2–3 weeks duration using *Selenastrum capricornutum* and the commercial triallate formulation Far-Go (10% ai), in either a natural water or the standard synthetic algal growth medium, were conducted by Turbak, Olson, and McFeters (1986). The EC_{50}, based on algal cell numbers, was 6.20 $\mu g \cdot L^{-1}$ for natural water and 11.2 $\mu g \cdot L^{-1}$ for the synthetic algal growth medium. The upper and lower confidence intervals for both EC_{50}s varied by an order of magnitude and overlapped to such an extent that the two EC_{50}s were not significantly different.

Aquatic Community Studies

In the only community study with triallate found, laboratory microcosms simulating northern prairie wetlands were used. Triallate was introduced as a soil slurry to obtain nominal solution concentrations of 10, 100, and 1000 $\mu g \cdot L^{-1}$ (Johnson 1986). Each 4-L glass microcosm contained 3.8 L of water and sediment from a permanent wetland (hydrosoil) at a

ratio of 9:1 (v/v). After the introduction of the triallate, the microcosms were placed in an environmental chamber (20°C, 1400 lux on a 16-h light, 8-h dark cycle) for a week prior to the introduction of naturally derived macrophytes (*Lemna*, *Ceratophyllum*, and *Elodea*). Natural communities of invertebrates and algae developed in each microcosm.

Prior to triallate additions, 25 mature, gravid daphnids (*Daphnia magna*) were introduced into each microcosm. If, at any time, five or fewer daphnids were observed in a microcosm, an additional 25 daphnids were introduced in an attempt to produce a viable population. Acute toxicity tests (48 h) using the waters recovered from the control and triallate-treated microcosms were conducted at 14 and 30 d posttreatment using first instar *Daphnia magna* and fourth instar *Chironomus riparius*. Forty-eight–hour *D. magna* acute toxicity tests, using microcosm waters at 14 d posttreatment, showed 0%, 60%, and 100% mortality at nominal 10-$\mu g \cdot L^{-1}$, 100-$\mu g \cdot L^{-1}$, and 1000-$\mu g \cdot L^{-1}$ treatments, respectively. Similar tests with chironomids showed that triallate was 100 times more toxic to daphnids than to chironomids. Even after 30 d, water from the 10-$\mu g \cdot L^{-1}$ treatment produced a 50% reduction in the number of adult daphnids surviving a 7-d chronic toxicity test.

This microcosm study demonstrated triallate toxic effects to daphnids and that these effects persisted even at low concentrations. Continued introduction of daphnids was necessary on days 1, 4, 7, 10, and 14 before a viable daphnid population was established in the 10-$\mu g \cdot L^{-1}$ treatment. Daphnid populations could not be established in the 100- and 1000-$\mu g \cdot L^{-1}$ treatments in these time periods. It should be noted that the aqueous concentration of these nominal concentrations is probably much lower, considering that a substantial amount of triallate might be soil-bound. Smith and Fitzpatrick (1970) reported a strong adsorption of triallate from aqueous solution onto soil particles (up to 95%).

The simulation of a drought cycle in the microcosms (i.e., removal of macrophytes, macroinvertebrates, and water, with subsequent replacement of fresh, uncontaminated water and new daphnids) did not change the time required to establish daphnid populations in the 10-$\mu g \cdot L^{-1}$ treatment.

Triallate effects on phytoplankton, as determined by short-term growth bioassays using *Selenastrum capricornutum*, demonstrated that the 100- and 1000-$\mu g \cdot L^{-1}$ treatments reduced algal growth (cell counts) by more than 40% even at 30 d posttreatment. There was no effect on algal growth at 10 $\mu g \cdot L^{-1}$. Aquatic vascular plants were not affected by any treatment. Dissolved oxygen production in microcosms was observed to increase (20% above control levels) at 100 and 1000 $\mu g \cdot L^{-1}$ at days 14, 21, and 28 during the 30-d experiment period. It was implied that this increase was due to a stimulation in photosynthetic productivity in microcosms due to the presence of triallate.

Microbial activity, as measured by respiratory electron transport, glucose metabolism, oxygen consumption, and alkaline phosphatase activity, was not disturbed by the triallate treatments in the microcosm study.

Guideline

The minimum toxicological data requirements for deriving a Canadian water quality guideline (CCME 1991) were not met with the current triallate data base. Derivation of an interim guideline value, however, was possible with the existing data. Mayer and Ellersieck (1986) reported a 48-h median lethal concentration of 57 $\mu g \cdot L^{-1}$ for the invertebrate *Daphnia magna*. A concentration of 2.4 $\mu g \cdot L^{-1}$ triallate was found to affect the reproduction of *Ceriodaphnia dubia*. This was the lowest concentration of triallate found causing a significant effect in an aquatic organism and was subsequently used as the basis for an interim guideline.

Therefore a safety factor of one order of magnitude is appropriate (CCME 1991). The resulting interim guideline is 0.24 $\mu g \cdot L^{-1}$.

Agricultural Uses

Livestock Waters

Toxicity to Livestock and Related Biota

Acute Toxicity—Several Russian studies cited by the U.S. EPA (1983) described the acute oral toxicity of triallate to laboratory and domestic animals. They report single oral dose LD_{50} values of 930 and 1471 $mg \cdot kg^{-1}$ body weight for mice and rats, respectively (Pestova 1968). Single oral dose LD_{50} values of 500 and 945 $mg \cdot kg^{-1}$ were also reported for rabbits and rats, respectively (Verkhovskii 1972). Other reported single oral dose LD_{50}s ranged from 1675–2165 $mg \cdot kg^{-1}$ for rats to >20 000 $mg \cdot kg^{-1}$ for dogs (Wiswesser 1976). The acute oral LD_{50} for the northern bobwhite quail (*Colinus virginianus*) was

reported to be >2251 mg•kg^{-1} body weight. The dietary concentration is >5000 mg•kg^{-1} feed to bobwhite quail and mallard duck (Smith 1987).

Subacute and Chronic Toxicity—Most of the data on triallate subacute and chronic toxicity comes from brief manufacturers' reports and abstracts of Russian research papers. The manufacturer of triallate (Monsanto) reports that dietary concentrations of 10, 30, or 100 mg•kg^{-1} of feed ingested by rats (approximately 0.5, 1.5, or 5 mg•kg^{-1}•d^{-1}) for three generations produced no treatment-related effects. A dietary concentration of 200 mg•kg^{-1} (about 10 mg•kg^{-1}•d^{-1}) produced depressed weight gain in female rats during a 2-yr study. However, neither gross pathological changes nor abnormal hematological indices were observed at this level (Johannsen *et al.* 1977). Detailed supporting data for these claims were not presented.

Abstracts of Russian papers report edema and plethora in the brains of rats that were fed triallate at 14.7 mg•kg^{-1} body weight for 4 months (Rappoport and Pestova 1974). The maximum tolerated single oral dose of 1000 mg•kg^{-1} caused decreased succinic and lactic dehydrogenase activity, decreased hepatic thiol content, and an increased hepatic pyruvic acid level (Pestova 1968). An increase in RNAase activity of the liver and spleen and disruption in normal thyroid gland function were also reported due to a single oral dose of 1000 mg•kg^{-1} (Voitenko *et al.* 1967). Other subacute reactions are the inhibition of acetylcholinesterase activity in the peripheral and central nervous system and decreased osmotic resistance of erythrocytes (Zhavoronkov, Verkhovskii, and Evdokimov 1973). Sheep and pigs administered a single oral dose of 300 mg•kg^{-1} exhibited altered hematological parameters including transient changes in total plasma protein content, increased albumin, decreased globulin, decreased RNA and DNA, and increased free nucleotide levels (Verkhovskii 1972; Verkhovskii, Zhavoronkov, and Evdokimov 1973; Zhavoronkov and Verkhovskii 1975).

Concern about a possible delayed neurotoxic effect of triallate, which has been observed with the similar compound diallate, led to studies using white leghorn hens. Hens given 300 mg•kg^{-1} twice a day for 3 d exhibited mild, transient ataxia and leg weakness at 19 d posttreatment. A similar dosage schedule using 400 mg•kg^{-1} produced moderate ataxia and lethargy at 5 d post-treatment. Recovery from these symptoms occurred in 4 d (Fisher and Metcalf 1983). Doses of 340–420 mg•kg^{-1}•d^{-1} administered to mature white leghorn hens in gelatin capsules for 25 d caused greater than 40% weight loss. The condition of these birds continued to decline until they were sacrificed on day 36. Gross examination of the gastrointestinal tract revealed a few 1- to 2-mm lesions in the gizzard. A dosage of 85–105 mg•kg^{-1}•d^{-1} for 25 d did not cause a decrease in weight or egg production in spite of a transient decrease in food consumption. As well, ataxia and narcosis were not evident (Hansen *et al.* 1985).

Uptake, Metabolism, and Elimination—Triallate is rapidly absorbed from the gastrointestinal tract. Ingested triallate at 1000 or 1471 mg•kg^{-1} appears in the blood 15 min after a single oral dose and attains a maximum level in 30 min (Khokhol'kova and Pestova 1969).

The metabolism of triallate in rats involves the formation of trichloroacrylic acid by the microsomal oxidases. Formation of the trichloroacrylic acid is thought to be via the NADPH-dependent S-methylene hydroxylation of triallate to unstable, highly reactive intermediate trichloroacroleins (Marsden and Casida 1981, 1982). Microsomal incubation of triallate results in the rapid formation and glutathione conjugation of trichloroacrolein (Hackett *et al.* 1990).

Complete elimination from rabbits of single oral doses of 500 mg•kg^{-1} triallate occurred in 7 d (Zhavoronkov, Polyakova, and Verkhovskii 1972). Single oral doses of 1000 or 1471 mg•kg^{-1} were completely eliminated from the bodies of rats in 1–3 d (Khokhol'kova and Pestova 1969).

Carcinogenicity, Mutagenicity and Teratogenicity—Manufacturer testing of triallate, using male and female rats consuming dietary concentrations of 50, 100, and 200 mg•kg^{-1}, did not indicate a tumorogenic response in terms of the number of rats with tumors, the number of tumors per rat, or the number of rats with malignant neoplasms. In addition, there were no gross pathological changes or differences in survival (Johannsen *et al.* 1977). Additional information concerning the carcinogenic potential of triallate was not found.

A large amount of mutagenicity information, obtained using a variety of test systems, is available in the published literature. A compilation and a review of these data were published by Carerer and Morpurgo (1981). Triallate produces a mutagenic

response in the *Salmonella typhimurium* strains TA100 and TA1535, both with and without metabolic activation (De Lorenzo, Silengo, and Cortese 1976; De Lorenzo *et al.* 1978; Carerer, Ortali, Cardamone, and Morpurgo 1978; Carerer, Ortali, Cardamone, Torracca, and Raschetti 1978; Sikka and Florcyzk 1978; Sandhu and Waters 1980; Douglas *et al.* 1981a, 1981b; Kasica, Sanhu, and Waters 1981; Sandhu *et al.* 1981, 1984; Shiau, Huff, and Felkner 1981; Wildeman and Nazar 1982).

Dose-related increases in base substitution and frameshift mutations were noted for triallate in *S. typhimurium* strains TA100, TA1535, and TA98. A positive mutagenic response, however, was not observed in strains TA1537, TA1536, and TA1538. For those strains exhibiting a positive reaction, triallate is considered to be a direct-acting, mutagen-inducing, base pair substitution (U.S. EPA 1983). Triallate also induced forward mutations in *Saccharomyces coelicolor* (Carerer, Ortali, Caramone, and Morpurgo 1978; Carerer, Ortali, Cardamone, Torracca, and Raschetti 1978) and in *Aspergillus nidulans* (Morpurgo *et al.* 1977).

Mutagenic responses were not found for *Escherichia coli* WP2, bacteriophages, and *Saccharomyces cerevisiae* D7 using reverse mutation criteria (Andersen, Leighty, and Takahashi 1972; Kasica, Sandhu, and Waters 1981; Sandhu *et al.* 1981). These authors, however, reported a significant increase in mitotic recombinations in *S. cerevisiae* D3 exposed to triallate with and without metabolic activation.

Triallate was shown to be mutagenic in tests using mammalian cells. Chinese hamster ovary cells exhibited dose-related increases in the frequency of chromosomal aberrations, sister chromatid exchanges, and cytotoxicity indicative of the clastogenic (i.e., breaking) effect that triallate has toward chromosomes (Douglas *et al.* 1981a, 1981b). The L5178Y mouse lymphoma thymidine kinase assay is also positive for triallate mutagenicity (Kasica, Sandhu, and Waters 1981; Sandhu *et al.* 1981). *In vitro* studies showed that triallate metabolism by the microsomal fraction of PCB-induced rat liver homogenate produced a mutagenic substance (Distlerath, Loper, and Tabor 1982, 1985). At a concentration of 100 $mg \cdot L^{-1}$, triallate caused 57% inhibition of DNA synthesis in rat thymocytes, and a 52% inhibition of DNA synthesis and a 5% inhibition of unscheduled DNA synthesis in human lymphocytes (Rocchi *et al.* 1980). The weight of evidence in the scientific literature implies that triallate is a potential mutagen that is capable of acting with or without metabolic activation. Triallate, however, does not demonstrate a positive mutagenic response in all tests (U.S. EPA 1983).

Data pertaining to the teratogenicity of triallate are scarce. A manufacturer's study with rabbits using orally administered doses of 3 and 10 $mg \cdot kg^{-1}$ body weight on days 6–18 of gestation reportedly did not induce teratogenic responses in the offspring (Johannsen *et al.* 1977). Access to experimental data was not possible since the report was written in abstract form.

Guideline

Insufficient data are available for the determination of a safe concentration of triallate in livestock watering supplies. The mammalian toxicity data used to derive the guideline for triallate in drinking water supplies were proprietary and not available for this report. In accordance with the procedure established by the CCREM (1987), the guideline for drinking water supplies (230 $\mu g \cdot L^{-1}$) (Health and Welfare Canada 1989) is used as the interim guideline for livestock watering supplies.

Irrigation Waters

Toxicity to Nontarget Plant Species

Various laboratory and field studies have detailed the toxicity of triallate to nontarget plants, especially the domestic oat (*Avena sativa*). These studies are presented in Appendix D. Sublethal reactions to nontarget plants have been demonstrated by triallate concentrations as low as 1 $mg \cdot L^{-1}$ in an irrigation application (Kratky and Warren 1971) and 0.28 $kg \cdot ha^{-1}$ and 0.11 $mg \cdot kg^{-1}$ as soil applications (McKercher and McGregor 1979). The phytotoxicity of triallate varies and is influenced by a variety of environmental and soil factors. For example, phytotoxicity increases as soil moisture increases. Water appears to compete with triallate for adsorption sites on soil particles and adsorbed triallate may be replaced by water to increase triallate bioavailability. Increased temperature also increases phytotoxicity. This may be due to either reduced triallate adsorption and/or increased herbicidal activity of the available triallate at higher temperatures (Miller and Nalewaja 1976). Soil organic matter is a major determinant of phytotoxicity, with increases in organic matter corresponding to decreases in phytotoxicity (McKercher, Ashford,

and Morgan 1975).

Triallate formulation also influences phytotoxicity, with greater growth inhibition occurring with liquid (i.e., emulsifiable concentrate) formulations than with similar application rates of the granular formulation (Miller and Nalewaja 1976).

Guideline

Various laboratory studies have established that concentrations as low as 1 $mg \cdot L^{-1}$ can cause decreased root and shoot growth in crop species (Kratky and Warren 1971). A definitive dose–response relationship between triallate water concentrations and phytotoxic responses by crop species, however, could not be established from the scientific literature as the concentration range for most of these studies was inadequate. A lowest-observed-effect-application rate (LOEAR) and a no-observed-effect application rate (NOEAR) were not available to derive a species maximum acceptable toxicant concentration (SMATC). Thus, a guideline value for triallate in irrigation water was not derived at this time.

Recreational Water Quality and Aesthetics

Organoleptic Effects

Reports dealing with triallate-caused taste and odour of water and tainting of fish flesh were not found.

Guideline

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

Industrial Water Supplies

Guideline

At present, the CCME lacks the necessary information to set water quality guidelines that will protect industrial water uses from most chemical compounds. A survey of industry water quality needs is being conducted, and upon completion, it should be possible to set guidelines for many chemicals, including triallate, to protect this water use.

SUMMARY

After an evaluation of the published information on the herbicide triallate, water quality guidelines were derived (Table 3). The background information on triallate 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.

Table 3. Recommended Water Quality Guidelines for Triallate

Uses	Guidelines
Raw water for drinking water supply	230 $\mu g \cdot L^{-1}$ (MAC)*
Freshwater aquatic life	0.24 $\mu g \cdot L^{-1}$ (interim)
Agricultural water uses	
Livestock waters	230 $\mu g \cdot L^{-1}$ (interim)
Irrigation waters	No recommended guideline
Recreational water quality and aesthetics	No recommended guideline
Industrial water supplies	No recommended guideline

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

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Appendix A

Environmental Concentration Ranges of Triallate Residues in Canadian Surface Water, Groundwater, Atmosphere, Sediment, and Biota

Table A-1. Environmental Concentration Ranges of Triallate Residues in Canadian Surface Water, Groundwater, Atmosphere, Sediment, and Biota

Location, years, and conditions	Matrix	Concentration range (& mean)	Samples with pesticide/ number of samples	Reference
Saskatchewan, 300 km north of Regina (Melfort) and Regina. Accumulative air samples, 24-h basis. First week of May till early mid-November for the years 1981 and 1982.	Air	1982 <1 $ng \cdot m^3$–160 $ng \cdot m^3$ 1981 <1 $ng \cdot m^3$–25 $ng \cdot m^3$	NR	Grover, Kerr, *et al.* 1988
Saskatchewan, 300 km north of Regina (Melfort) and Regina. Accumulative air samples, 24-h basis. First week of May till early mid-November for the years 1978 and 1979.	Air	1979 <1 $ng \cdot m^3$–104 $ng \cdot m^3$ 1978 <1 $ng \cdot m^3$–198 $ng \cdot m^3$	NR	Grover, Kerr, and Khan 1981
Ochre River, western Manitoba. 3.5-L grab sampling in duplicate on March 14, April 13, April 27, and at weekly intervals afterward until Sept. 5/84. Final collection on Oct. 10/84. Drains mainly noncropped land and forest.	Surface water	Detectable levels (T > 3 $ng \cdot L^{-1}$) found only in October. Avg. for Oct. was 6.4 $ng \cdot L^{-1}$.	NR	Muir and Grift 1987
Turtle River. As above. Drains mainly agricultural land.	Surface water	May - 10.4 $ng \cdot L^{-1}$ June - 9.9 $ng \cdot L^{-1}$ July - 2.7 $ng \cdot L^{-1}$ Sept. - 3.7 $ng \cdot L^{-1}$ Oct. - 5.5 $ng \cdot L^{-1}$ (detection limits = 3 $ng \cdot L^{-1}$)	NR	Muir and Grift 1987
La Salle River, Manitoba. One grab sample per 7 sampling sites at 30-d intervals from Aug. to Dec. 1984 at midstream. Drains agricultural land.	Surface water	ND (detection limit = 0.10 $\mu g \cdot L^{-1}$)	NR	Therrien-Richards and Williamson 1987
Sampling with dredge at 3 equidistant points across stream width at each sampling location on 1 occasion in Aug. 1984 (1 sample per sampling site) in above study area.	Sediment	16.9–119 $ng \cdot g^{-1}$ (detection limit = 2.7 $ng \cdot g^{-1}$)	9/21	Therrien-Richards and Williamson 1987
100 g sampled from each site on 1 occasion Aug. 1984 from 4 sampling locations in above study area.	Aquatic macrophyte *Myriophyllum* sp.	ND (detection limit = 2.7 $ng \cdot g^{-1}$)	NR	Therrien-Richards and Williamson 1987

ND = not detected
NR = not reported
T = trace

Table A-1. Continued

Location, years, and conditions	Matrix	Concentration range (& mean)	Samples with pesticide/ number of samples	Reference
Samples of small forage fish. Samples equal 100 g of each fish from species from 4 sampling sites and 3 sub-samples at 1 site for a total of 6 samples.	Fish tissue: brown bullhead (*Ictalurus nebulosus*),	<2.7–4.2 $ng \cdot g^{-1}$	NR	Thierren-Richards and Williamson 1987
	brook stickleback (*Culaea inconstans*),	3.3 $ng \cdot g^{-1}$	NR	
	central mudminnow (*Umbra limi*)	<2.7–9.2 $ng \cdot g^{-1}$ (detection limit = 2.7 $ng \cdot g^{-1}$)	NR	
LaSalle River, Manitoba. Sampling interval clustered during April 1983 to coincide with snowmelt water runoff and at monthly intervals from May 1983 to March 1984 (excluding Aug. 1983). Drains agricultural land, 2 sampling locations.	Spring runoff water	0.02–0.15 $\mu g \cdot L^{-1}$ (detection limit) = 0.05 $\mu g \cdot L^{-1}$	27/27	Williamson 1984
Assiniboine River, Manitoba. Sampling at monthly intervals from May 1983 to March 1984 (excluding Aug. 1983). Drains agricultural land, 2 sampling locations.	Surface water	(Detection limit = 0.05 $\mu g \cdot L^{-1}$)	7/15	Williamson 1984
April 11, 1983; samples collected on 1 day from 2 water pools.	Surface water	Trace (detectable but <0.05 $\mu g \cdot L^{-1}$)	2/3	Williamson 1984
June 1, 1983; 1 sample collected from 1 pool.		ND (<0.05 $\mu g \cdot L^{-1}$)		
Study area 2800 ha operated by 17 farmers and the City of Regina. Sampling on a daily basis for duration of runoff event at 4 culverts crossing into study area at a stream connecting 2 permanent sloughs and at a culvert exiting the lower slough; 7 sampling locations.	Spring water runoff	0.4678 $\mu g \cdot L^{-1}$ at one site on Mar 27; 0.6443 $\mu g \cdot L^{-1}$ at same site on March 28; below detection limit (0.1 $\mu g \cdot L^{-1}$) at all other sites and times	NR	Waite *et al.* 1986
Assiniboine River, Manitoba (downstream Trans-Canada Highway). One midstream grab sample per site at 30-d intervals from Aug. to Dec. 1984. Drains agricultural land.	Surface water	ND (detection limit = 0.1 $\mu g \cdot L^{-1}$)	NR	Therrien-Richards and Williamson 1987
Sampling by hand of fine-grained deposits on lee side of midstream obstructions (sand bars and rocks) on 1 occasion Aug. 1984. Data reported for only 1 sampling site; study area as above.	Sediment	ND (detection limit = 2.7 $ng \cdot g^{-1}$)	NR	Williamson 1984

Table A-1. Continued

Location, years, and conditions	Matrix	Concentration range (& mean)	Samples with pesticide/ number of samples	Reference
Samples of small forage fish. Samples equal 100 g of each fish species; study area as above.	Fish tissue: silver chub (*Hybopsis storeriana*), stone cat (*Noturus flavus*), channel catfish (*Ictalurus punctatus*), brown bullhead (*Ictalurus nebulosus*)	ND (detection limit = 2.7 $ng \cdot g^{-1}$)	NR	Therrien-Richards and Williamson 1987
Red Deer River, Bindless, Alberta, Emerson, Manitoba, Selkirk, Manitoba, from May 1960 to February 1988.	Surface water	0.1–0.08 $\mu g \cdot L^{-1}$	6/95	NAQUADAT 1991
Souris River, Manitoba, at Coulter to Wawanesca from May 1960 to February 1988.	Surface water	0.01–0.72 $\mu g \cdot L^{-1}$	4/28	NAQUADAT 1991
Qu'Appelle River, Saskatchewan, from November 1975 to December 1987.	Surface water	0.01–0.046 $\mu g \cdot L^{-1}$	2/44	NAQUADAT 1991
Canot River, Saskatchewan, from October 1973 to January 1978	Surface wter	0.028 $\mu g \cdot L^{-1}$	1/45	NAQUADAT 1991
Churchill River, Saskatchewan, from April 1974 to January 1988.	Surface water	0.024 $\mu g \cdot L^{-1}$	1/36	NAQUADAT 1991
Reservoirs receive snowmelt water from a 640-ha study area located 10 km north of Regina. Sampling was done on a weekly basis in 1985 and twice in 1987 from one location in each of 2 reservoirs.	Surface water	0.22 µg/L maximum with a mean of 0.11 µg/L (no range given)	23/64	Waite *et al.* 1990
Sampling in a 2800-ha study area located north of Regina during 2 brief periods of melt separated by a month of cold weather in 1987 from 7 sites in study area.	Spring runoff water	0.98 $\mu g \cdot L^{-1}$ maximum with a mean of 0.38 $\mu g \cdot L^{-1}$ (no range given)	19/22	Waite *et al.* 1990
Sampling on 9 sequential days in 1985 from 6 sites in above study area.	Spring runoff water	0.62 $\mu g \cdot L^{-1}$ maximum with a mean of 0.19 $\mu \cdot L^{-1}$ (no range given)	36/37	Waite *et al.* 1990

Table A-1. Continued

Location, years, and conditions	Matrix	Concentration range (& mean)	Samples with pesticide/ number of samples	Reference
Sampling from 4 locations, 10 km north of Regina in summers, 4 times in 1987 from 4 iron stand pipes installed in a surficial aquifer.	Groundwater	0.63 $\mu g \cdot L^{-1}$ maximum with a mean of 0.15 $\mu g \cdot L^{-1}$ (no range given)	7/105	Waite *et al.* 1990
80-ha study area located in Saskatchewan; groundwater samples from SIDC piezometers in summer of 1987 on 2 separate days.	Groundwater	0.13–0.39 $\mu g \cdot L^{-1}$ range, 0.10 $\mu g \cdot L^{-1}$ detection limit	3/13	Maathuis *et al.* 1988
Study area 11 km^2 located in township 30 in Saskatchewan; water samples taken from 3 piezometers and 2 canals near piezometers on 30 separate days.	Groundwater	0.13–0.15 $\mu g \cdot L^{-1}$ range, 0.1 $\mu g \cdot L^{-1}$ detection limit	5/18	Maathuis *et al.* 1988

Appendix B

Summary of Triallate Persistence Studies in Soil

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

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Begbroke Hill, Yarnton, Oxford. Soil type NR	1.7 kg•ha^{-1} Spring 1969	0–15 cm	1.35 kg•ha^{-1} (0 wks) 0.24 kg•ha^{-1} (22 wks)	These experiments were begun in 1963 when triallate was applied at 1.7 kg•ha^{-1} pre-emergence to wheat and barley.	Fryer, Smith, and Hance 1980
	Spring 1970		1.39 kg•ha^{-1} (0 wks) 0.26 kg•ha^{-1} (18 wks)		
	Spring 1971		1.61 kg•ha^{-1} (0 wks) 0.18 kg•ha^{-1} (22 wks)		
	Spring 1972		1.23 kg•ha^{-1} (0 wks) 0.21 kg•ha^{-1} (23 wks)	Herbicide as soon as possible after sowing and incorporated to 2.5–5 cm.	
	Spring 1973		1.20 kg•ha^{-1} (0 wks) 0.51 kg•ha^{-1} (21 wks)		
	Spring 1974		1.19 kg•ha^{-1} (0 wks) 0.50 kg•ha^{-1} (24 wks)		
	Spring 1975		0.99 kg•ha^{-1} (1 wk) 0.39 kg•ha^{-1} (27 wks)		
	Spring 1976		0.95 kg•ha^{-1} (0 wks) 0.39 kg•ha^{-1} (21 wks)		
	3.3 kg•ha^{-1} (twice annually)	0–15 cm	5.50 kg•ha^{-1} (after final application - Dec. 1968) 1.27 kg•ha^{-1} (6 mo) 0.62 kg•ha^{-1} (12 mo) 0.26 kg•ha^{-1} (18 mo) 0.24 kg•ha^{-1} (21 mo) 0.19 kg•ha^{-1} (34 mo) 0.09 kg•ha^{-1} (40 mo)	Herbicide applied twice annually from 1963 to 1968 to hand-weeded uncropped plots. Incorporation NR.	
Melfort, Sask. Melfort silty clay (11.7 OM, pH 5.2, field capacity 36%)	1.7 kg•ha^{-1} Oct. 1971 Oct. 1972 Oct. 1973 May 1972	0-5 cm	75 ± 3% (7 mo) 43 ± 3% (7 mo) 3 ± 1% (5 mo) 35 ± 3% (5 mo) 25 ± 4% (12 mo) 12 ± 4% (17 mo)	6.8 mg triallate added to 20 x 20 cm plots and thoroughly incorporated into top 5 cm of soil.	Smith 1975

NR = not reported
OM = organic matter

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Regina, Sask. Regina heavy clay (4.2 OM, pH 7.7, field capacity 40%)	1.7 kg•ha^{-1} Oct. 1971 Oct. 1972 May 1973 May 1972	0–5 cm	 75 ± 7% (5 mo) 23 ± 10% (5 mo) 11 ± 2% (5 mo) 18 ± 2% (5 mo) 16 ± 1% (12 mo) 12 ± 3% (17 mo)	Applications and sampling carried out during 3rd wk of October and 2nd wk of May.	Smith 1975
Jameson, Sask. Jameson sandy loam (3.2 OM, pH 7.5, field capacity 11%)	1.7 kg•ha^{-1} Oct. 1971 Oct. 1972 May 1973 May 1972	0–5 cm	 54 ± 6% (7 mo) 37 ± 8% (7 mo) 10 ± 6% (5 mo) 14 ± 3% (5 mo) 7 ± 2% (12 mo) 0 (17 mo)		Smith 1975
Regina, Sask. Heavy clay (4.2 OM, pH 7.7, field capacity 40%)	1.25 kg•ha^{-1} Sept. 1979 Oct. 1979 Nov. 1979 Sept. 1981 Oct. 1981 Nov. 1981	0–5 cm	 53% (8 mo) 64% (7 mo) 50% (6 mo) 22% (8 mo) 22% (7 mo) 23% (6 mo)	5 mg triallate added to 20 x 20 cm plots in triplicate and incorporated to 5 cm.	Smith and Hayden 1982a
White City, Sask. Sandy loam (4.0% OM, pH 7.6, field capacity 20%)	1.25 kg•ha^{-1} Sept. 1979 Oct. 1979 Nov. 1979 Sept. 1980 Nov. 1979 Sept. 1980 Oct. 1980 Nov. 1980 Sept. 1981 Oct. 1981 Nov. 1981	0–5 cm	 56% (8 mo) 62% (7 mo) 61% (6 mo) 23% (8 mo) 61% (6 mo) 23% (8 mo) 27% (7 mo) 29% (6 mo) 23% (8 mo) 20% (7 mo) 21% (6 mo)	Differences in carry-over between years considered to reflect differences in soil moisture and temperature following soil treatment. Applications made during 1st wk of each fall month and soil sampled during 2nd wk of May.	Smith and Hayden 1982a

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Regina, Sask. Heavy clay (physical characteristics NR)	1.7 kg•ha^{-1} May 1972	0–5 cm	18 ± 2% (5 mo) 16 ± 1% (12 mo) 12 ± 3% (17 mo)	7.8 mg triallate applied to 20 x 20 cm plots and incorporated.	Smith and Hayden 1976
	May 1973		11 ± 2% (5 mo) 9 ± 4% (12 mo) 2 ± 1% (17 mo)		
Melfort, Sask. Silty loam (physical characteristics NR)	1.7 kg•ha^{-1} May 1972	0–5 cm	35 ± 3% (5 mo) 25 ± 4% (12 mo) 12 ± 4% (17 mo)		Smith and Hayden 1976
	May 1973		3 ± 1% (5 mo) 5 ± 4% (12 mo) 0 (17 mo)		
Jameson, Sask. Asquith sandy loam (physical characteristics NR)	1.7 kg•ha^{-1} May 1972	0–5 cm	14 ± 3% (5 mo) 7 ± 2% (12 mo) 0 (17 mo)		Smith and Hayden 1976
	May 1973		10 ± 6% (5 mo) 6 ± 2% (12 mo) 0 (17 mo)		
Regina, Sask. (4.2% OM, pH 7.7)	2.8 kg•ha^{-1} (5 mg•kg^{-1})	0–5 cm	80 ± 6% (2 wk) 50 ± 7% (6 wk) 25 ± 3% (13 wk) 16 ± 5% (21 wk)	8 mg triallate as emulsifiable concentrate diluted with benzene applied to 18 x 18 cm plots immediately incorporated to 5 cm.	Smith 1971
Jameson, Sask. (3.2% OM, pH 7.5)			26 ± 3% (21 wk)		Smith 1971
Indian Head, Sask. (4.2% OM, pH 7.5)			20 ± 3% (21 wk)		Smith 1971
Melfort, Sask. (10.6% OM, pH 5.2)			27 ± 4% (21 wk)		Smith 1971
Tisdale, Sask. (6.7% OM, pH 6.2)			21 ± 7% (21 wk)	Little indication that soil type affects persistence of triallate under field conditions.	Smith 1971

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Laukaa, Finland Fine sand (2.5% OM, pH 5.6)	NR (sprayed 1973–1976)	NR	0.007 $mg \cdot kg^{-1}$ in 1978 (2 yr2 after final application)	Field procedures NR; commercial formulation applied.	Heinonen-Tanski *et al.* 1985
Regina, Sask. Regina heavy clay (4.0% OM, pH 7.5, field capacity 39.7%) and Weyburn, Sask. Weyburn loam (6.5% OM, pH 7.0%, field capacity 28.0%)	1, 2, and 4 $mg \cdot kg^{-1}$		50% (8–11 wk)	Lab study in which herbicide (emulsifiable concentrate of 0.4 $kg \cdot l^{-1}$) mixed with soil, weighed into bottles to make 20-g samples at field capacity. samples at field capacity.	Smith 1969
Saskatchewan Heavy clay (4.2% OM, pH 7.7, field capacity 40%)	1.5 $kg \cdot ha^{-1}$ May 1979 May 1980 May 1981 1.5 $kg \cdot ha^{-1}$ May 1979 May 1980 May 1981	0–5 cm	 34 ± 8% (22 wk) 64 ± 8% (22 wk) 15 ± 9% (22 wk) 46 ± 4% (22 wk) 58 ± 7% (22 wk) 16 ± 3% (22 wk)	6 mg triallate added to 20 x 20 cm plots and incorporated 5 cm. Differences in residue levels between years believed to reflect edaphic and soil moisture conditions.	Smith and Hayden 1982b
Sandy loam (4.0% OM, pH 7.6, field capacity 20%)	1.5 $kg \cdot ha^{-1}$ May 1979 May 1980 May 1981 1.5 $kg \cdot ha^{-1}$ May 1979 May 1980 May 1981	0–5 cm	 28 ± 4% (22 wk) 32 ± 3% (22 wk) 12 ± 1% (22 wk) 32 ± 1% (22 wk) 35 ± 0% (22 wk) 12 ± 2% (22 wk)		Smith and Hayden 1982b
Regina, Sask. Typic Boroll heavy clay (3.1% OM, pH 7.5, field capacity NR)	1.48 $kg \cdot ha^{-1}$ May 20, 1983	0–10 cm	91.2 ± 12.8% (1 d) 70.9 ± 8.8% (3 d) 64.9 ± 10.8% (5 d) 63.5 ± 20.3% (7 d) 54.1 ± 1.4% (28 d) 43.9 ± 13.5% (67 d) 20 ± 10% (160 d)	Shallow cultivation and harrowing of study area on April 27, seeded to wheat on May 9, and application of an emulsifiable concentrate incorporated into the top 5 cm.	Grover, Smith *et al.* 1988b

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Delta Junction, Alaska, Volkmar and Beales silt loams (physical characteristics NR)	0.7, 1.4, or 2.8 kg•ha^{-1} late May 1982	0–15 cm	84 ± 22% (4 wk) 61 ± 14% (17 wk) 54 ± 5% (49 wk) 27 ± 11% (70 wk) 36 ± 8% (103 wk) 14 ± 55% (155 wk)	Triallate incorporated within 2 h of application to a depth of 5.1 cm. Residue values for all rates are averages since application rate did not have an affect on residue persistence.	Conn and Cameron 1988
Regina, Sask. Heavy clay (4.2% OM, pH 7.5, field capacity 40%)	1.5 kg•ha^{-1} May 1977	0-5 cm	30 ± 1% (10 wk) 20 ± 0% (20 wk)	6.0 mg triallate applied to each plot (20 x 20 cm) and immediately incorporated into the top 5 cm	Smith 1979
	May 1978 1.5 kg•ha^{-1} triallate and 0.75 kg•ha^{-1} trifluralin		30 ± 1% (10 wk) 23 ± 1% (20 wk)		
	May 1977		36 ± 3% (10 wk) 27 ± 2% (20 wk)		
	May 1978		24 ± 0% (10 wk) 16 ± 1% (20 wk)		
White City, Sask. Sandy loam (4.0% OM, pH 7.6, field capacity 20%)	1.5 kg•ha^{-1} May 1977	0-5 cm	20 ± 1% (10 wk) 12 ± 0% (20 wk)		Smith 1979
	May 1978		27 ± 4% (10 wk) 14 ± 2% (20 wk)		
	1.5 kg•ha^{-1} triallate and 0.75 kg•ha^{-1} trifluralin May 1977		25 ± 2% (10 wk) 10 ± 1% (20 wk)		
	May 1978		32 ± 1% (10 wk) 20 ± 3% (20 wk)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Regina, Sask. Regina heavy clay (4.2% OM, pH 7.8, field capacity 40%)	2.24 kg•ha^{-1} (4 mg•kg^{-1})	0-5 cm	12 wk 51% (40% soil moisture) 54% (35% soil moisture) 63% (30% soil moisture) 85% (20% soil moisture)	Triallate as a commercial formulation of emulsifiable concentrate (0.4 kg•l^{-1}) incorporated into the top 5 cm of soil.	Smith 1970
Weyburn, Sask. Weyburn loam (6.5% OM, pH 6.5, field capacity 28%)	2.8 kg•ha^{-1} (4 mg•kg^{-1})	0-5 cm	12 wk 43% (30% soil moisture) 47% (25% soil moisture) 48% (20% soil moisture) 60% (15% soil moisture)		Smith 1970
Regina, Sask. Regina heavy clay (physical characteristics given above)	2.8 kg•ha^{-1} (5 mg•kg^{-1})	0-5 cm	14.3%–22.6% (33 wk)	8 mg triallate applied to field plots (18 x 18 cm) and thoroughly incorporated into the top 5 cm.	Smith 1970
Begbroke, Oxford, England coarse, sandy loam (2% OM, pH 7, field capacity NR)	1.68 kg•ha^{-1} May 4, 1963 April 11, 1964 April 1, 1965 March 17, 1966	 0-15 cm	 NR NR NR 1.4 kg•ha^{-1} (0 wk) 1.05 kg•ha^{-1} (6 wk) 0.84 kg•ha^{-1} (12 wk) 0.28 kg•ha^{-1} (22 wk) 0.28 kg•ha^{-1} (25 wk) 0.14 kg•ha^{-1} (33 wk) 0.14 kg•ha^{-1} (52 wk)	Triallate applied after sowing and incorporated within 2 h.	Fryer and Kirkland 1970
	March 21, 1967		0.98 kg•ha^{-1} (0 wk) 0.77 kg•ha^{-1} (6 wk) 0.49 kg•ha^{-1} (14 wk) 0.35 kg•ha^{-1} (22 wk) 0.14 kg•ha^{-1} (34 wk)		
	3.36 kg•ha^{-1} May 4 & Aug. 28, 1963 April 11 & Oct. 28, 1964 April 1 & Oct. 22, 1965	0-15 cm	NR NR NR 2.45 kg•ha^{-1} (21 wk)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
	March 17, 1966		4.13 $kg \cdot ha^{-1}$ (10 wk) 3.15 $kg \cdot ha^{-1}$ (6 wk) 2.24 $kg \cdot ha^{-1}$ (12 wk) 3.08 $kg \cdot ha^{-1}$ (22 wk) 1.26 $kg \cdot ha^{-1}$ (25 wk) 1.19 $kg \cdot ha^{-1}$ (33 wk)		
	Nov. 11, 1966		3.43 $kg \cdot ha^{-1}$ (0 wk) 2.59 $kg \cdot ha^{-1}$ (5 wk) 1.96 $kg \cdot ha^{-1}$ (19 wk)		
	March 21, 1967		4.69 $kg \cdot ha^{-1}$ (0 wk) 2.80 $kg \cdot ha^{-1}$ (6 wk) 2.45 $kg \cdot ha^{-1}$ (14 wk) 1.33 $kg \cdot ha^{-1}$ (22 wk) 0.91 $kg \cdot ha^{-1}$ (35 wk)		
	Nov. 21, 1967		4.20 $kg \cdot ha^{-1}$ (0 wk) 2.59 $kg \cdot ha^{-1}$ (15 wk)		
	March 8, 1968		5.04 $kg \cdot ha^{-1}$ (0 wk) 1.75 $kg \cdot ha^{-1}$ (15 wk)		
	Dec. 6, 1968		5.46 $kg \cdot ha^{-1}$ (0 wk)		
Regina, Sask. Rego Dark Brown Chernozemic (4.2% OM, pH 7.7, field capacity 40%)	1.4 $kg \cdot ha^{-1}$ (2nd wk May 1983)	0-7.5 cm	0.53 $\pm$ 0.03 $mg \cdot kg^{-1}$ (6 mo) 0.40 $\pm$ 0.02 $mg \cdot kg^{-1}$ (12 mo)	Triallate immediately incorporated to 5 cm after application of commercial formulation.	Smith and Milward 1985
			Aged 6 mo 50% (45 d) 50% (43 d) 50% (43 d) Aged 12 mo 50% (39 d) Fresh comparison 50% (37 d)	50-g samples of the soils with aged triallate residues (6 mo old) weighed into 175-mL cartons, moistened to 85% of field capacity, loosely capped incubated in the dark at 20 $\pm$ 1°C.	

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Braunschweig, West Germany Parabrown soil (pH 5.4, field capacity 36.2%, % OM NR)	1 $mg \cdot kg^{-1}$			Lab study.	Anderson 1981
	2.4% water content		91.2 ± 1.5% (0 wk) 60.2 ± 0.6% (10 wk)		
	9.0% water content		94.1 ± 3.2% (0 wk) 50% (7 wk) 34.4 ± 0.4% (10 wk)		
	12.3% water content		94.2 ± 1.9% (0 wk) 50% (6.4 wk) 34.7 ± 1.1% (10 wk)		
	16.4% water content		95.0 ± 1.7% (0 wk) 50% (5.5 wk) 29.8 ± 0.0% (10 wk)		
	19.0% water content		95.3 ± 3.1% (0 wk) 50% (4.9 wk) 20.8 ± 1.6% (10 wk)		
Regina, Sask. Regina heavy clay (4.0% OM, pH 7.5, field capacity 39.7%)	0.56 $kg \cdot ha^{-1}$		50% (12 d)	Lab study.	Banting 1967
	1.12 $kg \cdot ha^{-1}$		50% (20 d) 50% (49 d)		
Braunschweig, West Germany Agricultural soil (1.26% total C, pH 5.4, field capacity NR)	0.25 $mg \cdot kg^{-1}$		95.1% (0 wk) 47.0% (10 wk) 36.8% (20 wk) 20.6% (52 wk)	Lab study.	Anderson and Domsch 1980b
	0.5 $mg \cdot kg^{-1}$		95.9% (0 wk) 46.5% (10 wk) 37.2% (20 wk) 17.4% (52 wk)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
	1.0 mg•kg^{-1}		96.2% (0 wk) 55.7% (10 wk) 34.5% (20 wk) 13.8% (52 wk)		
	5.0 mg•kg^{-1}		96.2% (0 wk) 74.3% (10 wk) 57.8% (20 wk) 35.3% (52 wk)		
	50.0 mg•kg^{-1}		97.1% (0 wk) 77.1% (10 wk) 64.5% (20 wk) 44.6% (52 wk)		
Braunschweig, West Germany Parabrown soil (% OM NR, pH 5.4, field capacity NR)	1 mg•kg^{-1} Fresh soil (655 mg microbial C•kg^{-1} soil)		96.4% (0 wk) 63.5% (4 wk) 39.9% (10 wk)	Lab study.	Anderson 1984
	20°C (330 mg microbial C•kg^{-1} soil) 33°C (130 mg microbial C•kg^{-1} soil)		95.9% (0 wk) 69.7% (4 wk) 57.9% (10 wk) 97.2% (0 wk) 79.9% (4 wk) 68.3% (10 wk)		
	44.5°C (85 mg microbial C•kg^{-1} soil)		96.5% (0 wk) 82.0% (4 wk) 70.3% (10 wk)		
	1 mg•kg^{-1} Unamended soil		94.0% (0 wk) 65.8% (4 wk) 48.0% (10 wk)		
	Soil amended with glucose		94.6% (0 wk) 46.7% (4 wk) 17.1% (10 wk)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
	Soil amended with carbohydrate mixture		94.9% (0 wk) 40.0% (4 wk) 15.0% (10 wk)		
Braunschweig, West Germany Agricultural soil (total C = 1.26%, pH 5.4, field capacity NR)	1 $mg \cdot kg^{-1}$ 1 $mg \cdot kg^{-1}$ 1 $mg \cdot kg^{-1}$		95.2% (0 d) 50% (50 d) 39.1% (85 d) 50% (35 d) 50% (52 d)	Lab study.	Anderson and Domsch 1980a
Begbroke, Oxford, England soil (2% organic carbon, pH NR, field capacity 29%)	2.24 $kg \cdot ha^{-1}$		<u>Foil Dish</u> 50% (15.5 d) Granules 50% (1.5 d) Emulsifiable concentrate <u>Dry Soil</u> 50% (70 d) Granules 50% (69 d) Emulsifiable concentrate <u>Wet Soil</u> 50% (8.5 d) Granules 50% (3.0 d) Emulsifiable concentrate	Greenhouse experiment. Triallate was applied as either a spray (0.68% emulsifiable concentrate) or as 10% granules.	Hance, Holroyd, and McKone 1973
clay loam soil (physical characteristics NR)	2.24 $kg \cdot ha^{-1}$ (June 4)	5.7 cm	50% (11.5 d) 2.5% granules 50% (9.0 d) 5% granules 50% (10.0 d) 10% granules	Granules (containing 2.5%, 5% or 10% triallate) were applied to 5.5 m x 1.8 m field plots of spring barley.	

Table B-1. Continued

Location/soil type (% organic matter; pH: moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Regina, Sask. top 5 cm of soil (3.1% OM, pH 7.7, field capacity NR)	1.48 $kg \cdot ha^{-1}$	10 cm	1.35 $\pm$ 0.19 $mg \cdot kg^{-1}$ (0 d) 1.05 $\pm$ 0.13 $mg \cdot kg^{-1}$ (2 d) 0.94 $\pm$ 0.30 $mg \cdot kg^{-1}$ (6 d) 0.80 $\pm$ 0.20 $mg \cdot kg^{-1}$ (27 d) 0.65 $\pm$ 0.20 $mg \cdot kg^{-1}$ (66 d) 0.55 $\pm$ 0.15 $mg \cdot kg^{-1}$ (96 d) 0.30 $\pm$ 0.15 $mg \cdot kg^{-1}$ (159 d) 0.42 $\pm$ 0.07 $mg \cdot kg^{-1}$ (325 d)	Triallate (emulsifiable concentrate) applied and immediately incorporated to 5 cm. Initial residue levels measured immediately after application and incorporation.	Cessna *et al.* 1988

Appendix C

Acute Toxicity Values of Triallate for Aquatic Organisms

Table C-1. Acute Toxicity Values of Triallate for Aquatic Organisms

Species	Test conditions	Temperature (°C)	pH	Hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% ai)	LC_{50}/EC_{50} ($mg \cdot L^{-1}$) 24 h	48 h (confidence interval)	96 h	Reference
VERTEBRATES									
Oncorhynchus mykiss (Rainbow trout)	S, M	12	7.6	40	Technical (95.30)	1.3 (1.0–1.7)		0.62 (0.44–0.87)	Mayer and Ellersieck 1986
	S, M	12	7.6	40	EC (46.3)	1.3 (1.0–1.6)		1.0 (0.7–1.4)	
Ictalurus punctatus (Channel catfish)	S, M	22	7.0	40	Technical (95.30)	2.5 (1.9–3.3)		1.7 (1.1–2.5)	Mayer and Ellersieck 1986
	S, M	22	7.0	40	EC (46.3)	1.8 (1.3–2.5)		1.1 (0.8–1.6)	
INVERTEBRATES									
Daphnia magna (Cladoceran) (1st instar)	S, M	17	7.3	39	Technical (95.30)		0.08 (0.06–0.10)		Mayer and Ellersieck 1986
	S, M	17	7.2	43	EC (46.3)		0.057 (0.048–0.067)		
Chironomus plumosus (Midge larvae) (3rd instar)	S, U	22	7.5	40	Technical (95.30)		0.49 (0.36–0.67)		Johnson 1986
Chironomus riparius (Midge larvae) (4th instar)	S, U	NR	NR	NR	EC (40.7)	1.0			Johnson 1986

EC = emulsifiable concentrate
NR = not reported
S = static
M = measured
U = unmeasured

Appendix D

Summary of Triallate Phytotoxicity Data

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
Oat (*Avena sativa*) (seedlings)	1 $mg \cdot L^{-1}$	Decrease in root size; 50% decrease in shoot size; 4 d posttreatment	Lab study, no soil	Kratky and Warren 1971
	10 $mg \cdot L^{-1}$	50% decrease in root size; 50% decrease in shoot size; 4 d posttreatment		
Cucumber (*Cucumis sativus*) (seedlings)	1 $mg \cdot L^{-1}$	50% decrease in root size; 4 d posttreatment	Lab study, no soil	Kratky and Warren 1971
	10 $mg \cdot L^{-1}$	50% decrease in root size; 4 d posttreatment		
Sorghum (*Sorghum vulgare*) (seedlings)	1 $mg \cdot L^{-1}$	50% decrease in root size; 50% decrease in shoot size; 4 d posttreatment	Lab study, no soil	Kratky and Warren 1971
Oat (*Avena sativa*) (seedlings)	0.35 $kg \cdot ha^{-1}$	70% plant injury	Environmental Chamber	Chang *et al.* 1974
	0.70 $kg \cdot ha^{-1}$	86% plant injury		
Wheat (*Triticum aestivum*) (seeds)	2.2 $kg \cdot ha^{-1}$	9% increase in seed number	Field cultivated	Moyer and Dryden 1977
	1.1 $kg \cdot ha^{-1}$	14% increase in seed number		
	1.65 $kg \cdot ha^{-1}$	20% increase in seed number		
	1.4 $kg \cdot ha^{-1}$	18% fresh weight increase at harvest	Field cultivated	O'Sullivan *et al.* 1982
Mustard (*Brassica napus*) (seeds)	1.4 $kg \cdot ha^{-1}$	10% plant mortality	Field cultivated	Chow 1976
Potato (*Solanum tuberosum*) (mature plant)	305 $mg \cdot L^{-1}$	55% decrease in secondary metabolism	Lab study, no soil	Bolton and Harwood 1976

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	30.5 $mg \cdot L^{-1}$	55% decrease in secondary metabolism		
	3.05 $mg \cdot L^{-1}$	22% decrease in secondary metabolism		
Barley (*Hordeum* sp.) (seeds)	1.1 $kg \cdot ha^{-1}$	30% decrease in plant number	Field cultivated	Klose 1961
	1.7 $kg \cdot ha^{-1}$	47% decrease in plant number		
	2.2 $kg \cdot ha^{-1}$	66% decrease in plant number		
	2.8 $kg \cdot ha^{-1}$	66% decrease in plant number		
Flax ((*Linum usitatissimum*) (seeds)	1.1 $kg \cdot ha^{-1}$	17% decrease in plant number	Field cultivated	Klose 1961
	1.7 $kg \cdot ha^{-1}$	25% decrease in plant number		
	2.2 $kg \cdot ha^{-1}$	25% decrease in plant number		
	2.8 $kg \cdot ha^{-1}$	29% decrease in plant number		
Wheat (*Triticum aestivum*) (seeds)	1.1 $kg \cdot ha^{-1}$	28% decrease in plant number	Field cultivated	Klose 1961
	1.7 $kg \cdot ha^{-1}$	33% decrease in plant number		
	2.2 $kg \cdot ha^{-1}$	58% decrease in plant number		
	4 $mg \cdot L^{-1}$	10% root size increase; 8% shoot size increase; 5 d posttreatment	Lab study, no soil	Banting 1970
	8 $mg \cdot L^{-1}$	10% root size increase; 15% shoot size decrease; 5 d posttreatment		
	16 $mg \cdot L^{-1}$	5% root size decrease; 15% shoot size decrease; 5 d posttreatment		
	64 $mg \cdot L^{-1}$	32% increase in meristem mitotic rate; 3 d post-treatment		Banting 1970
	2.8 $kg \cdot ha^{-1}$	51%–174% increase in harvest yield; 15 wk posttreatment	Lab study, no soil	Carlson and Morrow 1986

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	1.4 $kg \cdot ha^{-1}$ yield; 15 wk posttreatment	72%–158% increase in harvest		
Oat (*Avena sativa*) (seeds - with hull)	1.5 $mg \cdot L^{-1}$	4%–10% decrease in germination; 13%–49% decrease in coleoptile length; 6%–35% decrease in shoot dry weight; 5 d posttreatment	Lab study, no soil	Heath, Ashford, and McKercher 1984
	3.0 $mg \cdot L^{-1}$	0%–6% decrease in germination; 13%–55% decrease coleoptile length; 7%–43% decrease in shoot dry weight; 5 d posttreatment		
Oat (*Avena sativa*) (seeds - without hull)	1.5 $mg \cdot L^{-1}$	22%–26% decrease in coleoptile length; 31%–33% decrease in shoot dry weight; 5 d posttreatment		Heath, Ashford, and McKercher 1984
	3.0 $mg \cdot L^{-1}$	31%–54% decrease in coleoptile length; 41%–54% decrease in shoot dry weight; 5 d posttreatment		
Oat (*Avena sativa*) (seedlings)	0.12 $mg \cdot kg^{-1}$	27%–59% decrease in plant number; 28 d posttreatment	Environmental chamber	McKercher and McGregor 1980
	0.22 $mg \cdot kg^{-1}$	40%–69% decrease in plant number; 28 d posttreatment with NH_4Cl, HNO_3, or HCl		
	0.36 $mg \cdot kg^{-1}$	72%–85% decrease in plant number; 28 d posttreatment with NH_4Cl, HNO_3, or HCl		
	0.12 $mg \cdot kg^{-1}$	15%–25% decrease in plant number; 28 d posttreatment with various soil moistures		

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	0.22 $mg \cdot kg^{-1}$	40%–49% decrease in plant number; 28 d posttreatment with various soil moistures		
	0.36 $mg \cdot kg^{-1}$	57%–76% decrease in plant number; 28 d posttreatment with various soil moistures		
	0.12 $mg \cdot kg^{-1}$	32%–59% decrease in plant number; 28 d posttreatment with various soil moistures and 350 $mg \cdot kg^{-1}$ N		
	0.22 $mg \cdot kg^{-1}$	52%–69% decrease in plant number; 28 d posttreatment with various soil moistures and 350 $mg \cdot kg^{-1}$ N		
	0.36 $mg \cdot kg^{-1}$	67%–85% decrease in plant number; 28 d posttreatment with various soil moistures and 350 $mg \cdot kg^{-1}$ N		
Oat (*Avena sativa*) (seeds)	0.57 $kg \cdot ha^{-1}$	7%–42% decrease in dry weight; 6 wk posttreatment with 0-6720 $kg \cdot ha^{-1}$ lime amendments	Field cultivated	McKercher and McGregor 1979
	0.84 $kg \cdot ha^{-1}$	20%–47% decrease in dry weight; 6 wk posttreatment with 0-6720 $kg \cdot ha^{-1}$ lime amendments		
	0.28 $kg \cdot ha^{-1}$	20%–40% decrease in plant number; 6 wk posttreatment with 0–6720 $kg \cdot ha^{-1}$ lime amendments		
	0.56 $kg \cdot ha^{-1}$	60%–72% decrease in plant number; 6 wk posttreatment with 0–6720 $kg \cdot ha^{-1}$ lime amendments		

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	0.84 $kg \cdot ha^{-1}$	74%–84% decrease in plant number; 6 wk posttreatment with 0–6720 $kg \cdot ha^{-1}$ lime amendments		
Oat (*Avena sativa*) (seedlings)	0.11 $mg \cdot kg^{-1}$	31%–47% decrease in plant number; 29%–53% decrease in plant dry weight; 25 d posttreatment with 1–3 meq Ca/100 g soil amendments	Environmental chamber	McKercher and McGregor 1979
	0.18 $mg \cdot kg^{-1}$	52%–60% decrease in plant number; 54%–70% decrease in plant dry weight; 25 d posttreatment with 1–3 meq Ca/100 g soil amendments		
	0.11 $mg \cdot kg^{-1}$	16%–26% decrease in plant number; 16% decrease in plant dry weight; 25 d posttreatment without amendments		
	0.18 $mg \cdot kg^{-1}$	32%–59% decrease in plant number; 54% decrease in plant dry weight; 25 d posttreatment without amendments		
Oat (*Avena sativa*) (seeds)	0.22 $kg \cdot ha^{-1}$	50% decrease in shoot length; in soil containing 1.8% organic matter; 7 d posttreatment	Greenhouse study	Grover, Banting, and Morse 1979
Dill (*Anethum graveolens*)	3 $kg \cdot ha^{-1}$	21%–32% decrease in plant fresh weight of mature plants; 26% decrease in dill oil yield from mature plants	Field cultivated	Wall and Friesen 1986
Oat (*Avena sativa*) (seeds)	1.15 $ug \cdot g^{-1}$	50% decrease in dry weight; 14 d posttreatment;	Greenhouse study	Nyffeler *et al.* 1982
	0.99 $ug \cdot g^{-1}$	50% decrease in fresh weight; 14 d posttreatment;		
	1.10 $ug \cdot g^{-1}$	50% decrease in shoot length		

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	0.55 $kg \cdot ha^{-1}$	50% decrease in shoot length; in soil containing 4.2% organic matter; 7 d posttreatment		
	1.19 $kg \cdot ha^{-1}$	50% decrease in shoot length; in soil containing 10.5% organic matter; 7 d posttreatment		

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Abstract

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

Résumé

On a examiné la documentation relative aux utilisations, au devenir et aux effets du triallate 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 lignes directrices sur la qualité de l'eau sont recommandées pour la protection d'utilisations particulières de l'eau.

Canadian Water Quality Guidelines for Triallate

R.A. Kent, M. Taché, P.-Y. Caux, S. De Silva and K. Lemky

INTRODUCTION AND BACKGROUND

Production and Uses

Triallate is the common name for the agricultural herbicide with the Chemical Abstracts Service (CAS) and IUPAC name N,N-diisopropyl-thiocarbamate 2,3,3-trichloroallyl. It is an amber oil with a molecular formula of $C_{10}H_{16}Cl_3NOS$ and a molecular weight of 304.7. The CAS registry number for triallate is 2303-17-5. It is also known as bis(1-methylethyl)-carbamothioic acid or S-(2,3,3-trichloro-2-propenyl) ester. Triallate was introduced into Canada in the early 1960s by Monsanto and is currently marketed under the trade names Avadex BW and Fortress (Agriculture Canada 1990). Triallate is not manufactured in Canada. Canadian import data for triallate are presented in Table 1. At present, three Avadex BW products, consisting of 400 and 480 g•L^{-1} active ingredient (ai) emulsifiable concentrates and a 10% ai granular formulation, are registered in Canada. Avadex granules have recently (September 1990) been registered for a fall surface treatment. (P. Marshall, 1991 Monsanto Canada, Ottawa, pers. com.). In this application, herbicide is spread in the fall just prior to freeze-up, and incorporation is delayed until spring. This treatment is intended for prairie soils that are erosion prone; the removal of a fall tillage operation can drastically decrease erosion vulnerability. A fourth product (Fortress) contains a 4% trifluralin, 10% triallate granular mixture. Triallate is a very popular preemergence herbicide highly effective in controlling certain monocots, particularly wild oats. It is recommended for control of wild oats in barley, durum wheat, spring wheat, and dry peas (Worthing and Walker 1987). It is also recommended for use on canola, flax, sugar beets, and mustard (Agriculture Canada 1982).

Preplant treatments require that triallate be sprayed on the soil surface and worked into the top 5–8 cm of soil with a disk or cultivator. Postplant treatment of cereals requires that triallate be sprayed on the soil surface and worked into the soil by harrowing (the crop must be seeded deep enough to prevent disturbance by harrowing). In both pre- and postplant treatments, triallate should be worked into the soil within 2 h after spraying (OMAF 1989). Normal applications range from 1.12 to 1.68 kg ai•ha^{-1} (Worthing and Walker 1987).

Table 1. Statistics Canada Import Data for Triallate

	1983	1984	1985	1986	1987
Triallate formulated herbicides (tonnes)	19 185	23 980	16 607	11 862	7009
Triallate technical (tonnes)	2 672	3 000	1 560	972	562

Source: Statistics Canada (1986, 1988).
Note: The quantities refer to the mass of the product (i.e., not the active ingredient) and likely include solvents and additives (e.g., surfactants, etc.). Secondary pesticide active ingredients may also be included.

Physical and Chemical Characteristics

The physical and chemical characteristics of triallate are presented in Table 2. The water solubility is reported to be 4 mg•L^{-1}. The structural formula for triallate is presented in Figure 1.

$$[(CH_3)_2CH]_2N.CO.SCH_2\overset{Cl}{\overset{|}{C}}=CCl_2$$

Figure 1. Structural formula for triallate.

Mode of Action

The major phytotoxic effect of triallate is inhibition of cell elongation or expansion. The effect is more pronounced on the stem and leaf meristematic tissue than on root tissue (Banting 1967, 1970; Thiele and Zimdahl 1976). In wild oats (*Avena fatua* L.), 63% mitotic inhibition occurred in stem and leaf meristematic tissues during a 3-d germination period when the plants were exposed to vapour from a 249.85-mg•L^{-1} triallate solution (Banting 1970). Inhibitory effects on elongation were observed at concentrations that did not affect mitosis. Thus inhibition of mitosis appears to be a secondary effect (Banting 1970). The herbicidal action of triallate apparently depends on the diffusion of the vapour phase into the coleoptile, resulting in the suppression of development of the first leaf and interference

Table 2. Physical and Chemical Characteristics of Triallate

Chemical formula	$C_{10}H_{16}Cl_3NOS$[1]
Molecular weight	304.7[1]
Physical state	Amber oil°C[2] (°C not given)
Henry's law constant	1.02 Pa·m^3·mol^{-1}[3]
Melting point	29–30°C[1]
Boiling point	148–149°C[1]
Vapour pressure	13.3 mPa at 20°C[4] 20.2 mPa at 23°C[4] 16.0 mPa at 25°C[2] 27.5 mPa at 25°C[4] 27.6 mPa at 25°C[1] 44.6 mPa at 30°C[4] 70.4 mPa at 35°C[4] 131.5 mPa at 40°C[4] 266.9 mPa at 45°C[4]
Log octanol/water partition coefficient (K_{ow})	4.6[5]
Log sediment/water distribution coefficient (K_{oc})	3.3[6] 3.45–3.53[7]
Solubility: Water	4 mg·L^{-1} at 25°C[1][2]
Half-life in topsoil*	3–88 d[8,9]
Bioconcentration factor	150[5]

Note: Half-life is strongly dependent on soil humidity i.e., 3 days in wet soil (greenhouse)[8], up to 70 d (greenhouse)[8], and 88 d (field study, Regina, Saskatchewan)[9] in dry soil.

[1]U.S. EPA 1983.
[2]Worthing and Walker 1987.
[3]Suntio *et al.* 1988.
[4]Grover *et al.* 1978.
[5]Estimated from Chiou *et al.* 1977.
[6]Kenaga 1980.
[7]Singh *et al.* 1990.
[8]Hance, Holroyd, and McKone 1973.
[9]Grover, Smith, *et al.* 1988.

in the maturation of the cells of the coleoptile (Miller and Nalewaja 1976). Thiocarbamates are known to interfere with lipid formation, resulting in decreased epicuticular wax formation and thinner cuticula, thus increasing leaf wettability and plant susceptibility to foliage–applied herbicides (Hess 1989). These symptoms and the production of necrotic lesions have also been observed by Billet and Ashford (1978). The effects of triallate on the elongation of shoot cells and disruption of wax formation appear to have a common cause in the inhibition of fatty acid synthesis, which reduces cuticular wax formation by inhibiting fatty acid elongation (Bolton and Harwood 1976). Thiocarbamates, for example, EPTC, have been shown to inhibit gibberellic acid synthesis, which eventually affects cell elongation (Wilkinson and Ashley 1979). Triallate, having a similar structure, is expected to act similarly.

Methods of Analysis

McKone and Hance (1967) described an extraction and gas chromatographic (GC) analysis of triallate in soil and vegetable matter that had a detection limit of 0.05 mg·kg^{-1}. The extractant used was a mixture of 2,2,4-trimethylpentane and isopropyl alcohol. Vegetable matter extracts required further cleanup techniques (i.e., thin layer or column chromatographic separation), which were not necessary for the soil extracts.

Several authors (Beestman and Deming 1976; Anderson and Domsch 1980a, 1980b; Anderson 1981) used a mixture of benzene and isopropanol (2:1, v/v) to extract triallate from soil. Benzene alone was used to extract triallate from water. Detection limits were not reported by these authors.

A second group of researchers (Smith 1970, 1979; Jury *et al.* 1980; Smith and Hayden 1982a, 1982b; Smith and Milward 1985) extracted triallate from soil samples using 30% aqueous acetonitrile containing 2.5%–3.0% glacial acetic acid. The extract was subsequently partitioned into n-hexane prior to GC analysis. Detection limits were not reported by these researchers.

Extraction of triallate from water was described by Muir and Grift (1987). Adjustment of pH to 2.0 with HCl was followed by extraction with dichloromethane. Final water removal was accomplished by passing the extract through an anhydrous sodium sulphate column. Florisil column cleanup was followed by ethyl acetate/hexane elution. The triallate detection limit for the GC/MS method was 3 ng·L^{-1}. Triallate was extracted from surface water samples, sediment, fish, and macrophytes using diethyl ether and analyzed by GC with electron capture detector (ECD) (Therrien-Richards and Williamson 1987). They reported an analytical detection limit of 0.10 µg·L^{-1} for water samples and 2.7 ng·g^{-1} for sediment, fish, and macrophytes. Environment Canada's National Water Quality Laboratory uses a gas-liquid chromatography method with ECD for the analysis of triallate in surface waters. A detection limit of 0.01 µg·L^{-1} was reported for river water samples (Environment Canada 1984).

Entry into the Environment

Triallate has the potential to leave the site of application and enter the nontarget environment by direct volatilization and subsequent atmospheric transport mechanisms, surface water runoff, and soil adsorption.

Concentrations in Atmosphere

A soil-applied herbicide such as triallate with a relatively high vapour pressure has a great potential for evaporation or volatilization (Grover 1983). Atmospheric concentrations as high as 198 $ng \cdot m^{-3}$ have been recorded in Regina and Melfort, Saskatchewan, where triallate is extensively used in the surrounding area (Grover, Kerr, *et al.* 1988). The seasonal occurrence of triallate in the air generally follows the seasonal use patterns for this herbicide. Soil moisture conditions and rainfall events, however, greatly influence the occurrence and concentration of triallate in the air (Grover, Kerr, *et al.* 1988). Reported maximum concentrations of triallate (200 $ng \cdot m^{-3}$) in Saskatchewan occurred during the spray season of May 1978 when the soil was relatively wet (Grover 1983). During the summer, when the soil was dry, or following freezing of the soil in the fall, airborne residues of triallate were less than 10 $ng \cdot m^{-3}$.

In the field, vapour losses are influenced by the nature of the target, atmospheric turbulence, and soil moisture (Grover 1983). Volatilization losses of triallate are increased when it is applied as an emulsifiable concentrate as opposed to the granular formulation (Hance, Holroyd, and McKone 1973; Smith and Hayden 1981), when the soil is moist (Beestman and Deming 1976; Hance, Holroyd, and McKone 1973; Smith 1983), and when the compound is not soil incorporated (Worthing and Walker 1987).

Concentrations in Water, Sediment, and Biota

A summary of triallate concentrations in Canadian surface water and biota is presented in Appendix A. Snowmelt runoff from fields treated the previous fall may be a significant factor in the presence of triallate in surface waters on the Canadian prairies. Support for this comes from the positive linear correlation ($r^2 = 0.713$) between the flow rates of the La Salle River (southern Manitoba) in the spring and the observed concentrations of triallate. When the river flow increased in June, the same correlation could not be found (Williamson 1984). In southern Saskatchewan, triallate entry into surface waters in spring runoff was also observed by Waite *et al.* (1986). They reported 0.47 and 0.64 $\mu g \cdot L^{-1}$ on March 27 and 28, 1984, respectively, in the runoff from 648 ha in the South Saskatchewan River basin. In 1985 and in 1987, on an agricultural watershed north of Regina, Waite *et al.* (1990) recorded maximum levels of 0.62 $\mu g \cdot L^{-1}$ and 0.98 $\mu g \cdot L^{-1}$, respectively. The presence of low triallate concentrations in rivers during spring and fall application periods suggests that transport and deposition of triallate vapours and triallate adsorbed to dust particles may be the cause of low concentrations of triallate in surface waters not contaminated by surface runoff (Muir and Grift 1987). This is further supported by a study in a Saskatchewan watershed by Grover, Kerr, *et al.* (1988), which showed that aerial transport is a significant path of herbicide input to surface waters.

In a shallow groundwater study, in the Outlook Irrigation District, Saskatchewan, Maathuis *et al.* (1988) recorded triallate concentrations in piezometers with ranges between 0.13 and 0.39 $\mu g \cdot L^{-1}$ and between 0.13 and 0.15 $\mu g \cdot L^{-1}$. These high concentrations of triallate could not be explained because triallate had not been applied in the region in the past few years. During a monitoring survey for triallate in the La Salle River in August–December 1984, triallate was not detected in the water column in an area where it was heavily used. The limit of detection was 0.10 $\mu g \cdot L^{-1}$. Triallate, however, was found in the river sediments at concentrations ranging from 16.9 to 119 $\mu g \cdot kg^{-1}$ (Therrien-Richards and Williamson 1987). Triallate is strongly adsorbed to soil particles. As a result, another major transport pathway from treated fields is by soil erosion via surface runoff and atmospheric suspension. Reports of triallate concentrations in edge-of-field runoff are relatively few. Triallate concentrations in runoff water would be expected to be reduced by soil incorporation through a reduction in the amount available for runoff loss. Triallate concentrations in rivers such as the Ochre, Turtle, La Salle, and Assiniboine in Manitoba, which drain areas where the herbicide is used, ranged from 3 $ng \cdot L^{-1}$ to 150 $ng \cdot L^{-1}$ (Muir and Grift 1987; Williamson 1984). Triallate concentrations ranging from 1.58 to 6.77 $\mu g \cdot L^{-1}$ were detected in spring runoff and snowmelt in Saskatchewan (Grover, Kerr, *et al.* 1988). In a long-term field experiment in Saskatchewan, triallate concentrations in irrigation tailwaters were reported to be 1.8 $\mu g \cdot L^{-1}$. The concentration of triallate in the drainage canal, which carried all tailwaters and return irrigation flows from the basin, however, was <0.1 $\mu g \cdot L^{-1}$ following the first irrigation event after triallate application (Cessna and Grover 1982).

Small forage fish collected from the La Salle River, Manitoba, were found to contain triallate.

Sufficient numbers of individual species (whole body samples) were composited to produce 100-g samples. Maximum triallate concentrations in brown bullhead (*Ictalurus nebulosus*), brook stickleback (*Culaea inconstans*), and the central mudminnow (*Umbra limi*) were reported to be 4.2, 3.3, and 9.2 $\mu g \cdot kg^{-1}$, respectively (Therrien-Richards and Williamson 1987). These data, together with the lack of detectable residues in aquatic macrophytes (*Myriophyllum* sp.) in the La Salle River, further support the rapid selective partitioning to sediment phases and subsequent incorporation into sediment-associated biota.

The U.S. national water quality monitoring data base, STORET, did not contain monitoring data for triallate (U.S. EPA 1983).

Environmental Fate, Persistence, and Degradation

Soil

Processes such as adsorption, leaching, chemical and biological degradation, volatilization, and photodecomposition (influenced by environmental conditions including soil temperature, moisture, and composition) affect the rate of triallate loss from soils (Smith 1970). Of these factors, soil adsorption, microbial degradation, and volatilization appear to be the most important to triallate dissipation (Smith 1970; Anderson 1981; Grover, Smith *et al.* 1988), with adsorption affecting the amount of triallate available in the soil solution for degradation and volatilization.

Persistence in Soil

Reported values of triallate persistence in soil are quite variable depending on the environmental conditions (see Appendix B). The 6-month carry-over of triallate residues from spring and fall applications for various locations in Saskatchewan is reported to range from 3% to 75% of the initial application (Smith 1970, 1971, 1975, 1979; Smith and Hayden 1976, 1982a, 1982b; Cessna *et al.* 1988; Grover, Smith, *et al.* 1988). The upper values of this range generally correspond to fall–spring carry-over rates, while the lower values typically represent spring–fall carry-over.

Half-life values for triallate persistence, obtained from various laboratory, greenhouse, and field studies, ranged from 3 to 88 d (Banting 1967; Smith 1969; Hance, Holroyd, and McKone 1973; Anderson 1981; Grover, Smith, *et al.* 1988). The lower portion of this range represents surface applications without incorporation on wet soil. Thorough incorporation of the herbicide into the soil typically produces half-lives in the upper portion of this range indicating the importance of volatilization to triallate dissipation.

In a soil at Oxford, England, to which triallate was applied and soil incorporated in the spring at a rate of 1.68 $kg \cdot ha^{-1}$, residues could not be detected at the end of the growing season. However, the domestic oat (*Avena sativa*) bioassay method that was used to detect triallate presence (detection limit of about 0.1 $mg \cdot kg^{-1}$) was concluded to be too imprecise and insensitive for persistence studies (Fryer and Kirkland 1970). Studies at the same location found little evidence for the accumulation of triallate in soils even after repeated applications and at rates above normal; plots treated twice annually for 6 years at 3.3 $kg \cdot ha^{-1}$ were found to contain 5.50 $kg \cdot ha^{-1}$ after the final application, but only 0.62 $kg \cdot ha^{-1}$ a year later and 0.09 $kg \cdot ha^{-1}$ (2% of initial value of 5.5 $kg \cdot ha^{-1}$) 3.5 years later (Fryer and Kirkland 1970; Fryer, Smith, and Hance 1980).

In Saskatchewan, climatic conditions are typically represented by long, cold winters and hot, dry summers. Triallate residues recovered from the top 5 cm of field plots in May 1972 represented 54% (for a sandy loam soil), 75% (for a heavy clay soil), and 75% (for a silty clay soil) of the initial 1.7-$kg \cdot ha^{-1}$ treatment applied the previous October. Comparable values for May 1973 were 37% (sandy loam), 23% (heavy clay), and 43% (silty clay) from an October 1972 application. Actual residue concentrations were not reported (Smith 1975).

Residues recovered in October 1972 following a May 1972 application of 1.7 $kg \cdot ha^{-1}$ were 14% (sandy loam), 18% (heavy clay), and 35% (silty clay). Comparable values for October 1973 were 10% (sandy loam), 11% (heavy clay), and 3% (silty clay) of a May 1973 application. Actual residue concentrations were not reported (Smith and Hayden 1976). Comparisons of percent triallate soil residues between fall–spring carry-overs and spring–fall carry-overs indicate that these were generally higher on the silty clay. Further, the fall–spring carry-over exhibits greater residue recoveries. Smith (1975) attributes this finding to triallate volatilization and biological degradation mechanisms being more significant over the spring–fall period.

The soils of the subarctic interior of Alaska are frozen for 6 months or longer each year; greater persistence is expected as a result of these colder conditions. In this region, average triallate carry-over of 54%, 36%, and 14% was reported after 1, 2, and 3 years (Conn and Cameron 1988). The average annual carry-over did not vary greatly, despite different initial spring application rates (0.7, 1.4, or 2.8 kg•ha-1).

Although the application rate of triallate is reported to have no effect on persistence (Conn and Cameron 1988), Banting (1967) found an increase in application rate from 0.56 to 1.12 $kg \cdot ha^{-1}$ corresponded to a half-life increase from 49 to 66 d in a laboratory study. In another laboratory study, persistence was very similar for triallate soil application rates ranging from 0.25 to 1.0 $mg \cdot kg^{-1}$. The absolute amounts of triallate dissipating from the soil were greater as the application rate increased from 5.0 to 50.0 $mg \cdot kg^{-1}$ (Anderson and Domsch 1980b).

There appears to be a relationship between the organic matter content of a soil and triallate persistence, however, the nature of this relationship is not clear. Various investigators report the following: (1) an increase in persistence with increasing soil organic matter from approximately 3.2% to 11.7% (Smith 1975, 1979); (2) no clear trend in triallate persistence over a range of 3.2%–10.6% organic matter (Smith 1971); (3) decreasing persistence with an increase in organic matter from 4.2% to 6.5% (Smith and Fitzpatrick 1970); and (4) little difference in the persistence of triallate among different soil types (Smith 1969). Persistence was reported to be greater in organic soils than in light-textured soils (Smith 1983).

The discrepancy in triallate persistence data in relation to soil organic matter may be due to variations in soil moisture and temperature (Smith and Hayden 1982a). Increased soil moisture and temperature result in a decrease in persistence (Smith 1970; Hance, Holroyd, and McKone 1973; Smith and Hayden 1976, 1982a, 1982b; Anderson 1981; Conn and Cameron 1988; Grover, Smith, *et al.* 1988), probably due to increased volatilization and/or biodegradation. Increased soil aeration, soil moisture content, and temperature also contribute to reducing the persistence of carbamate herbicides as a group by providing conditions conducive to increased microbial activity (Kaufman 1967). A decrease in triallate persistence is associated with both an increase in the biomass of soil microorganisms (Anderson 1981, 1984) and amendment of soils with glucose or a carbohydrate mixture (Anderson 1984). Triallate persistence in soil may also be due in part to its adsorption onto microbial cell walls (Cullimore and Smith 1972). Under controlled laboratory conditions, triallate adsorption on different adsorbents showed that triallate has a greater affinity for organic adsorbents (peat moss, straw wheat) than for inorganic adsorbents (clay). Triallate bound to montmorillonite is more easily desorbed with water than from peat moss, suggesting that weak physical forces (Van der Waals) are involved in the compound's binding to montmorillonite (Grover 1974). Leaching of triallate is shown to be higher in soils with high clay and low organic content than in soils with low clay and high organic content (Smith 1969).

Although the soil persistence of a number of herbicides may be affected when used in combination with other chemicals (Hurle and Walker 1980), several studies have shown that herbicidal combinations with triallate have little or no effect on the persistence of the compound. Anderson and Domsch (1980a) found triallate persistence to be reduced by the addition of chlorpyrifos to soils, but various combinations of other pesticides did not affect carry-over. As well, the addition of trifluralin (Smith 1979) and chloramben (Smith and Hayden 1982b) to soils had little effect on the persistence of triallate.

The availability of triallate in soil to various dissipation and degradation mechanisms also affects its persistence. The formation of bound or unextractable soil residues is an important process controlling the availability of triallate (Anderson 1981), but little information is available describing the formation or structure of these bound residues. In view of the information related to decreased dissipation of diallate with increased adsorption, triallate appears to be unavailable for short-term (i.e., hours) phytotoxic or biodegradation reactions when adsorption and bound residue formation are prominent processes. Over longer terms (i.e., months), however, these bound residues are apparently susceptible to biodegradation (Anderson and Domsch 1980b).

Triallate persistence in soil is greatly influenced by the formulation with which it is applied. Granular formulations of triallate are reported to be more persistent than the emulsifiable concentrates be-

cause of their slower release into the environment and their incorporation in soils (Hance, Holroyd, and McKone 1973; Smith and Hayden 1981; Qureshi 1987).

Dissipation

Three distinct phases of triallate dissipation in Canadian soils are described by Grover, Smith, *et al.* (1988) as (1) an initial rapid phase with volatilization as the major means of dissipation after application and incorporation, followed by (2) a slow and continuous dissipation over the entire growing season with volatilization and microbial degradation as the major pathways of dissipation, and (3) little or no dissipation in winter. Initial rapid volatilization losses, followed by slow dissipation, is congruent with field and laboratory investigations of triallate (Smith 1970, 1971; Anderson and Domsch 1980b; Jury *et al.* 1980; Cessna *et al.* 1988).

The reported rate kinetics values for triallate are quite variable. First-order kinetics were described for triallate soil dissipation by Banting (1967) and Smith and Milward (1985). Banting (1967), however, found a lag period in triallate dissipation of 28 and 45 d, which depended on the application rate between the time of triallate application and the onset of breakdown. The influence this lag period may have on the half-life range of 3–88 d generally attributed to triallate was not discussed.

The gross dissipation of triallate for the entire growing season, although described earlier by Grover, Smith, *et al.* (1988) as occurring in two distinct phases, was reported to follow first-order kinetics. Because triallate is lost from soil by three different routes (i.e., volatilization, biodegradation, and bound residue formation), a rate of loss between first- and second-order kinetics is considered nonetheless to be more representative than first-order kinetics (Anderson and Domsch 1980b).

Volatilization—Volatilization of triallate is considered to be the initial dominant route of soil dissipation from treated areas (Smith 1979, 1983; Grover 1983; Grover, Kerr, *et al.* 1988, Grover, Smith, *et al.* 1988). Since triallate is a very volatile substance, it must be incorporated into the soil shortly after application (Smith 1969, 1970; Cullimore and Smith 1972). Volatilization of triallate from deep incorporations is less than that from shallower incorporations (Smith 1983). In areas where triallate is used extensively, airborne residues (measured by using an air sampling train [tube, chamber, flow meter, and pump] with polyurethane foam as the adsorbent material) can be detected throughout the growing season (Grover 1983). However, over long periods and after soil incorporation, volatilization losses are considerably less than those due to biodegradation and bound residue formation (Anderson 1981, 1984; Anderson and Domsch 1980b). Extensive adsorption has been reported to substantially reduce losses due to volatilization (Smith 1970). Volatilization of preemergence, soil-incorporated herbicides is a function of vapour pressure, but under field conditions, loss due to volatilization is governed by (1) the rate of herbicide desorption from soil (adsorption/ desorption potential), (2) movement to the soil surface (diffusion and mass flow potential), (3) the rate of volatilization at the soil surface (vaporization potential), and (4) the rate of vapour movement away from the surface (atmospheric turbulence potential) (Jury *et al.* 1980; Grover 1983). In addition to soil-adsorbed and solution-phase triallate, the gaseous phase of the herbicide can also move to the soil surface by diffusion (Jury *et al.* 1980).

Under field conditions, maximum triallate vapour concentrations were typically found during peak application periods in May when soil moisture conditions were relatively high. During relatively dry springs, airborne residues were lower than those measured following summer rainfall events (Grover 1983; Cessna *et al.* 1988; Grover, Kerr, *et al.* 1988; Grover, Smith, *et al.* 1988). Although soil water was reported to have little influence on volatilization rates in closed systems without air exchange (Anderson 1981), several other investigators have reported increased triallate volatilization with increased soil moisture (Hance, Holroyd, and McKone 1973; Miller and Nalewaja 1976; Smith and Hayden 1982a; Grover 1983; Smith 1983; Cessna *et al.* 1988). For instance, appreciable volatilization losses were not found from dry soils kept in the laboratory at 50°C for 28 d (Smith 1970). Triallate volatilization losses were suggested to be minimal during summer months on the Canadian prairies where the top 5 cm of soil are often dry even though soil temperatures of 50°C and higher have been recorded.

Water is thought to displace triallate from soil adsorption sites as soil moisture levels increase beyond that necessary to produce a monolayer around the soil particles (Hance, Holroyd, and McKone 1973; Miller and Nalewaja 1976; Menzer and Nelson 1980). Triallate in the liquid phase moves upward

primarily by convection when evaporation occurs at the soil surface (Jury *et al.* 1980; Grover, Smith, *et al.* 1988). The mass flow of thiocarbamates to the soil surface has been referred to as the "wick effect" (Menzer and Nelson 1980), which is the capillary action of water flowing upward against gravity. Both a gas phase and a liquid phase (by convection) are contributing to the upward movement of water as evaporation from the surface occurs. Convection is the mechanism whereby triallate is resupplied at the surface soil layer as it is lost by diffusion to the air (Jury *et al.* 1980).

Volatilization of triallate from soils decreased with increasing organic matter content (Beestman and Deming 1976; Miller and Nalewaja 1976), which may reflect a higher adsorption in these soils (Hance, Holroyd, and McKone 1973). Similar triallate volatilization losses from two soils of different organic matter contents (1.24% and 5.1%), however, have also been reported under laboratory conditions (Jury *et al.* 1980). The higher adsorptive capacity of the more organic soil was thought to be offset by its lower bulk density and higher porosity, which resulted in a higher triallate diffusion coefficient (Jury *et al.* 1980).

Both the formulation and application rate affect triallate volatilization, with the volatilization rate decreasing from the emulsifiable concentrate to the unformulated technical grade triallate to the granular formulation (Hance, Holroyd, and McKone 1973; Miller and Nalewaja 1976; Smith and Hayden 1981). Volatilization increases with increasing application rate (Hance, Holroyd, and McKone 1973; Anderson and Domsch 1980b).

Under conditions favouring triallate volatilization from soils, maximum rates of loss are typically reached soon after application, followed by a rapid decrease, which is likely associated with a quick loss of the herbicide near the soil surface (Jury *et al.* 1980).

A volatilization loss equal to 17.6% of the amount of triallate applied was reported for a single growing season in southern Saskatchewan. Approximately 50% of the volatilization loss occurred during the first 4–5 d following application, with the subsequent vapour flux from the soil decreasing with time over the growing season (Grover, Smith, *et al.* 1988).

Jury *et al.* (1990) recently evaluated the volatilization of organic chemicals residing below the soil surface. Their model was designed as a screening tool to assess the volatilization potential of compounds under standard soil and environmental conditions. They found the soil cover thickness required to restrict volatilization to less than 0.7% of the triallate mass incorporated in soil was 3.6 cm for a sandy soil and 1.5 cm for a clay soil.

The vapour flux of triallate from a glass surface was successfully predicted using a mathematical model based on triallate vapour pressure and molecular weight (Grover *et al.* 1978). The average volatilization rate from glass plates was 5.71 $\mu g \cdot cm^{-2} \cdot h^{-1}$ at 25°C during a 4- to 6-h period. This value may be equalled or exceeded under field conditions when adsorptive processes are not operating in moist soils and air exchange rates are high. Since in practice triallate is incorporated into the soil, however, it is difficult to assess the exact relationship between volatilization rates from the nonadsorbing surfaces in this study to those expected in the field where adsorption is important (Grover *et al.* 1978). Another field study in Saskatchewan demonstrated a maximum volatilization rate of 0.04 $\mu g \cdot cm^{-2} \cdot h^{-1}$ during the 4–6 h following application of 1.5 $kg \cdot ha^{-1}$ triallate as an emulsifiable concentrate to a heavy clay soil (air temperature of 14.4°C) (Grover, Smith, *et al.* 1988).

Microbial Degradation—While volatilization is initially important, the breakdown of triallate by soil microorganisms is the most important factor affecting the dissipation of the herbicide from agricultural soils in the long term (Smith 1969, 1970; Anderson and Domsch 1980a, 1980b; Smith and Hayden 1981, 1982a; Anderson 1984; Smith and Milward 1985). This is particularly true when triallate is incorporated into the soil (Banting 1967; Kaufman 1967).

Most temperate agricultural soils contain microorganisms and/or systems of cell-free enzymes that can degrade triallate (Anderson and Domsch 1980b). The overall rate of metabolism of herbicides in soils is a function of (1) the amount of herbicide in the soil and its distribution, (2) the amount of the enzymatic material in the soil (both within and outside the microbial cells) and its distribution, and (3) the activity level of the enzymatic degradation systems. The herbicide bioavailability is influenced by variables such as soil moisture, temperature, aeration, pH, nutrient status, and organic content. Rates of triallate metabolism are expected to change

temporarily with changes in these factors. Thus, a linear relationship between microbial biomass and triallate degradation has not always been supported by the available data (Anderson 1981, 1984). Also, as the total amount of triallate in the soil decreases with time, the availability of the herbicide to the degradation systems is reduced and the rate of degradation declines.

For most herbicides, the pool of enzymatic material that accounts for the biodegradation potential usually requires no induction period for the initiation of biodegradation (Anderson and Domsch 1976). An exception is triallate; Banting (1967) reported a lag period for the initiation of triallate biodegradation.

Very little information is available concerning the metabolic pathways and metabolites of triallate degradation in soil. In a series of laboratory investigations, the major products of triallate degradation were reported to be CO_2 and soil-bound residues, the formation of which was related to the water content of the soil (Anderson and Domsch 1980a). Almost without exception, the quantity of the unextractable residues was initially greater than CO_2 production. Over longer periods of time, CO_2 production was found to increase relative to the unextractable residues as would be expected as the residues were biodegraded. In addition, degradation products also included traces of water- and benzene-soluble metabolites (Anderson and Domsch 1980b; Anderson 1981).

Climatic factors have been reported to strongly affect the degradation of triallate in soils (Heinonen-Tanski *et al.* 1985), with warm soil temperatures being more conducive to the breakdown process than cold soil temperatures (Smith 1970; Conn and Cameron 1988). Increasing soil moisture also appears to increase triallate breakdown. Soil moisture not only acts as a solvent making herbicides available for degradation, but also influences microbial biomass in the soil (Anderson 1981, 1984). Degradation appears to be retarded as soil moisture falls below field capacity (McKercher and Thangudu 1982); moisture levels in excess of the wilting point are considered to be required for effective microbial degradation (Smith 1970, 1971). During the summer months, soils of the Canadian prairies typically have moisture levels well below field capacity, and thus microbial activity and, consequently, triallate degradation are expected to be low (Smith 1969). In flooded soils, persistence of triallate suggests that anaerobic conditions are unfavourable for microbial degradation (McKercher and Thangudu 1982).

A review of the microbial breakdown of the general category of thiocarbamates failed to provide information concerning triallate degradation, but suggested the possible metabolic processes affecting this family of herbicides (Kaufman 1967). The possible sites of metabolic attack on the thiocarbamate molecule are the alkyl groups, the amide linkage, or the ester linkage. The initial site of attack is determined by the nature of the alkyl groups attached to the amide linkage; in the presence of relatively small alkyl groups at the ester linkage, the thiocarbamate molecule is likely to be hydrolyzed at the ester linkage. Triallate in aqueous solution, however, has been found to be resistent to hydrolysis over a pH range of 4–8. Only a maximum of 15% of the herbicide was degraded in this manner over 24 weeks (Smith 1969).

Mobility and Leaching— Adsorption of triallate to soil clay and organic matter combined with the low water solubility of the herbicide are considered important factors contributing to the low leachability of triallate in soils. The mobility or leaching of triallate in field soils can be expected to be minimal due to its strong adsorption to soils (Smith 1971; Grover, Banting, and Morse 1979; Grover 1983). This is supported by observations of negligible triallate residue movement beyond the depth of soil incorporation (Fryer and Kirkland 1970; Smith 1970, 1971, 1975; Fryer, Smith, and Hance 1980; Smith and Hayden 1982a, 1982b). Approximately 96% of the applied granular triallate remained in the upper 0–1 cm of laboratory soil columns after 15.2 cm of simulated rainfall was applied at 2.5 $cm \cdot h^{-1}$ (Beestman and Deming 1976). The addition of an emulsifier to the granules enhanced triallate movement through the soil; four times more triallate was moved beyond 1 cm, but 95% of it was concentrated in the upper 3 cm of the soil. In a similar experiment, only 5%–13% of the triallate applied to two soil types (Regina heavy clay and Weyburn loam) was eluted from columns with 23 cm of simulated rainfall (Smith 1969). Since the annual summer rainfall on the Canadian prairies is normally less than 25.4 cm, it is thought that excessive leaching of triallate in the field is unlikely (Smith 1969).

The extent of adsorption of a substance to various soils is often described by the Freundlich equation, $X/M = kC^n$, where X is the mass of

adsorbed solute, M is the adsorbent (sediment or soil) mass, C is the equilibrium concentration, and k (the adsorption coefficient) and n are estimated from the linear regression of log X/M vs. log C (Grover, Banting, and Morse 1979). X/M has units of soil-adsorbed concentration ($\mu g \cdot g^{-1}$) (B.T. Bowman, 1990, Agriculture Canada, London, Ont., pers. com.). The adsorption coefficients for triallate on various soils from England and Saskatchewan have been reported to range from 23 to 150 $\mu g^{(1-n)} \cdot mL^{-n} \cdot g^{-1}$ (concentration range at equilibrium 4–30 $\mu g \cdot L^{-1}$ soil and 0.03–0.9 $\mu g \cdot mL^{-1}$ solution, n ranging from 0.96 to 0.98) (Hance, Holroyd, and McKone 1973; Grover, Banting, and Morse 1979). Triallate is strongly adsorbed to soil colloids, and this may be the most important factor regulating its availability in soil. Between 79%–96% of the original amount of triallate in aqueous solutions, ranging in concentrations from 0.5 to 3 $mg \cdot L^{-1}$, was adsorbed by several Saskatchewan soils. As well, the soil solution concentrations of triallate at equilibrium were well below its solubility in water (Grover, Banting, and Morse 1979).

The structure of triallate (Fig. 1) supports the suggestion that adsorption will be by nonionic interactions (Grover, Banting, and Morse 1979). Thus, pH has little effect on adsorption of triallate to soils (Grover 1974). A report of triallate adsorption increasing with decreasing pH was attributed to the strong inverse relationship between organic matter content and pH of soils (Grover, Banting and Morse 1979).

Triallate is strongly adsorbed on hydrophobic, organic adsorbents, such as activated charcoal, peat moss, and cellulose, and is negligibly desorbed by water. Wheat straw, which is a mixture of cellulose, hemi-cellulose, lignin, and proteins, also exhibits strong adsorption of triallate coupled with minimal desorption by water (Grover 1974). Triallate mobility in soils, in contrast to volatization, is not substantially affected by emulsifying agents used in some triallate formulations.

Organic matter content appears to be one of the most important factors governing the adsorption of triallate in soils. A positive relationship between soil organic matter and adsorption of triallate has been found by various investigators (Smith 1970; Hance, Holroyd, and McKone 1973; Beestman and Deming 1976; Jury *et al.* 1980). Organic matter content is highly correlated ($r = 0.97$) with the triallate adsorption coefficients for several Saskatchewan soils and is considered to be the most important factor affecting the behaviour of triallate in these soils (Grover, Banting, and Morse 1979).

Khan (1973) studied the nature of a triallate-montmorillonite complex and showed that triallate adsorption onto clay is by complexation of the triallate carbonyl group to the exchangeable cations on the clay. The triallate-montmorillonite complex was stable even on heating to 50°C under dry conditions, but when shaken with distilled water, it was completely displaced from the clay (Khan 1973). The affinity of triallate for clay explains its higher persistence in clay-enriched soils at field capacity moisture levels (Smith and Fitzpatrick 1970).

Photodecomposition—The dissipation of triallate from soil occurring as a result of photodecomposition does not appear important (WSSA 1983). The ultraviolet absorption spectrum of triallate does not indicate absorption at wavelengths greater than 280 nm. Since the spectrum of solar radiation at the earth's surface has a minimum wavelength of about 290 nm, photodecomposition is not expected to be a determining component in the dissipation of triallate from the soil (Beestman and Deming 1976). Minimal losses of triallate from photodecomposition were reported by Grover, Banting, and Morse (1979).

Water and Sediment

Compared to soil studies, information related to the fate and persistence of triallate in the aquatic environment is scarce. Although triallate might react with available free radicals and be subjected to photochemical reactions, specific data supporting this hypothesis were not found (U.S. EPA 1983). Based on the previously discussed work of Smith (1969), who found low (10%–15%, pH 4–8) hydrolyzation values, this mode of action for triallate dissipation is not expected to be a significant degradation factor in the aquatic environment.

Studies of triallate biodegradation in water or sediments were not found. Retention of triallate in flooded soils suggests that anaerobic conditions in sediments are not favourable for microbial degradation (McKercher and Thangudu 1982).

The measured half-life of triallate in aquatic systems is available from only one study. Monsanto Company (1987) measured the half-life of triallate in water to range between 3 and 15 days under various

laboratory conditions. A major portion of the loss, however, was due to the volatilization. More details of this study were not provided (P. Marshall, 1991, Monsanto Canada, Ottawa, pers. com.). A Henry's law constant has been estimated by Suntio *et al.* (1988) at 1.02 Pa $m^3 \cdot mol^{-1}$. Volatilization from water may or may not be significant depending on the rates of competitive processes (Suntio *et al.* 1988). The half-life of triallate in water due to volatilization has been estimated to be "several days" (U.S. EPA 1983). This estimate was based on the known vapour pressure and water solubility of triallate and data for the volatilization from water of the closely related herbicide diallate. Muir (in press) has predicted that triallate will volatilize rapidly from shallow waters based on its high transfer coefficient and has estimated that the half-life for volatilization from water of 1-m depth (20°C) would be 8 d.

The strong adsorption of triallate from aqueous solution onto soil particles (95% to a Regina heavy clay and Weyburn loam) (Smith and Fitzpatrick 1970) indicates that adsorption onto particulate material in the aquatic environment is a major fate process. The sediment detections reported by Therrien-Richards and Williamson (1987) in the La Salle River in Manitoba (16.9–119 $ng \cdot g^{-1}$, Appendix A) support this assumption.

RATIONALE

Raw Water for Drinking Water Supply

Guideline

The Federal–Provincial Subcommittee on Drinking Water has recommended a maximum acceptable concentration of 230 $\mu g \cdot L^{-1}$ as the Canadian drinking water quality guideline for triallate (Health and Welfare Canada 1989).

Concentrations in Drinking Water

Published measurements of triallate in treated (municipal and private) water in Canada were not found (Hiebsch 1988).

Freshwater Aquatic Life

Bioaccumulation

Published studies on the experimental bioaccumulation of triallate in aquatic animals were not found in the scientific literature. However, several unpublished studies provide preliminary bioaccumulation data. Monsanto Company (1982) found that the daily bioconcentration factors during the exposure phase ranged from 210 to 574 for channel catfish *(Ictalurus punctatus)*, and from 282 to 778 for bluegill sunfish (*Lepomis macrochirus).* In both cases, rapid elimination occurred within 2 weeks during the depuration period. Environment Canada (1990) has a preliminary report on the bioconcentration potential of triallate in rainbow trout (*Oncorhynchus mykiss*). In this study, trout were exposed to triallate at a mean measured concentration of 0.14 $\mu g \cdot L^{-1}$ in a continuous flow system; steady state body burdens of 0.069 $\mu g \cdot L^{-1}$ were achieved after 3 d of exposure; and BCFs of 789 to 838 were generated by the static model (mean fish concentration divided by mean water concentration). An estimated bioconcentration factor of 150 was published by Kenaga (1980) based on equations developed by Kenaga and Goring (1980). Using the equations published by Chiou *et al.* (1977), a log octanol/water partition coefficient of 4.6 can be calculated. This value would seem to suggest a higher bioconcentration factor than 150. Triallate, however, is known to be easily metabolized and excreted by terrestrial animals (Khokhol'kova and Pestova 1969; Zhavoronkov, Polyakova, and Verkhovskii 1972; Marsden and Casida 1982). The same would be expected of aquatic animals, thus limiting an organism's ability to retain (i.e., bioaccumulate) triallate.

Although triallate could not be detected in the water of the La Salle River, Manitoba, with a detection limit of 0.10 $\mu g \cdot L^{-1}$, it was detected in three species of forage fish (brown bullhead, brook stickleback, and central mudminnow). The tissue concentrations ranged from 3.3 to 9.2 $ng \cdot g^{-1}$, with a detection limit of 2.7 $ng \cdot g^{-1}$ (Therrien-Richards and Williamson 1987). If the detection limit for triallate in water (0.10 $\mu g \cdot L^{-1}$) is used with the maximum tissue concentration reported (9.2 $ng \cdot g^{-1}$), a bioaccumulation factor of 92 results. Water concentrations below 0.10 $\mu g \cdot L^{-1}$ would produce higher bioaccumulation factors, which could be similar to the value of 150 as predicted by Kenaga (1980).

At four sampling locations in the La Salle River, Therrrien-Richards and Williamson (1987) found no bioaccumulation of triallate in an aquatic macrophyte *Myriophyllum* sp. (detection limit 2.7 $ng \cdot g^{-1}$).

Toxicity to Aquatic Organisms

Acute Lethal Toxicity

Vertebrate acute toxicity data for technical triallate (95.3% ai) consists of 24-h LC_{50}s of 1300 $\mu g \cdot L^{-1}$ for rainbow trout (*Oncorhynchus mykiss*) and 2500 $\mu g \cdot L^{-1}$ for channel catfish (*Ictalurus punctatus*). The 96-h LC_{50}s are 620 and 1700 $\mu g \cdot L^{-1}$ for the respective species. Tests conducted with the formulated emulsifiable concentrate (46.3% ai) produced 24-h LC_{50}s of 1300 and 1800 $\mu g \cdot L^{-1}$ for rainbow trout and channel catfish, respectively. The 96-h LC_{50}s are 1000 and 1100 $\mu g \cdot L^{-1}$ for the respective species (Mayer and Ellersieck 1986).

Invertebrate aquatic organisms were considerably more sensitive to triallate. Invertebrate acute toxicity testing using technical triallate produced 48-h LC_{50}s of 80 $\mu g \cdot L^{-1}$ for first instar *Daphnia magna* (Mayer and Ellersieck 1986) and a 48-h EC_{50} of 2300 $\mu g \cdot L^{-1}$ for fourth instar *Chironomus riparius* (Buhl and Faerber 1989). A 96-h test using third instar *Chironomus plumosus* produced an LC_{50} of 490 $\mu g \cdot L^{-1}$ (Mayer and Ellersieck 1986).

Acute toxicity of the emulsifiable concentrate formulation ranged from a 48-h LC_{50} of 57 $\mu g \cdot L^{-1}$ for first instar *D. magna* (Mayer and Ellersieck 1986) to an EC_{50} of 1230 $\mu g \cdot L^{-1}$ for *C. riparius* (Buhl and Faerber 1989). A summary of the limited acute toxicity data of triallate to aquatic vertebrates and invertebrates is presented in Appendix C. A solvent carrier was not used in the development of toxicity data by Mayer and Ellersieck (1986). An acetone solvent carrier was used in one of the controls to simulate the formulation additive in the tests conducted by Buhl and Faerber (1989). They found that immobilization and mortality in the untreated control and solvent control did not exceed 10% in any of the tests.

Chronic Toxicity and Sublethal Reactions

Fathead minnow (*Pimephales promelas*) larvae used in 7-d survival and growth tests with triallate demonstrated a sharp dose–response relationship. Mortality was not observed at 202 $\mu g \cdot L^{-1}$, but 100% mortality occurred at 531 $\mu g \cdot L^{-1}$. A 7-d LC_{50} of 330 $\mu g \cdot L^{-1}$ was estimated from the data. Fathead minnow growth (based on the dry weight of fry) was reduced (33%) at 202 $\mu g \cdot L^{-1}$ (lowest-observed-effect concentration), but not at 125 $\mu g \cdot L^{-1}$ (no-observed-effect concentration) producing an estimated MATC (maximum acceptable toxic concentration) of 160 $\mu g \cdot L^{-1}$ (Environment Canada 1989).

As with acute lethality data, the limited chronic data also indicate the greater sensitivity of aquatic invertebrates when compared to vertebrates. Standard 7-d survival and reproduction bioassays conducted using *Ceriodaphnia dubia* produced a more gradual dose–response relationship with mortalities observed over almost the entire exposure range (0.35–531 $\mu g \cdot L^{-1}$). The 7-d LC_{50} was 12 $\mu g \cdot L^{-1}$. Reproduction (measured as the daily production of young) was reduced (by 59%) at a concentration of 2.4 $\mu g \cdot L^{-1}$ (the lowest-observed-effect concentration), but not at 1.3 $\mu g \cdot L^{-1}$. The resulting estimated MATC was calculated to be 1.8 $\mu g \cdot L^{-1}$ (Environment Canada 1989).

Aquatic Plants

Information related to the acute toxicity of triallate to aquatic plants is also scarce. An algal bioassay of 18–36 h duration resulted in less than 50% inhibition of chlorophyll production in *Chlorella pyrenoidosa* when tested with 1000- and 10 000-$\mu g \cdot L^{-1}$ triallate concentrations (Kratky and Warren 1971). More specific data were not generated by these authors. The 10 000-$\mu g \cdot L^{-1}$ triallate solution was produced with either an acetone or methanol solvent carrier; the report did not specify which solvent was used.

Algal bioassays of 2–3 weeks duration using *Selenastrum capricornutum* and the commercial triallate formulation Far-Go (10% ai), in either a natural water or the standard synthetic algal growth medium, were conducted by Turbak, Olson, and McFeters (1986). The EC_{50}, based on algal cell numbers, was 6.20 $\mu g \cdot L^{-1}$ for natural water and 11.2 $\mu g \cdot L^{-1}$ for the synthetic algal growth medium. The upper and lower confidence intervals for both EC_{50}s varied by an order of magnitude and overlapped to such an extent that the two EC_{50}s were not significantly different.

Aquatic Community Studies

In the only community study with triallate found, laboratory microcosms simulating northern prairie wetlands were used. Triallate was introduced as a soil slurry to obtain nominal solution concentrations of 10, 100, and 1000 $\mu g \cdot L^{-1}$ (Johnson 1986). Each 4-L glass microcosm contained 3.8 L of water and sediment from a permanent wetland (hydrosoil) at a

ratio of 9:1 (v/v). After the introduction of the triallate, the microcosms were placed in an environmental chamber (20°C, 1400 lux on a 16-h light, 8-h dark cycle) for a week prior to the introduction of naturally derived macrophytes (*Lemna*, *Ceratophyllum*, and *Elodea*). Natural communities of invertebrates and algae developed in each microcosm.

Prior to triallate additions, 25 mature, gravid daphnids (*Daphnia magna*) were introduced into each microcosm. If, at any time, five or fewer daphnids were observed in a microcosm, an additional 25 daphnids were introduced in an attempt to produce a viable population. Acute toxicity tests (48 h) using the waters recovered from the control and triallate-treated microcosms were conducted at 14 and 30 d posttreatment using first instar *Daphnia magna* and fourth instar *Chironomus riparius*. Forty-eight–hour *D. magna* acute toxicity tests, using microcosm waters at 14 d posttreatment, showed 0%, 60%, and 100% mortality at nominal 10-$\mu g \cdot L^{-1}$, 100-$\mu g \cdot L^{-1}$, and 1000-$\mu g \cdot L^{-1}$ treatments, respectively. Similar tests with chironomids showed that triallate was 100 times more toxic to daphnids than to chironomids. Even after 30 d, water from the 10-$\mu g \cdot L^{-1}$ treatment produced a 50% reduction in the number of adult daphnids surviving a 7-d chronic toxicity test.

This microcosm study demonstrated triallate toxic effects to daphnids and that these effects persisted even at low concentrations. Continued introduction of daphnids was necessary on days 1, 4, 7, 10, and 14 before a viable daphnid population was established in the 10-$\mu g \cdot L^{-1}$ treatment. Daphnid populations could not be established in the 100- and 1000-$\mu g \cdot L^{-1}$ treatments in these time periods. It should be noted that the aqueous concentration of these nominal concentrations is probably much lower, considering that a substantial amount of triallate might be soil-bound. Smith and Fitzpatrick (1970) reported a strong adsorption of triallate from aqueous solution onto soil particles (up to 95%).

The simulation of a drought cycle in the microcosms (i.e., removal of macrophytes, macroinvertebrates, and water, with subsequent replacement of fresh, uncontaminated water and new daphnids) did not change the time required to establish daphnid populations in the 10-$\mu g \cdot L^{-1}$ treatment.

Triallate effects on phytoplankton, as determined by short-term growth bioassays using *Selenastrum capricornutum*, demonstrated that the 100- and 1000-$\mu g \cdot L^{-1}$ treatments reduced algal growth (cell counts) by more than 40% even at 30 d posttreatment. There was no effect on algal growth at 10 $\mu g \cdot L^{-1}$. Aquatic vascular plants were not affected by any treatment. Dissolved oxygen production in microcosms was observed to increase (20% above control levels) at 100 and 1000 $\mu g \cdot L^{-1}$ at days 14, 21, and 28 during the 30-d experiment period. It was implied that this increase was due to a stimulation in photosynthetic productivity in microcosms due to the presence of triallate.

Microbial activity, as measured by respiratory electron transport, glucose metabolism, oxygen consumption, and alkaline phosphatase activity, was not disturbed by the triallate treatments in the microcosm study.

Guideline

The minimum toxicological data requirements for deriving a Canadian water quality guideline (CCME 1991) were not met with the current triallate data base. Derivation of an interim guideline value, however, was possible with the existing data. Mayer and Ellersieck (1986) reported a 48-h median lethal concentration of 57 $\mu g \cdot L^{-1}$ for the invertebrate *Daphnia magna*. A concentration of 2.4 $\mu g \cdot L^{-1}$ triallate was found to affect the reproduction of *Ceriodaphnia dubia*. This was the lowest concentration of triallate found causing a significant effect in an aquatic organism and was subsequently used as the basis for an interim guideline.

Therefore a safety factor of one order of magnitude is appropriate (CCME 1991). The resulting interim guideline is 0.24 $\mu g \cdot L^{-1}$.

Agricultural Uses

Livestock Waters

Toxicity to Livestock and Related Biota

Acute Toxicity—Several Russian studies cited by the U.S. EPA (1983) described the acute oral toxicity of triallate to laboratory and domestic animals. They report single oral dose LD_{50} values of 930 and 1471 $mg \cdot kg^{-1}$ body weight for mice and rats, respectively (Pestova 1968). Single oral dose LD_{50} values of 500 and 945 $mg \cdot kg^{-1}$ were also reported for rabbits and rats, respectively (Verkhovskii 1972). Other reported single oral dose LD_{50}s ranged from 1675–2165 $mg \cdot kg^{-1}$ for rats to >20 000 $mg \cdot kg^{-1}$ for dogs (Wiswesser 1976). The acute oral LD_{50} for the northern bobwhite quail (*Colinus virginianus*) was

reported to be >2251 mg•kg^{-1} body weight. The dietary concentration is >5000 mg•kg^{-1} feed to bobwhite quail and mallard duck (Smith 1987).

Subacute and Chronic Toxicity—Most of the data on triallate subacute and chronic toxicity comes from brief manufacturers' reports and abstracts of Russian research papers. The manufacturer of triallate (Monsanto) reports that dietary concentrations of 10, 30, or 100 mg•kg^{-1} of feed ingested by rats (approximately 0.5, 1.5, or 5 mg•kg^{-1}•d^{-1}) for three generations produced no treatment-related effects. A dietary concentration of 200 mg•kg^{-1} (about 10 mg•kg^{-1}•d^{-1}) produced depressed weight gain in female rats during a 2-yr study. However, neither gross pathological changes nor abnormal hematological indices were observed at this level (Johannsen *et al.* 1977). Detailed supporting data for these claims were not presented.

Abstracts of Russian papers report edema and plethora in the brains of rats that were fed triallate at 14.7 mg•kg^{-1} body weight for 4 months (Rappoport and Pestova 1974). The maximum tolerated single oral dose of 1000 mg•kg^{-1} caused decreased succinic and lactic dehydrogenase activity, decreased hepatic thiol content, and an increased hepatic pyruvic acid level (Pestova 1968). An increase in RNAase activity of the liver and spleen and disruption in normal thyroid gland function were also reported due to a single oral dose of 1000 mg•kg^{-1} (Voitenko *et al.* 1967). Other subacute reactions are the inhibition of acetylcholinesterase activity in the peripheral and central nervous system and decreased osmotic resistance of erythrocytes (Zhavoronkov, Verkhovskii, and Evdokimov 1973). Sheep and pigs administered a single oral dose of 300 mg•kg^{-1} exhibited altered hematological parameters including transient changes in total plasma protein content, increased albumin, decreased globulin, decreased RNA and DNA, and increased free nucleotide levels (Verkhovskii 1972; Verkhovskii, Zhavoronkov, and Evdokimov 1973; Zhavoronkov and Verkhovskii 1975).

Concern about a possible delayed neurotoxic effect of triallate, which has been observed with the similar compound diallate, led to studies using white leghorn hens. Hens given 300 mg•kg^{-1} twice a day for 3 d exhibited mild, transient ataxia and leg weakness at 19 d posttreatment. A similar dosage schedule using 400 mg•kg^{-1} produced moderate ataxia and lethargy at 5 d post-treatment. Recovery from these symptoms occurred in 4 d (Fisher and Metcalf 1983). Doses of 340–420 mg•kg^{-1}•d^{-1} administered to mature white leghorn hens in gelatin capsules for 25 d caused greater than 40% weight loss. The condition of these birds continued to decline until they were sacrificed on day 36. Gross examination of the gastrointestinal tract revealed a few 1- to 2-mm lesions in the gizzard. A dosage of 85–105 mg•kg^{-1}•d^{-1} for 25 d did not cause a decrease in weight or egg production in spite of a transient decrease in food consumption. As well, ataxia and narcosis were not evident (Hansen *et al.* 1985).

Uptake, Metabolism, and Elimination—Triallate is rapidly absorbed from the gastrointestinal tract. Ingested triallate at 1000 or 1471 mg•kg^{-1} appears in the blood 15 min after a single oral dose and attains a maximum level in 30 min (Khokhol'kova and Pestova 1969).

The metabolism of triallate in rats involves the formation of trichloroacrylic acid by the microsomal oxidases. Formation of the trichloroacrylic acid is thought to be via the NADPH-dependent S-methylene hydroxylation of triallate to unstable, highly reactive intermediate trichloroacroleins (Marsden and Casida 1981, 1982). Microsomal incubation of triallate results in the rapid formation and glutathione conjugation of trichloroacrolein (Hackett *et al.* 1990).

Complete elimination from rabbits of single oral doses of 500 mg•kg^{-1} triallate occurred in 7 d (Zhavoronkov, Polyakova, and Verkhovskii 1972). Single oral doses of 1000 or 1471 mg•kg^{-1} were completely eliminated from the bodies of rats in 1–3 d (Khokhol'kova and Pestova 1969).

Carcinogenicity, Mutagenicity and Teratogenicity—Manufacturer testing of triallate, using male and female rats consuming dietary concentrations of 50, 100, and 200 mg•kg^{-1}, did not indicate a tumorogenic response in terms of the number of rats with tumors, the number of tumors per rat, or the number of rats with malignant neoplasms. In addition, there were no gross pathological changes or differences in survival (Johannsen *et al.* 1977). Additional information concerning the carcinogenic potential of triallate was not found.

A large amount of mutagenicity information, obtained using a variety of test systems, is available in the published literature. A compilation and a review of these data were published by Carerer and Morpurgo (1981). Triallate produces a mutagenic

response in the *Salmonella typhimurium* strains TA100 and TA1535, both with and without metabolic activation (De Lorenzo, Silengo, and Cortese 1976; De Lorenzo *et al.* 1978; Carerer, Ortali, Cardamone, and Morpurgo 1978; Carerer, Ortali, Cardamone, Torracca, and Raschetti 1978; Sikka and Florcyzk 1978; Sandhu and Waters 1980; Douglas *et al.* 1981a, 1981b; Kasica, Sanhu, and Waters 1981; Sandhu *et al.* 1981, 1984; Shiau, Huff, and Felkner 1981; Wildeman and Nazar 1982).

Dose-related increases in base substitution and frameshift mutations were noted for triallate in *S. typhimurium* strains TA100, TA1535, and TA98. A positive mutagenic response, however, was not observed in strains TA1537, TA1536, and TA1538. For those strains exhibiting a positive reaction, triallate is considered to be a direct-acting, mutagen-inducing, base pair substitution (U.S. EPA 1983). Triallate also induced forward mutations in *Saccharomyces coelicolor* (Carerer, Ortali, Caramone, and Morpurgo 1978; Carerer, Ortali, Cardamone, Torracca, and Raschetti 1978) and in *Aspergillus nidulans* (Morpurgo *et al.* 1977).

Mutagenic responses were not found for *Escherichia coli* WP2, bacteriophages, and *Saccharomyces cerevisiae* D7 using reverse mutation criteria (Andersen, Leighty, and Takahashi 1972; Kasica, Sandhu, and Waters 1981; Sandhu *et al.* 1981). These authors, however, reported a significant increase in mitotic recombinations in *S. cerevisiae* D3 exposed to triallate with and without metabolic activation.

Triallate was shown to be mutagenic in tests using mammalian cells. Chinese hamster ovary cells exhibited dose-related increases in the frequency of chromosomal aberrations, sister chromatid exchanges, and cytotoxicity indicative of the clastogenic (i.e., breaking) effect that triallate has toward chromosomes (Douglas *et al.* 1981a, 1981b). The L5178Y mouse lymphoma thymidine kinase assay is also positive for triallate mutagenicity (Kasica, Sandhu, and Waters 1981; Sandhu *et al.* 1981). *In vitro* studies showed that triallate metabolism by the microsomal fraction of PCB-induced rat liver homogenate produced a mutagenic substance (Distlerath, Loper, and Tabor 1982, 1985). At a concentration of 100 $mg \cdot L^{-1}$, triallate caused 57% inhibition of DNA synthesis in rat thymocytes, and a 52% inhibition of DNA synthesis and a 5% inhibition of unscheduled DNA synthesis in human lymphocytes (Rocchi *et al.* 1980). The weight of evidence in the scientific literature implies that triallate is a potential mutagen that is capable of acting with or without metabolic activation. Triallate, however, does not demonstrate a positive mutagenic response in all tests (U.S. EPA 1983).

Data pertaining to the teratogenicity of triallate are scarce. A manufacturer's study with rabbits using orally administered doses of 3 and 10 $mg \cdot kg^{-1}$ body weight on days 6–18 of gestation reportedly did not induce teratogenic responses in the offspring (Johannsen *et al.* 1977). Access to experimental data was not possible since the report was written in abstract form.

Guideline

Insufficient data are available for the determination of a safe concentration of triallate in livestock watering supplies. The mammalian toxicity data used to derive the guideline for triallate in drinking water supplies were proprietary and not available for this report. In accordance with the procedure established by the CCREM (1987), the guideline for drinking water supplies (230 $\mu g \cdot L^{-1}$) (Health and Welfare Canada 1989) is used as the interim guideline for livestock watering supplies.

Irrigation Waters

Toxicity to Nontarget Plant Species

Various laboratory and field studies have detailed the toxicity of triallate to nontarget plants, especially the domestic oat (*Avena sativa*). These studies are presented in Appendix D. Sublethal reactions to nontarget plants have been demonstrated by triallate concentrations as low as 1 $mg \cdot L^{-1}$ in an irrigation application (Kratky and Warren 1971) and 0.28 $kg \cdot ha^{-1}$ and 0.11 $mg \cdot kg^{-1}$ as soil applications (McKercher and McGregor 1979). The phytotoxicity of triallate varies and is influenced by a variety of environmental and soil factors. For example, phytotoxicity increases as soil moisture increases. Water appears to compete with triallate for adsorption sites on soil particles and adsorbed triallate may be replaced by water to increase triallate bioavailability. Increased temperature also increases phytotoxicity. This may be due to either reduced triallate adsorption and/or increased herbicidal activity of the available triallate at higher temperatures (Miller and Nalewaja 1976). Soil organic matter is a major determinant of phytotoxicity, with increases in organic matter corresponding to decreases in phytotoxicity (McKercher, Ashford,

and Morgan 1975).

Triallate formulation also influences phytotoxicity, with greater growth inhibition occurring with liquid (i.e., emulsifiable concentrate) formulations than with similar application rates of the granular formulation (Miller and Nalewaja 1976).

Guideline

Various laboratory studies have established that concentrations as low as 1 $mg \cdot L^{-1}$ can cause decreased root and shoot growth in crop species (Kratky and Warren 1971). A definitive dose–response relationship between triallate water concentrations and phytotoxic responses by crop species, however, could not be established from the scientific literature as the concentration range for most of these studies was inadequate. A lowest-observed-effect-application rate (LOEAR) and a no-observed-effect application rate (NOEAR) were not available to derive a species maximum acceptable toxicant concentration (SMATC). Thus, a guideline value for triallate in irrigation water was not derived at this time.

Recreational Water Quality and Aesthetics

Organoleptic Effects

Reports dealing with triallate-caused taste and odour of water and tainting of fish flesh were not found.

Guideline

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

Industrial Water Supplies

Guideline

At present, the CCME lacks the necessary information to set water quality guidelines that will protect industrial water uses from most chemical compounds. A survey of industry water quality needs is being conducted, and upon completion, it should be possible to set guidelines for many chemicals, including triallate, to protect this water use.

SUMMARY

After an evaluation of the published information on the herbicide triallate, water quality guidelines were derived (Table 3). The background information on triallate 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.

Table 3. Recommended Water Quality Guidelines for Triallate

Uses	Guidelines
Raw water for drinking water supply	230 $\mu g \cdot L^{-1}$ (MAC)*
Freshwater aquatic life	0.24 $\mu g \cdot L^{-1}$ (interim)
Agricultural water uses	
Livestock waters	230 $\mu g \cdot L^{-1}$ (interim)
Irrigation waters	No recommended guideline
Recreational water quality and aesthetics	No recommended guideline
Industrial water supplies	No recommended guideline

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

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Appendix A

Environmental Concentration Ranges of Triallate Residues in Canadian Surface Water, Groundwater, Atmosphere, Sediment, and Biota

Table A-1. Environmental Concentration Ranges of Triallate Residues in Canadian Surface Water, Groundwater, Atmosphere, Sediment, and Biota

Location, years, and conditions	Matrix	Concentration range (& mean)	Samples with pesticide/ number of samples	Reference
Saskatchewan, 300 km north of Regina (Melfort) and Regina. Accumulative air samples, 24-h basis. First week of May till early mid-November for the years 1981 and 1982.	Air	1982 <1 $ng \cdot m^3$–160 $ng \cdot m^3$ 1981 <1 $ng \cdot m^3$–25 $ng \cdot m^3$	NR	Grover, Kerr, *et al.* 1988
Saskatchewan, 300 km north of Regina (Melfort) and Regina. Accumulative air samples, 24-h basis. First week of May till early mid-November for the years 1978 and 1979.	Air	1979 <1 $ng \cdot m^3$–104 $ng \cdot m^3$ 1978 <1 $ng \cdot m^3$–198 $ng \cdot m^3$	NR	Grover, Kerr, and Khan 1981
Ochre River, western Manitoba. 3.5-L grab sampling in duplicate on March 14, April 13, April 27, and at weekly intervals afterward until Sept. 5/84. Final collection on Oct. 10/84. Drains mainly noncropped land and forest.	Surface water	Detectable levels (T > 3 $ng \cdot L^{-1}$) found only in October. Avg. for Oct. was 6.4 $ng \cdot L^{-1}$.	NR	Muir and Grift 1987
Turtle River. As above. Drains mainly agricultural land.	Surface water	May - 10.4 $ng \cdot L^{-1}$ June - 9.9 $ng \cdot L^{-1}$ July - 2.7 $ng \cdot L^{-1}$ Sept. - 3.7 $ng \cdot L^{-1}$ Oct. - 5.5 $ng \cdot L^{-1}$ (detection limits = 3 $ng \cdot L^{-1}$)	NR	Muir and Grift 1987
La Salle River, Manitoba. One grab sample per 7 sampling sites at 30-d intervals from Aug. to Dec. 1984 at midstream. Drains agricultural land.	Surface water	ND (detection limit = 0.10 $\mu g \cdot L^{-1}$)	NR	Therrien-Richards and Williamson 1987
Sampling with dredge at 3 equidistant points across stream width at each sampling location on 1 occasion in Aug. 1984 (1 sample per sampling site) in above study area.	Sediment	16.9–119 $ng \cdot g^{-1}$ (detection limit = 2.7 $ng \cdot g^{-1}$)	9/21	Therrien-Richards and Williamson 1987
100 g sampled from each site on 1 occasion Aug. 1984 from 4 sampling locations in above study area.	Aquatic macrophyte *Myriophyllum* sp.	ND (detection limit = 2.7 $ng \cdot g^{-1}$)	NR	Therrien-Richards and Williamson 1987

ND = not detected
NR = not reported
T = trace

Table A-1. Continued

Location, years, and conditions	Matrix	Concentration range (& mean)	Samples with pesticide/ number of samples	Reference
Samples of small forage fish. Samples equal 100 g of each fish from species from 4 sampling sites and 3 sub-samples at 1 site for a total of 6 samples.	Fish tissue: brown bullhead (*Ictalurus nebulosus*),	<2.7–4.2 $ng \cdot g^{-1}$	NR	Thierren-Richards and Williamson 1987
	brook stickleback (*Culaea inconstans*),	3.3 $ng \cdot g^{-1}$	NR	
	central mudminnow (*Umbra limi*)	<2.7–9.2 $ng \cdot g^{-1}$ (detection limit = 2.7 $ng \cdot g^{-1}$)	NR	
LaSalle River, Manitoba. Sampling interval clustered during April 1983 to coincide with snowmelt water runoff and at monthly intervals from May 1983 to March 1984 (excluding Aug. 1983). Drains agricultural land, 2 sampling locations.	Spring runoff water	0.02–0.15 $\mu g \cdot L^{-1}$ (detection limit) = 0.05 $\mu g \cdot L^{-1}$	27/27	Williamson 1984
Assiniboine River, Manitoba. Sampling at monthly intervals from May 1983 to March 1984 (excluding Aug. 1983). Drains agricultural land, 2 sampling locations.	Surface water	(Detection limit = 0.05 $\mu g \cdot L^{-1}$)	7/15	Williamson 1984
April 11, 1983; samples collected on 1 day from 2 water pools.	Surface water	Trace (detectable but <0.05 $\mu g \cdot L^{-1}$)	2/3	Williamson 1984
June 1, 1983; 1 sample collected from 1 pool.		ND (<0.05 $\mu g \cdot L^{-1}$)		
Study area 2800 ha operated by 17 farmers and the City of Regina. Sampling on a daily basis for duration of runoff event at 4 culverts crossing into study area at a stream connecting 2 permanent sloughs and at a culvert exiting the lower slough; 7 sampling locations.	Spring water runoff	0.4678 $\mu g \cdot L^{-1}$ at one site on Mar 27; 0.6443 $\mu g \cdot L^{-1}$ at same site on March 28; below detection limit (0.1 $\mu g \cdot L^{-1}$) at all other sites and times	NR	Waite *et al.* 1986
Assiniboine River, Manitoba (downstream Trans-Canada Highway). One midstream grab sample per site at 30-d intervals from Aug. to Dec. 1984. Drains agricultural land.	Surface water	ND (detection limit = 0.1 $\mu g \cdot L^{-1}$)	NR	Therrien-Richards and Williamson 1987
Sampling by hand of fine-grained deposits on lee side of midstream obstructions (sand bars and rocks) on 1 occasion Aug. 1984. Data reported for only 1 sampling site; study area as above.	Sediment	ND (detection limit = 2.7 $ng \cdot g^{-1}$)	NR	Williamson 1984

Table A-1. Continued

Location, years, and conditions	Matrix	Concentration range (& mean)	Samples with pesticide/ number of samples	Reference
Samples of small forage fish. Samples equal 100 g of each fish species; study area as above.	Fish tissue: silver chub (*Hybopsis storeriana*), stone cat (*Noturus flavus*), channel catfish (*Ictalurus punctatus*), brown bullhead (*Ictalurus nebulosus*)	ND (detection limit = 2.7 $ng \cdot g^{-1}$)	NR	Therrien-Richards and Williamson 1987
Red Deer River, Bindless, Alberta, Emerson, Manitoba, Selkirk, Manitoba, from May 1960 to February 1988.	Surface water	0.1–0.08 $\mu g \cdot L^{-1}$	6/95	NAQUADAT 1991
Souris River, Manitoba, at Coulter to Wawanesca from May 1960 to February 1988.	Surface water	0.01–0.72 $\mu g \cdot L^{-1}$	4/28	NAQUADAT 1991
Qu'Appelle River, Saskatchewan, from November 1975 to December 1987.	Surface water	0.01–0.046 $\mu g \cdot L^{-1}$	2/44	NAQUADAT 1991
Canot River, Saskatchewan, from October 1973 to January 1978	Surface wter	0.028 $\mu g \cdot L^{-1}$	1/45	NAQUADAT 1991
Churchill River, Saskatchewan, from April 1974 to January 1988.	Surface water	0.024 $\mu g \cdot L^{-1}$	1/36	NAQUADAT 1991
Reservoirs receive snowmelt water from a 640-ha study area located 10 km north of Regina. Sampling was done on a weekly basis in 1985 and twice in 1987 from one location in each of 2 reservoirs.	Surface water	0.22 µg/L maximum with a mean of 0.11 µg/L (no range given)	23/64	Waite *et al.* 1990
Sampling in a 2800-ha study area located north of Regina during 2 brief periods of melt separated by a month of cold weather in 1987 from 7 sites in study area.	Spring runoff water	0.98 $\mu g \cdot L^{-1}$ maximum with a mean of 0.38 $\mu g \cdot L^{-1}$ (no range given)	19/22	Waite *et al.* 1990
Sampling on 9 sequential days in 1985 from 6 sites in above study area.	Spring runoff water	0.62 $\mu g \cdot L^{-1}$ maximum with a mean of 0.19 $\mu \cdot L^{-1}$ (no range given)	36/37	Waite *et al.* 1990

Table A-1. Continued

Location, years, and conditions	Matrix	Concentration range (& mean)	Samples with pesticide/ number of samples	Reference
Sampling from 4 locations, 10 km north of Regina in summers, 4 times in 1987 from 4 iron stand pipes installed in a surficial aquifer.	Groundwater	0.63 $\mu g \cdot L^{-1}$ maximum with a mean of 0.15 $\mu g \cdot L^{-1}$ (no range given)	7/105	Waite *et al.* 1990
80-ha study area located in Saskatchewan; groundwater samples from SIDC piezometers in summer of 1987 on 2 separate days.	Groundwater	0.13–0.39 $\mu g \cdot L^{-1}$ range, 0.10 $\mu g \cdot L^{-1}$ detection limit	3/13	Maathuis *et al.* 1988
Study area 11 km^2 located in township 30 in Saskatchewan; water samples taken from 3 piezometers and 2 canals near piezometers on 30 separate days.	Groundwater	0.13–0.15 $\mu g \cdot L^{-1}$ range, 0.1 $\mu g \cdot L^{-1}$ detection limit	5/18	Maathuis *et al.* 1988

Appendix B

Summary of Triallate Persistence Studies in Soil

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

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Begbroke Hill, Yarnton, Oxford. Soil type NR	1.7 kg•ha^{-1} Spring 1969	0–15 cm	1.35 kg•ha^{-1} (0 wks) 0.24 kg•ha^{-1} (22 wks)	These experiments were begun in 1963 when triallate was applied at 1.7 kg•ha^{-1} pre-emergence to wheat and barley.	Fryer, Smith, and Hance 1980
	Spring 1970		1.39 kg•ha^{-1} (0 wks) 0.26 kg•ha^{-1} (18 wks)		
	Spring 1971		1.61 kg•ha^{-1} (0 wks) 0.18 kg•ha^{-1} (22 wks)		
	Spring 1972		1.23 kg•ha^{-1} (0 wks) 0.21 kg•ha^{-1} (23 wks)	Herbicide as soon as possible after sowing and incorporated to 2.5–5 cm.	
	Spring 1973		1.20 kg•ha^{-1} (0 wks) 0.51 kg•ha^{-1} (21 wks)		
	Spring 1974		1.19 kg•ha^{-1} (0 wks) 0.50 kg•ha^{-1} (24 wks)		
	Spring 1975		0.99 kg•ha^{-1} (1 wk) 0.39 kg•ha^{-1} (27 wks)		
	Spring 1976		0.95 kg•ha^{-1} (0 wks) 0.39 kg•ha^{-1} (21 wks)		
	3.3 kg•ha^{-1} (twice annually)	0–15 cm	5.50 kg•ha^{-1} (after final application - Dec. 1968) 1.27 kg•ha^{-1} (6 mo) 0.62 kg•ha^{-1} (12 mo) 0.26 kg•ha^{-1} (18 mo) 0.24 kg•ha^{-1} (21 mo) 0.19 kg•ha^{-1} (34 mo) 0.09 kg•ha^{-1} (40 mo)	Herbicide applied twice annually from 1963 to 1968 to hand-weeded uncropped plots. Incorporation NR.	
Melfort, Sask. Melfort silty clay (11.7 OM, pH 5.2, field capacity 36%)	1.7 kg•ha^{-1} Oct. 1971 Oct. 1972 Oct. 1973 May 1972	0-5 cm	 75 ± 3% (7 mo) 43 ± 3% (7 mo) 3 ± 1% (5 mo) 35 ± 3% (5 mo) 25 ± 4% (12 mo) 12 ± 4% (17 mo)	6.8 mg triallate added to 20 x 20 cm plots and thoroughly incorporated into top 5 cm of soil.	Smith 1975

NR = not reported
OM = organic matter

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Regina, Sask.	1.7 kg•ha^{-1}	0–5 cm		Applications and sampling	Smith 1975
Regina heavy clay	Oct. 1971		75 ± 7% (5 mo)	carried out during 3rd wk	
(4.2 OM, pH 7.7,	Oct. 1972		23 ± 10% (5 mo)	of October and 2nd wk	
field capacity 40%)	May 1973		11 ± 2% (5 mo)	of May.	
	May 1972		18 ± 2% (5 mo)		
			16 ± 1% (12 mo)		
			12 ± 3% (17 mo)		
Jameson, Sask.	1.7 kg•ha^{-1}	0–5 cm			Smith 1975
Jameson sandy loam	Oct. 1971		54 ± 6% (7 mo)		
(3.2 OM, pH 7.5,	Oct. 1972		37 ± 8% (7 mo)		
field capacity 11%)	May 1973		10 ± 6% (5 mo)		
	May 1972		14 ± 3% (5 mo)		
			7 ± 2% (12 mo)		
			0 (17 mo)		
Regina, Sask.	1.25 kg•ha^{-1}	0–5 cm		5 mg triallate added to	Smith and Hayden 1982a
Heavy clay	Sept. 1979		53% (8 mo)	20 x 20 cm plots in triplicate	
(4.2 OM, pH 7.7,	Oct. 1979		64% (7 mo)	and incorporated to 5 cm.	
field capacity 40%)	Nov. 1979		50% (6 mo)		
	Sept. 1981		22% (8 mo)		
	Oct. 1981		22% (7 mo)		
	Nov. 1981		23% (6 mo)		
White City, Sask.	1.25 kg•ha^{-1}	0–5 cm			
Sandy loam (4.0%	Sept. 1979		56% (8 mo)	Differences in carry-over	Smith and Hayden 1982a
OM, pH 7.6, field	Oct. 1979		62% (7 mo)	between years considered	
capacity 20%)	Nov. 1979		61% (6 mo)	to reflect differences in	
	Sept. 1980		23% (8 mo)	soil moisture and temper-	
	Nov. 1979		61% (6 mo)	ature following soil treatment.	
				Applications made during 1st wk	
	Sept. 1980		23% (8 mo)	of each fall month and soil	
	Oct. 1980		27% (7 mo)	sampled during 2nd wk of May.	
	Nov. 1980		29% (6 mo)		
	Sept. 1981		23% (8 mo)		
	Oct. 1981		20% (7 mo)		
	Nov. 1981		21% (6 mo)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Regina, Sask. Heavy clay (physical characteristics NR)	1.7 kg•ha^{-1} May 1972	0–5 cm	18 ± 2% (5 mo) 16 ± 1% (12 mo) 12 ± 3% (17 mo)	7.8 mg triallate applied to 20 x 20 cm plots and incorporated.	Smith and Hayden 1976
	May 1973		11 ± 2% (5 mo) 9 ± 4% (12 mo) 2 ± 1% (17 mo)		
Melfort, Sask. Silty loam (physical characteristics NR)	1.7 kg•ha^{-1} May 1972	0–5 cm	35 ± 3% (5 mo) 25 ± 4% (12 mo) 12 ± 4% (17 mo)		Smith and Hayden 1976
	May 1973		3 ± 1% (5 mo) 5 ± 4% (12 mo) 0 (17 mo)		
Jameson, Sask. Asquith sandy loam (physical characteristics NR)	1.7 kg•ha^{-1} May 1972	0–5 cm	14 ± 3% (5 mo) 7 ± 2% (12 mo) 0 (17 mo)		Smith and Hayden 1976
	May 1973		10 ± 6% (5 mo) 6 ± 2% (12 mo) 0 (17 mo)		
Regina, Sask. (4.2% OM, pH 7.7)	2.8 kg•ha^{-1} (5 mg•kg^{-1})	0–5 cm	80 ± 6% (2 wk) 50 ± 7% (6 wk) 25 ± 3% (13 wk) 16 ± 5% (21 wk)	8 mg triallate as emulsifiable concentrate diluted with benzene applied to 18 x 18 cm plots immediately incorporated to 5 cm.	Smith 1971
Jameson, Sask. (3.2% OM, pH 7.5)			26 ± 3% (21 wk)		Smith 1971
Indian Head, Sask. (4.2% OM, pH 7.5)			20 ± 3% (21 wk)		Smith 1971
Melfort, Sask. (10.6% OM, pH 5.2)			27 ± 4% (21 wk)		Smith 1971
Tisdale, Sask. (6.7% OM, pH 6.2)			21 ± 7% (21 wk)	Little indication that soil type affects persistence of triallate under field conditions.	Smith 1971

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Laukaa, Finland Fine sand (2.5% OM, pH 5.6)	NR (sprayed 1973–1976)	NR	0.007 $mg \cdot kg^{-1}$ in 1978 (2 yr2 after final application)	Field procedures NR; commercial formulation applied.	Heinonen-Tanski *et al.* 1985
Regina, Sask. Regina heavy clay (4.0% OM, pH 7.5, field capacity 39.7%) and Weyburn, Sask. Weyburn loam (6.5% OM, pH 7.0%, field capacity 28.0%)	1, 2, and 4 $mg \cdot kg^{-1}$		50% (8–11 wk)	Lab study in which herbicide (emulsifiable concentrate of 0.4 $kg \cdot l^{-1}$) mixed with soil, weighed into bottles to make 20-g samples at field capacity. samples at field capacity.	Smith 1969
Saskatchewan Heavy clay (4.2% OM, pH 7.7, field capacity 40%)	1.5 $kg \cdot ha^{-1}$ May 1979 May 1980 May 1981 1.5 $kg \cdot ha^{-1}$ May 1979 May 1980 May 1981	0–5 cm	 34 ± 8% (22 wk) 64 ± 8% (22 wk) 15 ± 9% (22 wk) 46 ± 4% (22 wk) 58 ± 7% (22 wk) 16 ± 3% (22 wk)	6 mg triallate added to 20 x 20 cm plots and incorporated 5 cm. Differences in residue levels between years believed to reflect edaphic and soil moisture conditions.	Smith and Hayden 1982b
Sandy loam (4.0% OM, pH 7.6, field capacity 20%)	1.5 $kg \cdot ha^{-1}$ May 1979 May 1980 May 1981 1.5 $kg \cdot ha^{-1}$ May 1979 May 1980 May 1981	0–5 cm	 28 ± 4% (22 wk) 32 ± 3% (22 wk) 12 ± 1% (22 wk) 32 ± 1% (22 wk) 35 ± 0% (22 wk) 12 ± 2% (22 wk)		Smith and Hayden 1982b
Regina, Sask. Typic Boroll heavy clay (3.1% OM, pH 7.5, field capacity NR)	1.48 $kg \cdot ha^{-1}$ May 20, 1983	0–10 cm	91.2 ± 12.8% (1 d) 70.9 ± 8.8% (3 d) 64.9 ± 10.8% (5 d) 63.5 ± 20.3% (7 d) 54.1 ± 1.4% (28 d) 43.9 ± 13.5% (67 d) 20 ± 10% (160 d)	Shallow cultivation and harrowing of study area on April 27, seeded to wheat on May 9, and application of an emulsifiable concentrate incorporated into the top 5 cm.	Grover, Smith *et al.* 1988b

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Delta Junction, Alaska, Volkmar and Beales silt loams (physical characteristics NR)	0.7, 1.4, or 2.8 kg•ha^{-1} late May 1982	0–15 cm	84 ± 22% (4 wk) 61 ± 14% (17 wk) 54 ± 5% (49 wk) 27 ± 11% (70 wk) 36 ± 8% (103 wk) 14 ± 55% (155 wk)	Triallate incorporated within 2 h of application to a depth of 5.1 cm. Residue values for all rates are averages since application rate did not have an affect on residue persistence.	Conn and Cameron 1988
Regina, Sask. Heavy clay (4.2% OM, pH 7.5, field capacity 40%)	1.5 kg•ha^{-1} May 1977	0-5 cm	30 ± 1% (10 wk) 20 ± 0% (20 wk)	6.0 mg triallate applied to each plot (20 x 20 cm) and immediately incorporated into the top 5 cm	Smith 1979
	May 1978 1.5 kg•ha^{-1} triallate and 0.75 kg•ha^{-1} trifluralin		30 ± 1% (10 wk) 23 ± 1% (20 wk)		
	May 1977		36 ± 3% (10 wk) 27 ± 2% (20 wk)		
	May 1978		24 ± 0% (10 wk) 16 ± 1% (20 wk)		
White City, Sask. Sandy loam (4.0% OM; pH 7.6, field capacity 20%)	1.5 kg•ha^{-1} May 1977	0-5 cm	20 ± 1% (10 wk) 12 ± 0% (20 wk)		Smith 1979
	May 1978		27 ± 4% (10 wk) 14 ± 2% (20 wk)		
	1.5 kg•ha^{-1} triallate and 0.75 kg•ha^{-1} trifluralin				
	May 1977		25 ± 2% (10 wk) 10 ± 1% (20 wk)		
	May 1978		32 ± 1% (10 wk) 20 ± 3% (20 wk)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Regina, Sask. Regina heavy clay (4.2% OM, pH 7.8, field capacity 40%)	2.24 kg•ha^{-1} (4 mg•kg^{-1})	0-5 cm	12 wk 51% (40% soil moisture) 54% (35% soil moisture) 63% (30% soil moisture) 85% (20% soil moisture)	Triallate as a commercial formulation of emulsifiable concentrate (0.4 kg•l^{-1}) incorporated into the top 5 cm of soil.	Smith 1970
Weyburn, Sask. Weyburn loam (6.5% OM, pH 6.5, field capacity 28%)	2.8 kg•ha^{-1} (4 mg•kg^{-1})	0-5 cm	12 wk 43% (30% soil moisture) 47% (25% soil moisture) 48% (20% soil moisture) 60% (15% soil moisture)		Smith 1970
Regina, Sask. Regina heavy clay (physical characteristics given above)	2.8 kg•ha^{-1} (5 mg•kg^{-1})	0-5 cm	14.3%–22.6% (33 wk)	8 mg triallate applied to field plots (18 x 18 cm) and thoroughly incorporated into the top 5 cm.	Smith 1970
Begbroke, Oxford, England coarse, sandy loam (2% OM, pH 7, field capacity NR)	1.68 kg•ha^{-1} May 4, 1963		NR	Triallate applied after sowing and incorporated within 2 h.	Fryer and Kirkland 1970
	April 11, 1964		NR		
	April 1, 1965		NR		
	March 17, 1966	0-15 cm	1.4 kg•ha^{-1} (0 wk) 1.05 kg•ha^{-1} (6 wk) 0.84 kg•ha^{-1} (12 wk) 0.28 kg•ha^{-1} (22 wk) 0.28 kg•ha^{-1} (25 wk) 0.14 kg•ha^{-1} (33 wk) 0.14 kg•ha^{-1} (52 wk)		
	March 21, 1967		0.98 kg•ha^{-1} (0 wk) 0.77 kg•ha^{-1} (6 wk) 0.49 kg•ha^{-1} (14 wk) 0.35 kg•ha^{-1} (22 wk) 0.14 kg•ha^{-1} (34 wk)		
	3.36 kg•ha^{-1} May 4 & Aug. 28, 1963	0-15 cm	NR		
	April 11 & Oct. 28, 1964		NR		
	April 1 & Oct. 22, 1965		NR 2.45 kg•ha^{-1} (21 wk)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
	March 17, 1966		4.13 kg•ha^{-1} (10 wk) 3.15 kg•ha^{-1} (6 wk) 2.24 kg•ha^{-1} (12 wk) 3.08 kg•ha^{-1} (22 wk) 1.26 kg•ha^{-1} (25 wk) 1.19 kg•ha^{-1} (33 wk)		
	Nov. 11, 1966		3.43 kg•ha^{-1} (0 wk) 2.59 kg•ha^{-1} (5 wk) 1.96 kg•ha^{-1} (19 wk)		
	March 21, 1967		4.69 kg•ha^{-1} (0 wk) 2.80 kg•ha^{-1} (6 wk) 2.45 kg•ha^{-1} (14 wk) 1.33 kg•ha^{-1} (22 wk) 0.91 kg•ha^{-1} (35 wk)		
	Nov. 21, 1967		4.20 kg•ha^{-1} (0 wk) 2.59 kg•ha^{-1} (15 wk)		
	March 8, 1968		5.04 kg•ha^{-1} (0 wk) 1.75 kg•ha^{-1} (15 wk)		
	Dec. 6, 1968		5.46 kg•ha^{-1} (0 wk)		
Regina, Sask. Rego Dark Brown Chernozemic (4.2% OM, pH 7.7, field capacity 40%)	1.4 kg•ha^{-1} (2nd wk May 1983)	0-7.5 cm	0.53 ± 0.03 mg•kg^{-1} (6 mo) 0.40 ± 0.02 mg•kg^{-1} (12 mo)	Triallate immediately incorporated to 5 cm after application of commercial formulation.	Smith and Milward 1985
			Aged 6 mo 50% (45 d) 50% (43 d) 50% (43 d) Aged 12 mo 50% (39 d) Fresh comparison 50% (37 d)	50-g samples of the soils with aged triallate residues (6 mo old) weighed into 175-mL cartons, moistened to 85% of field capacity, loosely capped incubated in the dark at 20 ± 1°C.	

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Braunschweig, West Germany Parabrown soil (pH 5.4, field capacity 36.2%, % OM NR)	1 mg•kg^{-1}			Lab study.	Anderson 1981
	2.4% water content		91.2 ± 1.5% (0 wk)		
			60.2 ± 0.6% (10 wk)		
	9.0% water content		94.1 ± 3.2% (0 wk)		
			50% (7 wk)		
			34.4 ± 0.4% (10 wk)		
	12.3% water content		94.2 ± 1.9% (0 wk)		
			50% (6.4 wk)		
			34.7 ± 1.1% (10 wk)		
	16.4% water content		95.0 ± 1.7% (0 wk)		
			50% (5.5 wk)		
			29.8 ± 0.0% (10 wk)		
	19.0% water content		95.3 ± 3.1% (0 wk)		
			50% (4.9 wk)		
			20.8 ± 1.6% (10 wk)		
Regina, Sask. Regina heavy clay (4.0% OM, pH 7.5, field capacity 39.7%)	0.56 kg•ha^{-1}		50% (12 d)	Lab study.	Banting 1967
	1.12 kg•ha^{-1}		50% (20 d)		
			50% (49 d)		
Braunschweig, West Germany Agricultural soil (1.26% total C, pH 5.4, field capacity NR)	0.25 mg•kg^{-1}		95.1% (0 wk)	Lab study.	Anderson and Domsch 1980b
			47.0% (10 wk)		
			36.8% (20 wk)		
			20.6% (52 wk)		
	0.5 mg•kg^{-1}		95.9% (0 wk)		
			46.5% (10 wk)		
			37.2% (20 wk)		
			17.4% (52 wk)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
	1.0 mg•kg^{-1}		96.2% (0 wk) 55.7% (10 wk) 34.5% (20 wk) 13.8% (52 wk)		
	5.0 mg•kg^{-1}		96.2% (0 wk) 74.3% (10 wk) 57.8% (20 wk) 35.3% (52 wk)		
	50.0 mg•kg^{-1}		97.1% (0 wk) 77.1% (10 wk) 64.5% (20 wk) 44.6% (52 wk)		
Braunschweig, West Germany Parabrown soil (% OM NR, pH 5.4, field capacity NR)	1 mg•kg^{-1}			Lab study.	Anderson 1984
	Fresh soil (655 mg microbial C•kg^{-1} soil)		96.4% (0 wk) 63.5% (4 wk) 39.9% (10 wk)		
	20°C (330 mg microbial C•kg^{-1} soil)		95.9% (0 wk) 69.7% (4 wk) 57.9% (10 wk)		
	33°C (130 mg microbial C•kg^{-1} soil)		97.2% (0 wk) 79.9% (4 wk) 68.3% (10 wk)		
	44.5°C (85 mg microbial C•kg^{-1} soil)		96.5% (0 wk) 82.0% (4 wk) 70.3% (10 wk)		
	1 mg•kg^{-1}				
	Unamended soil		94.0% (0 wk) 65.8% (4 wk) 48.0% (10 wk)		
	Soil amended with glucose		94.6% (0 wk) 46.7% (4 wk) 17.1% (10 wk)		

Table B-1. Continued

Location/soil type (% organic matter; pH; moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
	Soil amended with carbohydrate mixture		94.9% (0 wk) 40.0% (4 wk) 15.0% (10 wk)		
Braunschweig, West Germany Agricultural soil (total C = 1.26%, pH 5.4, field capacity NR)	1 $mg \cdot kg^{-1}$ 1 $mg \cdot kg^{-1}$ 1 $mg \cdot kg^{-1}$		95.2% (0 d) 50% (50 d) 39.1% (85 d) 50% (35 d) 50% (52 d)	Lab study.	Anderson and Domsch 1980a
Begbroke, Oxford, England soil (2% organic carbon, pH NR, field capacity 29%)	2.24 $kg \cdot ha^{-1}$		Foil Dish 50% (15.5 d) Granules 50% (1.5 d) Emulsifiable concentrate Dry Soil 50% (70 d) Granules 50% (69 d) Emulsifiable concentrate Wet Soil 50% (8.5 d) Granules 50% (3.0 d) Emulsifiable concentrate	Greenhouse experiment. Triallate was applied as either a spray (0.68% emulsifiable concentrate) or as 10% granules.	Hance, Holroyd, and McKone 1973
clay loam soil (physical characteristics NR)	2.24 $kg \cdot ha^{-1}$ (June 4)	5.7 cm	50% (11.5 d) 2.5% granules 50% (9.0 d) 5% granules 50% (10.0 d) 10% granules	Granules (containing 2.5%, 5% or 10% triallate) were applied to 5.5 m x 1.8 m field plots of spring barley.	

Table B-1. Continued

Location/soil type (% organic matter; pH: moisture content)	Application rate (as % ai) and date	Soil depths measured	Residues (time posttreatment)	Results and comments	Reference
Regina, Sask. top 5 cm of soil (3.1% OM, pH 7.7, field capacity NR)	1.48 kg•ha^{-1}	10 cm	1.35 ± 0.19 mg•kg^{-1} (0 d) 1.05 ± 0.13 mg•kg^{-1} (2 d) 0.94 ± 0.30 mg•kg^{-1} (6 d) 0.80 ± 0.20 mg•kg^{-1} (27 d) 0.65 ± 0.20 mg•kg^{-1} (66 d) 0.55 ± 0.15 mg•kg^{-1} (96 d) 0.30 ± 0.15 mg•kg^{-1} (159 d) 0.42 ± 0.07 mg•kg^{-1} (325 d)	Triallate (emulsifiable concentrate) applied and immediately incorporated to 5 cm. Initial residue levels measured immediately after application and incorporation.	Cessna *et al.* 1988

Appendix C

Acute Toxicity Values of Triallate for Aquatic Organisms

Table C-1. Acute Toxicity Values of Triallate for Aquatic Organisms

Species	Test conditions	Temperature (°C)	pH	Hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% ai)	LC_{50}/EC_{50} ($mg \cdot L^{-1}$) 24 h	48 h (confidence interval)	96 h	Reference
VERTEBRATES									
Oncorhynchus mykiss (Rainbow trout)	S, M	12	7.6	40	Technical (95.30)	1.3 (1.0–1.7)		0.62 (0.44–0.87)	Mayer and Ellersieck 1986
	S, M	12	7.6	40	EC (46.3)	1.3 (1.0–1.6)		1.0 (0.7–1.4)	
Ictalurus punctatus (Channel catfish)	S, M	22	7.0	40	Technical (95.30)	2.5 (1.9–3.3)		1.7 (1.1–2.5)	Mayer and Ellersieck 1986
	S, M	22	7.0	40	EC (46.3)	1.8 (1.3–2.5)		1.1 (0.8–1.6)	
INVERTEBRATES									
Daphnia magna (Cladoceran) (1st instar)	S, M	17	7.3	39	Technical (95.30)		0.08 (0.06–0.10)		Mayer and Ellersieck 1986
	S, M	17	7.2	43	EC (46.3)		0.057 (0.048–0.067)		
Chironomus plumosus (Midge larvae) (3rd instar)	S, U	22	7.5	40	Technical (95.30)		0.49 (0.36–0.67)		Johnson 1986
Chironomus riparius (Midge larvae) (4th instar)	S, U	NR	NR	NR	EC (40.7)	1.0			Johnson 1986

EC = emulsifiable concentrate
NR = not reported
S = static
M = measured
U = unmeasured

Appendix D

Summary of Triallate Phytotoxicity Data

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
Oat (*Avena sativa*) (seedlings)	1 $mg \cdot L^{-1}$	Decrease in root size; 50% decrease in shoot size; 4 d posttreatment	Lab study, no soil	Kratky and Warren 1971
	10 $mg \cdot L^{-1}$	50% decrease in root size; 50% decrease in shoot size; 4 d posttreatment		
Cucumber (*Cucumis sativus*) (seedlings)	1 $mg \cdot L^{-1}$	50% decrease in root size; 4 d posttreatment	Lab study, no soil	Kratky and Warren 1971
	10 $mg \cdot L^{-1}$	50% decrease in root size; 4 d posttreatment		
Sorghum (*Sorghum vulgare*) (seedlings)	1 $mg \cdot L^{-1}$	50% decrease in root size; 50% decrease in shoot size; 4 d posttreatment	Lab study, no soil	Kratky and Warren 1971
Oat (*Avena sativa*) (seedlings)	0.35 $kg \cdot ha^{-1}$	70% plant injury	Environmental Chamber	Chang *et al.* 1974
	0.70 $kg \cdot ha^{-1}$	86% plant injury		
Wheat (*Triticum aestivum*) (seeds)	2.2 $kg \cdot ha^{-1}$	9% increase in seed number	Field cultivated	Moyer and Dryden 1977
	1.1 $kg \cdot ha^{-1}$	14% increase in seed number		
	1.65 $kg \cdot ha^{-1}$	20% increase in seed number		
	1.4 $kg \cdot ha^{-1}$	18% fresh weight increase at harvest	Field cultivated	O'Sullivan *et al.* 1982
Mustard (*Brassica napus*) (seeds)	1.4 $kg \cdot ha^{-1}$	10% plant mortality	Field cultivated	Chow 1976
Potato (*Solanum tuberosum*) (mature plant)	305 $mg \cdot L^{-1}$	55% decrease in secondary metabolism	Lab study, no soil	Bolton and Harwood 1976

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	30.5 $mg \cdot L^{-1}$ metabolism	55% decrease in secondary		
	3.05 $mg \cdot L^{-1}$	22% decrease in secondary metabolism		
Barley (*Hordeum* sp.) (seeds)	1.1 $kg \cdot ha^{-1}$	30% decrease in plant number	Field cultivated	Klose 1961
	1.7 $kg \cdot ha^{-1}$	47% decrease in plant number		
	2.2 $kg \cdot ha^{-1}$	66% decrease in plant number		
	2.8 $kg \cdot ha^{-1}$	66% decrease in plant number		
Flax ((*Linum usitatissimum*) (seeds)	1.1 $kg \cdot ha^{-1}$	17% decrease in plant number	Field cultivated	Klose 1961
	1.7 $kg \cdot ha^{-1}$	25% decrease in plant number		
	2.2 $kg \cdot ha^{-1}$	25% decrease in plant number		
	2.8 $kg \cdot ha^{-1}$	29% decrease in plant number		
Wheat (*Triticum aestivum*) (seeds)	1.1 $kg \cdot ha^{-1}$	28% decrease in plant number	Field cultivated	Klose 1961
	1.7 $kg \cdot ha^{-1}$	33% decrease in plant number		
	2.2 $kg \cdot ha^{-1}$	58% decrease in plant number		
	4 $mg \cdot L^{-1}$	10% root size increase; 8% shoot size increase; 5 d posttreatment	Lab study, no soil	Banting 1970
	8 $mg \cdot L^{-1}$	10% root size increase; 15% shoot size decrease; 5 d posttreatment		
	16 $mg \cdot L^{-1}$	5% root size decrease; 15% shoot size decrease; 5 d posttreatment		
	64 $mg \cdot L^{-1}$	32% increase in meristem mitotic rate; 3 d post-treatment		Banting 1970
	2.8 $kg \cdot ha^{-1}$	51%–174% increase in harvest yield; 15 wk posttreatment	Lab study, no soil	Carlson and Morrow 1986

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	1.4 $kg \cdot ha^{-1}$ yield; 15 wk posttreatment	72%–158% increase in harvest		
Oat (*Avena sativa*) (seeds - with hull)	1.5 $mg \cdot L^{-1}$	4%–10% decrease in germination; 13%–49% decrease in coleoptile length; 6%–35% decrease in shoot dry weight; 5 d posttreatment	Lab study, no soil	Heath, Ashford, and McKercher 1984
	3.0 $mg \cdot L^{-1}$	0%–6% decrease in germination; 13%–55% decrease coleoptile length; 7%–43% decrease in shoot dry weight; 5 d posttreatment		
Oat (*Avena sativa*) (seeds - without hull)	1.5 $mg \cdot L^{-1}$	22%–26% decrease in coleoptile length; 31%–33% decrease in shoot dry weight; 5 d posttreatment		Heath, Ashford, and McKercher 1984
	3.0 $mg \cdot L^{-1}$	31%–54% decrease in coleoptile length; 41%–54% decrease in shoot dry weight; 5 d posttreatment		
Oat (*Avena sativa*) (seedlings)	0.12 $mg \cdot kg^{-1}$	27%–59% decrease in plant number; 28 d posttreatment	Environmental chamber	McKercher and McGregor 1980
	0.22 $mg \cdot kg^{-1}$	40%–69% decrease in plant number; 28 d posttreatment with NH_4Cl, HNO_3, or HCl		
	0.36 $mg \cdot kg^{-1}$	72%–85% decrease in plant number; 28 d posttreatment with NH_4Cl, HNO_3, or HCl		
	0.12 $mg \cdot kg^{-1}$	15%–25% decrease in plant number; 28 d posttreatment with various soil moistures		

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	0.22 $mg \cdot kg^{-1}$	40%–49% decrease in plant number; 28 d posttreatment with various soil moistures		
	0.36 $mg \cdot kg^{-1}$	57%–76% decrease in plant number; 28 d posttreatment with various soil moistures		
	0.12 $mg \cdot kg^{-1}$	32%–59% decrease in plant number; 28 d posttreatment with various soil moistures and 350 $mg \cdot kg^{-1}$ N		
	0.22 $mg \cdot kg^{-1}$	52%–69% decrease in plant number; 28 d posttreatment with various soil moistures and 350 $mg \cdot kg^{-1}$ N		
	0.36 $mg \cdot kg^{-1}$	67%–85% decrease in plant number; 28 d posttreatment with various soil moistures and 350 $mg \cdot kg^{-1}$ N		
Oat (*Avena sativa*) (seeds)	0.57 $kg \cdot ha^{-1}$	7%–42% decrease in dry weight; 6 wk posttreatment with 0-6720 $kg \cdot ha^{-1}$ lime amendments	Field cultivated	McKercher and McGregor 1979
	0.84 $kg \cdot ha^{-1}$	20%–47% decrease in dry weight; 6 wk posttreatment with 0-6720 $kg \cdot ha^{-1}$ lime amendments		
	0.28 $kg \cdot ha^{-1}$	20%–40% decrease in plant number; 6 wk posttreatment with 0–6720 $kg \cdot ha^{-1}$ lime amendments		
	0.56 $kg \cdot ha^{-1}$	60%–72% decrease in plant number; 6 wk posttreatment with 0–6720 $kg \cdot ha^{-1}$ lime amendments		

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	0.84 $kg \cdot ha^{-1}$	74%–84% decrease in plant number; 6 wk posttreatment with 0–6720 $kg \cdot ha^{-1}$ lime amendments		
Oat (*Avena sativa*) (seedlings)	0.11 $mg \cdot kg^{-1}$	31%–47% decrease in plant number; 29%–53% decrease in plant dry weight; 25 d posttreatment with 1–3 meq Ca/100 g soil amendments	Environmental chamber	McKercher and McGregor 1979
	0.18 $mg \cdot kg^{-1}$	52%–60% decrease in plant number; 54%–70% decrease in plant dry weight; 25 d posttreatment with 1–3 meq Ca/100 g soil amendments		
	0.11 $mg \cdot kg^{-1}$	16%–26% decrease in plant number; 16% decrease in plant dry weight; 25 d posttreatment without amendments		
	0.18 $mg \cdot kg^{-1}$	32%–59% decrease in plant number; 54% decrease in plant dry weight; 25 d posttreatment without amendments		
Oat (*Avena sativa*) (seeds)	0.22 $kg \cdot ha^{-1}$	50% decrease in shoot length; in soil containing 1.8% organic matter; 7 d posttreatment	Greenhouse study	Grover, Banting, and Morse 1979
Dill (*Anethum graveolens*)	3 $kg \cdot ha^{-1}$	21%–32% decrease in plant fresh weight of mature plants; 26% decrease in dill oil yield from mature plants	Field cultivated	Wall and Friesen 1986
Oat (*Avena sativa*) (seeds)	1.15 $ug \cdot g^{-1}$	50% decrease in dry weight; 14 d posttreatment;	Greenhouse study	Nyffeler *et al.* 1982
	0.99 $ug \cdot g^{-1}$	50% decrease in fresh weight; 14 d posttreatment;		
	1.10 $ug \cdot g^{-1}$	50% decrease in shoot length		

Table D-1. Summary of Triallate Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
	0.55 $kg \cdot ha^{-1}$	50% decrease in shoot length; in soil containing 4.2% organic matter; 7 d posttreatment		
	1.19 $kg \cdot ha^{-1}$	50% decrease in shoot length; in soil containing 10.5% organic matter; 7 d posttreatment		