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Canadian Water Quality Guidelines for Dinoseb

R.A. Kent, B.D. Pauli and P.-Y. Caux

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WATER QUALITY BRANCH
OTTAWA, ONTARIO, 1991

(Disponible en français sur demande)

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Abstract

A literature review was conducted on the uses, fate, and effects of dinoseb 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 à l'utilisation, au devenir et aux effets du dinoseb sur l'eau broute utilisée comme eau potable, 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 l'eau utilisée à des fins industrielles. Ces renseignements sont résumés dans cette publication. À partir de cette étude, on recommande des concentrations maximales afin de protéger les diverses utilisations particulières de l'eau.

Canadian Water Quality Guidelines for Dinoseb

R.A. Kent, B.D. Pauli, and P.-Y. Caux

SOURCES, OCCURRENCE, AND CHARACTERISTICS

Uses and Production

Dinoseb is the common name for a group of highly toxic dinitrophenol herbicides that includes the parent chemical, various salt derivatives, and a phenol form. The salts include the alkanolamine, the triethanolamine, the sodium, and the ammonium. The acetate form, dinoseb acetate, is also a herbicide. The parent compound has the chemical name 2-*sec*-butyl-2,4-dinitrophenol (IUPAC) and is a dark amber crystalline compound with a molecular weight of 240.21 and a chemical formula of $C_{10}H_{12}O_5N_2$ (Fig. 1). The Chemical Abstracts Service (CAS) name is 2-(1-methylpropyl)4, 6-dinitrophenol. The CAS registry numbers for dinoseb and dinoseb acetate are 88-85-7 and 2813-95-8, respectively. Other non-proprietary names for dinoseb are dinitrobutylphenol and DNBP. Dinoseb trade names and commercial formulations used in Canada are listed in Table 1. Dinoseb was introduced in 1945 by the Dow Chemical Company for herbicidal and insecticidal uses and has been registered for use in Canada since 1949 (Agriculture Canada 1989a). The chemical was manufactured or distributed by at least five companies in Canada (Table 1).

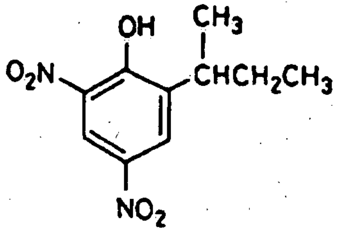

Figure 1. Structural formula for dinoseb.

As a result of a recommendation by Health and Welfare Canada, Agriculture Canada recently suspended the registration of all non-essential uses of dinoseb (Agriculture Canada 1990). The recommendation was based upon an unacceptable risk to dinoseb applicators of teratogenic effects, cataract formation, and male reproductive effects (Agriculture Canada 1989b). A suspension amounts to termination of the sale of dinoseb-containing products by registrants. Registration of dinoseb-containing products has been retained in Canada for only the essential uses of early cane control in raspberries in British Columbia and weed control in beans and peas in British Columbia and the Atlantic provinces. All non-essential uses of dinoseb were cancelled (which amounts to an end to the sale and use of dinoseb-containing products) as of 1 November 1990. The essential registrations for dinoseb will be withdrawn when acceptable alternatives become available. The U.S. Environmental Protection Agency suspended the registration of all pesticide products containing dinoseb under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) on 7 October 1986 (U.S. EPA 1986), based upon similar conclusions regarding dinoseb toxicology.

Prior to its recent suspension in Canada, dinoseb was used principally (70% of total Canadian use) as a pre-harvest aid (top killer) in potatoes (Agriculture Canada 1990). This use allowed the skin of the tuber to mature so that less feathering and bruising occurred during harvest.

Dinoseb is a selective contact herbicide that was commonly used for controlling the growth of annual grassy and broadleaf weeds and the top growth of perennial grassy and broadleaf weeds (Table 1); the compound also has fungicidal and insecticidal properties (Weed Science Society of America 1983). Target weeds included most broadleaf weeds and grasses, pigweed, lamb's-quarters, ragweed, purslane, mustards, barnyard grass, crabgrass, and foxtail. Dinoseb was used for pre-emergence weed control in a variety of agricultural crops, including corn, beans, green peas, potatoes, cucumbers, and gladiolus. It was also used as a post-emergence herbicide on grapes, berry crops, hops, alfalfa, and some clovers. As a pre-harvest foliage desiccant, dinoseb was used in seed

Table 1. **Dinoseb Formulations Used in Canada**[1]

Formulation[2]	Manufacturer	Use
Dinitro General Weed Killer General Weed Killer 600	Van Waters and Rogers Cedar	May be used to kill tiny weeds that emerge before corn, beans, potatoes, or gladiolus and for the control of weeds in grapes and bush fruits (blackberries, blueberries, currants, gooseberries, and raspberries).
VW and R Guardsman Weed and Top Killer Potato Top Killer 300 Potato Top Killer[3] Topper Potato Top Killer[3] Later's Dinoseb General[3] Dytop Potato Top Killer[3]	Van Waters and Rogers Cedar Green Cross/Ciba–Geigy Uniroyal Later May and Baker/Rhone–Poulenc	Potato top killing allows the skin of the mature so that less feathering or bruising will occur at harvest. Killing of the tops is also necessary if the potatoes are severely infected with late blight. Guardsman Weed and Top Killer is also recommended for weed and grass control in peas and preharvest spraying of alfalfa, trefoil, clovers, flax, and soybeans to facilitate harvest or seed crops. Later's Dinoseb General is recommended for all these uses as well as for the control of annual grasses and broadleaf weeds in potatoes.
Pfizer Dinoseb	United–Agri Products	Pre–emergence application for most broadleaf weeds and grasses in the Atlantic provinces.[4]
Yellow Stuff G[3] Sinox General Herbicide[3]	Uniroyal May and Baker/Rhone–Pulenc	A contract spray used for general weed killing purposes, it burns the top growth of all non-woody plants. Also used for pre-harvesting drying (defoliation) of alfalfa, alsike, ladino, red clover, and trefoil for seed production and for weed control in certain clovers. Sinox General can also be used for orchard floors and ditch banks, etc., for grapes, and for potato top killing.
Tank Mixtures Dyanap Liquid Weed Killer[3] $220\ g \cdot L^{-1}$ naptalm $110\ g \cdot L^{-1}$ dinoseb	Uniroyal	A pre-emergence herbicide combination for the control of pigweed, lamb's quarters, ragweed, purslane, mustards, barnyard grass, crabgrass, and foxtail in cucumbers.

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[1] As of 14 February 1990, all Canadian registrations of dinoseb are suspended except for early cane control in raspberries in British Columbia and weed control in beans and peas in British Columbia and the Atlantic provinces; in the United States, dinoseb was available in a variety of commercial formulations, including Caldron, Vertac General Killer, Vertac Selective Weed Killer, Vertac Dinitro Weed Killer 5, Basanite, Chemox General, Chemox PE, Chemsect, Dinitrex, Dinitro–3, Drexel Dynamite 3, Dynamite, Elgetol 318, Gebutox, Hel–Kiloseb, Nitropone C, Subitex, Unicrop DNEP, Premerge Plus with Dinitro, Supersevtox, and Klean Krop (Farm Chemicals Handbook 1987; Worthing and Walker 1987; U.S. EPA 1988).

[2] All formulations are emulsifiable concentrates or emulsions.

[3] Production voluntarily discontinued by registrant.

[4] A recommendation of the Atlantic Weed Science Sub-Committee under the authority of the Ministers of Agriculture of Newfoundland, Prince Edward Island, Nova Scotia and New Brunswick for the 1990 growing season.

Sources: Agricultur Canada 1989a; product labels.

production in flax, legumes, soybeans, alfalfa, alsike, ladino, red clover, and trefoil. Other uses of dinoseb in Canada included the control of plant growth in drainage ditches and brush control/conifer release in silviculture. In the United States, dinoseb was used as an insecticide (for the control of mites, aphids, and other insects on fruits and nuts) and as a fungicide (for control of white mould on peanuts); however, these applications constituted minor uses (Farm Chemicals Handbook 1987).

In 1984, 204 t of the formulated dinoseb herbicide were imported into Canada (Statistics Canada 1984). In 1985, 1986, and 1987, 294, 247, and 112 t, respectively, were imported (Statistics Canada 1985, 1986, 1987).

Dinoseb was sold as a liquid herbicide or a liquid emulsifiable concentrate. An application rate of 1.5–8.0 kg active ingredient (ai)•ha^{-1} was recommended for its use as a pre-emergence herbicide. For weed control in vineyards, orchards, drainage ditches, and berry fields, an application rate of 0.6–2.1 kg ai•ha^{-1} was recommended. Pre-harvest drying of seed crops required similar application rates. Top killing of potatoes prior to harvest required application of slightly higher doses (1.5–3.0 kg ai•ha^{-1}) (application rates calculated from manufacturers' label information). Crop setback distances from farm ditches vary, as these are established by provincial ministries.

Limited Canadian use-pattern information is available for dinoseb. Based upon its predominant use in potato cultivation prior to suspension, the Maritime provinces (Prince Edward Island being the largest potato producer, followed by New Brunswick) accounted for the majority of dinoseb use in Canada. After the Atlantic provinces, Quebec, Ontario, and Manitoba followed, in decreasing order of potato production.

The second most important use prior to suspension was in raspberry production, which is practiced almost exclusively in British Columbia (over 90% of Canadian production). Of this, the majority of dinoseb was used in the Fraser River valley around Abbotsford. Potato culture in the Kettle River valley also accounted for a small amount of dinoseb use in British Columbia (M. Edwards, 1989, Agriculture Canada, Vancouver, pers. com.). Use of dinoseb in early cane control in raspberries is one of the retained essential uses, and raspberry production in the Fraser River valley, B.C., will likely account for the bulk of future dinoseb use in Canada until a suitable replacement is found.

In the Prairie provinces, dinoseb use has been limited since 1985. Very limited amounts of the herbicide were used in Alberta, largely in potato farming (M. Constable, 1989, Environment Canada, Edmonton, pers. com.). In Saskatchewan, dinoseb was used in field pea production, for weed control, and as a desiccant. In Manitoba, it was used in potato farming as a top killer prior to harvest. Prior to its suspension, dinoseb was recently recommended as a cucumber herbicide in Manitoba (Manitoba Agriculture 1989).

Dinoseb applications in peas, beans, and soybeans accounted for a large portion of its Ontario usage (R. Frank, 1989, Ministry of Agriculture and Food, Guelph, Ont., pers. com.). A pesticide use survey conducted in Ontario in 1983 (McGee 1984) also indicated that the herbicide was employed to a limited extent in cucumber production. Use of the herbicide was low, however, with only 790 kg ai used on all field crops, fruits, vegetables, and roadsides in 1983. In 1988, 90 kg were used for the same purposes (Moxley 1989). As a guide for the 1990 growing season, prior to its restricted use, dinoseb was registered for weed control in peas and as a pre-harvest spray to facilitate harvest of soybean seed crops (Ontario Ministry of Agriculture and Food 1989). Because of the restricted use of this compound, no quantitative information is available on dinoseb usage in Quebec; however, because of similar agricultural practices, use patterns were likely similar to those in Ontario.

No information exists on the sales or use of dinoseb in the Northwest Territories or in Yukon Territory, but it is assumed that there is no significant use of the chemical in these areas (J. Jasper, 1989, Indian and Northern Affairs Canada, Yellowknife, N.W.T., pers. com.).

Physical and Chemical Characteristics

The reported physical and chemical properties of dinoseb and dinoseb acetate are summarized in Tables 2 and 3, respectively. Dinoseb is a dark brown solid or viscous liquid with a melting point range of about 38°C–42°C (Table 2). The phenol form of dinoseb, only slightly soluble in water, is soluble in oil and is formulated as an emulsifiable concentrate. The amine and ammonium salts of dinoseb are much more soluble in water than the phenol form.

Table 2. Physical and Chemical Characteristics of Dinoseb

Property	Value	Reference
Chemical formula	$C_{10}H_{12}O_5N_2$	Worthing and Walker 1987
Molecular weight	240,21	Worthing and Walker 1987
Physical state	Dark amber, monoclinic crystals	Worthing and Walker 1987 Hayes 1982
Henry's law constant (K_h)	51.11 (20°C)	Suntio *et al.* 1988
Dissociation constant (pK_a)	4.62 4.0 4.03	Cessna and Grover 1978 McLeese *et al.* 1979 Call *et al.* 1989
Melting point	41°C–42°C 40°C 37.9°C–39.3°C	Wallnöfer *et al.* 1978 Vlassak *et al.* 1976 Hayes 1982
Vapour pressure	130 Pa (151.5°C) 10 Pa (20°C)	Kan 1980 Suntio *et al.* 1988
Sediment/water distribution coefficient	Not reported	
Octanol/water coefficient (log P)	3.69 3.69 3.88[1]	Call *et al.* 1989 McLeese *et al.* 1979
Organic carbon/water partition coefficient (log K_{ow})	3.77 ($cm^3 \cdot g^{-1}$)	Gustafson 1989
Solubility		
Water	47 $mg \cdot L^{-1}$ (20°C) 52 $mg \cdot L^{-1}$ (25°C) ≈100 $mg \cdot L^{-1}$ (≈25°C)	Suntio *et al.* 1988 Kan 1980 Worthing and Walker 1987
Ethanol/methanol	Not reported	
Chloroform	Not reported	
Half-life in soils		Dinoseb Task Force 1985a
Photolytic degradation	14–30 h	Howard *et al.* 1982
Microbial degradation	43–123 d	

[1] Using the equation in Banerjee *et al.* 1980.

Dinoseb and its various salts may be prepared through a two-step process involving the sulphonation of 6-alkylphenol or 2,4-dinitrophenol with concentrated sulphuric acid. The 2,4-dinitro-6-alkylphenol formed in the first step is dissolved in water with sufficient nitric acid added to convert the sulphonic acid derivative to the desired nitrophenol (Monnig and Zweidinger 1980). Otherwise, the sulphonated 2,4-dinitrophenol is butylated and the sulphonic group removed (Kaufman 1976). Dinoseb can also be prepared by controlled nitration of o-*sec*-butylphenol, which is made by reacting butylene with phenol (Kaufman 1976).

Analytical Techniques

The most commonly employed technique for measuring concentrations of dinoseb in water involves extraction of acidified samples with dichloromethane, followed by rotary evaporation and methylation using diazomethane (Yip and Howard 1968; Wan 1989). The derivatized extract is then combined with isooctane and analyzed with a gas–liquid chromatograph (GLC) equipped with an electron capture detector (ECD). Wan (1989) used the same procedure for sediment samples; limits of detection were 1 $\mu g \cdot kg^{-1}$ for sediment and 0.02 $\mu g \cdot L^{-1}$ for water. GLC with ECD was also used to measure residues of dinoseb in tissues and soils by Gardner and McKellar (1980); they could quantify residues of dinoseb to 20 pg. A colorimetric method employing an ultraviolet–visible spectrophotometer running at a wavelength of 376 nm was also used to determine levels of dinoseb in water (detection limit 100 $\mu g \cdot L^{-1}$) (Skelley 1989).

Table 3. Physical and Chemical Characteristics of Dinoseb Acetate

Property	Value	Reference
Chemical formula	$C_{12}H_{14}O_6N_2$	Worthing and Walker 1987
Molecular weight	282.25	Zitko *et al.*, 1976
Physical state	Not reported	
Henry's law constant (K_h)	Not reported	
Dissociation constant (pK_a)	Not reported	
Melting point	Not reported	
Vapour pressure	Not reported	
Sediment/water dsitribution coefficient	Not reported	
Octanol/water partition coefficient (log P)(log P)	3.72	Zitko *et al.* 1976
Solubility:		
Water	Not reported	
Ether	Not reported	
Chloroform	Not reported	
Methanol	Not reported	
Half-life in soils	Not reported	

Mode of Action

Movement of dinoseb into plant leaves may occur either by diffusion through leaf surfaces or as a vapour through leaf stomates. Movement into the roots is by diffusion or absorption. Once in the plant, little or no translocation of the herbicide occurs (Kaufman 1976).

The Weed Science Society of America (1983) listed the symptomatic effect of dinoseb as direct cell necrosis. Kaufman (1976), in a detailed review of the mode of action of the phenolic herbicides, mentioned that dinoseb is an inhibitor of metabolism. The work of Simon (1953) revealed that there are several different mechanisms by which dinitrophenols exert their toxic action: inhibition of oxidative and glycolytic phosphorylation, inhibition of respiration and fermentation, and protein denaturation. The dinitrophenols may also inhibit or retard transpiration, mineral uptake, and glyceride synthesis and degrade chlorophyll. Dinoseb may uncouple and inhibit the oxidative phosphorylation system in plant and animal tissues, which leads to decreased adenosine triphosphate (ATP) levels (Simon 1953; Kaufman 1976). A study by St. John and Hilton (1974) revealed that dinoseb inhibited synthesis of glycerides in intact wheat seedlings. They suggested that dinoseb altered membrane structure and inhibited membrane lipid synthesis.

Entry into the Environment

Agricultural applications of dinoseb have the potential to contaminate the environment through a variety of transport routes. Direct contamination of surface water may occur after applications of dinoseb for weed control in drainage ditches or may result from aerial or ground-boom spraying operations. Indirect contamination of surface waters can occur as a result of runoff from treated areas or surface recharge with contaminated groundwater. Additional contamination may result from spills, deliberate dumping of tank residues, or improper equipment washing operations. Contamination of groundwater with dinoseb residues may occur as a result of leaching from treated areas. Other sources of groundwater contamination include spills and infiltration of equipment wash water.

Environmental Concentrations

Surface Water

Little information is available on the occurrence of dinoseb in Canadian surface water; dinoseb is not included in routine monitoring programs conducted by Environment Canada (Table 4). Special studies have been undertaken at sites where dinoseb contamination has been suspected (i.e., after spills or near application sites). In addition, several provinces (British Columbia, Ontario, Alberta, New Brunswick) and federal agencies (Health and Welfare Canada, Agriculture Canada) have conducted studies to determine levels of dinoseb in surface water and groundwater. Data concerning surface water concentrations of dinoseb are available for only British Columbia and Alberta. In British Columbia, Wan (1989) found dinoseb contamination in farm ditches where the crop setbacks were 3 m or less from the ditches. From May 1985 until February 1986, 14 of 25 (56%) of the samples contained dinoseb, with a maximum concentration of 18.6 µg•L^{-1}: samples were collected in May (prior to application, 0 detections), July (shortly after spraying, 3 detections), October (4 detections), December (4 detections), and the following February (3 detections). In October of 1986, dinoseb (at 5.0 µg•L^{-1}) was detected in 1 of 25 samples. In Alberta, dinoseb was not detected (detection limit 0.15 µg•L^{-1}) in 283 samples of surface water from 15 municipalities in 1978–1985 (Hiebsch 1988).

Groundwater

A summary of the available information on levels of dinoseb in Canadian groundwater sources is presented in Table 5. Although Frank *et al.* (1987) reported a relatively high incidence of contamination in Ontario farm wells (3 of 7, or 43% contaminated), the wells investigated were suspected to be contaminated through spills, spray drift, or surface water runoff carrying pesticides directly into the wells rather than by infiltration of contaminated groundwater. In fact, all of the contaminated wells received their contamination as a result of spills. In one of these wells, a drum of herbicide was spilled 3 m from the sandpoint wellhead. The dinoseb concentration in the well peaked 237 d later at 36 mg•L^{-1}, and the well was abandoned as a water source a year after the spill. The concentration was still 3.7 mg•L^{-1} 382 d after the spill. This spill also revealed that dinoseb can leach through soil and be laterally transported in the subsurface zone; 1 year after the spill, traces of dinoseb at 0.1 and 0.3 µg•L^{-1} were detected in two of four neighbouring wells.

Dinoseb (at 0.8 and 1.1 µg•L^{-1}) (detection limit 0.02 µg•L^{-1}) was detected in the subsurface drainage of a potato field (depth to tile 1 m) in New Brunswick 11 months after the last application (O'Neill *et al.* 1989, 1990). This field study revealed that dinoseb is mobile and persistent in groundwater and can contaminate this water source as a result of agricultural applications. At this site, 37 other detections were made from a collection of 133 samples. The maximum concentration found was 44 µg•L^{-1}. Most of the concentrations, however, were below 1.0 µg•L^{-1}, and most were closer in time to an application of dinoseb.

A broad review of the use of pesticides in the Atlantic region (Gillis and Walker 1986) identified dinoseb as a major agricultural pesticide and, therefore, a priority pesticide that is potentially hazardous to groundwater in the region. Very little information on

Table 4. Summary of Data on the Occurrence of Dinoseb in Surface Water in Canada

Location	Detection limit (µg·L^{-1})	Frequency of detection	Maximum concentration (µg·L^{-1})	Mean concentration (µg·L^{-1})	Reference
British Columbia					
Lower Fraser River basin, ditches in the vicinity of five farms	0.1	14 of 25 (1985) 1 of 25 (1986)	18.6 5.0	4.9 —	Wan 1989
Alberta					
15 municipalities using surface water supplies (1978–1985)	0.15	0 of 283	< 0.15	NA	Hiebsch 1988
Saskatchewan		No data			
Manitoba		No data			
Northwest Territories		No data			
Yukon		No data			
Ontario		No data			
Quebec		No data			
New Brunswick		No data			
Nova Scotia		No data			
Prince Edward Island		No data			
Newfoundland		No data			

NA = not applicable

environmental occurrences was available. In Prince Edward Island, dinoseb was detected in 11 of 40 wells sampled in 1985, at a maximum concentration of 16.4 µg•L^{-1} (Hiebsch 1988).

For other provinces for which there are data, no dinoseb has been found in groundwater in Alberta or Manitoba (Hiebsch 1988), and low levels were found in wells in British Columbia (Agriculture Canada 1989c) (Table 5).

Atmospheric Transport and Precipitation

No data are available on levels of dinoseb in dry or wet atmospheric fallout. Aerial applications of the pesticide in particular, and ground-boom applications as well, may result in spray drift (U.S. EPA 1986), and areas located downwind of treated areas may receive significant quantities of dinoseb. This dry fallout could result in coincidental exposures of non-target organisms. From 1981 to 1985 in California, approximately 6% of all dinitrophenol poisoning incidents resulted from coincidental exposure due to spray drift (U.S. EPA 1986). It is unlikely, however, that significant long-range atmospheric transport or wet precipitation of dinoseb occurs as a result of agricultural applications.

Sediment

Limited information exists on the levels of dinoseb in sediments. One reason may be the difficulty in recovering and detecting dinoseb residues in sediment samples (Wan 1989). A sampling program undertaken in Holmes Brook, N.B., in 1980 (B. Ernst, 1989, Environment Canada, Dartmouth, N.S., pers. com.) revealed elevated levels in stream sediments. The maximum and mean concentrations of dinoseb in seven sediment samples taken at three sites in the river were 0.086 and 0.033 mg•kg^{-1}, respectively. Slightly lower levels were reported at three sites located on the Dunk River, P.E.I. Here, maximum and mean concentrations of dinoseb in five sediment samples were 0.030 and 0.019 mg•kg^{-1}, respectively. No data on the concentrations of dinoseb in water were available for either of these locations.

Table 5. Summary of Data on the Occurrence of Dinoseb in Groundwater in Canada

Location (date)	Detection limit (µg·L^{-1})	Frequency of detection	Maximum concentration (µg·L^{-1})	Mean concentration (µg·L^{-1})	Reference
British Columbia					
Lower Fraser River basin – 51 wells from Langley South Abbotsford (Fall 1988)	0.02	8 of 51	0.02 Trace 0.09 0.06 Trace 0.85 0.02 0.10		Agriculture Canada 1989c
– Aldergrove	0.02				Environment Canada 1989
(Oct. 1988)		1 of 5	4.7		
(Nov. 1988)		2 of 4	6.0 6.0		
Kettle River basin					Environment Canada 1989
– Grand Forks	0.02				
(Oct. 1988)		2 of 4	1.6 0.5		
(Nov. 1988)		0 of 7	NA	NA	
Yukon Territory		No data			
Alberta					
13 municipalities using groundwater supplies (1978–1985)	0.15	0 of 26	NA	NA	Hiebsch 1988
Saskatchewan		No data			
Manitoba					
49 sources of drinking water (Oct. 1986)	0.05	0 of 49	NA	NA	Hiebsch 1988
Northwest Territories		No data			
Ontario					
– 3 rural wells (1979–1984)	0.05	3 of 7	36 000[1]	NR	Frank *et al.* 1987
Quebec		No data			
New Brunswick					
– 73 wells		15 of 73	12.4	2.84	Agriculture Canada 1989c
Drainage from five tile-drained potato fields (1987–1988)	0.02	39 of 133	44	NR	O'Neill *et al.* 1989
Nova Scotia		No data			
Prince Edward Island					
– 40 wells (1985)	NR	11 of 40	16.4	NR	Hiebsch 1988
Newfoundland		No data			

NR = not reported
NA = not applicable
[1] As a result of a spill

In British Columbia, where dinoseb was found in ditches draining farms (Wan 1989), the herbicide was also found in the ditch bank sediment. Residues were found in one sample at 22.9 µg•kg^{-1} shortly after herbicide application in July 1985 but were not detected a year later despite a more intensive sampling effort. At two sites sampled in 1987 and 1988, mean levels of dinoseb in sediments were found to be 81.2 and 108.6 µg•kg^{-1}, respectively. The elevated levels occurred during the wet season, which led Wan (1989) to speculate that the herbicide was being transported from the treated areas to the ditches via surface runoff.

Biota

Few data are available on the levels of dinoseb in aquatic biota in Canada. In Holmes Brook, N.B., and the Dunk River, P.E.I. (B. Ernst, 1989, Environment Canada, Dartmouth, N.S., pers. com.), mean levels of dinoseb in fish livers (species not identified) ranged from 0.110 mg•kg^{-1} (n = 1) to 0.175 mg•kg^{-1} (n = 4), respectively. A maximum level of 0.37 mg•kg^{-1} was recorded in fish from the Dunk River. As mentioned above, no data were available on the concentrations of dinoseb in the water at either of these locations.

Environmental Fate, Persistence, and Degradation

Three modes of application are typically used during dinoseb treatments. These include backpack, land boom, and aerial spray. The method and timing of application to a great extent influence the environmental fate and persistence of dinoseb. The major processes that determine the fate of dinoseb in the environment include aqueous stability, photolysis, adsorption, and microbial degradation. In addition, the frequency and magnitude of rain events and irrigation and wind conditions during application may further modify the environmental fate and persistence of dinoseb.

Volatilization

The rate of volatilization of dinoseb from plant and soil surfaces may depend on the method of application and the type of formulation (Kaufman 1976). Cohen *et al.* (1984) listed a volatilization half-life for dinoseb of 26 d based on a laboratory experiment in which dinoseb was applied to the surface of moist loam soil at 25°C with a simulated airspeed of 1 km•h^{-1}. Kaufman (1976) reported that some loss of dinoseb may occur given specific conditions of soil acidity, high temperature, and surface soil moisture. Volatilization is expected to occur more readily under acidic conditions because the herbicide exists as a more volatile free acid. The significance of dinoseb volatilization has been illustrated by the killing of plants by dinoseb vapours (Davis *et al.* 1954); volatilization was implied by the pattern of necrosis, which involved primarily the lower leaves (Kaufman 1976).

The water/air partitioning behaviour of a low concentration of solute in water is reflected by Henry's law constants and becomes important for the non-dissociated nitrophenol. Schwarzenbach *et al.* (1988) showed that the calculated Henry's law constant at 20°C for some substituted 2-nitrophenols was smaller than 5×10^{-2} atm•mol^{-1}•L^{-1}. This signifies that the water/air exchange will be mostly gas film controlled and that evaporation from natural water bodies will be insignificant compared with other processes. The authors attributed this phenomenon to the fact that the vapours of the compounds show ideal gas behaviour and the solute/solute interactions do not play a determining role. Intramolecular hydrogen bonding between the hydroxyl and nitro groups is not affected by proximity effects (Schwarzenbach *et al.* 1988). Small air/water ratios for these compounds translate into an efficient scavenging from the atmosphere by wet precipitation (Leuenberger *et al.* 1985).

Photodegradation on Soil and Plants

As dinoseb is a contact herbicide and is sprayed directly on foliage, photodegradative loss of dinoseb applications may be substantial. A study by Hazelton Laboratories America Inc. (Dinoseb Task Force 1985a) indicated that dinoseb was unstable in natural sunlight; a half-life of 14 h was calculated for a California sandy loam soil. In artificial light (unspecified wavelength), dinoseb was slightly more stable, with a half-life of 30 h reported for the same soil type. Rapid photodegradation (half-life < 1 h) in natural sunlight was also reported for dinoseb applied to bean leaves (Matsuo and Casida 1970). The photodegradation of dinoseb on plant surfaces was confirmed in studies with beans: any dinoseb formed after photoalteration of 2-*sec*-butyl-4,6-dinitrophenyl isopropyl carbonate (dinobuton) was photodecomposed within 7 d (Bandal and Casida 1972). On apples, a half-life of less than 8 h was reported (Hawkins and Saggers 1974). In a compilation of the rates of environmental degradation for various chemicals (Syracuse Research Corp. 1989), the atmospheric photooxidation half-life of dinoseb was estimated to be between 12.2 and 122 h. This estimate was based on structure–activity relationships for gas-phase reactions of hydroxyl radicals with organic compounds (Atkinson 1987). The above data suggest that photodegradation is a major factor in determining the fate of dinoseb in the environment.

Photodegradation in Water

Dinoseb appears to be more resistant to photolytic degradation in water than on agricultural soils or plant surfaces. In aqueous solutions exposed to natural sunlight, dinoseb had a half-life of 14–18 d (Dinoseb Task Force 1985b). Increased stability in artificial light (unspecified wavelength) was indicated by a half-life of 42–58 d. Kaufman (1976) reported that dinitrophenols are stable in acidic solutions but are susceptible to photodecomposition by ultraviolet radiation (wavelength not reported) in alkaline solution.

Aqueous Hydrolysis

Dzialo (1984) reported that dinoseb was stable to hydrolysis in solutions of pH 5, 7, and 9 held at 25°C for 30 d. Woodward (1976) observed similar toxicities of dinoseb to fish exposed to fresh solutions and solutions "aged" for 4 weeks. These results suggested little contaminant decay over the 4-week period, further indicating the stability of dinoseb in water.

Adsorption on Soils

A number of factors control the adsorption of dinoseb onto agricultural soils. These include the composition of the soils (i.e., particle size and organic matter content), the ambient temperature, and the soil pH. Adsorption, assessed by calculating the ratio of the solute concentrations (adsorbed concentration/solution concentration) at equilibrium, is reported as a K_d value. K_d values are measured in $\mu g^{(1-N)} \cdot g^{-1} \cdot mL^{-N}$, where N is the exponent expressing isotherm curvature. A low K_d value indicates little adsorption, whereas higher values indicate more adsorption onto soil particles. A single study (Dinoseb Task Force 1985c) reported K_d values of less than 5 for dinoseb in four soil types, including silt loam, sand, sandy loam, and silty clay loam soils. These results suggest that dinoseb has a relatively high potential for leaching out of areas dominated by these types of soil. Unfortunately, the units of measurement of the dinoseb in soil and in solution were not provided, so it is not possible to compare its K_d with those of other herbicides (Bowman 1981). Although little information is available concerning the adsorption of dinoseb to organic soil fractions, Kaufman (1976) stated that the influence of pH would presumably strongly affect adsorption to soil organic matter. He concluded that phenols exist as free acids in acidic soils and would be strongly adsorbed in the presence of clays.

Mobility in Soils

As a result of its adsorptive behaviour, dinoseb may be highly mobile in certain agricultural soils. For instance, leaching occurs more readily in alkaline soils than in acidic soils (Kaufman 1976). Experiments using thin-layer chromatography indicated that dinoseb exhibited intermediate to high mobility in silt loam, sand, sandy loam, and silty clay loam soils (Dinoseb Task Force 1985d). Field studies have confirmed that significant leaching will occur in some soils; in northwestern New Brunswick, high levels of dinoseb (maximum 44 $\mu g \cdot L^{-1}$) were measured in the effluent of tile-drained potato fields (unspecified soil types) by O'Neill *et al.* (1989). In the model presented by Gustafson (1989), dinoseb would be classified as a "leacher" based on its water solubility and the soil organic carbon/water partition coefficient (K_{ow}), but Gustafson classified it as a "transition" compound because contradictory evidence exists as to its leaching potential. In Gustafson's (1989) model, a leachability index was calculated by an examination of a plot of two pesticide properties: the soil half-life ($t_{½}$) and K_{oc}. The plot was constructed by calculating a "groundwater ubiquity score (GUS)," where GUS = $\log_{10}(t_{½}) \circ [4-\log_{10}(K_{oc})]$.

Microbial Degradation

Information on the effects of soil microorganisms on dinoseb persistence suggests that there is significant potential for microbial degradation of dinoseb residues in agricultural soils. Kaufman (1976) stated that two mechanisms function in the microbial degradation of dinitrophenols. The first involves reduction of a nitro group to an amine, and the second includes an oxidative elimination of the nitro group with subsequent formation of dihydric phenol. Wallnöfer *et al.* (1978) reported that dinoseb was transformed (50% transformation in 3 d) to 6-acetoamido-2-*sec*-butyl-4-nitrophenol by *Azotobacter* sp. in agricultural soils. When no other sources of organic matter were provided, pure cultures of *Pseudomonas aeruginosa* and *P. putida* degraded 90% and 50% of a dinoseb application, respectively, in 20 d (Douros and Reid 1956). The Syracuse Research Corp. (1989) estimated a soil half-life of 43–123 d for dinoseb. Details were not provided, but the estimate was based on aerobic soil mineralization data for ^{14}C-labelled dinoseb incubated in a silt loam soil that had been amended with sewage sludge and manure and monitored for release of $^{14}CO_2$ for 60 d (Doyle *et al.* 1978).

Summary of Environmental Fate

A summary of the environmental fate of dinoseb is presented in Table 6. This table identifies potential degradation pathways of dinoseb in surface water and groundwater, soil/sediment, and biota. Dinoseb herbicides applied directly onto soils and plants during warm, dry conditions are likely rapidly decomposed through photodegradation (half-life < 1 d). Subsequent bacterial degradation of much of the remaining residue

Table 6. Summary of Dinoseb Degradation in Soil/Sediment, Water, and Biota

Dinoseb degradation in soil/sediment	Dinoseb degradation in water	Dinoseb degradation in biota
PHOTOLYSIS · photolytic degradation with natural sunlight on a California sandy loam soil, t½ = 14 h; with artificial light t½ = 30 h; (Dinoseb Task Force 1985a) OXIDATION · no data AEROBIC METABOLISM · established role in dinoseb degradation (Kaufman 1976) · no transformation products isolated from treated soils (Smith 1988) · 50% transformation in 3 d by Azotobacter in agricultural soils (Wellnöfer *et al.* 1978) ANAEROBIC METABOLISM · no data VOLATILIZATION · some loss by co-distillation under specific conditions of soil acidity, high temperature, and surface soil moisture (WSSA 1983) MOBILITY · water-soluble salts of dinoseb leach readily and move much more than do oil-soluble and water-miscible formulations; movement affected by soil texture, precipitation, and formulation (Kaufman 1976) ADSORPTION/DESORPTION · not tightly adsorbed on most soils; should not be leached from the top 30 cm of soil by rainfall in the first year after application (WSSA 1983) · adsorption is pH- and temperature-dependent; more adsorption on acid soils, especially in the presence of mineral clays (Kaufman 1976) · intermediate to high mobility in silt loam, and silty clay loam (organic matter 0.8%—3%), K_d <5 (Dinoseb Task Force 1985d) PERSISTENCE · t½ = 43—123 d (estimate) (Syracuse Research Corp. 1989) · 50% transformation in 3 d by *Azotobacter* in agricultural soils (Wallnöfer *et al.* 1978) · measured dissipation rate of 0.027 per day ("moderately short lived") from laboratory soil surface (Nash 1988)	PHOTOLYSIS · stable in acid solutions but susceptible to decomposition by ultraviolet radiation in alkaline solution (Kaufan 1976) · in water with natural sunlight, t½ = 14—18 d (not stated whether photolytic degradation was only cause of dissipation) (Dinoseb Task Force 1985b) OXIDATION · no data AEROBIC METABOLISM · "unacclimated" aerobic aqueous biodegradation t½ = 43—123 d (estimate) (Syracuse Research Corp. 1989) HYDROLYSIS · stable to hydrolysis at pH 5, 7, and 9 at 25°C for 30 d (Dzialo 1984) ANAEROBIC METABOLISM · "unacclimated" aerobic aqueous biodegradation t½ = 4—15 d (estimate) (Syracuse Research Corp. 1989) VOLATILIZATION · no data PERSISTENCE · t½ = 43—123 d in surface water (estimate), t½ = 4d in groundwater (estimate) (Syracuse Research Corp. 1989) · monitoring data indicate long persistence in groundwater Frank *et al.*1987; O'Neill *et al.* 1989)	· in mammals, phenolic pesticides readily assimilated and excreted slowly over a period of many weeks; possible formation of dinoseb–albumin complexes; reduction of nitro groups to amine groups principal route of metabolism in animals; 2–amino–4–nitro–6–*sec*–butyphenol isolated from the urine of rats and rabbits fed dinoseb side–chain oxidation yields 3–methyl–3–(2–hydroxy–3,5–dinitrophenyl)propionic acid (Kaufman 1976) in fish, rapid elimination of ^{14}C–labelled dinoseb;: fathead minnows, 71% eliminated in 24 h and 96% after 14 d; trout, 90% eliminated within 24 h; metabolites not identified (Call *et al.*1984)

WSSA = Weed Science Society of America

on the soil surface also takes place relatively rapidly (i.e., within 8–10 d). The Weed Science Society of America (1983) concluded that the average persistence of dinoseb phytotoxicity when the herbicide is applied at recommended rates is 14–28 d. Applications prior to rain events or irrigation, however, may result in the leaching of dinoseb into the subsurface soil. In the cool, moist, and dark conditions therein, dinoseb is likely to be stable for extended periods of time; monitoring of a dinoseb spill that contaminated wells in Prince Edward Island in 1984 has revealed that during the 6 years since the spill, contamination levels have remained at or above the level measured immediately following the spill (D. Jardine, 1990, P.E.I. Department of the Environment, pers. com.). Although the Syracuse Research Corp. (1989) estimated the groundwater half-life of dinoseb to be between 4 and 246 d, this would appear to be a low estimate.

The mobile nature of dinoseb in some soil types may lead to contamination of groundwater after agricultural applications. Dinoseb was found in 6 of 66 wells sampled, at levels of 4.5 $\mu g \bullet L^{-1}$ or less, at Long Island, N.Y. (Cohen *et al.* 1984), and has been found in the subsurface drainage from potato fields in New Brunswick (O'Neill *et al.* 1989). Application of dinoseb prior to rain events or irrigation can result in direct contamination of surface water via runoff from treated areas. The highest concentrations of dinoseb in aquatic ecosystems, however, are likely to occur as a result of herbicide spills (at manufacturing, packaging, and treatment sites), backflows into wells during loading, cleaning spraying equipment near watercourses, and spray drift during application (Monnig and Zweidinger 1980; Frank *et al.* 1987).

Substituted 2-nitrophenols such as dinoseb show small Henry's law constants and large water/air ratios, making these compounds mobile and found at appreciable concentrations in aqueous phases and rainwater (Tereda 1981; Schwarzenbach *et al.* 1988).

RATIONALE

Raw Water for Drinking Water Supply

Guideline

There is no recommended limit for the concentration of dinoseb in drinking water listed in the Guidelines for Canadian Drinking Water Quality (Health and Welfare Canada 1989). A drinking water guideline for the compound is currently under development (G. Wood, 1990, Health and Welfare Canada, pers. com.).

Summary of Existing Guidelines

The U.S. EPA (1987) published human health advisories for dinoseb in drinking water. The 1-d and 10-d health advisories for dinoseb are 300 $\mu g \bullet L^{-1}$. These values are based on a teratology study in which dinoseb produced neural tube defects in rabbits at doses greater than 3 $mg \bullet kg^{-1} \bullet d^{-1}$. The longer-term (7-year) health advisories for a 10-kg child and a 70-kg adult are 10 and 35 $\mu g \bullet L^{-1}$, respectively. These were based on a two-generation reproduction study with rats, which produced a lowest-observed-effect level (LOEL) of 1 $mg \bullet kg^{-1} \bullet d^{-1}$, based on a decrease in pup weight. The lifetime health advisory for dinoseb (considered protective of non-carcinogenic adverse health effects over a lifetime of exposure) is 7 $\mu g \bullet L^{-1}$. This level was based on a 2-year rat dietary study, which produced compound-related decreases in mean thyroid weights of all male animals exposed to dinoseb (U.S. EPA 1987), and a relative contribution from drinking water to the total daily dinoseb exposure of 20%. Dinoseb was not included in the drinking water guidelines for selected herbicides published by the World Health Organization (WHO 1987).

Concentrations in Drinking Water

Dinoseb has been found in drinking water sources in New Brunswick, Prince Edward Island, and British Columbia (Table 5). The contaminated samples came from private wells in agricultural areas. The contamination may be a result of the fact that the water was not treated; no dinoseb was found in samples of treated drinking water collected in Manitoba and Alberta (Heibsch 1988; see also below). In many cases, the contamination in the private wells can be traced to mishandling of the herbicide; how-ever, some aquifer contamination (above 10 $\mu g \bullet L^{-1}$) in Prince Edward Island (Don Jardine, 1990, P.E.I. Department of the Environment, pers. com.) and New Brunswick (O'Neill *et al.* 1989) has occurred as a result of agricultural applications. The dinoseb found in rural wells (Frank *et al.* 1987) in Ontario was the result of spills or mishandling of the herbicide around the wells. Dinoseb was found in 6 of 66 wells sampled in Long Island, N.Y. (concentrations were 4.5 $\mu g \bullet L^{-1}$ or less), but no further details were provided (Cohen *et al.* 1984).

Removal by Water Treatment Operations

No information was found on the mechanisms available for removing dinoseb from contaminated water during treatment of drinking water supplies. Monnig and Zweidinger (1980), however, while investigating means to remove dinoseb from the wastewater of dinoseb manufacturing processes, found that a treatment system involving activated carbon filtration removed the herbicide. After passage through a carbon-filled column 142 cm in height by 2.5 cm in diameter, no dinoseb was detected in the water collected from the column even when the input samples contained dinoseb at a concentration of 750 mg•L^{-1}.

Freshwater Aquatic Life

Bioaccumulation

A limited amount of information on the accumulation of dinoseb in aquatic biota indicates rapid uptake and elimination of dinoseb by freshwater fish. In fathead minnows (*Pimephales promelas*), 24-d exposures to high (7.22 µg•L^{-1}) and low (0.62 µg•L^{-1}) concentrations of dinoseb resulted in equilibrium ^{14}C bioconcentration factors (BCFs) of 64.1 and 61.5, respectively (Call *et al.* 1984). Exposures for 28 d resulted in a mean total (dinoseb plus metabolites) BCF of 56.2 measured as ^{14}C. However, only 2.3% of the total ^{14}C was extracted as parent herbicide, for a mean BCF of 1.4 for the dinoseb itself. Lorz *et al.* (1979) reported that the spleen, gall bladder, liver, and kidney appeared to be the major sites of dinoseb accumulation in coho salmon (Table 7).

Elimination of ^{14}C was rapid from dinoseb-exposed fathead minnows (Call *et al.* 1984). Within 24 h after transfer to uncontaminated water, the fish had eliminated 71% of the ^{14}C originating from labelled dinoseb. After 14 d, an average of 96% of the ^{14}C had been eliminated. Although no data were found on the biomagnification of dinoseb in freshwater ecosystems, the bioconcentration information suggests that ingestion of contaminated food organisms is not likely to lead to significant biomagnification. Direct toxicity and sublethal effects (see below) are much more likely to result in adverse effects on fish and aquatic life.

Toxicity to Aquatic Organisms

Fish

Mode of Toxic Action—Although dinoseb has been shown to be highly toxic to freshwater fish (Woodward 1976; Call *et al.* 1984; Gersich and Mayes 1986), the exact mechanism of toxicity has not been established. Research on mammals has shown that dinitrophenols produce acute toxicity through the disruption of oxidative metabolism. This toxic action may result from actions at both the systemic and cellular levels of organization. Evidence for systemic action lies in reports of methemoglobinemia (oxidation of the hemoglobin in erythrocytes resulting in reduced capacity to bind oxygen reversibly) in affected animals. Elevated levels of methemoglobin in the blood can cause symptoms such as cyanosis and tissue hypoxia. At the cellular level, dinoseb can affect energy metabolism by uncoupling oxidative phosphorylation by preventing the formation of adenosine triphosphate (ATP) from adenosine diphosphate (ADP) in mitochondria. Cellular metabolic processes are ATP-dependent, and ATP depletion stimulates the glycolytic breakdown of storage polysaccharides to lactate (Campbell 1973). These cellular changes, in turn, result in acidosis, oxygen debt, and anoxic stress. It is likely that the impairment of energy-requiring metabolic processes is the primary mode of toxic action of dinoseb.

Acute Toxicity—Limited evidence from salmonid species (Woodward 1976) indicates that the acute toxicity of dinoseb to freshwater fish may be modified by certain characteristics of the aquatic environment. These include, but are not necessarily limited to, pH, water hardness, and water temperature. In addition, the toxicity of dinoseb is also dependent on the species and life history stage of the exposed organisms.

Investigations into the effects of dinoseb on two salmonid species indicate that the acute toxicity is pH-dependent within the range considered acceptable for freshwater aquatic life (pH 6–9). For instance, a trend can be seen in the toxicity data collected by Woodward (1976) from two fish species (cutthroat trout, *Salmo clarki*, and lake trout, *Salvelinus namaycush*) tested under similar experimental conditions (water hardness = 35 mg $CaCO_3$•L^{-1}, temperature = 10°C) (Fig. 2). In these experiments, the toxicity of dinoseb was reduced at higher pH values. Ionization of dinoseb, a weak acid, at higher pH could decrease its ability to be transported across the gill, thus reducing its toxicity (Woodward 1976). Dinoseb may also lose its phenolic characteristics and become less toxic under alkaline conditions. Regression analysis of the pooled data set for cutthroat trout and lake trout indicates that the relationship between pH and the

Table 7. Concentration of Dinoseb in Tissues of Yearling Coho Salmon *(Oncorhynchus kisutch)* Following Exposure to Dinoseb

	Dinoseb concentration ($mg \cdot kg^{-1}$)[1]						
Exposure concentration (duration)	Skin	Muscle	Gill	Spleen	Gall bladder	Liver	Kidney
20 $\mu g \cdot L^{-1}$ (384 h)	<0.02	<0.01	0.09	1.4	<0.15	0.40	0.37
60 $\mu g \cdot L^{-1}$ (144 h)	<0.02	<0.01	<0.09	0.61	0.77	<0.07	<0.04

[1] Based on the wet weight of tissue examined; generally, 3–5 fish were combined in one sample.

Source: Lorz *et al.* 1979.

acute toxicity of dinoseb (Fig. 2) can be expressed by the linear equation:

$$\ln 96\text{-h } LC_{50} = 1.96(pH) - 9.71$$

which is significant ($r = 0.93$, $F = 64.9$, $p < 0.001$), although the sample size is small ($n = 12$). (Woodward's [1976] data on cutthroat trout and lake trout also suggest a slight but non-significant mitigation of dinoseb toxicity with increasing water hardness.) The limited data on channel catfish (*Ictalurus punctatus*) and rainbow trout (*Salmo gairdneri*) also tend to support this relationship between pH and acute toxicity of dinoseb (Lipschuetz and Cooper 1961; McCorkle *et al.* 1977; Skelley 1989).

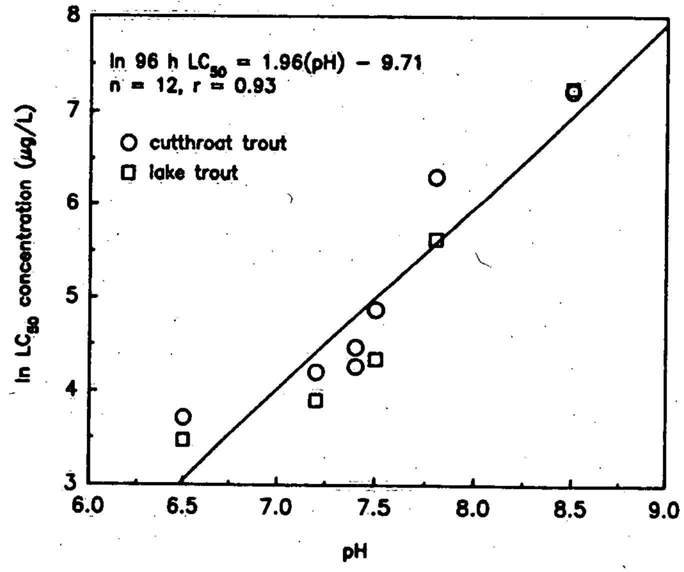

Figure 2. Influence of pH on the acute toxicity of dinoseb to cutthroat trout (*Salmo clarki*) and lake trout (*Salvelinus namaycush*) (pooled data from Woodward 1976).

Because freshwater fish are poikilothermic, their metabolic rate is determined to a large extent by ambient water temperatures. Water temperature thus has the potential to alter dinoseb toxicity through effects on uptake rates, rates of aerobic metabolism (biotransformation), or the chemistry of the chemical itself. The limited data for two species of salmonid (Woodward 1976) suggest an increase in the acute toxicity of dinoseb when water temperature increases from 5°C to 15°C ($r = 0.77$, $n = 6$), but the relationship was not significant ($p = 0.073$).

The toxicity of dinoseb also varies with species (Fabacher and Chambers 1974; Woodward 1976; Nishiuchi 1977; Juhnke and Lüdemann 1978; Lorz *et al.* 1979; Hashimoto and Nishiuchi 1981; Call *et al.* 1984; Geiger *et al.* 1985; Gersich and Mayes 1986) (Appendix A). Median lethal values (96-h LC_{50}s) for native fish species range from 32 and 41 $\mu g \cdot L^{-1}$ for lake trout and cutthroat trout, respectively, to 1400 $\mu g \cdot L^{-1}$ for lake trout.

The formulation of the herbicide can also affect its toxicity. Skelley (1989), for instance, found that different dinoseb products resulted in different levels of toxicity to channel catfish and fathead minnows even when the concentrations were based on the amount of active ingredient in the formulations. For the channel catfish, technical dinoseb (% ai not reported) was the least toxic formulation of five tested, with a 96-h LC_{50} of 58 $\mu g \cdot L^{-1}$ (95% confidence interval [C.I.] = 52–63), whereas Premerge 3® (50.7% ai) was the most toxic, with a 96-h LC_{50} of 28 $\mu g \cdot L^{-1}$ (95% C.I. = 24–33). Conversely, for the fathead minnow, technical dinoseb was the most toxic (88 $\mu g \cdot L^{-1}$, C.I. = 78–98), whereas Premerge 3® was the least toxic, with an LC_{50} of 150 $\mu g \cdot L^{-1}$ (C.I. = 110–200).

Insufficient data were found to evaluate the effects of dinoseb on various life stages of freshwater fish.

Studies concerning other waterborne toxicants suggest that the alevins and newly emerged fry stages are the most sensitive to environmental contaminants (Mayer and Ellersieck 1986). The studies noted in the above evaluation of dinoseb toxicity to fish (e.g., Woodward 1976) were conducted on the early life stages of sensitive fish species. Therefore, water quality guidelines subsequently developed should adequately protect all life stages without incorporation of additional margins of safety.

Chronic Toxicity—Data on the chronic toxicity of dinoseb are available for four freshwater fish species (Table 8). For salmonids, long-term chronic mortality values (6- to 81-d LC_{50}s) ranged from 12 to 125 µg•L^{-1}. Similar end points (8- and 64-d LC_{50}s) for fathead minnows were higher (16 and 500 µg•L^{-1}, respectively). Variability in the responses was related to water hardness, water temperature, duration of exposure, and species tested, with lake trout being the most sensitive species.

Studies on sublethal chronic effects of dinoseb are presented in Table 9. Long-term sublethal studies on the hatchability, development, survival, and growth of fathead minnows exposed to dinoseb for 64 d yielded a LOEL of 48.5 µg•L^{-1} based on the significant effects of a 91% decrease in mean number of survivors and 14% decrease in wet weight compared with controls at 60 d post-hatch (Call *et al.* 1984). In addition, some fish exhibited swollen abdomens and abdominal hemorrhaging at concentrations as low as 4.3 µg•L^{-1}.

Significant reductions in the growth of lake trout were observed at even lower concentrations of dinoseb (0.5 µg•L^{-1}) in soft water (30 mg $CaCO_3$•L^{-1}) (Woodward 1976). Following an exposure of 81 d (21 d pre-hatching to 60 d post-hatching), a LOEL of 0.5 µg•L^{-1} was recorded. This was based on significant reductions of 34.7% and 9.1% in weight and length, respectively, of lake trout fry 60 d old.

Limited data are also available on the short-term sublethal effects of dinoseb on fish (Table 9). Qualitative changes in protein composition and enzyme activity were reported in goldfish (*Carassius auratus*) exposed to 100 and 200 µg•L^{-1} dinoseb for 96 h (Paulov 1980b).

Invertebrates

Available information on the toxicity of dinoseb to freshwater invertebrates indicates that these animals are generally less sensitive than fish. Median lethal concentrations (LC_{50}s) reported in the literature (Sanders 1970; Zitko *et al.* 1976; Paulov 1979; Hashimoto and Nishiuchi 1981) ranged from 100 to 2800 µg•L^{-1} (Table 10).

Incomplete information on the experimental conditions under which the bioassays were conducted prevents detailed comparison of the relative sensitivity of the various taxonomic groups of freshwater invertebrates. Nevertheless, the data suggest that the sensitivities of freshwater snails and *Daphnia* are similar. Studies with lobsters (*Homarus americanus*) indicated that the larvae are extremely susceptible to the toxic effects of dinoseb (96-h LC_{50} = 7.5 µg•L^{-1}) (Zitko *et al.* 1976). These studies, however, cannot infer that the early life stages of aquatic invertebrates may be more sensitive to dinoseb than the adult forms.

More information is required to completely assess the effects of dinoseb on freshwater invertebrates. The available data suggest, however, that fish species should be the focus for guideline development. Therefore, guidelines derived for the protection of freshwater fish species should also protect freshwater invertebrates.

Aquatic Plants

As dinoseb is a potent uncoupler of oxidative phosphorylation, it could have adverse effects on primary productivity in aquatic ecosystems. In turn, inhibition of autotrophic production may translate into impacts on higher trophic levels that depend either directly or indirectly on algae as an energy source.

Research on the effects of dinoseb has focused on three classes of freshwater algae: the green algae (Chlorophyceae), blue-green algae (Cyanophyceae), and diatoms (Bacillariophyceae) (Table 11). Reported EC_{50}s for green algae ranged from 1032 to 3897 µg•L^{-1} when inhibition of growth was taken as the experimental end point (e.g., Hawxby *et al.* 1977; Hess 1980). Median effective concentrations for inhibition of photosynthesis (based on O_2 evolution) were 432, 745, >2400, and >2400 µg•L^{-1} for *Chlorella*, *Lyngbya*, *Chlorococcum*, and *Anabaena* spp., respectively (Hawxby *et al.* 1977). No significant toxic effects were observed in two species of diatoms exposed to 2000 µg•L^{-1} dinoseb (Palmer and Maloney 1955). Insufficient data were found to evaluate the influence

Table 8. Chronic Toxicity of Dinoseb to Freshwater Fish

Species	Test type	Formulation, % ai	Life stage	Duration (d)	LC_{50} ($\mu g \bullet L^{-1}$)	Temperature (°C)	pH	Hardness mg $CaCO_3 \bullet L^{-1}$	Reference
CYPRINIDAE									
Fathead minnow	F,M	Technical,	NR	8	500	25	7.5	48	Call *et al.* 1984
(Pimephales promelas)	F,M	98%		64	16	25	7.5	48	
SALMONIDAE									
Cutthroat trout	F,M	Technical,	30 d	10	125	10	7.4	162	Woodward 1976
(Salmo clarki)		95.8%							
Lake trout	F,M	Technical,	30 d	10	65	10	7.4	162	Woodward 1976
(Salvelinus namaycush)	F,M	95.8%		40	12	10	7.4	162	
	F,M			81	12	10	7.4	162	
Coho salmon	NR	Premerge	Yearling	6	88	NR	NR	NR	Lorz *et al.* 1979
(Oncorhynchus kisutch)									

F = flow-through
M = measured concentrations
NR = not reported

Table 9. Sublethal Effects of Dinoseb on Freshwater Fish

Species	Concentration ($\mu g L^{-1}$)	Duration (d)	Effect	Reference
CYPRINIDAE				
Fathead minnow	48.5	64	Reduced growth rate	Call *et al.* 1984
(Pimephales promelas)	(LOEL)		(14% compared with controls)	
Goldfish	100	4	Qualitative changes in enzyme activity	Paulov 1980b
(Carassius carassius)	200	4	Qualitative changes in enzyme activity	
SALMONIDAE				
Lake trout	0.5 (LOEL)[1]	81	Weight 65% of controls at 81 d	Woodward 1976
(Salvelinus namaycush)	1.6	81	Weight 64% of controls at 81 d	
	2.3	81	Weight 65% of controls at 81 d	
	4.9	81	Weight 55% of controls at 81 d	
	10.0	81	Weight 40% of controls at 81 d	

[1] Length and weight of fry (at 60 d post-hatching) significantly different from those of control group at 81 d ($p = 0.05$).

of chemical or physical parameters on the toxicity of dinoseb to freshwater algae.

One study was found concerning the effects of dinoseb on aquatic vascular plants. O'Brien and Prendeville (1979) examined the effects of dinoseb on the cell membrane permeability of duckweed (*Lemna minor*). Exposures of the plants for 6–9 h to dinoseb concentrations of 240 $\mu g \bullet L^{-1}$ or more resulted in leakage of electrolytes into a deionized solution 17 h after transfer to the dinoseb-free medium. In these experiments, the specific conductance of the ambient solution was measured. The leakage of electrolytes, measured by changes in electrical conductance, was taken as an indicator of cell membrane permeability; quantities of individual ions leaking were not recorded, and EC_{50}s were not calculated. Similar conductivity changes in the ambient solution were observed after 29 h at lower concentrations (24 $\mu g \bullet L^{-1}$).

In summary, comparison of available dinoseb toxicity data for aquatic plants and fish indicates that the latter should be the focus during development of water quality guidelines for the protection of freshwater aquatic life.

Summary of Existing Guidelines

The Michigan Department of Natural Resources (1988) has developed an aquatic life guideline equivalent for dinoseb, referred to as an aquatic chronic value (ACV). The department has deemed that the toxicity of dinoseb to sensitive aquatic biota is significantly influenced by ambient pH and as a result has recommended an ACV based upon the following linear

Table 10. Summary of Available Information on the Effects of Dinoseb on Freshwater Invertebrates

Species	Test type	Formulation % ai	Duration (h)	LC_{50} ($\mu g \cdot L^{-1}$)	Hardness (°C)	pH	Hardness (mgCaC03•L-1)	End point	Reference
CRUSTACEA									
Daphnia magna	S,U	Dinoseb acetate, NR	24	100	NR	NR	NR	LC_{50}	Paulov 1979
	S,U		1	1000	NR	NR	NR	LC_{50}	
	S,U	Technical, >99%	48	240	20	7.9	77	LC_{50}	Gersich and Mayes 1986
Daphnia pulex	NR	Technical, NR	3	2300	NR	NR	NR	LC_{50}	Hashimoto and Nishiuchi 1981
Gammarus fasciatus	NR	Technical, NR	24	2800	NR	NR	NR	LC_{50}	Sanders 1970
	NR		48	2500	NR	NR	NR	LC_{50}	
	NR		96	1800	NR	NR	NR	LC_{50}	
Orconectes limosus	SR,M	NR	215	1000	8	NR	NR	NOEL	Zitko *et al.* 1976
	SR,M		144	10 000	12	NR	NR	NOEL	
MOLLUSCA									
Indoplanorbis exustus	NR	Technical, NR	48	160	NR	NR	NR	LC_{50}	Hashimoto and Nishiuchi 1981
Semisulcospira libertina	NR	Technical, NR	48	130	NR	NR	NR	LC_{50}	Hashimoto and Nishiuchi 1981

S = static bioassay
SR = static renewal
F = flow-trough
M = measured concentrations
U = unmeasured concentrtions
NR = not reported

Table 11. Summary of Available Information on the Effects of Dinoseb on Freshwater Algae

Species	Test type	Duration (h)	EC ($\mu g L^{-1}$)	Temperature (°C)	pH	Effect[1]	Reference
CHLOROPHYCEAE							
Chlorella pyrenoidosa	S,U	24	1032	25	6.6	50% growth	Hawxby *et al.* 1977[2]
	S,U	24	432	25	6.6	50% photosynthesis	
Chlorella variegata	S,U	72	2000	22	NR	No toxic effects	Palmer and Maloney 1955[3]
Chlorococcum sp.	S,U	24	>2400	25	6.6	50% growth	Hawxby *et al.* 1977
	S,U	24	>2400	25	6.6	50% photosynthesis	
Chlamydomonas eugametos	S,U	48	3897	25	NR	50% growth	Hess 1980[4]
	S,U	48	1193	25	NR	15% growth	
Scenedesmus obliquus	S,U	72	2000	22	NR	No toxic effects	Palmer and Maloney 1955
CYANOPHYCEAE							
Lyngbya sp.	S,U	24	1417	25	6.6	50% growth	Hawxby *et al.* 1977
	S,U	24	745	25	6.6	50% photosynthesis	
Anabaena variabilis	S,U	24	>2400	25	6.6	50% growth	Hawxby *et al.* 1977
	S,U	24	>2400	25	6.6	50% photosynthesis	
Cylindrospernum licheniforme	S,U	72	2000	22	NR	No toxic effects	Palmer and Maloney 1955
Microcystis aeruginosa	S,U	72	2000	22	NR	No toxic effects	Palmer and Maloney 1955
BACILLARIOPHYCEAE							
Gomphonema parvulum	S,U	72	2000	22	NR	No toxic effects	Palmer and Maloney 1955
Nitzschia palea	S,U	72	2000	22	NR	No toxic effects	Palmer and Maloney 1955

S = static bioassay
U = unmeasured concentrations
EC = concentration of dinitrophenol that resulted in the specified effect
NR = not reported

[1] Growth = inhibition of growth rate; Photosynthesis = inhibition of photosynthesis as measured by O_2 evolution.
[2] Technical-grade dinoseb, amount of active ingredient not reported.
[3] Commercial-grade dinoseb, amount of active ingredient not reported.
[4] Technical-grade dinoseb, amount of active ingredient not reported.

equation: ACV = 1.5837 pH - 12.8931 (µg•L^{-1}). The data used to derive this regression were not available or evaluation. It is, however, similar to the relationship calculated for pH and median lethal values for cutthroat trout and lake trout (Fig. 2).

Guideline

Analysis of the available data on the toxicity of dinoseb to aquatic biota indicates that fish are the most sensitive organisms. Accordingly, the recommended guidelines for dinoseb are based on dose/response data available for freshwater fish. The quantity and quality of the aquatic toxicity and fate data meet the minimum data requirements established for developing Canadian water quality guidelines (CCME 1991). In accordance with the CCME guideline development approach, when suitable information exists on chronic toxicity, the lowest LOEL for an aquatic species is multiplied by a safety factor of 0.1 to arrive at the guideline value. Review of the dinoseb toxicity information reveals that the most sensitive LOEL reported was 0.5 µg•L^{-1}, which caused a significant reduction in the growth of early life stages of lake trout (Woodward 1976) (Table 9). Multiplication of this LOEL by a safety factor of 0.1 results in a guideline value of 0.05 µg•L^{-1} for the herbicide dinoseb. The range of acute and chronic toxicity data for selected taxonomic groups of freshwater aquatic life is compared against this guideline value in Figures 3 and 4.

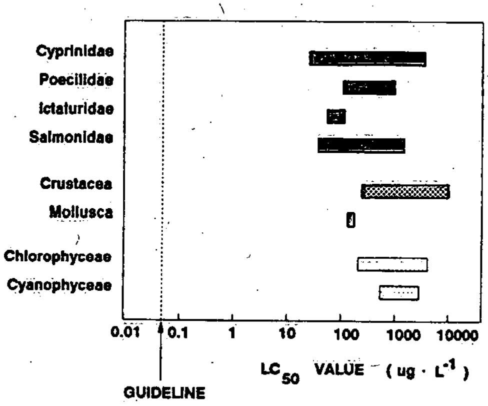

Figure 3. Acute toxicity range of dinoseb to fish (▮), invertebrates (▨), and algae (░).

Agricultural Uses

In previous Canadian water quality guideline documents, recommended guidelines for livestock and irrigation water were usually derived by either evaluating and adapting existing guidelines, objectives, standards, or criteria from other jurisdictions or, in the case of livestock water, recommending the Canadian drinking water quality guideline as an interim guideline for livestock watering. The livestock watering and irrigation water guidelines for dinoseb are derived following guideline development protocols that have recently been drafted for the Canadian Council of Ministers of the Environment (Environment Canada 1990).

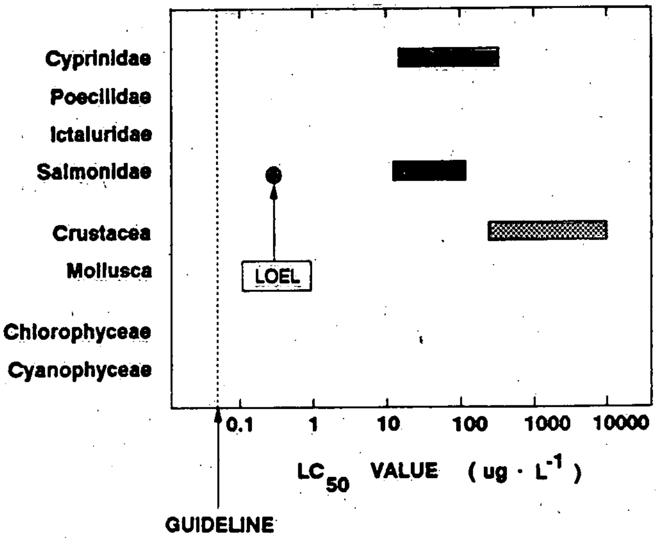

Figure 4. Chronic toxicity range of dinoseb to fish (▮) and invertebrates (▨).

Livestock Watering

Toxicity to Livestock and Related Biota

Dinoseb can potentially affect mammals, birds, and other animals through direct and indirect routes of exposure. Direct dermal exposures may result when non-target organisms inhabit treated areas or areas affected by spray drift. Indirect dermal exposure may result from contact with treated vegetation or soils in treated areas. Oral exposure may result from ingestion of contaminated vegetation, animals, or water.

Mammals—The U.S. EPA's FIFRA suspension of registrations of dinoseb-based products (U.S. EPA 1986) was based on a review of the mammalian toxicity of these chemicals. One reason dinoseb is hazardous to mammals is the high potential for exposure together with the high dermal and oral toxicity of the compound. One study documenting an accidental exposure of an applicator who was dermally exposed through a leaking backpack sprayer indicated that dinoseb is acutely toxic to humans at relatively low dosages (dermal LD_{50} of approximately 75 mg•kg^{-1} for one human) (U.S. EPA 1986). Studies with laboratory animals also revealed that dinoseb has the potential to cause cataracts (Gosselin *et al.* 1981; Hayes 1982), male sterility (Brown 1981), and damage to the immune system (Dandliker *et al.* 1980). Recent studies on the teratological effects of dinitrophenol compounds in rabbits (Leist 1986a, 1986b) have identified serious developmental effects. These latter data suggested that exposure of pregnant women to dinoseb may pose a significant risk of birth defects (U.S. EPA 1986). The U.S. EPA and Health and Welfare Canada have reached the common conclusion that pesticide applicators are exposed to significant and unacceptable health risks while handling the herbicide (Agriculture Canada 1989b). It was judged that risk of potential male reproductive effects, teratogenic effects, and cataract formation is still unacceptably high for dinoseb applicators, even if measures are taken to minimize exposure (protective clothing, gloves, etc.) (Agriculture Canada 1989b).

An assessment of human contamination and toxicity related to dinoseb application, secondary contact, and ingestion was presented by the U.S. EPA (1986). The results indicated that mixing and loading result in the highest rates of exposure. At the highest application rates (used for pre-emergence treatments), those employed in mixing and loading may have daily exposures of approximately 39 mg•kg^{-1} body weight, or roughly 50% of the estimated dermal LD_{50}. Pilots, flaggers (those directing locations for aerial spray operations), ground-boom applicators, and hand sprayers may also be exposed to unsafe levels of dinoseb under certain circumstances. Other farm workers may be exposed to pesticide drift during application or to pesticide residues on vegetation. Additional exposure may result from contact with contaminated farm equipment or clothing. Contamination of groundwater sources may also represent a source of exposure to dinoseb.

Livestock may be directly exposed to dinoseb drift during aerial or ground-boom application. In addition, oral and dermal exposures may occur if livestock are permitted to graze in recently treated fields or adjacent areas or consume contaminated water. Wildlife species inhabiting nearby areas may receive even higher exposures to dinoseb as a result of uncontrolled grazing in treated fields. The U.S. EPA (1986) reported that application rates of dinoseb of 11.2 kg•ha^{-1} could result in residue levels on long grass, leaves, and leafy crops of over 1000 mg•kg^{-1}. At the maximum application rate (8.0 kg•ha^{-1}) recommended for products that were registered in Canada, residue levels of 700 mg•kg^{-1} could be expected. Assuming that the dietary concentration/dose relationship developed for rats by Linder *et al.* (1982) applies in the field, dietary doses of dinoseb could reach 50 mg•kg^{-1}•d^{-1} (using a value of 70 g food intake•kg^{-1} body weight•d^{-1}). Dermal exposures and exposures due to ingestion of contaminated water would further augment daily dietary exposure.

A. Acute and Chronic Toxicity

Dinoseb is acutely toxic to mammals at low levels (10–90 mg•kg^{-1}) relative to other herbicides (paraquat LD_{50} = 236 mg•kg^{-1}; glyphosate LD_{50} = 7940 mg•kg^{-1}; diuron LD_{50} > 10 000 mg•kg^{-1}). At least one human death has been directly attributable to an accidental dermal exposure to dinoseb (U.S. EPA 1986). The available data on the acute toxicity of dinoseb to mammals, including livestock, are presented in Table 12. The effects of sublethal exposures to dinoseb in humans include systemic poisoning, injury to the eyes and skin, cataracts, and weight loss. In other mammals (rodents), weight loss and reduced growth rates are the most commonly reported effects of chronic exposure, although methemoglobinemia has also been observed in sheep (Table 13).

B. Reproductive Effects

Long-term exposures (> 200 d) of mice and rats to sublethal levels (1 mg•kg^{-1}•d^{-1}) of dinoseb have resulted in adverse effects on the testes (Brown 1981) and decreased weight of pups (Irvine 1981). Shorter exposures (3–70 d) to higher levels of the chemical have produced similar effects (Preache and Gibson 1975a, 1975b; Linder *et al.* 1982; Giavini *et al.* 1986a, 1986b; Leist 1986b). Reduced fecundity (Preache and Gibson 1975b) and decreased fetal survival (Spencer and Sing 1982) have been observed in mice and rats exposed to dinoseb. A summary of mammalian reproductive effects of dinoseb is presented in Table 14.

C. Teratogenicity

Teratogenic effects of dinoseb in rats, mice, and rabbits have been documented. Skeletal abnormalities have been reported in fetuses exposed to dinoseb during various stages of gestation (Preache and Gibson 1975a, 1975b; Kavlock *et al.* 1985; Giavini *et al.* 1986a, 1986b; Leist 1986a, 1986b). In general,

Table 12. Acute Toxicity of Dinoseb[1] to Mammals

Species	Dose $(mg \cdot kg^{-1} \cdot d^{-1})$	Exposure period (d)	Method of administration	Effect	Reference
Man	NR[2,3]	1	Dermal	Mortality	U.S. EPA 1986
Man	75 (est.)[4]	1	Dermal	LD$_{50}$	U.S. EPA 1986
Sheep	45.0	1	Oral	50% mortality	Frøslie 1976
Rat	9.0	3	IP	20% mortality	McCormack *et al.* 1980
Rat	11.2	3	IP	100% mortality	McCormack *et al.* 1980
Rat	12.5	3	IP	100% mortality	McCormack *et al.* 1980
Rat	15.8	3	IP	100% mortality	McCormack *et al.* 1980
Rat	22 (est.)	21	Oral	14% mortality	Hall *et al.* 1978
Rat	38 (est.)	21	Oral	100% mortality	Hall *et al.* 1978
Rat	40.0	1	Oral	Acute LD$_{50}$	U.S. EPA 1985
Rat	59.0	1	Oral	Acute LD$_{50}$ for females	U.S. EPA 1986
Rat	89.0	1	Oral	Acute LD$_{50}$ for males	U.S. EPA 1986
Mouse	14.1	1	IP	LD$_{50}$ at 32°C	Preache and Gibson 1975a
Mouse	17.7	4	SC	13% maternal mortality	Preache and Gibson 1975b
Mouse	18.8	3	IP	45% maternal mortality	Preache and Gibson 1975b
Mouse	20.2	1	IP	LD$_{50}$ at 25°C	Preache and Gibson 1975b
Mouse	22.5	1	IP	LD$_{50}$ at 6°C	Preache and Gibson 1975b
Mouse	32.0	4	Oral	18% maternal mortality	Preache and Gibson 1975b
Mouse	41.0	1	Oral	Acute LD$_{50}$	U.S. EPA 1985
Rabbit	75.0	1	Dermal	LD$_{50}$	U.S. EPA 1986
Rabbit	356.0[2]	1	Dermal	LD$_{50}$	U.S. EPA 1986
Guinea pig	25.0	1	Oral	Acute LD$_{50}$	U.S. EPA 1985

IP = intraperitoneal injection
SC = subcutaneous injection
NR = not reported

[1] 2-*sec*-butyl-4,6-dinitrophenol (technical grade, 97% ai) unless otherwise specified.
[2] Alkanolamine salt formulation.
[3] Accidental exposure.
[4] Estimated from accidental exposure immediately above.

Table 13. Chronic Toxicity of Dinoseb[1] to Mammals

Species	Dose $(mg \cdot kg^{-1} \cdot d^{-1})$	Exposure period (d)	Method of administration	Effect	Reference
Man	NR	NR	Dermal	Systemic poisoning	U.S. EPA 1986
Man	NR	NR	Dermal	Injury to eyes and skin	U.S. EPA 1986
Man	NR	NR	Oral	Cataracts	U.S. EPA 1986
Sheep	45.0	1	Oral	Methemoglobinemia	Frøslie, 1976
Rat	1.0	730	Oral	Decreased thyroid weights	Hazelton Inc. 1977
Rat	2.5 (est.)	60	Oral	Reduced growth rate	Hall *et al.* 1978
Rat	6.9	5	Oral	Maternal weight loss	Spencer and Sing 1982
Rat	9.0	3	IP	Maternal weight loss	McCormack *et al.* 1980
Rat	9.1	70	Oral	No increase in body weight	Linder et al. 1982
Rat	10.0	10	Oral	Rate of growth reduced	Giavini *et al.* 1986b
Rat	15.6	70	Oral	Rapid weight loss (19% of BW)	Linder *et al.* 1982
Rat	17.9	10	Oral	Maternal weight loss	Giavini *et al.* 1986a
Rat	22.2	70	Oral	Rapid weight loss (38% of BW)	Linder *et al.* 1982
Hamster	50% of LD$_{50}$	1	Oral	Decreased antibody production	Dandliker *et al.* 1980
Rabbit	10.0	10	Oral	Maternal weight loss	Leist 1986a

IP = intraperitoneal injection
BW = body weight
NR = not reported

[1] 2-*sec*-butyl-4,6-dinitrophenol (technical grade, 97% ai).

Table 14. Summary of Information on the Effects of Dinoseb[1] on Mammalian Reproduction

Species	Dose (mg•kg^{-1}•d^{-1})	Exposure period (d)	Method of administration	Effect	Reference
Rat	1.0	203	Oral	Decreased pup body weight	Irvine 1981
Rat	8.0	3	IP	Decreased fetal body weight	McCormack *et al.* 1980
Rat	9.1	70	Oral	Atypical sperm morphology	Linder *et al.* 1982
Rat	9.1	70	Oral	17% reduction in sperm count	Linder *et al.* 1982
Rat	9.2	5	Oral	40% reduction in fetal survival	Spencer and Sing 1982
Rat	10.0	2	IP/SC	Fetal development rate reduced	Beaudoin and Fisher 1981
Rat	13 (est.)	60	Oral	Diffuse atrophy of testes	Hall *et al.* 1978
Rat	15.0	10	Oral	Decreased fetal body weight	Giavini *et al.* 1986b
Rat	15.6	70	Oral	Infertility in males	Linder *et al.* 1982
Rat	15.6	20	Oral	Decrease in weight of gonads	Linder *et al.* 1982
Rat	15.6	30	Oral	70% reductin in sperm count	Linder *et al.* 1982
Rat	15.6	50	Oral	94% reduction in sperm count	Linder *et al.* 1982
Rat	17.9	10	Oral	Decreased fetal body weight	Giavini *et al.* 1986b
Rat	22.2	70	Oral	Infertility in males	Linder *et al.* 1982
Mouse	1.0	700	Oral	Adverse effects on testes	Brown 1981
Mouse	7.5	3	IP	Decreased fetal body weight	Preache and Gibson 1975a
Mouse	17.7	4	SC	Decreased fecundity	Preache and Gibson 1975b
Rabbit	10.0	10	Oral	Decreased fetal body weight	Leist 1986b

IP = intraperitoneal injection
SC = subcutaneous injection

[1] 2-*sec*-butyl-4,6-dinitrophenol (technical grade, 97% ai).

Table 15. Summary of Information on the Teratogenic Effects of Dinoseb[1] on Mammals

Species	Dose (mg•kg^{-1}•d^{-1})	Exposure period (d)	Method of administration	Effect	Reference
Rat	15.0	10	Oral	Fetal skeletal abnormalities	Giavini *et al.* 1986a
Mouse	7.5	3	IP	Increased rate of hydrocephaly	Preache and Gibson 1975b
Mouse	7.5	3	IP	Fetal skeletal abnormalities	Preache and Gibson 1975a
Mouse	17.7	4	SC	Fetal skeletal abnormalities	Preache and Gibson 1975b
Mouse	20.0	9	Oral	Fetal skeletal abnormalities	Preache and Gibson 1975b
Mouse	26.0	1	Oral	Fetal skeletal abnormalities	Kavlock *et al.* 1985
Mouse	33.0	1	Oral	Fetal skeletal abnormalities	Kavlock *et al.* 1985
Rabbit	10.0	13	Oral	Fetal neural tube defects	Leist 1986a
Rabbit	10.0	13	Oral	Fetal skeletal abnormalities	Leist 1986a
Rabbit	10.0	10	Oral	Fetal skeletal abnormalities	Leist 1986b

IP = intraperitoneal injection
SC = subcutaneous injection

[1] 2-*sec*-butyl-4,6-dinitrophenol (technical grade, 97% ai).

it appears that higher dosages of dinoseb are required to produce teratogenicity (7.5–33.0 mg•kg^{-1}•d^{-1}) than are required to produce more generalized reproductive effects (1–22 mg•kg^{-1}•d^{-1}) (Table 14). To complicate matters, levels of dinoseb known to be teratogenic in mammals are similar to dosages that resulted in maternal toxicity (Table 15).

A summary of data on exposures of mammals that resulted in no adverse effects is provided in Table 16. The no-observed-adverse-effect level (NOAEL) is in the 3–15 mg•kg^{-1}•d^{-1} range.

D. Carcinogenicity and Mutagenicity

According to the U.S. EPA (1987), no evidence of a carcinogenic response was observed in a 2-year chronic feeding study in which dinoseb was administered to rats at levels as high as 10 mg•kg^{-1}•d^{-1}. It was also noted that, with the exception of an

Table 16. Summary of Information on Dinoseb[1] Exposures that Resulted in No Adverse Effects on Mammals

Species	Dose ($mg \cdot kg^{-1} \cdot d^{-1}$)	Exposure period (d)	Method of administration	Effect	Reference
Rat	3.0	NR	Oral	No teratogenic effects	U.S. EPA 1986
Rat	3.8	70	Oral	No effect on growth	Linder *et al.* 1982
Rat	3.8	70	Oral	No effect on male fertility	Linder *et al.* 1982
Rat	3.8	70	Oral	No atypical sperm morphology	Linder *et al.* 1982
Rat	5.0	10	Oral	No effect on growth rate	Giavini *et al.* 1986b
Rat	9.1	70	Oral	No effect on male fertility	Linder *et al.* 1982
Rat	10.0	10	Oral	No fetal skeletal malformations	Giavini *et al.* 1986a
Rat	10.0	10	Oral	No fetal visceral malformations	Giavini *et al.* 1986a
Rat	10.0	10	Oral	No fetal visceral abnormalities	Giavini *et al.* 1986a
Rat	10.0	2	IP or SC	No effect on embryo survival	Beaudoin and Fisher 1981
Rat	10.0	730	Oral	No carcinogenic response	Hazelton Inc. 1977
Rat	13.8	5	Oral	No effect on growth rate	Spencer and Sing 1982
Rat	15.0	10	Oral	No effect on reproduction	Giavini *et al.* 1986b
Mouse	6.3	10	IP	No adverse effects on fetuses	Preache and Gibson 1975b
Mouse	15.0	1	Oral	No adverse effects on fetuses	Chernoff and Kavlock 1983
Rabbit	3.0	13	Oral	No teratogenic effects	Leist 1986a, 1986b
Rabbit	10.0	13	Oral	No adverse effects on females femelles	Leist 1986a

IP = intraperitoneal injection
SC = subcutaneous injection
NR = not reported

[1] 2-sec-butyl-4,6-dinitrophenol (technical grade, 97% ai).

Table 17. Maximum Daily Intakes (MDIs) of Dinoseb for Mammals

Species	LD_{50}[1] ($mg \cdot kg^{-1}$)	LOEL[2] ($mg \cdot kg \cdot d^{-1}$)	NOEL[3] ($mg \cdot kg \cdot d^{-1}$)	MDI[4] ($mg \cdot kg \cdot d^{-1}$)
Man	75.0	NA	NA	NA
Rat	<40–90	1.0	0.0	1.0
Mouse	14–40	1.0	0.0	1.0
Rabbit	75.0	10.0	3.0	5.5
Guinea pig	25.0	NA	NA	NA
Mean				1.8[5]

NA = not available

[1] Lethal dose via oral exposure route, except for man, which is via dermal exposure.
[2] Lowest-observed-effect level (published from dose–response studies) (U.S. EPA 1987).
[3] No-observed-effect level (highest exposure level that is less than LOEL).
[4] Maximum daily intake = geometric mean of LOEL and NOEL.
[5] Geometric mean of rat, mouse, and rabbit MDIs.

Source: Information in text and tables

increase in DNA damage in bacteria, dinoseb was not mutagenic in a number of organisms, including *Salmonella typhimurium, E scherichia coli, Saccharomyces cerevisiae, Drosophila melanogaster,* and *Bacillus subtilis.* The dinoseb formulations Premerge (dinoseb) and Dyanap (dinoseb plus naptalam) were not mutagenic in the Ames mutagen assay (Eisenbeis *et al.* 1981). Recently, however, Xu and Schurr (1990) labelled dinoseb a "strongly positive" genotoxic compound as a result of its ability to cause bacterial DNA damage.

The available information on the effects of dinoseb on mammals can be used to calculate maximum daily intake (MDI) levels for each species. MDIs for rats, mice, and rabbits are 1.0, 1.0, and 5.5 $mg \cdot kg^{-1} \cdot d^{-1}$, respectively (Table 17). Calculation of the geometric mean of these MDIs results in an MDI for mammals of 1.8 $mg \cdot kg^{-1} \cdot d^{-1}$. Assuming that the sensitivities of ungulates and rodents are similar (as suggested by limited acute toxicity data), this generalized MDI may be used as a basis for calculating acceptable levels of dinoseb for wildlife and livestock watering.

Table 18. Summary of Information on the Effects of Dinoseb on Birds

Species	Dose (mg•kg^{-1}•d^{-1})	Formulation % ai	Exposure period (d)	Method of administration	Type of effect	Reference
Mallard	11.5	Dinoseb acid, NR	1	Oral	Acute LD$_{50}$	U.S. EPA 1986
Mallard	27[1]	Technical, 97.6%	14	Oral	Acute LD$_{50}$	Hudson *et al.* 1984
Bobwhite quail	42.5	Dinoseb acid, NR	1	Oral	Acute LD$_{50}$	U.S. EPA 1986
Bobwhite quail	122	Alkanolamine salt[2]	1	Oral	Acute LD$_{50}$	U.S. EPA 1986
Japanese quail	354[3]	Technical, 95.8%	5	Oral	Acute LD$_{50}$	Hill and Camardese 1986
Ring-necked pheasant	26.4	Technical, 97.6%	14	Oral	Acute LD$_{50}$	Hudson *et al.* 1986
Ring-necked pheasant	515[1]	Technical, NR	NR	Oral	Subacute LD$_{50}$	U.S. EPA 1986

NR = not reported

[1] Only a single dose was administered.

[2] Alkanolamine salt (51% emulsifiable concentrate).

[3] LC$_{50}$ as mg·kg^{-1} ai in a 5-d ad libitum diet derived by probit analysis.

Table 19. Summary of Information on the Effects of Dinoseb on Other Non-target Organisms

Species	Dose[1]	Effect	Reference
Fasciola hepatica (liver fluke)	4.3[2] 2.0[2]	20-d LC$_{50}$ for embryos Reduced embryonic developmental rate	Christian *et al.* 1985
Coleomegilla maculata (beetle)	1.3[3]	LD$_{50}$ from topical application	Stam *et al.* 1978
Geocoris punctipes (spider)	0.2[3]	LD$_{50}$ from topical application locale	Stam *et al.* 1978
Deroceras reticulatum (slug)	1.6[4]	25% reduction in population size	Barry 1969

[1] Concentration, dose administered, or application rate.

[2] Concentration in µg·L^{-1}, formulation used not reported.

[3] Dose in µg per individual, formulation used not reported.

[4] Application rate in kg ai·ha^{-1}, formulation used not reported.

Birds—The limited data available concerning the effects of dinoseb on birds (Table 18) indicate that these animals exhibit a wide range of sensitivity to the herbicide. An acute LD$_{50}$ of 11.5 mg•kg^{-1}•d^{-1} indicates that mallards are quite susceptible to the toxic effects of dinoseb. Upland gamebirds, such as the bobwhite quail and the ring-necked pheasant, appear to be more resistant. Documented kills of gamebirds and songbirds in treated agricultural areas attest to the toxicity of dinoseb to birds (U.S. EPA 1986).

Microbes—The effects of dinoseb on soil microorganisms have been documented in a number of studies. Hegazi *et al.* (1979) and Vlassak *et al.* (1976) demonstrated that dinoseb is a specific inhibitor of nitrogen fixation in soil, primarily because of its effect on the N$_2$-fixing *Azotobacter* bacteria. Torstensson (1975) reported broader effects, including acute toxicity, on a variety of microbiota (including various species of *Azotobacter* and *Rhizobium*) in pure cultures and in soils; reduction in dry weight and number of organisms was measured, but EC$_{50}$s were not reported. Stojanovic *et al.* (1972) observed severe effects on populations of bacteria (99% reduction), streptomycetes (67% reduction), and fungi (100% reduction) at extremely high concentrations of technical dinoseb in soils (11 227 kg•ha^{-1}). Conversely, Lewis *et al.* (1978) reported no effects on soil microbial activity, as measured by CO$_2$ evolution and dehydrogenase activity, when dinoseb was applied at recommended rates (3.4 kg ai•ha^{-1}). Further, no effects on the rate of alfalfa decomposition were observed when dinoseb was applied at 100 times the recommended rate (Lewis *et al.* 1978).

Other Non-target Organisms—Information on the effects of dinoseb on non-target organisms not already addressed above is presented in Table 19. These data are difficult to interpret because of atypical exposure units. Results with the liver fluke (*Fasciola hepatica*) (Christian *et al.* 1985) suggest that dinoseb treatment of areas destined for future livestock grazing may be beneficial in terms of controlling the transmission of liver flukes. Data from ladybird beetles and spiders suggest low toxicity of dinoseb relative to insecticides (Stam *et al.* 1978). Dinoseb was rated as relatively ineffective at controlling slugs in cornfields in Ohio (Barry 1969).

Guideline

Information available on the acute toxicity of dinoseb to mammals (Frøslie 1976; U.S. EPA 1986) indicates that toxicity is similar across broad taxonomic groups. It can be postulated, therefore, that the maximum daily intake (MDI) calculated for mammals using data on rabbits, rats, and mice could also apply to livestock. Thus, this MDI was used to develop water quality guidelines for livestock watering.

Maximum daily intakes are expressed in milligrams per kilogram per day. Calculation of water quality guidelines, therefore, requires information on livestock body weights and daily water intakes. Water consumption varies considerably with ambient air temperature, humidity, and levels of activity, and with milk production in mammals. Table 20 provides a summary of information on livestock body weights and daily water intakes.

Table 20. Livestock Body Weights and Daily Water Intakes

Species	Body weight (kg)	Daily water intake ($L{\cdot}d^{-1}$)
Lactating dairy cattle	320–820	70–200
Beef cattle	180–730	15–80
Sheep	40–120	8–15
Goat	10–100	NA
Horse	200–600	20–45
Swine	23–180	2–4.5
Poultry	0.5–30	NA

NA = not available

Source: Dr. W. Buckley, Animal Scientist, Agassiz Research Station, Agriculture Canada, pers. com.

The following equation (from U.S. EPA 1987) was used to calculate livestock watering guidelines for dinoseb:

$$\text{RfD} = \text{MDI}/\text{SF}$$

where:

RfD = reference dose ($\mu g{\cdot}kg^{-1}{\cdot}d^{-1}$)
MDI = 1800 $\mu g{\cdot}kg^{-1}{\cdot}d^{-1}$ (from Table 17), and
SF = safety factor (=0.1).

The safety factor applied above was selected in accordance with the U.S. National Academy of Sciences/U.S. EPA Office of Drinking Water guidelines for long-term exposures to toxic substances in drinking water. This safety factor reflects the difference between the uncertainty factors used in calculating long-term and 10-d health advisories (i.e., 1000/100 = 10, or × 0.1) for humans consuming dinoseb-contaminated drinking water (U.S. EPA 1987). Thus, the RfD is 180 $\mu g{\cdot}kg^{-1}{\cdot}d^{-1}$.

In mammals, a drinking water equivalent level (DWEL) is calculated as follows:

$$\text{DWEL} = (\text{RfD} \cdot \text{BW})/\text{WIR}$$

where:

RfD = reference dose = 180 $\mu g{\cdot}kg^{-1}{\cdot}d^{-1}$
BW = body weight = 820 kg (dairy cow)
WIR = water intake rate = 200 $L{\cdot}d^{-1}$.

As their high water consumption maximizes dietary exposures in dairy animals, a DWEL was developed using lactating dairy cattle. DWELs for livestock watering were calculated to be approximately 740 $\mu g{\cdot}L^{-1}$. This calculation assumes that 100% of the exposure to dinoseb results from the ingestion of drinking water.

Unfortunately, no information is available on the relative contributions of drinking water, food, and dermal exposures for livestock. In the absence of this information, the assumed percentage (20%) of daily exposure contributed through ingestion of drinking water (the relative source contribution, or RSC) was used in the calculation of the water quality guideline (U.S. EPA 1987):

$$\begin{aligned}\text{Livestock WQG} &= \text{DWEL} \cdot \text{RSC}\\ &= 740\ \mu g{\cdot}L^{-1} \cdot 20\%\\ &= 150\ \mu g{\cdot}L^{-1}\end{aligned}$$

Thus, the recommended water quality guideline for dinoseb for livestock watering is 150 $\mu g{\cdot}L^{-1}$. The guideline was developed to protect the most sensitive livestock watering use (i.e., lactating dairy animals) and should be appropriate for other livestock watering uses.

Irrigation

Toxicity to Non-target Plant Species

Because of its relatively high toxicity and rapid uptake, dinoseb has the potential to adversely affect non-target crops if residues are present in irrigation waters. Contamination of irrigation waters may occur as a result of return flows from dinoseb-treated fields, contamination of groundwater, or direct application of the herbicide to drainage ditches. In addition to direct toxicity, secondary effects of dinoseb on non-target crop species may affect their productivity. Significant reductions of bacterial populations have been reported in dinoseb-treated soils (Stojanovic *et al.* 1972; Vlassak *et al.* 1976). The former study, however, had a view toward the disposal of large quantities of pesticides in soils. The reductions in the numbers of soil bacteria have the potential to affect nitrification (Torstensson 1975) and nitrogen fixation (Hegazi *et al.* 1979; Lindström *et al.* 1985). Inhibition of nitrogenase activity in soils or in root nodules can result in specific effects on plant growth.

Table 21. Summary of Information on Irrigation Water Uses in Canada

Province	Area under irrigation in 1985 (ha)	Major irrigated crops (% of provincial total)
British Columbia	109 000	Hay and pasture (65.8%), tree fruits (14.2%)
Alberta	454 000	Hay and pasture (41.8), cereals (38.9%), sugar beets (6.9%)
Saskatchewan	101 000	Hay and pasture (74.4%), cereals (19.2%)
Manitoba	11 330	Cereals (37%), vegetables (21.3%), potatoes (14.5%)
Ontario	40 000	Tobacco (63.8%), vegetables (10.8%)
Quebec	14 700	Hay and pasture (53.3%), cereals (21.2%)
Atlantic Canada	1 900	Potatoes (30.8%)

Source: Modified from CCREM 1987.

To develop an irrigation guideline, information regarding irrigation patterns (i.e., which crops are irrigated and the rates used) and crop-specific toxicity is required. Information on irrigation patterns in Canada (CCREM 1987) is presented in Table 21. In the Canadian Water Quality Guidelines (CCREM 1987), an annual irrigation rate of 1000 mm or 10^7 L•ha^{-1} was assumed. This rate is considerably higher than that used in most parts of Canada and provides a margin of safety in dinoseb exposure estimates.

Vascular plants exhibit a wide range of sensitivities to dinoseb. There is a suggestion in the literature that the susceptibility of various plant species to pre-emergence applications of dinoseb is correlated with seed size (Barrons and Watson 1969). Plants with large seeds were generally more tolerant of dinoseb than small-seeded plants (Schroeder and Warren 1971). Differences between families were also evident, with the legumes being the most resistant and mustards and solanoids (e.g., eggplants, tomatoes, peppers) being the most sensitive (Table 22).

Table 22. Sensitivity of Selected Crops to Pre-emergence Treatments of Dinoseb

Species	I_{50} shoot growth (kg•ha^{-1})	I_{50} root growth (kg•ha^{-1})
GRAMINEAE		
Timothy *(Phleum pratense)*	3.4	2.9
Rice *(Oryza sativa)*	5.9	7.9
Barley *(Hordeum vulgare)*	14.0	12.4
Wheat *(Triticum aestivum)*	14.9	11.1
Oats *(Avena sativa)*	17.0	19.5
Sorghum *(Sorghum bicolor)*	26.3	13.0
Corn *(Zea mays)*	38.5	23.0
CHENOPODIACEAE		
Sugar beet *(Beta vulgaris)*	2.8	1.5
COMPOSITAE		
Lettuce *(Lactuca sativa)*	2.2	1.9
CRUCIFERAE		
Cabbage *(Brassica oleracea)*	3.9	3.8
CUCURBITACEAE		
Cucumber *(Cucumis sativus)*	10.0	9.7
Squash *(Cucurbita pepo)*	23.9	22.3
LEGUMINOSAE		
Ladino clover *(Trifolium repens)*	8.5	8.2
Alfalfa *(Medicago sativa)*	12.4	8.6
Soybean *(Glycine max)*	26.2	21.1
Snap bean *(Phaseolus vulgaris)*	34.0	32.2
Pea *(Pisum sativum)*	>60.0	34.0
LILIACEAE		
Onion *(Allium cepa)*	1.2	1.3
Asparagus *(Asparagus officinalis)*	4.6	4.8
SOLANACEAE		
Eggplant *(Solanum melongena)*	2.7	2.7
Tomato *(Lycopersicon esculentum)*	2.9	2.4
Pepper *(Capsicum frutescens)*	7.8	6.9
UMBELLIFERAE		
Carrot *(Daucus carota)*	2.1	2.1

I_{50} = concentration of herbicide that caused a 50% reduction in the fresh weight of shoots and roots under greenhouse conditions.

Guideline

No irrigation water quality guidelines for dinoseb were available from provincial or federal agencies in Canada, from state or federal agencies in the United States, or from international agencies (International Joint Commission, World Health Organization). Water quality guidelines for irrigation waters were, therefore, developed using available information relating to dinoseb toxicity and crop irrigation patterns.

No data on the chronic toxicity or sublethal effects of dinoseb on terrestrial non-target plants were found in the literature. To take into account the variability in the toxicity of dinoseb to terrestrial non-target plant species, family final acute values (FFAVs) for dinoseb were estimated by taking the geometric mean of the species I_{50} values (the concentration of herbicide that caused a 50% reduction in the fresh weight of both shoots and roots) for the two most sensitive species in each group. Maximum acceptable application rates (MAARs) for each group were calculated by dividing the FFAV by a safety factor of one order of magnitude (MacDonald 1990). The MAARs for cereals/hays, legumes, and other crops are presented in Table 23.

Table 23. Maximum Acceptable Dinoseb Application Rates for Selected Groups of Terrestrial Plants

Plant group	FFAV (kg•ha^{-1})	MAAR (kg•ha^{-1})
Leguminosae	9.29	0.247
Graminae	4.63	0.123
Other crops	1.62	0.043

FFAV = family final acute value (geometric mean of concentrations toxic to two sensitive species of each family (see text)

MAAR = maximum acceptable application rate (see text)

The MAAR was then divided by the irrigation rate (10^7 L•ha^{-1}•yr^{-1}) to calculate the maximum acceptable toxicant concentration (MATC) for each group of terrestrial plants. For most of the irrigation water uses in Canada, the MATC calculated for cereals and hays (Graminae) of 46 µg•L^{-1} could be adopted as the Canadian irrigation water quality guideline. For legume culture, the guideline for dinoseb in irrigation waters is 93 µg•L^{-1}. A guideline of 16 µg•L^{-1} is recommended for the protection of all other agricultural crops. These water quality guidelines were developed under the assumption that annual exposures to the equivalent of a single non-toxic dose of dinoseb would not have adverse impacts on non-target plant species.

In accordance with the above derivation, a guideline of 16 µg•L^{-1} is recommended for irrigation water uses in Canada.

Recreational Water Quality and Aesthetics

Organoleptic Effects

No information was found relating to the ability of dinoseb to impart a taste or odour to water. In addition, information related to the tainting of fish flesh by accumulated dinoseb was not found.

Guideline

At present, there is no evidence to indicate that recreational water quality and aesthetics would be adversely affected by pesticide residues when pesticides are used according to label instructions. Therefore, water quality guidelines are not recommended at this time. A guideline for recreational water quality should consider health effects due to exposure from dermal absorption in addition to aesthetic considerations. Absorption through the skin may equal or exceed intake from food and drinking water for compounds with high dermal absorption rates. Unfortunately, quantitative data are usually lacking.

Industrial Water Supplies

Guideline

At present there is no evidence to indicate that industrial water supplies would be adversely affected by pesticide residues when pesticides are used according to label instructions. Therefore, water quality guidelines are not recommended at this time.

SUMMARY

After an evaluation of the published information on the herbicide dinoseb, Canadian water quality guide-lines were derived (Table 24). The background information on dinoseb 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 24. Recommended Water Quality Guidelines for Dinoseb

Water use	Recommended guidelines
Raw water for drinking water supply	No recommended guideline
Freshwater aquatic life	0.05 µg•L^{-1}
Agricultural uses	
Livestock watering	150 µg•L^{-1}
Irrigation	16 µg•L^{-1}
Recreational water quality and aesthetics	No recommended guideline
Industrial water supplies	No recommended guideline

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Appendix A
Acute Toxicity of Dinoseb to Freshwater Fish

Table A-1. Acute Toxicity of Dinoseb to Freshwater Fish

Spice	Test type	Duration (h)	Formulation % ai	Life stage	LC_{50} ($\mu g \cdot L^{-1}$)	Temperature (°C)	pH	Hardness (mg $CaCO_3$)	Reference
CYPRINIDAE									
Goldfish (*Carassius auratus*)	NR	48	Technical, NR	NR	65	NR	NR	NR	Hashimoto and Nishiuchi, 1981
Common Carp (*Cyprinus carpio*)	NR	48	Technical, NR	NR	70	NR	NR	NR	Hashimoto and Nishiuchi, 1981
Blacknose dace (*Rhinichthys atratulus*)	F,U	24	Technical, NR	NR	240	21	8.0	79	Lipschuetz and Cooper, 1961
Fathead minnow (*Pimephales promelas*)	S,U	96	Technical, > 99 %	NR	230	17	7.2	77	Gersich and Mayes, 1986
	S,U	96			130	17	7.5	77	
	S,U	96			160	17	7.6	77	
Fathead minnow (*Pimephales promelas*)	F,M	96	Technical, 98 %	30 d	410	24	7.3	50	Geiger *et al.* 1985
	F,M	96			700	25	7.3	50	
Fathead minnow (*Pimephales promelas*)	F,M	24	Technical, 98 %	30 d	800	25	7.5	48	Call *et al.* 1984
	F,M	48			700	25	7.5	48	
	F,M	96			700	25	7.5	48	
Fathead minnow (*Pimephales promelas*)	S,U	96	Technical, NR	1 yr	88	12	7.4	44	Skelley, 1989
	S,U	96	Premerge 50.7 %	1 yr	150	12	7.4	44	
Golden orfe (*Leuciscus idus*)	NR	NR	NR	NR	2 000	NR	NR	NR	Juhnke and Lüdemann, 1978
Pond loach (*Misgurnus anguillicaudatus*)	NR	48	Technical, NR	NR	180	25	NR	NR	Hashimoto and Nishiuchi, 1981

S = static bioassay
N = measured concentrations
U = unmeasured concentrations
F = flow-through
NR = not reported
SR = static renewal

Table A-1. Continued

Spice	Test type	Duration (h)	Formulation % ai	Life stage	LC$_{50}$ (µg•L^{-1})	Temperature (°C)	pH	Hardness (mg CaCO$_3$)	Reference
Medaka	NR	NR	Commercial,	NR	23	25	5.0	NR	Nishiuchi, 1977
(*Oryzias latipes*)	NR	NR	NR[1]		83	25	6.0	NR	
	NR	NR			240	25	7.0	NR	
	NR	NR			280	25	8.0	NR	
	NR	NR			420	25	9.0	NR	
	NR	NR			630	25	10.0	NR	
Medaka	NR	48	Technical,	NR	150	25	NR	NR	Hashimoto and Nishiuchi, 1981
(*Oryzias latipes*)			NR						
Harlequin fish	NR	24	Tubotox,	NR	3 400	NR	NR	NR	Alabaster, 1969
(*Rasbora heteromorpha*)	NR	48	90 %		3 000	NR	NR	NR	
POECILIDAE									
Mosquitofish	S,U	24			870	21	NR	NR	Fabacher and Chambers, 1974
(*Gambusia affinis*)	S,U	24			960	21	NR	NR	
Guppy	S,U	96	Technical,	Fingering	106	26	6.0	90	Saarikoski and Viluksela, 1981
(*Poecilia reticulata*)	S,U	96	NR		353	26	7.0	90	
	S,U	96			984	26	8.0	90	
Guppy	NR	48	Aretit,	NR	800 (est)	NR	NR	NR	Paulov, 1980
(*Poecilia reticulata*)			47 %						
ICTALURIDAE									
Channel catfish	S,U	96	NR	1 yr	118	20	8.2	80	McCorkle *et al.* 1977
(*Ictalurus punctatus*)									
Channel catfish	S,U	96	Technical,	1 yr	58	12	7.4	44	Skelley, 1989
(*Ictalurus punctatus*)			95 %						
Channel catfish	S,U	96	Premerge,	1 yr	28	12	7.4	44	
(*Ictalurus punctatus*)			50.7 %						

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[1] Unspecified commercial formulations of Dormant or Premerge.

Table A-1. Continued

Spice	Test type	Duration (h)	Formulation % ai	Life stage	LC_{50} ($\mu g \cdot L^{-1}$)	Temperature (°C)	pH	Hardness (mg $CaCO_3$)	Reference
SALMONIDAE									
Rainbow trout	F,U	24	Technical,	NR	300	18	8.0	79	Lipschuetz and Cooper, 1961
(*Salmo gairdneri*)	F,U	24	NR		73	18	6.9	79	
Atlantic Salmon (*Salmo salar*)	SR,M	96			70[2]	9	NR	NR	Zitko *et al.*, 1976
Cutthroat trout	S,M	96	Technical,	Finger-	58	5	7.2	35	Woodward, 1976
(*Salmo clarki*)	S,M	96	95.8 %	ing	67	10	7.2	35	
	S,M	96			42	15	7.2	35	
	S,M	96			41	10	6.5	35	
	S,M	96			130	10	7.5	35	
	S,M	96			1 350	10	8.5	35	
	S,M	96			550	10	7.8	35	
	S,M	96			340	10	7.8	120	
	S,M	96			280	10	7.8	240	
	S,M	96			152	10	7.4	162	
	S,M	96			71	10	7.4	35	
	S,M	96			87	10	7.4	35	
Lake trout	S,M	96	Technical,	Finger-	135	5	7.2	35	Woodward, 1976
(*Salvelinus*	S,M	96	95.8 %	ing	44	10	7.2	35	
namaycush)	S,M	96			36	15	7.2	35	
	S,M	96			32	10	6.5	35	
	S,M	96			77	10	7.5	35	
	S,M	96			1 400	10	8.5	35	
	S,M	96			280	10	7.8	35	
	S,M	96			140	10	7.8	120	
	S,M	96			155	10	7.8	240	
	FC,M	96			79	10	7.4	162	

[2] Lethal threshold