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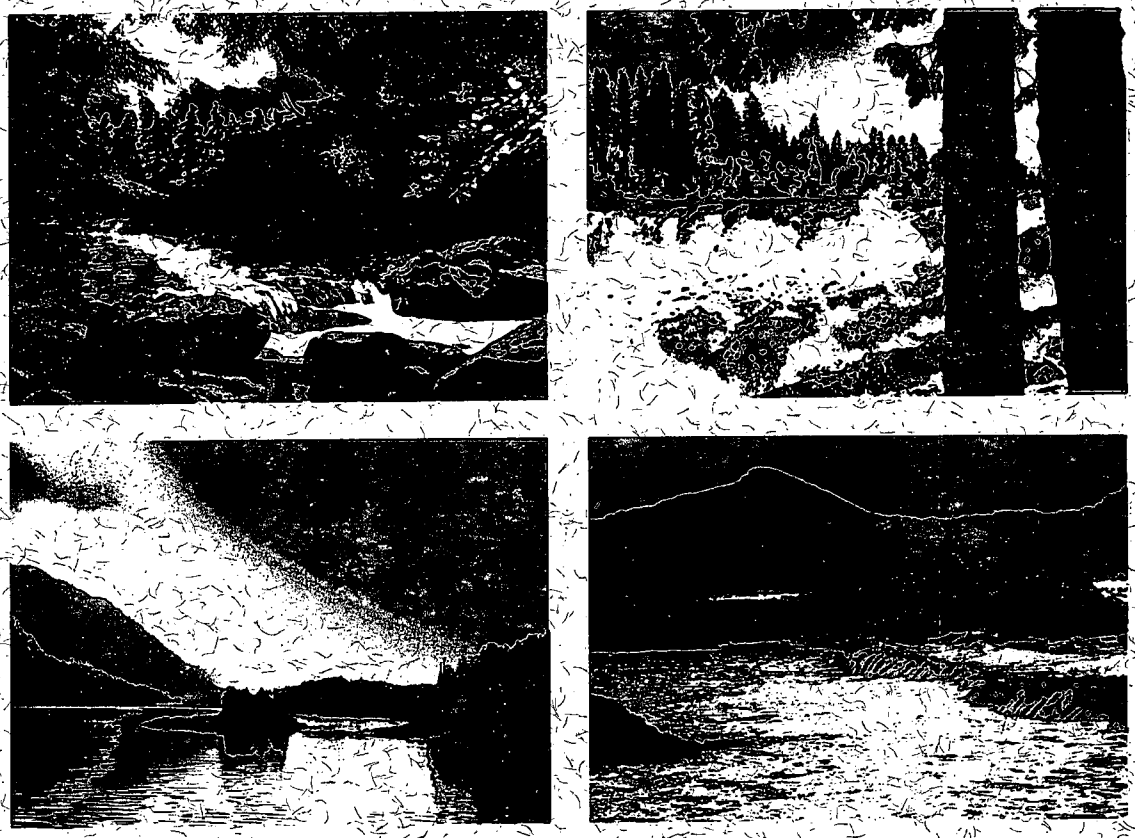
Canadian Water Quality Guidelines for Captan

R.A. Kent and B.D. Pauli



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INLAND WATERS DIRECTORATE
WATER QUALITY BRANCH
OTTAWA, ONTARIO, 1991

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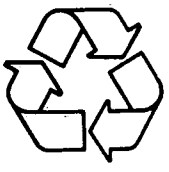

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Abstract

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

Résumé

On a examiné la documentation relative aux utilisations, au devenir et aux effets du captan sur l'eau naturelle utilisée comme eau potable non traitée, sur la vie aquatique en eau douce, sur l'utilisation de l'eau pour l'agriculture, sur la qualité de l'eau pour les loisirs et l'esthétique, ainsi que sur les approvisionnements en eau pour l'industrie. Ces renseignements sont résumés dans cette publication. À partir de cette étude, des lignes directrices sur la qualité de l'eau sont recommandées pour la protection d'utilisations particulières de l'eau.

Canadian Water Quality Guidelines for Captan

J. Gareau, D.M. Trotter, R.A. Kent, and B.D. Pauli

SOURCES, OCCURRENCE, AND CHARACTERISTICS

Uses and Production

Captan, the common name for the fungicide N-(trichloromethylthio)cyclohex-4-ene-1,2-dicarboximide (IUPAC), is a yellow or white crystal or powder, depending on purity. The Chemical Abstracts Service (CAS) name is N-[(trichloromethyl)thio]-4-cyclo-hexene-1,2-dicarboximide (Worthing and Walker, 1987), and the CAS registry number is 133–06–2. The structural formula for captan can be seen in Figure 1. Captan is manufactured by the action of ammonia on tetrahydrophthalic anhydride, which in turn is produced by the imide, which, through interaction with perchloromethyl mercaptan, results in captan (Agriculture Canada, 1982). Captan was first registered in Canada in 1953 (Agriculture Canada, 1990). Synonyms for captan are given in Table 1; names of some of the 82 different captan formulations and mixtures registered in Canada for agricultural and home use are presented in Table 2.

Captan is a broad-spectrum, nonsystemic fungicide. It is used as a seed treatment, foliage spray, post-harvest spray or dip, and preplant soil treatment to control disease in vegetables, fruit, seeds, nuts, berries, cereal grains, forage, ornamentals, and packing boxes. Its main use is as a seed treatment and for protection against mildews, late blight, and fungal pathogens.

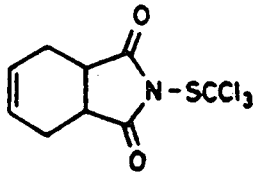


Figure 1. Structural formula for captan.

Table 1. Captan Synonyms

Synonym	Reference
Trichloromethylthiocyclohex-4-ene-1, 2-dicarboximide	NIOSH, 1979
N-trichloromethylthio-cis-delta4-cyclohexene-1, 2-dicarboximide	NIOSH, 1979
N-(trichloromethylthio)-4-cyclohexene-1,2-dicarboximide	NIOSH, 1979
N-[(trichloromethyl)thio] tetrahydrophthalimide	NIOSH, 1979
3a,4,7,7a-tetrahydro-2-[(trichloromethyl)thio]-1H-isoindole-1,3-(2H)-dione	Windholz *et al.*, 1983
N-trichloromethylthio-3a,4,7, 7a-tetrahydrophthalimide	Windhloz *et al.*, 1983
N-(trichloromethylmercapto)-delta4-tetrahydrophthalimide	Windholz *et al.*, 1983
cis-N(trichloromethyl)thio-gamma-cyclohexene-1,2-dicarboximide	Hermanutz *et al.*, 1973
N-trichloromethylthiotetra-hydrophthalimide	Metcalf, 1971

Captan is available as 90%–95% of the compound as the active ingredient (ai) in technical products and 3.5%–80% ai in end-use products (Agriculture Canada, 1990). Captan products are most widely used as wettable powders, flowable powders, and dusts. The wettable powder will not dissolve in water, but is formulated as a suspension at various concentrations of the active ingredient per litre. Other formulations are available as granules (U.S. EPA, 1984). Methods of captan application include dusting, spraying, misting, dipping, mixing, and low-pressure bomb aerosols (U.S. EPA, 1986). Rates of application are reported to be 0.22–11.2 $kg \cdot ha^{-1}$ for fields and 0.47–6.25 $g \cdot kg^{-1}$ for seed treatments (Goring, 1972; Agriculture Canada, 1982). For fruit production, the Ontario Ministry of Agriculture and Food (OMAF, 1989) recommended rates of application between 1.7 and 4.5 $kg \cdot ha^{-1}$.

Table 2. Trade Names and Formulations for Some Canadian Registered Captan Products

Trade name	Formulation
Argox B-3 Dual Purpose Seed Treatment	30% captan, 15% diazinon, 16.6% lindane
Agrox D-L Plus Insecticide/Fungicide Seed Treatment Powder	15% captan, 15% diazinon, 25% lindane
Captan 7.5 Dust	7.5% captan
Captan 10 Dust	10% captan
Captan 10 Wettable Powder	10% captan
Captan 5001 Seed Protectant	32% captan
Captan SP-4 Flowable	48% captan
Captan 4 Flowable	48% captan
Captan 50 WG Fungicide	50% captan
Captan 80 WP Fungicide	80% captan
Captan Technical	92% captan
Captan 75 Seed Protectant	75% captan
Chevron Captan Technical	92% captan
Chipman Agrox I.F. Dual Purpose Seed Treatment	30% captan, 15% diazinon
Chipman Captan Flowable Seed Treatment Fungicide	30% captan
Chipman Captan Flowable Fungicide	35% captan
Chipman Captan–Benomyl Fungicide	50% captan, 10% benomyl
Chipman Captan 30 Methoxychlor 3 Flowable Seed Treatment	30% captan, 3% methoxychlor
Chipman Captan–Methoxychlor 75-3 Seed Protectant	75% captan, 3% methoxychlor
C-I-L Fruit & Garden Fungicide	10% captan, 2% benomyl
C-I-L Soil & Bulb Dust	5% captan, 5% carbaryl
Clean Crop 5% Captan Fungicide	5% captan
Clean Crop Captan 50WP Fungicide	50% captan
Co-op Captan 50% WP Fungicide	50% captan
Co-op D-L+C Drillbox Seed Treatment Powder	15% captan, 15% diazinon, 25% lindane
Co-op Flower & Garden Dust	5% captan, 4% malathion, 3% methoxychlor
Co-op Potato Seed Piece Treatment	7.5% captan, 0.1% diazinon
Co-op Rose Dust Insecticide–Fungicide	5% captan, 2% malathion, 5% methoxychlor, 20% sulphur
Co-op Tomato Dust	5% captan, 5% carbaryl
DCL Dual Purpose Seed Treatment	22% captan, 7% diazinon, 12% lindane
DCT Dual Purpose Seed Treatment	18% captan, 6% diazinon, 14% thiophanate-methyl
Drillbox Diazinon–Lindane plus Captan Seed Treatment	10% captan, 10% diazinon, 17% lindane
Flowable Captan Seed Protectant	39.1% captan
Fruit Plus Fruit Tree Spray	10% captan, 6.1% phosmet
Gammasan Seed Treatment Powder	10% captan, 6% benomyl, 50% lindane
Green Cross Drillbox Lindasan Combination Treatment	10% captan, 37.5% lindane
Green Cross Fruit Tree & Garden Spray	10% captan, 10% carbaryl, 5% malathion
Green Cross Multi-Purpose Flower & Vegetable Dust	5% captan, 2% malathion, 5% methoxychlor, 20% sulphur
Green Cross Garden Rose & Evergreen Dust	5% captan, 5% carbaryl, 4% malathion, 3% thiophanate-methyl
Green Cross Rose Dust Insecticide Fungicide	5% captan, 5% carbaryl, 4% malathion, 20% sulphur
Green Leaf Bulb Dust Insecticide–Fungicide	5% captan, 5% carbaryl, 0.75% rotenone
Green Leaf Golden Garden Dust	5% captan, 5% methoxychlor, 0.75% rotenone
Gustafson Evershield II C-M Seed Protectant	34.7% captan, 0.9% malathion
Later's Bulb Dust	5% captan, 5% carbaryl
Later's Fruit-Guard Fruit Tree & Berry Spray	10% captan, 10% carbaryl
Later's Golden Garden Dust	5% captan, 5% methoxychlor, 0.75% rotenone
Martan 50WP Fungicide	50% captan
Orthocide 50WP	50% captan
Pfizer Captan 50W	50% captan
Pfizer D-iazinon L-indane C-aptan Drillbox Seed Treatment	15% captan, 15% diazinon, 25% lindane
Pfizer Potato Seed Piece Dual Purpose Treatment Powder	7.5% captan, 0.1% diazinon
Plant Products 7.5% Captan Greenhouse Fungicide Dust	7.5% captan
Potato Seed Piece Treatment Dust	7.5% captan
Scott's Cure Insecticide–Fungicide Dust or Spray	5% captan, 2% malathion, 5% methoxychlor, 20% sulphur
Soil & Bulb Dust	5% captan, 5% carbaryl
Stauffer Captan	92% captan
Vitavax–Captan 30W	24% captan, 6% carbathiin
VW&R Guardsman Captan 5% Fungicide	5% captan
Wilson's Bulb & Soil Dust	5% captan, 5% carbaryl

Source: Agriculture Canada, 1990.

In the United States, regulatory action has recently been taken against various captan-containing products. On 24 February 1989, the U.S. Environmental Protection Agency (U.S. EPA, 1989a) announced its intention to "cancel registrations and to deny registration applications for all pesticide products containing captan as an active ingredient" except for certain seed treatments and some fruit and vegetable applications. All uses of captan on crabapples, cranberries, grapefruit, lemons, limes, oranges, pineapples, quinces, rhubarb, and tangerines have been cancelled (U.S. EPA, 1990).

The reason for the action against captan stemmed from the conclusion of the U.S. EPA that pesticide products containing captan posed a potential risk related to the oncogenicity and mutagenicity of the compound. The agency classified captan as a possible human carcinogen based on data that revealed a statistically and biologically significant oncogenic response in both sexes of mice and in male rats; the EPA's principal concern was the risk of cancer to humans resulting from dietary exposure to captan (U.S. EPA, 1989a). In Canada, Agriculture Canada has taken no recent regulatory action with captan since a consultative report was published in 1982 (S. Keating, 1990, Agriculture Canada, pers. com.). In this report (CCIBP, 1982), the authors concluded that, although the toxicological properties of captan could not be fully characterized, the evidence available at the time indicated that captan was not a human carcinogen.

Quantitative information on the agricultural use of captan in Canada is available for Ontario and New Brunswick. In 1983, captan was used on fruit and vegetables in Ontario; 104 240 kg were used on fruit, and an additional 20 kg were used on vegetables (McGee, 1984). In 1988, 10 kg of captan were used on vegetables, 260 kg were used on field corn, and 71 140 kg were used on fruit (Moxley, 1989).

Captan usage in New Brunswick totalled 5079, 6413, 6953, and 2848 kg during the years 1984, 1985, 1986, and 1987, respectively (Shanks, 1984, 1985, 1986, 1987). In 1988, 4915 kg of the active ingredient were sold (Carr, 1988).

In the province of Quebec, the use of captan *per se* was not listed, but it was reported that 93 753 and 64 403 kg of phthalimides, the group of chemicals that includes captan, were used in the province in 1978 and 1982, respectively (Godon *et al.*, 1983). Captan is not used extensively in Alberta. Its primary usage in Alberta is for agricultural and horticultural seed treatments in amounts of less than 1 t per year (H.P. Sims, 1990, Alberta Environment, pers. com.). Information concerning captan use from other provinces was not available.

Physical and Chemical Characteristics

Physical and chemical characteristics of captan are presented in Table 3. Reported melting points for captan range from 160°C to 178°C, apparently as a result of differences in the purity of the product tested. Differences also exist for the reported water solubilities, which range from <0.5 to 10 $mg \cdot L^{-1}$, and for the octanol–water partition coefficient (K_{ow}). The variation in the reported water solubilities may result from differing degrees of captan hydrolysis as a result of different experimental procedures (Lukens, 1969) or different methods used to calculate captan solubility (U.S. Department of Agriculture, 1986). Further, captan's lack of stability in water has made it difficult to obtain good water solubility and partitioning data (B. Bowman, 1990, Agriculture Canada, pers. com.).

Analytical Techniques

Worthing and Walker (1987) listed the methods of product analysis for captan as gas–liquid chromatography (GLC), high-performance liquid chromatography (HPLC), infrared spectrometry, and total chlorine content analysis after alkaline hydrolysis; residues may be determined by GLC or spectrophotometry of a derivative. Atwood *et al.* (1987) used GLC and HPLC to examine the stability of captan suspensions in aqueous media. Captan in soil can be determined colorimetrically after reaction with resorcinol or with pyridine and tetraethylammonium hydroxide (Burchfield and Schechtman, 1958; Agriculture Canada, 1982). Fungal spore bioassays (Munnecke, 1958; Agnihotri, 1971) and microbial bioassays (Chinn, 1973) have also been used to quantify captan residues in soil. Captan and metabolites in goat milk and meat have been analyzed by GLC, whereas *in situ* fluorometry has been used to analyze captan and captafol simultaneously (Agriculture Canada, 1982). A Russian review (Chircova, 1982) recommended thin-layer chromatography (TLC) after benzene extraction for analysis of captan in foodstuffs. Mattern *et al.* (1990) recently used a multi-residue extraction procedure, separation by capillary column gas chromatography, and detection by mass chromatography with an ion trap mass spectrometer in the chemical ionization mode (GC/CIMS) to examine residues of captan and its metabolite tetrahydrophthalimide in apple, peach,

Table 3. Physical and Chemical Characteristics of Captan

Property	Value	Reference
Chemical formula	$C_9H_8Cl_3NO_2S$	Windholz *et al.*, 1983
Molecular weight	300.57	Windholz *et al.*, 1983
Physical state	Technical (90%–95% purity): pungent, yellow to buff amorphous powder; Pure: odourless, white crystals	U.S. EPA, 1984
Henry's law constant	>4.7 x 10^{-5} atmo·m^3·mol^{-1} at 25°C 0.6 Pa·m^3·mol^{-1} at 20°C	U.S. EPA, 1981 Suntio *et al.*, 1988
Melting point	178°C (pure) 175° (pure) 160°C–170°C (93%–95% pure) 178°C (crystals from CCl_4)	U.S. EPA, 1984 Agriculture Canada, 1982 Agriculture Canada, 1982 Windholz *et al.*, 1983
Vapour pressure	<1.3 mPa at 25°C <7.9 mPa at 25°C 0.038 Pa Not volatile	U.S. EPA, 1984; Worthing and Walker, 1987; Royal Society of Chemistry, 1987 Midwest Research Institute, 1975 Suntio *et al.*, 1988 U.S. EPA, 1984; U.S. Department of Agriculture, 1986
Log octanol–water partition coefficient (log K_{ow})	2.35 2.45 1.8	Leo *et al.*, 1971; Verschueren, 1983 Briggs, 1981 Suntio *et al.*, 1988
Olelyl alcholol–water partition coefficient	140 at 25°C	U.S. EPA, 1980
Specific gravity	1.73 (temperature unspecified)	U.S. EPA, 1980
Log soil–water distribution coefficient standardized for soil organic matter content (log K_{oc})	2.06	Briggs, 1981
Soil retention factor (R_f)	0.39 (soil = silty clay loam, 2.5% organic matter, 39.5% clay)	Dragun and Helling, 1981
Solubility		
Water	<0.5 mg·L^{-1} at 20°C 4.5 mg·L^{-1} 10 mg·L^{-1} 3.3 mg·L^{-1}	Verschueren, 1983 Herzel and Murty, 1984 Lukens, 1969 Worthing and Walker, 1987
Petroleum oils	Practically insoluble	Agriculture Canada, 1982
Chloroform	7.78 g·L^{-1}	Windholz *et al.*, 1983
Tetrachloroethane	8.15 g·L^{-1} at 20°C	Windholz *et al.*, 1983
Cyclohexanone	4.96 g·L^{-1} at 26°C	Windholz *et al.*, 1983
Dioxane	4.70 g·L^{-1} at 26°C	Windholz *et al.*, 1983
Benzene	2.13 g·L^{-1} at 26°C	Windholz *et al.*, 1983
Toluene	0.69 g·L^{-1} at 26°C	Windholz *et al.*, 1983

tomato, and potato samples. The detection limit was 0.1 mg·kg^{-1}. After extraction with dichloromethane, Frank *et al.* (1983) used GLC with a ^{63}Ni-electron capture detector to achieve detection limits of 0.01 µg·g^{-1} in apple tissue and 0.25 µg·L^{-1} in water.

Stephenson (1990) recently summarized the analytical methods for captan and the development of these methods over time. Many of the methods have been developed for the analysis of captan residues on fruit and vegetables. The residues are often confirmed with liquid chromatography and photoconductivity techniques (Gilvydis *et al.*, 1986).

Mode of Action

The principal mode of action of captan in fungal cells is due to its reaction with sulfhydryl groups

(Lukens, 1969). This results in the inhibition of many of the enzyme systems responsible for cellular energy processes, incorporation of inorganic phosphates, and metabolism and synthesis of amino acids (Owens and Novotny, 1959; Goring, 1972). Enzymes that are affected are those containing the –SH radical. Ultimately, captan reduces fungal spore germination, growth, and oxygen uptake (Owens and Novotny, 1959; Richmond and Somers, 1963). At concentrations well above those used in normal agricultural practice (approximately 1000 $mg \cdot kg^{-1}$), the nitrifying and ammonifying abilities of soil microflora are inhibited.

The $SCCl_3$ moiety of captan is fungitoxic and reacts with insoluble thiols. Initially, captan reacts with, and is detoxified by, less vital soluble sulfhydryl groups. Once these sites are exhausted, more vital insoluble or stable sulfhydryl groups are attacked (Lukens and Sisler, 1958; Richmond and Somers, 1963, 1966; Lukens *et al.*, 1965; Lukens, 1969). The rate at which captan reacts with sulfhydryl groups appears to be pH-dependent near the pK_a of the group, but pH-independent below this range. Captan fungitoxicity is reported to increase as the pH is decreased from 7.5 to 4.5 (Lukens and Sisler, 1958).

The reaction of captan with sulfhydryl compounds produces thiophosgene, tetrahydrophthalimide, disulphide, and hydrogen chloride. Thiophosgene is highly reactive and may also attack sulfhydryl, amino, hydroxy, and carboxy groups (Lukens and Sisler, 1958; Owens and Blaak, 1960a, 1960b; Lukens, 1963, 1969; Corbett *et al.*, 1984).

Corbett *et al.* (1984) listed the biochemical effects of the N-trichloromethylthio fungicides as inhibition of thiol enzymes, interactions with membranes, and disruption of mitochondrial reactions, including oxidative phosphorylation and NADH oxidation. Although Lukens and Sisler (1958) reported that thiophosgene is probably the ultimate toxicant, Owens and Novotny (1959) concluded that captan toxicity is due to reactions involving the intact molecule and not its decomposition products. In any case, captan may kill the fungal cells or inhibit their activity, depending on the extent of sulfhydryl exhaustion, sulfhydryl replenishment, and the captan dose (excess quantities of captan prevent sulfhydryl repair). Captan fungitoxicity follows a typical dose–response pattern (Lukens and Sisler, 1958; Owens and Novotny, 1959).

Evidence exists for the translocation of captan and its metabolic products from the roots to the leaves of several plant species grown in captan-treated soil. Evidence of translocation includes damaged leaf margins (Lukens, 1969), captan residues in the foliage of broad beans (Somers and Richmond, 1962), and increased fungal control on the foliage (Stoddard, 1954; Wallen and Hoffman, 1959; Somers and Richmond, 1962). The uptake of captan by fungal spores is rapid and increases linearly with temperature between 0°C and 40°C (Richmond and Somers, 1962a, 1962b). There is generally a direct relationship between uptake and spore sulfhydryl content (Richmond and Somers, 1963). Toxicity is correlated with the amount of captan absorbed by the spores (Owens and Novotny, 1959).

Sources and Pathways for Entering the Aquatic Environment

After application to soil and crop plants, captan has little potential to leave the site and contaminate the nontarget environment. Captan is nonvolatile, is unlikely to exhibit substantial leaching in soil, and rapidly hydrolyzes in water (see Environmental Fate, Persistence, and Degradation below). As a result, environmental concentrations of captan following normal agricultural applications are expected to be low. Other potential routes of contamination include accidental spills, misuse and mishandling, back-siphoning near wells, and washing or loading spray equipment near streams or ponds.

Environmental Concentrations

Few investigations of captan contamination of Canadian groundwater and surface waters have been conducted. Between 1979 and 1984, wells in rural Ontario that were suspected of being contaminated with pesticides were sampled. Captan was not detected (detection limit 0.005 $\mu g \cdot L^{-1}$) in any of the wells suspected of contamination by either runoff and drift (34 wells) or spills (4 wells) (Frank *et al.*, 1987a). In a follow-up study conducted in 1986 and 1987 (Frank *et al.*, 1990a), a further 179 farm wells were analyzed. Captan had been used on 17 farms, but no captan contamination of farm wells was found (detection limit 0.5 $\mu g \cdot L^{-1}$). No captan was detected (detection limit 0.002 $\mu g \cdot L^{-1}$) in 894 water samples collected from the mouths of the Grand, Saugeen, and Thames rivers (southern Ontario) over the period 1981–1985 (Frank and Logan, 1988). Between 1971 and 1985, 211 rural ponds in Ontario suspected of being contaminated with pesticides were sampled by Frank *et al.* (1990b). No captan was detected in any of the ponds (detection limit not given).

In 1989, the Nova Scotia government sampled water in 98 randomly selected rural wells in King's County, Nova Scotia. One well was found to be contaminated with a captan concentration near the detection limit of 0.01 $\mu g \cdot L^{-1}$ (NSDOE, 1990).

Data concerning captan contamination of U.S. surface waters and groundwater are also scarce. Near areas of high pesticide use in California, Maddy *et al.* (1982) failed to detect captan in 54 municipal and private wells monitored for the fungicide above a detection limit of 5.0 $\mu g \cdot L^{-1}$. The U.S. national water quality data base (STORET) to August 1983 records 183 water samples (mainly surface water and effluent) analyzed for captan. Maximum and mean captan concentrations of 0.04 and 0.002 $\mu g \cdot L^{-1}$ were reported, but the number of samples containing captan was not given (U.S. EPA, 1984).

Contamination resulting from washing or loading of spray equipment was reported on a tributary of the Cornwallis River, Nova Scotia, in 1982. Although concentrations of captan in the water were not reported, a fish kill was apparently the result of the spill (Eaton *et al.*, 1986). Environment Canada's national water quality data base (NAQUADAT) for the years 1960–1990 contains one record of captan occurrence in Canadian surface water. This single detection (<0.01 $\mu g \cdot L^{-1}$) was recorded in a farm pond in New Brunswick in 1971.

No information was found in the available literature on captan contamination in precipitation, sediment, or aquatic and terrestrial biota. Captan residues were found quite frequently on cherries, peaches, and strawberries collected at farmers' markets in southern Ontario between 1980 and 1984 (Frank *et al.*, 1987b); 29 of 36 sweet cherry samples, 29 of 36 peach samples, and 96 of 107 strawberry samples were contaminated with captan residues. Strawberries had the highest captan residue concentrations: in 1982, the average concentration was 2.81 $mg \cdot kg^{-1}$.

Environmental Fate, Persistence, and Degradation

Degradation in Soil

Half-lives of captan in soil are quite variable and range from 1 to >65 d (Munnecke, 1958; Burchfield, 1959; U.S. EPA, 1985). As hydrolysis is apparently the primary mechanism of breakdown in soil (Lukens, 1969; Goring, 1972), captan stability increases with decreasing pH and soil moisture content (Goring, 1972; Agriculture Canada, 1982). Rapid captan dissipation (half-life of 3.5 d) was observed in a moist (17.5% water) and slightly acidic (pH 6.4) soil (Burchfield, 1959). The half-life increased to >50 d with a decrease in pH to 6.2 and a reduction in soil moisture content to 1.6%. Kluge (1969), however, reported that captan degradation was not influenced by soil pH changes in the pH range 3.6–7.4. Persistence in soil is also reported to increase with increasing soil organic matter and decreasing temperature (Domsch, 1958; Hermanutz *et al.*, 1973; Frank *et al.*, 1983; U.S. EPA, 1985).

Using sterilized and unsterilized soils, Munnecke (1958) studied the persistence of captan as indicated by the fungicidal activity of the soil at various times following treatment. Both soils, tested 65 d after treatment, exhibited fungicidal activity similar to that of soil assayed 1 d after treatment. Thus, captan appeared to be very stable under the experimental conditions used. Captan was applied to the soil at a rate well above present agricultural applications (approximately 1000 $mg \cdot kg^{-1}$) as a soil drench. In this study, chemical analyses were not conducted to determine the presence of fungitoxic degradation products in the soil during the latter assays. In a forest soil, Kluge (1969) measured a half-life for captan of 3 weeks using a fungal bioassay.

The half-life of captan in soil also varies according to the manner in which it is applied and the application rate used. Half-lives of 1–2 d were observed when captan was uniformly mixed with soil at concentrations ranging from 31.25 to 1000 $mg \cdot kg^{-1}$ (Griffith and Matthews, 1969). When introduced on the surface of glass beads (to simulate its use as a seed coat dressing), the captan concentration varied little from the initial concentration for more than 21 d. The increased persistence was possibly the result of decreased surface area of the seed coat application available for chemical or microbial attack. Fungal spore bioassays were used to determine the almost complete degradation of captan in forest nursery soil 7 d following applications equivalent to 140, 280, and 560 $kg \cdot ha^{-1}$ (Agnihotri, 1971). Captan persistence of <4 and >32 weeks after initial applications of 100 and 1000 $mg \cdot kg^{-1}$, respectively, were based on microbial bioassays (Chinn, 1973).

Only 13% of an initial dose of 1.5 mg ^{14}C-labelled captan applied to the soil of a microcosm (the application rate was equivalent to 1.12 $kg \cdot ha^{-1}$) remained after 20 d. Of the amount remaining (0.195 mg), only 0.02 mg represented the original captan; the remain-

der consisted of unidentified captan degradation products (Cole and Metcalf, 1980).

Field studies have shown captan to be rapidly degraded in soils (U.S. Department of Agriculture, 1986). Even at sites that received 21 $kg \cdot ha^{-1}$ (over two orders of magnitude higher than typical application rates, applied as a drench to sandy loam and loam soils), there were no detectable residues of captan in the top 15 cm of soil 1 week after application (Li and Nelson, 1985). The detection limit of this study was not given.

The available information related to the microbial degradation of captan in soil was briefly reviewed by Sisler (1982). The limited number of studies available indicated that microorganisms play a significant role in the decomposition of captan, with hydrolysis as the important initial degradation mechanism (Lukens, 1969). Captan decomposition also proceeds by means of direct reactions with components of fungal cells, especially the cellular thiol constituents (U.S. Department of Agriculture, 1986). By contrast, bio-degradation may not be a significant fate process in natural waters (U.S. EPA, 1984).

Little information was found concerning the products of captan degradation in soil. Apart from hydrolysis products, captan will presumably react with sulfhydryl groups in living or dead organic material to produce tetrahydrophthalimide, thiophosgene, disulphide, and hydrogen chloride. During a study of the fate of ^{14}C-labelled captan applied to sorghum seedlings in a model ecosystem, Metcalf and Sanborn (1975) found seven unidentified degradation products in small amounts (<0.1 $\mu g \cdot L^{-1}$) in the water of the microcosm.

Mobility in Soil

Captan has limited mobility in soil and is unlikely to leach to any great extent (Goring, 1972; U.S. EPA, 1985). Various investigators have studied the penetration of surface-applied captan into soil by assessing the fungicidal activity of the soil at various depths. Even at high concentrations (2500 $mg \cdot L^{-1}$ of formulation), captan showed little movement downward into the soil (Kendrick and Middleton, 1954; Zentmyer, 1955; Newhall, 1958; Cetas and Whidden, 1960). Munnecke (1961) found that the extent to which captan leaches in various soils depends on the type of soil, the type of soil treatment, and the inherent properties of the fungicide formulation. Captan was found to be confined to the top 1.27 cm of columns of pre-wetted peat moss after application of 20 mL of a 2000 $mg \cdot L^{-1}$ captan suspension. The author noted that other fungicides prepared as solutions (e.g., nabam) penetrated the soil more readily than fungicides prepared as suspensions (e.g., captan, ferbam, zineb). A captan formulation prepared with 1.05-µm particles penetrated soil further than a formulation of 14.5-µm particles. The smaller particle size of the captan suspension increased movement in a loamy sand soil, but had no effect in either a loamy soil or a mixture of 50% peat moss and 50% loamy sand. Captan leaching occurred to a greater extent in a loamy sand than in a loam soil, and in dry soil than in previously wetted soil. Addition of water increased the leaching of captan through the loamy sand column until almost complete removal of the fungicide had occurred after 4800 mL of water leached through the 10.2-cm-high column. Leaching with 4000 mL of water to the soil columns containing the peat moss/loamy sand mixture did not substantially alter the distribution of captan in the column. The author speculated that the restricted mobility of captan in the peat moss was the result of sorption processes.

In a terrestrial model ecosystem, Gile and Gillett (1979) found that 13% of the radioactivity from applied ^{14}C-labelled captan was distributed in the soil, and only 0.01% of the radioactivity reached the groundwater of the microcosm 45 d after planting of captan-treated alfalfa and ryegrass seeds.

No soil sorption partition coefficient (K_d) for captan was found in the scientific literature. A soil retention factor (R_f) of 0.39 was reported by Dragun and Helling (1981) using soil TLC. The authors mentioned that this would mean the compound has "intermediate mobility" in the soil (Hagerstown silty clay loam with 2.5% organic matter and 39.5% clay) used in the TLC runs according to an earlier categorization of general mobility based on R_f values (Helling and Turner, 1968).

Volatilization

Captan has a low vapour pressure (see Table 3) and is considered to be nonvolatile (Munnecke, 1958; Munnecke *et al.*, 1962; Goring, 1972; U.S. Department of Agriculture, 1986). The fungicidal activity of air passed through captan-treated soil columns (100 $mg \cdot kg^{-1}$ soil) indicated that captan was not volatilized from the soil in fungitoxic amounts (Munnecke *et al.*, 1962). The loss of captan from aluminum weighing dishes by volatilization was less than 1% after 100 d

at 40°C. Approximately 0.5% was lost over the same period at 30°C (Whitehouse, 1967).

In other studies of captan volatilization, a recovery of 73% of ^{14}C-labelled captan in the air phase of a microcosm study from an original application as a seed coating was reported by Gile and Gillett (1979), but the authors did not indicate the relative amounts of radioactive captan and CO_2 collected. Specific compounds were not identified during a 36-d study by Ebing and Schuphan (1979) in which ^{14}C-labelled captan was applied to spinach and the volatilized compounds were collected.

Captan did not volatilize "to any great extent" in a diffusion chamber (Latham and Linn, 1965). This led the U.S. Department of Agriculture (1986) to conclude that the volatilization of captan from soil is probably not a significant dissipation process for the compound.

Degradation in Water

Hydrolysis is the primary route of captan degradation in water (U.S. Department of Agriculture, 1986). Several investigators (Burchfield, 1959; Hermanutz *et al.*, 1973; Wolfe *et al.*, 1976a; U.S. EPA, 1985; Dalvi, 1989) have found the reaction to be very rapid. Wolfe *et al.* (1976a, 1976b), for instance, determined the hydrolytic half-life of captan in river water to be 170 min. Frank *et al.* (1983) reported a half-life of 1 h for technical captan at pH 8.5 in water at 22°C. Atwood *et al.* (1987), on the other hand, found a maximum captan loss over 48 h from a 50% wettable powder suspension of only 27%, and they suggested that captan formulated as a wettable powder is more stable than technical captan in water.

Hydrolysis rates increase with increasing temperature (Frank *et al.*, 1983; U.S. EPA, 1984). Hermanutz *et al.* (1973) reported that the half-life of captan in Lake Superior water with a pH of 7.6 is about 7 h at 12°C and about 1 h at 25°C.

Over a pH range of 2–6, Wolfe *et al.* (1976a) reported aqueous hydrolysis to be pH-independent. Above pH 7, however, the reaction was pH-dependent, with persistence decreasing with increasing pH. Using an equation derived from hydrolysis rate constant experiments $\{t_{1/2} = 0.693/[k_{H_2O} + k_{OH}(OH)]\}$, the U.S. EPA (1984) calculated the maximum hydrolitic half-life of captan at various pH levels. In acidic pH (actual pH not given), the half-life was calculated to be approximately 12 h; at pH 7 (and 28°C), the hydrolytic half-life was calculated to be about 155 min; and at pH 10, a half-life of about 10 s was estimated.

The short persistence of captan in water was confirmed by a laboratory fish bioassay study (Hermanutz *et al.*, 1973). Fathead minnows (*Pimephales promelas*) suffered no apparent effects during a 10-d exposure to 550 $\mu g \cdot L^{-1}$ captan, introduced into static (no water exchange) test chambers 3 h prior to the introduction of the fish. This study also showed that the transformation products of captan were not toxic to the fish. When a group of fathead minnows was exposed to the same captan concentration in the static chambers immediately following the addition of the toxicant, all fish died within 8 h.

The reported products of captan hydrolysis in water include tetrahydrophthalimide, tetrahydrophthalmic acid, thiophosgene, hydrogen chloride, 4-cyclohexene-1,2-dicarboximide, CO_2, and sulphur (Fukuto and Sims, 1971; Hermanutz *et al.* 1973; Wolfe *et al.*, 1976a; U.S. EPA, 1984). As mentioned above, Hermanutz *et al.* (1973) commented that these products were not toxic to fathead minnows. Lukens (1969) briefly reported that products of captan hydrolysis were not toxic.

Although isolated microorganisms have the ability to biodegrade captan (Kluge, 1969), Paris *et al.* (1975) found only a slight increase in the degradation rate of captan in an aqueous solution in the presence of microorganisms compared with uninoculated control solutions over a pH range of 5.6–8.0. The U.S. EPA (1984) concluded that biodegradation may not be a significant fate process for captan in natural waters.

Direct photolysis does not appear to be an important determinant of the fate of captan in water. Photolysis studies with a mercury vapour lamp filtered to remove wavelengths below 280 nm indicated a photolytic half-life of >83 d after the data were normalized to conditions equivalent to mid-day sunlight at 30°N latitude (Wolfe *et al.*, 1976b). Laboratory experiments using methylene blue (37.4 $mg \cdot L^{-1}$) as a photosensitizer indicated that photooxidation involving singlet oxygen might be important in the degradation of captan. It has been suggested, however, that humic substances in natural water might quench this reaction (U.S. EPA, 1984). Experiments in natural waters with other similar compounds support this assumption (Wolfe *et al.*, 1976b).

The U.S. EPA (1984) noted that particle-mediated precipitation of captan from water has not been com-

prehensively studied. However, from the octanol–water partition coefficient (log K_{ow} = 1.8) (Suntio *et al.*, 1988) and the soil-water distribution coefficient (log K_{oc} = 2.06) (Briggs, 1981), the U.S. EPA (1984) was able to predict that captan may be moderately removed from water through sorption by particulate matter.

Summary of Environmental Fate

A summary of captan degradation in soil/sediment, water, and biota is presented in Table 4. In soil, the persistence of captan can range from a half-life of 1 d in a thoroughly mixed soil, to little degradation in 21 d under localized application conditions (seed treatments) (Griffith and Matthews, 1969), to 65 d with high application rates (Munnecke, 1958). Little captan remained after 1 week in a forest nursery soil treated with 250 $mg \cdot kg^{-1}$. Both hydrolysis and microbial degradation are significant fate processes for captan in soil, whereas volatilization is not important (U.S. EPA, 1984). The compound is not very mobile in soil and should not leach in appreciable quantities to ground-

Table 4. Summary of Captan Degradation in Soil/Sediment, Water, and Biota

Captan Degradation in Soil/Sediment	Captan Degradation in Water	Captan Degradation in Biota
PHOTOLYSIS • no data	PHOTOLYSIS • not significant (Wolfe *et al.*, 1976b)	• after ingestion in rats, absorption into bloodstream, hydrolytic cleavage in the blood or gastrointestinal tract via cleavage of the N–S bond to form tetrahydrophthalimide and a derivative of the trichloromethylthio side chain
OXIDATION • no data	OXIDATION • no data	
HYDROLYSIS • primary mechanism of degradation (Lukens, 1969; Goring, 1972)	HYDROLYSIS • primary mechanism of degradation (USDA, 1986) • first-order hydrolytic t½ = 1.8 h (estimate) (Syracuse Research Corp., 1989)	• reaction is pH–dependent • major route of excretion is via the urine, with some excretion in the feces and in the expired CO_2
AEROBIC METABOLISM • captan readily degraded in biological systems (USDA, 1986) • major metabolite tetrahydrophthalimide (USDA, 1986)	AEROBIC METABOLISM • not significant (Paris *et al.*, 1975) • t½ = 2–60 d (estimate) (Syracuse Research Corp., 1989)	• approximately 80%–92% of an orally administered dose excreted in 4 d, 50% of the total excretion occurring in the first 48 h (U.S. EPA, 1984)
ANAEROBIC METABOLISM • no data	ANAEROBIC METABOLISM • no data • t½ = 8–240 d (estimate) (Syracuse Research Corp., 1989)	
VOLATILIZATION • not significant (U.S. EPA, 1984)	VOLATILIZATION • no data (U.S. EPA, 1984)	
MOBILITY • little mobility in soil, and little leaching occurs (Goring, 1972; U.S. EPA, 1985) • movement increases with sand content of the soil (Munnecke, 1961)	PERSISTENCE • short persistence because of rapid hydrolysis (USDA, 1986) • t½ = 7 h at 12°C, pH 7.6 (USDA, 1986) • t½ = 0.18–10.3 h (estimate) in surface water and groundwater (Syracuse Research Corp., 1989)	
ADSORPTION/DESORPTION • soil retention factor (R_f) = 0.39 (Dragun and Helling, 1981)		
PERSISTENCE • relatively short half-life (USDA, 1986) • dependent on solubilization, moisture, pH, and temperature (Sisler, 1982) • t½ = 1–2 d (Griffith and Matthews, 1969) • t½ > 50 d in dry soil (Munnecke, 1958) • t½ = 2–60 d (estimate) (Syracuse Research Corp., 1989)		

USDA = U.S. Department of Agriculture

water. In water, hydrolysis is a rapid and important mechanism in captan dissipation and is probably the fate-determining step in natural waters (Wolfe *et al.*, 1976a, 1976b). The rate of hydrolysis increases with temperature and with alkaline pH (U.S. EPA, 1984). Conversely, photolysis and biodegradation are not significant processes in the fate of captan in natural waters. The compound is not likely to volatilize significantly from water, but it may be removed to a certain extent from water through sorption to particulate matter (U.S. EPA, 1984).

RATIONALE

Raw Water for Drinking Water Supply

Guideline

No guideline for captan in drinking water supplies has been developed by the Federal–Provincial Subcommittee on Drinking Water of the Federal–Provincial Advisory Committee on Environmental and Occupational Health for publication in the Guidelines for Canadian Drinking Water Quality (Health and Welfare Canada, 1989a).

Summary of Existing Guidelines

The U.S. EPA (1989) proposed an acceptable level of 15 $\mu g \cdot L^{-1}$ for captan in drinking water supplies. This concentration corresponds to an increased cancer risk of 10^{-6}. No guideline for captan was included in the drinking water guidelines published by the World Health Organization (WHO, 1987). The California State Department of Health Services developed an action level of 350 $\mu g \cdot L^{-1}$ for captan in drinking water, the U.S. EPA produced a "suggested no-adverse-effects level" of 17 $\mu g \cdot L^{-1}$, and New York State published a groundwater quality standard for potable water of 17.5 $\mu g \cdot L^{-1}$ captan (OMOE, 1989).

A maximum residue limit (MRL) of 5.0 ppm captan in fruits and vegetables was established by Health and Welfare Canada (1989b) for the protection of human consumers.

Concentrations in Drinking Water Supply

No information was found on the concentrations of captan in drinking water. Because of the rapid hydrolysis of this compound in water, high concentrations of captan are not expected in drinking water supplies. During studies of surface water and groundwater sources, rural wells, and municipal water supplies (see Environmental Concentrations above), very little contamination by captan was found (Maddy *et al.*, 1982; Spittler *et al.*, 1984; U.S. EPA, 1984; Frank *et al.*, 1987; Frank and Logan, 1988).

Removal by Water Treatment Operations

No information was found on the removal of captan by water treatment operations.

Freshwater Aquatic Life

Summary of Existing Guidelines

In an unpublished report, the U.S. EPA guideline development procedures for water quality criteria (Stephan *et al.*, 1985) were used by the U.S. EPA (1989b) to derive an aquatic life advisory concentration of 0.44 $\mu g \cdot L^{-1}$. This value was based on the lowest mean acute toxicity value derived from standardized tests for eight genera of freshwater and estuarine animals and an experimentally derived acute/chronic ratio.

Toxicity to Aquatic Organisms

Acute Lethal Toxicity

Vertebrates—Captan is highly toxic to fish and to some invertebrates (U.S. Department of Agriculture, 1986). Data concerning the acute toxicity of captan to aquatic organisms are summarized in Appendix A and in Mayer and Ellersieck (1986). Technical-grade captan produced 24-h LC_{50}s of 26.2–139 $\mu g \cdot L^{-1}$ for salmonids (Mayer and Ellersieck, 1986) and 96-h LC_{50}s of 26.2–200 $\mu g \cdot L^{-1}$ for salmonids and other species (Hermanutz *et al.*, 1973; Johnson and Finley, 1980; Mayer and Ellersieck, 1986). A wettable powder solution of 50% captan produced a 72-h LC_{50} of 320 $\mu g \cdot L^{-1}$ for rainbow trout *(Salmo gairdneri)* (Holland *et al.*, 1960). Tooby *et al.* (1975) calculated static LC_{50} values for the harlequin fish *(Rasbora heteromorpha)*. For an 89% solution of captan, the 24-h LC_{50} was 460 $\mu g \cdot L^{-1}$, the 48-h LC_{50} was 330 $\mu g \cdot L^{-1}$, and the 96-h LC_{50} was 300 $\mu g \cdot L^{-1}$. Hashimoto and Nishiuchi (1981) calculated a 48-h LC_{50} of 37 $\mu g \cdot L^{-1}$ for the goldfish (species not named) and a 48-h LC_{50} of 340 $\mu g \cdot L^{-1}$ for the pond loach (*Misgurnus anguilicaudatus*), and a 48-h LC_{50} of 1000 $\mu g \cdot L^{-1}$ for the medaka (*Oryzias latipes*) exposed to technical captan.

Concentrations of 250 and 500 $\mu g \cdot L^{-1}$ of 50% wettable powder solution of captan were reported to kill rainbow trout within 6 and 5 h, respectively (Van Hoof, 1980). In these experiments, conducted at 7°C, pH 8.0, and a water hardness of 250 mg $CaCO_3 \cdot L^{-1}$, the test animals were continuously dosed with the test concentrations. The same species was reported to survive a 3-d exposure to 180 $\mu g \cdot L^{-1}$ without apparent harm (Holland *et al.*, 1960). Under flow-through conditions, exposure to 63.5 $\mu g \cdot L^{-1}$ captan produced 100% mortality in 1-d-old fathead minnow (*Pimephales promelas*) larvae within 24 h. Under static conditions, 550 $\mu g \cdot L^{-1}$ resulted in 100% mortality within 8 h in 90-d-old fathead minnows (Hermanutz *et al.*, 1973). A captan concentration of 1000 $\mu g \cdot L^{-1}$ caused dramatic eye and head damage in larval zebrafish (*Brachydanio rerio*), followed by death after 1.5 h of exposure (Abedi and McKinley, 1967). Less severe eye damage occurred in zebrafish larvae exposed to the same concentration (the duration of the exposure was not specified) in a study by Hermanutz *et al.* (1973). In the same experiment, however, similar injuries to fathead minnow larvae were not found, suggesting species-specific captan sensitivity (Hermanutz *et al.*, 1973). In studies with amphibians, a 48-h LC_{50} of 3000 $\mu g \cdot L^{-1}$ was determined for *Bufo bufo japonicus* tadpoles (Hashimoto and Nishiuchi, 1981).

In summary, the freshwater vertebrate acute toxicity data base for captan contains tests with seven salmonid species, which provided toxicity data from 24-h exposures (10 values), 72-h exposures (1 value), and 96-h exposures (13 values). Additional test species include the fathead minnow *(Pimephales promelas)*, yellow perch *(Perca flavescens)*, carp *(Cyprinus carpio)*, goldfish *(Carassius auratus)*, channel catfish *(Ictalurus punctatus)*, and bluegill *(Lepomis macrochirus)*. The remaining test fish are resident of nontemperate regions.

The data of Abedi and McKinley (1967) resulted from tests with 1% acetone as a solvent carrier. It should be noted that Burrell *et al.* (1980) found that acetone inhibited the toxicity of captan to the aquatic fungus *Pythium ultimum*. At concentrations of acetone above 0.8% v/v of the exposure solution, the acetone was significantly antagonistic to captan toxicity; fungal inhibition was only 4.5% at 1.0% acetone, whereas it was 32% at acetone concentrations below 0.8%. The authors warned that such interactions can lead to underestimations of fungicide toxicity. The acute toxicity data reported by Bowman *et al.* (1982) are not presented in Appendix A, as the test water contained 20 000 $mg \cdot L^{-1}$ methanol.

The possibility that water hardness may increase captan toxicity has been mentioned (U.S. EPA, 1989), but data are lacking; for three species of fish, increasing water hardness appeared to increase toxicity, whereas changes in hardness did not appear to influence the toxicity for a fourth species. Examination of the available toxicity data in Appendix A did not reveal any relationship between captan toxicity and the hardness of the test water.

Invertebrates—Several short-term toxicity tests were available for freshwater invertebrates (Appendix A). Information concerning the test procedures, the formulation used, and the purity of the technical product could not be determined for all of these tests. The lowest LC_{50} (1000 $\mu g \cdot L^{-1}$) resulted from a 48-h test using the snail *Physa acuta*. Additional 48-h LC_{50}s of 1200, 1400, and 1500 $\mu g \cdot L^{-1}$ were reported for the snails *Semisulcospira libertina* and *Indoplanorbis exustus* and the mayfly *Cloeon dipterum*, respectively. LC_{50}s for daphnids include two 3-h values of 1500 and 6800 $\mu g \cdot L^{-1}$ for *Daphnia pulex* and *Moina macrocopa*, respectively (Hashimoto and Nishiuchi, 1981), and a 26-h value of 1300 $\mu g \cdot L^{-1}$ captan in acetone for *Daphnia magna* (Frear and Boyd, 1967). The formulation used was not reported for the latter study. A wettable powder formulation containing 80% captan produced a 96-h LC_{50} of 15 631 $\mu g \cdot L^{-1}$ in juvenile crayfish (*Procambarus clarkii*) (Cheah *et al.*, 1980).

Chronic Toxicity and Sublethal Reactions

Hermanutz *et al.* (1973) studied the chronic toxicity and sublethal effects of captan to the fathead minnow (*Pimephales promelas)* in a measured, flow-through system. The fish were exposed to technical-grade captan in 98.01% acetone with 0.05% of the surfactant Triton X-100. Survival of the fish was significantly reduced (100% mortality vs. 4.5% mortality in the controls) after a 315-d exposure to a captan concentration of 63.5 $\mu g \cdot L^{-1}$. After 51 d, all but one of the fish had died at 63.5 $\mu g \cdot L^{-1}$. The fish also experienced significant reductions in growth (55.6 mm mean length vs. 62.5 mm for the control) after 315 d of exposure to 39.5 $\mu g \cdot L^{-1}$ captan, although mean weight did not differ. Mean number of spawnings and mean eggs spawned per female were adversely affected by both 16.8 and 39.5 $\mu g \cdot L^{-1}$, but these effects were not statistically significant. The authors concluded that, based on survival and growth, these data indicated a maximum acceptable toxicant concentration (MATC) of between 16.8 and 39.5 $\mu g \cdot L^{-1}$ for the fathead minnow (based on no significant effect at 16.8

$\mu g \cdot L^{-1}$ and growth reduction at 39.5 $\mu g \cdot L^{-1}$. Thus, the lowest-observed-effect level (LOEL) was 39.5 $\mu g \cdot L^{-1}$. However, no positive controls were studied during these experiments (no acetone or Triton X-100 was added to the control water).

No chronic toxicity data for freshwater invertebrates were found in the available literature.

Information concerning the toxicity of captan to aquatic plants is limited. Exposure to captan concentrations as high as 500 $mg \cdot L^{-1}$ for 30 d caused reductions of only 0%–14% in the growth of the blue-green algae *Nostoc* sp., *Calothrix* sp., *Westiellopsis prolifica*, *Aulosira fertilissima*, and *Tolpothrix tenuis* (Babu and Bhalla, 1979). A captan concentration of 1 $mg \cdot L^{-1}$ reduced photosynthesis in the green alga *Chlorella vulgaris* by 90%, and 10 $mg \cdot L^{-1}$ caused complete inhibition (Malewicz and Borowski, 1979).

Accumulation and Elimination in Aquatic Organisms

The information that exists concerning the bioaccumulation or biomagnification of captan in terrestrial and aquatic ecosystems indicates that this compound will not bioaccumulate to any great extent (U.S. EPA, 1989a). Some information on the bioaccumulative potential of captan comes from the work of Metcalf and Sanborn (1975), who used a terrestrial–aquatic model ecosystem to study the environmental fate of captan. The ecosystem consisted of a 75.7-L aquarium with a sloping sand shelf entering a 7-L pond maintained at 26.5°C with 12 h of light per day. Sorghum (*Sorghum halopense*) was planted along the top of the sand shelf. After plankton, *Daphnia magna*, algae (*Oedogonium cardiacum*), and snails (*Physa* spp.) were added to the water. Between 0.22 and 1.1 $kg \cdot ha^{-1}$ (exact rate not specified) of ^{14}C-labelled captan was applied to the sorghum foliage. Salt-marsh caterpillars (*Estigmene acrea*) were immediately placed on the sorghum plants. At 26 d posttreatment, mosquito (*Culex pipiens*) larvae were added. Mosquitofish (*Gambusia affinis*) were added 30 d after the treatment. Upon termination of the experiment at 33 d, none of the parent captan was detected in any of the organisms.

^{14}C-labelled captan, introduced into a terrestrial microcosm at 1.12 $kg \cdot ha^{-1}$, produced a total residue level of 0.119 $mg \cdot kg^{-1}$ in a vole (*Microtus ochrogaster*) exposed to the contaminated environment for 5 d (Cole and Metcalf, 1980). Only 2% of this residue was the parent captan. Additional residues of the parent compound in terrestrial animals were reported to be 0.181 $mg \cdot kg^{-1}$ for earthworms (*Lumbricus terrestris*) and 0.069 $mg \cdot kg^{-1}$ for slugs (*Limex maximus*). After 20 d, the plants and animals were removed, and the microcosm was flooded with water and maintained as an aquatic microcosm for 7 d with introduced snails (*Physa* spp.) and mosquitofish. After 7 d, the snails contained a residue of 0.825 $mg \cdot kg^{-1}$, of which 0.048 $mg \cdot kg^{-1}$ was the parent captan. Total residues in the fish were 0.378 $mg \cdot kg^{-1}$, of which 0.0212 $mg \cdot kg^{-1}$ was the parent captan. Total ^{14}C residue in the water was 2.94 $\mu g \cdot L^{-1}$ (of which 0.1% was the parent captan), from which a bioaccumulation factor (BCF) of approximately 128 for total ^{14}C-pesticide residues could be calculated.

In one other study, the uptake of captan by golden ide (*Leuciscus idus melanotus*) and green algae (*Chlorella fusca* var. *vacuolata*) from water was followed for 3 and 1 d, respectively. Bioaccumulation factors of 10 and 20 were calculated for the fish and algae (Freitag *et al.*, 1985). Although the ^{14}C-labelled carbon of the captan was found in various components of both systems, the total radioactivity of the samples did not necessarily represent the parent captan, but may have also included labelled metabolic products.

Guideline

In the aquatic toxicity data base for captan, sufficient chronic, nonlethal responses for fish were not found (CCREM, 1987: Appendix IX). Further, no chronic toxicity data were found for freshwater invertebrates. Because of these deficiencies, only an interim freshwater aquatic life water quality guideline can be developed for captan (CCREM, 1987: Appendix IX). (Hermanutz *et al.*, [1973], calculated a LOEL of 39.5 $\mu g \cdot L^{-1}$ for the fathead minnow. During these studies, however, fish used in the control group were not exposed to the same surfactant and solvent used to deliver captan to the treatment groups; in other words, no solvent controls were tested.)

The existing guideline development procedure (CCREM, 1987: Appendix IX) advocates the use of acute toxicity data and application factors when sufficient chronic toxicity data are not available. Because of the deficiencies in the captan aquatic toxicity data base, the lowest acute toxicity value derived from acceptable testing methods, the 96-h LC_{50} for brown trout (26.2 $\mu g \cdot L^{-1}$) (Mayer and Ellersieck, 1986), was selected for the development of an interim freshwater aquatic life guideline. Because captan degrades quite

rapidly to nontoxic metabolites in water (half-life <12 h) (Hermanutz *et al.*, 1973; Frank *et al.*, 1983), an application factor for nonpersistent compounds (0.05) was used. Accordingly, an interim guideline of 1.3 $\mu g \cdot L^{-1}$ is derived for the protection of freshwater aquatic life. Because fish appear to be the aquatic organisms most sensitive to the toxic effects of captan, this guideline concentration should be protective of all freshwater biota (see Figs. 2 and 3).

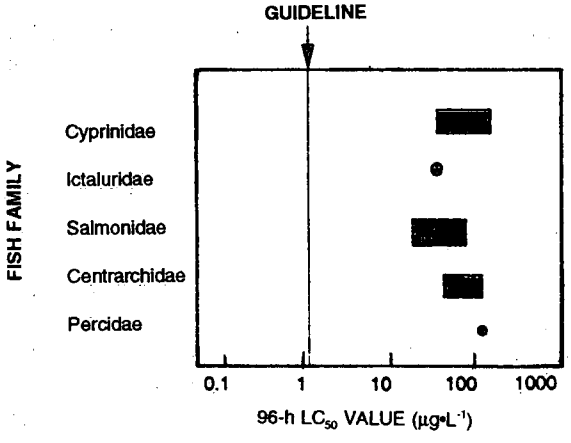
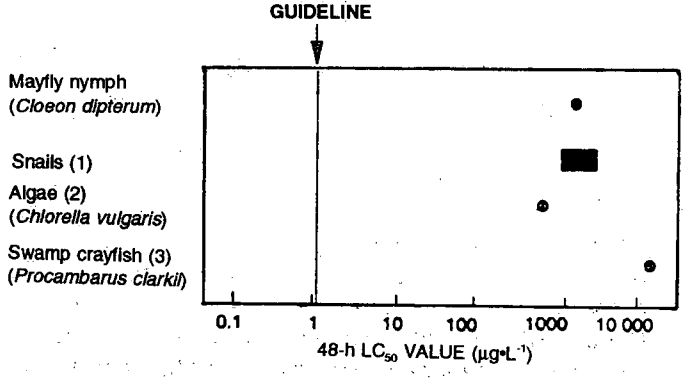


Figure 2. Acute toxicity of captan to freshwater fish



(1) *Physa acuta, Semisulcospira libertina, Indoplanorbis exustus*
(2) 90% reduction in photosynthesis
(3) Immature, 96-h LC_{50}

Figure 3. Acute toxicity of captan to freshwater invertebrates and algae

Agricultural Uses

Livestock Watering

Toxicity to Livestock and Related Biota

Acute Toxicity—Captan typically exhibits low acute oral toxicity to mammals and birds. Acute oral LD_{50} values for rats are reported to be above 9000 $mg \cdot kg^{-1}$ (Boyd and Krijnen, 1968; Metcalf, 1971; Goring, 1972; Agriculture Canada, 1982; NIOSH, 1983; U.S. EPA, 1986). An oral LD_{50} of 7000 $mg \cdot kg^{-1}$ was reported for mice (U.S. EPA, 1984). The ring-necked pheasant (*Phasianus colchicus*), Japanese quail (*Coturnix japonica*), and mallard (*Anas platyrhynchos*) are reported to have LD_{50}s of greater than 5000 $mg \cdot kg^{-1}$ captan in an *ad libitum* diet; there were no overt signs of toxicity at this concentration (Hill *et al.* 1975; Hill and Camardese, 1986). Hudson *et al.* (1984) reported an LD_{50} of greater than 2000 $mg \cdot kg^{-1}$ body weight for male mallards at 3–4 months of age. Two-week-old pheasants, mallards, and Japanese quail ingesting treated feed for 5 d followed by untreated feed for 3 d had LD_{50}s above 5000 $mg \cdot kg^{-1}$ (Pimentel, 1971). Bobwhite quail (*Colinus virginica*) had LD_{50}s of 2000–4000 $mg \cdot kg^{-1}$ in the same experiment. Captan was not acutely toxic to either red-winged blackbirds (*Agelaius phoeniceus*) or European starlings (*Sturnus vulgaris*) at oral doses of 100 $mg \cdot kg^{-1}$ (Schafer, 1972).

Dietary protein (casein) content has been shown to influence the acute oral toxicity of captan (Boyd and Krijnen, 1968; Krijnen and Boyd, 1970). The LD_{50} in male Wistar rats fed diets devoid of casein was 6.15 $mg \cdot kg^{-1}$. The LD_{50} was 12 600 $mg \cdot kg^{-1}$ at 26% casein (the optimal casein concentration) and 5320 $mg \cdot kg^{-1}$ at three times the optimal concentration (78%). The data therefore indicated that either a protein deficiency or excessive protein intake may increase the toxicity of captan.

In one other acute study, liver microsome samples prepared from rats given oral captan doses of 100 and 650 $mg \cdot kg^{-1}$ exhibited decreased aniline hydroxylase activity of 10% and approximately 50%, respectively. The response from the 650 $mg \cdot kg^{-1}$ dose was reported to have taken place within 24 h of treatment (Peeples and Dalvi, 1978).

Subacute and Chronic Toxicity—Many studies have been conducted on the long-term effects of captan ingestion by mammals and birds. A lowest-observed-adverse-effect level (LOAEL) of 100 $mg \cdot kg^{-1} \cdot d^{-1}$ was

determined in rats fed dietary captan concentrations of up to 250 $mg \cdot kg^{-1} \cdot d^{-1}$. End points for toxicity in this study were hepatocellular hypertrophy, increased kidney weight and decreased body weight. The no-observed-effect level (NOEL) using the same criteria for toxicity was 25 $mg \cdot kg^{-1} \cdot d^{-1}$ (U.S. EPA, 1985). Continuous feeding for 28 d of a diet containing 10 or 50 $mg \cdot kg^{-1}$ captan had little or no effect on rat liver enzyme activities (Mikol *et al.*, 1980). A diet containing 1000 $mg \cdot kg^{-1}$ fed over 56 d, however, produced increased microsomal p-nitroanisol-O-demethylase activity and decreased mean liver microsomal protein (Urbanek-Karlowska, 1977).

Gastric intubation delivery of captan doses of 6000–25 000 $mg \cdot kg^{-1}$ body weight, given over a 28-d period, produced a number of toxic effects in rats (Boyd and Krijnen, 1968). Clinical signs of intoxication included irritability, epistaxis, listlessness, hypothermia, anorexia, and changes in the urine. Mortality apparently occurred through cardiac or respiratory failure. Histopathological observations of dead animals showed marked meningocerebral congestion and capillary hemorrhage. Necropsy also revealed a high incidence (13%–87%) of lesions in the gastrointestinal tract, liver, thymus gland, brain, and spleen. A lower incidence (6%) of lesions in the cecum, colon, and lung was also observed (Boyd and Krijnen, 1968). However, according to the National Cancer Institute (NCI, 1977), the importance of Boyd and Krijnen's (1968) observations on gross pathology is equivocal, as these lesions were also frequently observed in untreated rats.

Rats fed captan at levels of 2525 and 6050 $mg \cdot kg^{-1}$ in the diet for 2 years exhibited lower body weights than controls. No relationship was observed between dose and decrease in survival. During the second year of the study, both low and high dose groups exhibited rough hair coats, loss of hair, pale mucous membranes, dermatitis, tachypnea, and hematuria (blood in the urine) (NCI, 1977).

A total oral captan dose of only 78 $mg \cdot kg^{-1}$ body weight over a 2-month period was reported to affect blood hemoglobin concentration, leucocyte and erythrocyte counts, prothrombin levels, serum cholesterol, and sperm motility in treated rats. Intermittent dosing with captan produced a more pronounced toxic response than did continuous dosing (Dekanozishvili, 1975).

Oral doses of captan at 500 $mg \cdot kg^{-1} \cdot d^{-1}$ for 14 d produced dystrophic changes in the kidneys, lungs, spleen, stomach, and intestinal mucosa of rabbits. As well, liver and muscle glycogen storage was inhibited (Szuperski and Grabarska, 1972).

Steers given free access to a feed mixture containing captan at levels ranging from 185.1 to 742.2 $mg \cdot kg^{-1}$ for 140 d did not exhibit any adverse effects. At the higher doses, weight gains were greater than those recorded for control steers (Dowe *et al.*, 1957). Ill effects were not reported for cattle and pigs fed rations containing captan at 500–4000 $mg \cdot kg^{-1}$ for approximately 6 months. It was suggested that captan is probably nontoxic in the amounts likely to be consumed in feed corn. Pigs fed a mixed ration containing 480 $mg \cdot kg^{-1}$ captan for 96 d were reported to be free of detectable pathological tissue changes. Similarly, average weight changes in two trials indicated that both weight losses and weight gains occur in pigs exposed to captan (Link *et al.*, 1956). Pigs refused to eat feed containing 8000 $mg \cdot kg^{-1}$ captan (Johnson, 1954).

Martin and Lewis (1979) injected chicken embryos at day 4 of incubation with captan at 12 $mg \cdot kg^{-1}$ egg weight. The biosynthesis of DNA, RNA, and various proteins in the developing limbs was monitored on days 8–14 of incubation. Peak ^{3}H-thymidine incorporation into DNA was inhibited by 33%. There was also lower incorporation of ^{3}H-uridine into RNA throughout the incubation period. Further, RNA synthesis was reduced by 32%, incorporation of ^{3}H-valine into protein was delayed, and there was lower total protein concentration in the developing embryos.

Chicks fed captan-treated seed corn in a mixed ration (producing a final level of 320 $mg \cdot kg^{-1}$ in the feed) for 74 d suffered no harmful effects. These conclusions were based on "favourable" weight gains and a lack of pathological changes in tissues (Link *et al.*, 1956). In a similar study, chicks fed the captan formulation Orthocide (430 $mg \cdot kg^{-1}$ of a 50% wettable powder) in a mixed ration for 28 d showed slower early growth than controls, but both were equal in weight by the end of the study. Evidence of external abnormalities was not reported (Ackerson and Mussehl, 1955).

Uptake, Metabolism, and Elimination—It is not clear whether captan is readily absorbed from the gastrointestinal tract. Studies conducted usually involve radiolabelled captan administered and followed in the organism. Therefore, the results do not necessarily reflect the pharmacokinetics of the unmetabolized captan, but may also represent those of the metabolic

products. Large oral doses of captan may result in nonabsorbed, unmetabolized captan being excreted in the feces. After an oral dose (650 mg·kg^{-1}) of trichloromethyl-^{14}C-labelled captan, 28.7% of the administered dose was excreted in the feces, and unaltered captan was also detected in the feces. With a smaller dose (12–134 mg·kg^{-1}), only 7.2%–11.3% of the administered radioactivity was detected in the feces, and unaltered captan was not detected (U.S. EPA, 1985).

Orally administered captan is rapidly metabolized in the gut of the rat. Initially, hydrolysis of the nitrogen–sulphur bond results in tetrahydrophthalimide and a derivative of the trichloromethylthio side chain. Both metabolites are further broken down into secondary metabolites. The metabolic reactions are facilitated by the presence of sulphite or thiosulphite radicals, sodium sulphite, cysteine, and glutathione. The hydrolysis of captan is pH-dependent and accelerates as the pH increases from the stomach to the small intestine (DeBaun *et al.*, 1974; U.S. EPA, 1985). Hydrolysis of captan is also reported to occur in the blood (U.S. EPA, 1985).

As part of the special review of captan, the U.S. EPA (1989a) discussed toxicological concerns with respect to captan metabolites. The agency suggested that certain lesions seen in long-term rat studies (e.g., renal cortical tubular adenomas and carcinomas) may be the result of the metabolism of captan to the tetrahydrophthalimide moiety. The EPA concluded that the evidence is sufficient to ascribe carcinogenic potential to tetrahydrophthalimide. Thiophosgene may be of less concern because the compound is so rapidly metabolized.

Captan with a ^{14}C-labelled trichloromethylthio group was administered by gavage to rats as a single dose of 100 mg·kg^{-1} (DeBaun *et al.*, 1974). Nine hours after treatment, 50% of the initial radiolabelled carbon had been excreted in the urine, feces, and expired air. At 1 d posttreatment, most of the assimilated radioactivity was found concentrated in the gastrointestinal tract (65.1%), the kidneys (11.6%), and the bladder (6.1%). By day 4, a total of only 0.6% of the original dose remained in all the tissues. Of this small amount, the highest proportion remained in the kidneys (21.3%); the bladder and lungs contained 19.9% and 10.2%, respectively. All of these organs are involved in the excretion of captan metabolites. Other tissues did not exhibit unusual localizations of radioactivity (DeBaun *et al.*, 1974).

By using ^{35}S-labelled captan in a single oral dose of either 143 or 390 mg·kg^{-1} administered to rats, Seidler *et al.* (1971) found 93% of the radioactivity excreted in the first 24 h (38% in the feces and 55% in the urine). An additional 5% was excreted during the second 24 h of the study. In another study with rats, the fate of captan ^{14}C-labelled on the carbonyl group was followed after single oral doses of 77.4–91.9 mg·kg^{-1}. Within 48 h of dosing, 92% of the radioactivity had been excreted, and an additional 4.8% was excreted within 96 h. The urine accounted for almost all of the excreted radioactivity (85.5%), none of which was unaltered captan. Only 12.3% of the radioactivity was excreted in the feces. At 96 h posttreatment, tissue residues were less than 0.1% of the total administered ^{14}C (Hoffman *et al.*, 1973).

Carcinogenicity, Mutagenicity, and Teratogenicity—The evidence concerning the carcinogenicity of captan has been controversial (CCIBP, 1982; U.S. EPA, 1989a). An early assay with mice, for instance, did not show evidence of carcinogenicity (Innes *et al.*, 1969). The NCI (1977) reported a weak carcinogenic response in mice and no response in rats. The results of low dose (Bio/dynamics Laboratories, 1983) and high dose (Chevron Chemical Co., 1981) chronic feeding studies in mice and a chronic feeding study in rats (Stauffer/Chevron, 1982) indicated a relationship between captan dose and tumour incidence. After a detailed review of the data and the responses of the companies with registered products, the U.S. EPA (1985) suggested that the data provide a "weight of evidence" for the classification of captan as a probable human carcinogen. In 1989, the U.S. EPA classified captan as a Group B_2 (probable human) carcinogen (U.S. EPA, 1989a).

The mutagenicity of captan appears to depend largely on the test procedures used. Several investigators have reported positive mutagenic responses in a variety of *in vitro* test systems. Examples include eight strains of the *Salmonella typhimurium* assay, five strains of the *Escherichia coli* assay, the *Aspergillus nidulans* assay, the Chinese hamster ovary V-79 cell assay, and the lung fibroblast cell assay (Legator *et al.*, 1969; Seiler, 1973; Kada *et al.*, 1974; Shirasu *et al.*, 1976; Moriya *et al.*, 1978, 1983; U.S. EPA, 1985).

Mutagenesis assays have indicated that captan induces DNA repair mechanisms, produces chromosome aberrations in mammalian cell cultures (U.S. EPA, 1985), and increases the number of X-chromo-

some breaks in human embryo cell cultures (Legator *et al.*, 1969). Xu and Schurr (1990) labelled captan a "strongly positive" genotoxic compound.

One explanation for the equivocal data concerning captan carcinogenicity comes from several studies that have shown a reduced or inactivated mutagenic response in bacterial strains or mammalian cell cultures that received captan pretreated with blood, sulfhydryl compounds, or drug-metabolizing enzyme systems (Marshall *et al.*, 1976; Swenberg *et al.*, 1976; Ficsor *et al.*, 1977; Moriya *et al.*, 1978; De Flora *et al.*, 1984; Xu and Schurr, 1990). Metabolic deactivation of the compound is assumed, which may explain the reported lack of carcinogenicity in mice whole-animal bioassays (Xu and Schurr, 1990). As an example, the positive mutagenic responses to captan observed in *S. typhimurium* TA1535 and *E. coli* could be largely inactivated if the medium contained blood, cysteine, or rat liver homogenate. Further, chromosomal aberrations in rat bone marrow cells were not induced with single oral doses of 500–2000 $mg \cdot kg^{-1}$ or five consecutive doses of 200–800 $mg \cdot kg^{-1}$ (Tezuka *et al.*, 1978). Finally, investigations of the mutagenic potential of captan *in vivo* typically produce negative results (Ficsor *et al.*, 1977; Simmon *et al.*, 1977; Tezuka *et al.*, 1978).

Captan may produce mutagenic toxicity. A significant increase in fetal mortality was caused by five daily oral doses of 100 $mg \cdot kg^{-1}$ administered to males 2 and 4 weeks prior to mating in mice and rats, respectively (Collins, 1972a); this was evidence of mutagenic potential by induction of major chromosomal aberrations. The dominant lethal and polygenic mutagenicity was also demonstrated in a two-generation reproduction study conducted by the same investigator (Collins, 1972b). A number of toxic effects were observed in F_1 and F_2 animals as the result of daily doses of 50 or 100 $mg \cdot kg^{-1}$ for 5 d given to the original parents. These effects included decreases in the percentage of liveborn, weaning weight, and survival to day 4. The results of this study were criticized as being unreliable, however, because of difficulties with reproducibility of the assay and the method of captan administration (gavage) (U.S. EPA, 1985). Further, a study by Tezuka *et al.* (1978), designed to verify the previous dominant lethal study using a similar protocol, found no dominant lethal effects in male and female mice receiving oral gavage doses of 200 or 600 $mg \cdot kg^{-1} \cdot d^{-1}$ for 5 d.

In other studies, captan has shown the potential to produce embryological and maternal toxic effects. Weight losses were observed in pregnant mice receiving oral doses of captan at 100 $mg \cdot kg^{-1} \cdot d^{-1}$ on gestation days 6–15, but prominent signs of fetal toxicity or abnormalities were not observed (Bionetics Research Laboratories, 1968). The same dose administered to another strain of mice on gestation days 6–15 also failed to produce teratogenic effects. Captan, however, did decrease maternal body weights and slightly increased fetal mortality rates (Courtney *et al.*, 1978). Insufficient data existed in this report for a proper evaluation by the U.S. EPA, (1985).

Oral administration of captan to Golden Syrian hamsters on gestation days 6–10 (cumulative doses to 1500 $mg \cdot kg^{-1} \cdot d^{-1}$) or as a single dose of 300 $mg \cdot kg^{-1} \cdot d^{-1}$ on gestation day 7 or 8 resulted in reduced fetal weight, increased maternal mortality, and teratogenic effects (i.e., fused ribs and exencephally) (Robens, 1970). Oral captan doses of 200 $mg \cdot kg^{-1} \cdot d^{-1}$ on gestation days 5–10 have also been reported to cause maternal weight loss and death, fetal weight loss and death, increased early and late resorptions, and post-implantation losses (Goldenthal, 1978). Fetal rib abnormalities observed at 400 $mg \cdot kg^{-1} \cdot d^{-1}$ were attributed to maternal stress.

Reproductive effects data for female rabbits are quite variable. Captan at 80 $mg \cdot kg^{-1} \cdot d^{-1}$, administered by intubation on days 7–12 of gestation, failed to produce maternal toxicity, fetotoxicity, or teratogenicity (Fabro *et al.*, 1966). On the other hand, doses of 12 $mg \cdot kg^{-1} \cdot d^{-1}$ reduced maternal, litter, and fetal weights (Chevron Chemical Co., 1981). Fetal teratogenicity was observed in rabbits dosed with captan in gelatin capsules on gestation days 6–16. At 75 $mg \cdot kg^{-1} \cdot d^{-1}$, 9 out of 75 implantations experienced some deformity, including deformed limbs, cleft lips, and fused upper lips. One fetus out of 49 experienced acephally at a dose of 37.5 $mg \cdot kg^{-1} \cdot d^{-1}$ (McLaughlin *et al.*, 1969) (control data were not provided).

Two reproduction studies with rats were submitted to the U.S. EPA (1985). During a three-generation study, rats were fed dietary captan levels of 25, 100, 250, and 500 $mg \cdot kg^{-1} \cdot d^{-1}$. Treatment-related effects included reduced weight gain of the parents at the three highest doses, reduced pup litter weights at all dosage levels, and reduced food consumption at most treatment levels. During a one-generation rat study, captan in the diet at 6, 12.5, and 25 $mg \cdot kg^{-1} \cdot d^{-1}$ caused no treatment-related effects. The U.S. EPA (1985) combined these two studies to satisfy their reproduction testing requirements and concluded that the NOAEL for toxic effects was 12.5 $mg \cdot kg^{-1} \cdot d^{-1}$.

Daily doses of 30 $mg \cdot kg^{-1}$ administered in gelatin capsules to beagle dogs throughout gestation increased the percentage of stillborn pups and produced a low incidence of terata (Earl *et al.,* 1973). These teratogenic effects were not dose-dependent and did not show any consistent pattern.

Several studies that failed to demonstrate terata when captan was administered to pregnant hamsters, beagle dogs, rhesus monkeys, and stump-tailed macaques (Kennedy *et al.,* 1968, 1975; Vondruska *et al.,* 1971) were declared invalid as a result of a 1979 Canadian/U.S. audit of the testing laboratory (U.S. EPA, 1985).

Guideline

In the absence of adequate information concerning the toxicity to livestock of compounds consumed in their drinking water, the Canadian drinking water quality guideline is usually used as a surrogate interim guideline for livestock watering (CCREM, 1987). However, a Canadian drinking water quality guideline for captan has not been developed (Health and Welfare Canada, 1989a). Thus, the NOAEL of 12.5 $mg \cdot kg^{-1} \cdot d^{-1}$ for reproductive effects in rats (U.S. EPA, 1985) was used as the basis for guideline development. This level is much lower than levels that have been shown to produce no adverse effects in livestock (e.g., Link *et al.*, 1856; Dowe *et al.*, 1957). For a conservative estimate, an uncertainty factor of 0.001 is applied to the NOAEL for the rat to produce an estimated NOAEL of 0.0125 $mg \cdot kg^{-1} \cdot d^{-1}$ for livestock. This safety factor was chosen, following the U.S. EPA (1987) procedure for developing a draft drinking water quality guideline for captan, to provide protection when extrapolating from a rodent species to another mammal. Using the weight of a lactating dairy cow (820 kg) and daily water consumption of 160 L (CCREM, 1987), the estimated NOAEL results in an allowable daily consumption of 64.1 $\mu g \cdot L^{-1}$. This value is multiplied by 0.2, because 20% of the total daily dose is assumed to result from consuming contaminated drinking water. Thus, a value of 13 $\mu g \cdot L^{-1}$ is suggested as an interim guideline for livestock watering supplies.

Irrigation

Toxicity to Nontarget Plant Species

Most captan plant toxicity data deal with its toxic effects to fungi (see Appendix B). There is also information on a large number of microorganisms, in most cases plant pathogens. As captan is a broad-spectrum fungicide (Lukens, 1969; Hassall, 1982), it is also likely to have adverse effects on nontarget microorganisms.

Captan is not reported to show evidence of phytotoxicity toward vascular plants (Agriculture Canada, 1982), and it can be used on these plants with a high degree of safety (Metcalf, 1971). It also does not appear to influence nitrogen fixation and nodulation (Schnelle and Hensley, 1990). Where phytotoxic effects due to captan have been observed, the hydrochloric acid or hydrogen ions formed during captan breakdown are believed to be at least partly responsible for the toxicity (Daines *et al.*, 1957; Miller, 1957). Thus, cells of vascular plants may be adversely affected if exposed to captan (Sisler and Cox, 1960). Captan has inhibitory effects on various steps of the citric acid cycle at doses as low as 100 $mg \cdot L^{-1}$ (Appendix B). At 1–5 $mg \cdot L^{-1}$, captan applied to the roots of bean and tomato plants in liquid culture is toxic to both plants (Silber, 1957; Lukens and Sisler, 1958). Under field conditions, however, captan levels would likely need to be much higher to produce similar responses.

Different varieties of apples and pears are reported to be injured by captan treatments, but doses were not provided (Thomson, 1979). The utilization of ^{14}C-labelled sugars in captan-treated corn and pea root tips was also inhibited by captan treatment (Dugger *et al.*, 1958). The phytotoxicity of captan is reported to increase with decreasing light intensity and increasing concentration, diluent, wetting agent, and temperature (Daines *et al.,* 1957).

Fungitoxicity of captan can occur at concentrations as low as 0.01 $mg \cdot L^{-1}$. Many important sulfhydryl-containing enzyme systems are inhibited, which could result in decreased cellular energy processes (Byrde *et al.,* 1956; Hochstein and Cox, 1956; Silber, 1957; Dugger *et al.,* 1959; Owens and Novotny, 1959; Owens and Blaak, 1960b; Montie and Sisler, 1962; Goring, 1972), incorporation of inorganic phosphate into organic molecules (Byrde *et al.* 1956; Owens and Novotny, 1959), or the metabolism of amino acids (Byrde *et al.*, 1956; Owens and Novotny, 1959). The incorporation of ^{14}C-labelled formate into RNA pyrimidines and purines was also inhibited by captan concentrations as low as 0.4 $mg \cdot L^{-1}$ in the fungus *Candida albicans* (Gale *et al.,* 1971).

The reported toxic effects of captan on fungi include reductions in spore germination (McCallan *et al.,* 1954; Owens and Novotny, 1959; Lukens *et al.*,

1965), growth (Lukens and Sisler, 1958; Rich, 1959; Montie and Sisler, 1962; Richmond and Somers, 1963; Ruch and Bland, 1979), and oxygen uptake (McCallan *et al.*, 1954; Owens and Novotny, 1959).

The effects of captan on soil microflora were observed to vary with dose between 400 and 600 $mg \cdot kg^{-1}$. Populations of soil algae, actinomycetes, and fungi were reported to be reduced, whereas bacterial numbers remained constant at these concentrations (Domsch 1959). At 1000 $mg \cdot kg^{-1}$, captan inhibits nitrifying bacteria and slightly affects ammonifying bacteria (Lukens, 1969).

Guideline

Reports of studies that have used captan-contaminated water for crop irrigation were not found. There was, in fact, very little information available on the effects of captan on vascular plants after application to foliage or soil. In addition, specific information regarding captan toxicity to nontarget terrestrial and aquatic vascular plants could not be found in the literature. Further, there were no data to suggest that captan residues in irrigation water that result from registered uses of the fungicide are harmful to crop plants. Therefore, no guideline is recommended at this time.

Recreational Water Quality and Aesthetics

Organoleptic Effects

Information on the concentrations of captan in water that might impart a taste or odour to the water was not found. The low volatility and rapid hydrolysis of the compound would appear to prevent captan from accumulating to levels that might cause these effects. As well, published information related to the production of a taste or odour in fish flesh exposed to dissolved captan was not found.

Guideline

At present, there is no information available to evaluate whether this water use would be adversely affected by captan residues when the fungicide is used according to label instructions. In addition, water containing captan residues at concentrations affecting recreational water uses would presumably already be severely impaired for other water uses (e.g., water for the protection of aquatic life). Thus, a water quality guideline has not been determined for recreation and aesthetics.

Industrial Water Supplies

Guideline

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

DATA GAPS

With respect to the aquatic toxicity of captan, more information is needed on the chronic toxicity of the compound to both freshwater invertebrates and vertebrates. No studies are currently available for inclusion in the data base; no freshwater invertebrate chronic toxicity studies were found, and the single fish study found did not include positive controls to investigate the possible effects of the acetone solvent. The influence of acetone, often used as a solvent for captan dilution, on the possible inhibition of the aquatic toxicity of captan needs further investigation. The toxicity of captan to aquatic vascular plants should also be addressed.

Some fate data are missing for captan, particularly data on the anaerobic metabolism of the compound. In addition, the persistence of the compound in groundwater has not been reported.

Although captan is probably not toxic to livestock species in the amounts that might be consumed in feed, no studies of livestock consuming captan-contaminated drinking water were found in the available literature. There is also no information on the residues of captan in meat, milk, and eggs after animals had consumed the compound.

Finally, no specific data were found on the toxic effects of captan on crop plants when the crops are irrigated with captan-contaminated irrigation water. In the absence of this information, an irrigation guideline for captan must rely on data collected during laboratory phytotoxicity studies.

SUMMARY

After an evaluation of the published information on the fungicide captan, Canadian water quality guidelines were derived (Table 5). The background informa-

Table 5. Recommended Water Quality Guidelines for Captan

Uses	Recommended guidelines
Raw water for drinking water supply	No recommended guideline
Freshwater aquatic life	1.3 $\mu g \cdot L^{-1}$ (interim)
Agricultural uses	
Livestock watering	13 $\mu g \cdot L^{-1}$ (interim)
Irrigation	No recommended guideline
Recreational water quality and aesthetics	No recommended guideline
Industrial water supplies	No recommended guideline

tion on captan in terms of uses and production, occurrence in the aquatic environment, and persistence and degradation was reviewed. The rationale employed for the development of the recommended guidelines was summarized.

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Appendix A

Acute Toxicity of Captan to Aquatic Organisms

Table A-1. Acute Toxicity of Captan to Aquatic Organisms

Species	Test conditions*	Temperature (°C)	pH	Water hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% ai)	LC_{50} ($\mu g \cdot L^{-1}$) 24 h	48 h	96 h	Reference
VERTEBRATES									
Lepomis macrochirus (bluegill)	F,M	24.9	NR	NR	Stock acetone solution			72 (47–111)[2]	Hermanutz *et al.*, 1973
Lepomis macrochirus (bluegill)	S,M	17.0	7.1	44	Technical (90)	145 (122–172)		141 (119–167)	Mayer and Ellersieck, 1986[3]
Salvelinus fontinalis (brook trout)	F,M	11.8	NR	NR	Stock acetone solution			34 (22–52)	Hermanutz *et al.*, 1973
Salmo trutta (brown trout)	S,M	12.0	7.5	44	Technical (90)	81 (69.8–94.0)		80 (63.8–100)	Mayer and Ellersieck, 1986
Salmo trutta (brown trout)	F,M	12.0	7.5	314	Technical (90)	26.2 (21.8–31.3)		26.2 (21.8–31.3)	Mayer and Ellersieck, 1986
Ictalurus punctatus (channel catfish)	S,M	20.0	7.4	44	Technical (90)	79.8 (72.6–87.8)		77.5 (70.5–85.2)	Mayer and Ellersieck, 1986
Oncorhynchus tshawytscha (chinook salmon) (fingerling)	S,M	12.0	7.5	44	Technical (90)	139 (115–168)		120 (103–140)	Mayer and Ellersieck, 1986
Oncoryhnchus tshawytscha (chinook salmon)	S,NR	12	7.2–7.5	40–50	Technical (90–100)			56.5 (52.3–61.0)	Johnson and Finley, 1980
Oncoryhnchus kisutch (coho salmon)	S,M	12.0	7.5	44	Technical (90)	137 (117–160)		137 (117–160)	Mayer and Ellersieck, 1986
Oncorhynchus kisutch (coho salmon)	F,M	12.0	7.5	314	Technical (90)	75.0 (64.9–85.5)		56.6 (52.3–61.0)	Mayer and Ellersieck, 1986

NR = not reported
S = static
F = flow-through
M = measured
U = unmeasured

[1]Stock acetone solution of technical-grade captan (1.9% by weight, 8.4% purity).

[2]Confidence interval in parentheses.

[3]All or part of the toxicity data cited by Mayer and Ellersieck (1986) have been previously cited by Johnson and Finley (1980) and Mauck and Oleon (1972); Mauck and Oleon (1972) has also been referenced as Mauck (1972) in U.S. Department of Agriculture (1986).

[4]Text of paper in Japanese; translation of test parameters unavailable.

Table A-1. Continued

Species	Test conditions*	Temperature (°C)	pH	Water hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% ai)	LC_{50} ($\mu g \cdot L^{-1}$) 24 h	48 h	96 h	Reference
Oncorhynchus kisutch (coho salmon)	S,NR	12	7.2–7.5	40–50	Technical (90–100)			138 (118–161)	Johnson and Finley, 1980
Salmo clarkii (cutthroat trout)	S,M	12.0	7.4	44	Technical (90)	74.1 (55.4–99.1)		56.4 (42.2–75.4)	Mayer and Ellersieck, 1986
Pimephales promelas (fathead minnow)	F,M	25.2	NR	NR	Stock acetone solution			65 (59–72	Hermanutz *et al.*, 1973
Pimephales promelas (fathead minnow)	F,M	12.0	7.5	314	Technical (90)	152 (123–186)		134 (100–178)	Mayer and Ellersieck, 1986
Pimephales promelas (fathead minnow)	S,M	12.0	7.5	44	Technical (90)	290 (211–398)		200 (168–238)	Mayer and Ellersieck, 1986
Salvelinus namaycush (lake trout)	S,M	12.0	7.5	44	Technical (90)	53 (44.5–63.1)		49 (40.1–59.9)	Mayer and Ellersieck, 1986
Salvelinus namaycush (lake trout)	S,M	12.0	7.4	162	Technical (90)	63.2 (49.6–80.5)		63.2 (49.6–80.5)	Mayer and Ellersieck, 1986
Salvelinus namaycush (lake trout)	F,M	12.0	7.5	314	Technical (90)	75.0 (51.7–109)		51.0 (39.2–66.2)	Mayer and Ellersieck, 1986
Salmo gairdneri (rainbow trout)	S,M	12.0	7.4	44	Technical (90)	76.4 (69.5–83.0)		73.2 (66.6–80.4)	Mayer and Ellersieck, 1986
Perca flavescens (yellow perch)	F,M	17.0	7.5	314	Technical (90)	>154		120 (97.3–147)	Mayer and Ellersieck, 1986
Cyprinus carpio (carp)	NR	NR	NR	NR	Technical		250		Hashimoto and Nishiuchi, 1981[49]
Misgurnus anguillicaudatus (pond loach)	NR	NR	NR	NR	Technical		340		Hashimoto and Nishiuchi, 1981
Oryzias latipes (medaka)	NR	NR	NR	NR	Technical		1000		Hashimoto and Nishiuchi, 1981
Goldfish (species not named)	NR	NR	NR	NR	Technical		37		Hashimoto and Nishiuchi, 1981

Table A-1. Continued

Species	Test conditions*	Temperature (°C)	pH	Water hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% ai)	LC_{50} ($\mu g \cdot L^{-1}$) 24 h	48 h	96 h	Reference
Rasbora heteromorpha (harlequin fish)	F,NR	NR	NR	NR	89%	460	330	300	Tooby *et al.*, 1975
Bufo bufo japonicus (tadpole)	NR	NR	NR	NR	Technical		3000		Hashimoto and Nashiuchi, 1981
INVERTEBRATES									
Procambarus clarkii (red swamp crayfish) (immature)	S,U	20±3	NR	NR	Captan 80 WP (80%)			15 631 (10 390–21 100)	Cheah *et al.*, 1980
Cloen dipterum (mayfly nymph)	NR	NR	NR	NR	Technical		1500		Hashimoto and Nishiuchi, 1981
Indoplanorbis exustus (snail)	NR	NR	NR	NR	Technical		1400		Hashimoto and Nishiuchi, 1981
Semisulcospira libertina (snail)	NR	NR	NR	NR	Technical		1200		Hashimoto and Nishiuchi, 1981
Physa acuta (snail)	NR	NR	NR	NR	Technical		1000		Hashimoto and Nishiuchi, 1981

Appendix B

Summary of Captan Phytotoxicity Data

Table B-1. Summary of Captan Phytotoxicity Data

Species	Dosage	Response	Conditions	Reference
PLANTS				
Pea (*Pisum sativum*) (excised root tips)	1000 $mg \cdot L^{-1}$	69% increase in pyruvate concentration and 62% decrease in acetaldehyde concentration after 24 h	Laboratory, no soil	Dugger *et al.*, 1959
Pea (*Pisum sativum*) (cotyledons)	0–1000 $mg \cdot L^{-1}$	No effect on hexokinase activity	Laboratory, no soil	Dugger *et al.*, 1959
Wheat germ	10–1000 mg·L-1	No effect on hexokinase activity	Laboratory, no soil	Dugger *et al.*, 1959
Lupine (*Lupinus alba*) (seedling mitochondria)	100 $mg \cdot L^{-1}$	91% decrease in pyruvate oxidation	Laboratory, no soil	Dugger *et al.*, 1959
Lupine (*Lupine alba*) (seedling mitochondria)	100 $mg \cdot L^{-1}$	70% decrease in alpha-ketoglutarate oxidation	Laboratory, no soil	Dugger *et al.*, 1959
FUNGI				
Fusareum roseum (conidia)	30.1 $mg \cdot L^{-1}$	7-fold increase in pyruvate concentration under aerobic conditions and 1.5-fold increase under anaerobic conditions	Laboratory, no soil	Hochstein and Cox, 1956
Saccharomyces cerevisiae	15 $mg \cdot L^{-1}$	28% inhibition of cocarboxylase activity	Laboratory, no soil	Hochstein and Cox, 1956
Saccharomyces cerevisiae	30.1 $mg \cdot L^{-1}$	38% inhibition of cocarboxylase activity	Laboratory, no soil	Hochstein and Cox, 1956
Saccharomyces cerevisiae	150.3 $mg \cdot L^{-1}$	49% inhibition of cocarboxylase activity	Laboratory, no soil	Hochstein and Cox, 1956
Saccharomyces pastorianus	0.01 $mg \cdot L^{-1}$	No effect on growth after 48 h	Laboratory, no soil, pH 4.5	Lukens and Sisler, 1958
Saccharomyces pastorianus	0.1 $mg \cdot L^{-1}$	47.6% reduction in growth after 48 h	Laboratory, no soil, pH 4.5	Lukens and Sisler, 1958

Table B-1. Continued

Species	Dosage	Response	Conditions	Reference
Saccharomyces pastorianus	1.0 $mg \cdot L^{-1}$	99.8% reduction in growth after 48 h	Laboratory, no soil, pH 4.5	Lukens and Sisler, 1958
Saccharomyces pastorianus	0.01 $mg \cdot L^{-1}$	8% reduction in growth after 48 h	Laboratory, no soil, pH 6.0	Lukens and Sisler, 1958
Saccharomyces pastorianus	0.1 $mg \cdot L^{-1}$	4% reduction in growth after 48 h	Laboratory, no soil, pH 6.0	Lukens and Sisler, 1958
Saccharomyces pastorianus	1.0 $mg \cdot L^{-1}$	99.8% reduction in growth after 48 h	Laboratory, no soil, pH 6.0	Lukens and Sisler, 1958
Saccharomyces cerevisiae	0.01 $mg \cdot L^{-1}$	20.3% reduction in growth after 48h	Laboratory, no soil, pH 7.5	Lukens and Sisler, 1958
Saccharomyces cerevisiae	0.1 $mg \cdot L^{-1}$	28.8% reduction in growth after 48 h	Laboratory, no soil, pH 7.5	Lukens and Sisler, 1958
Saccharomyces cerevisiae	1.0 $mg \cdot L^{-1}$	35.6% reduction in growth after 48 h	Laboratory, no soil, pH 7.5	Lukens and Sisler, 1958
Stemphylium sarcinaeforme (spores)	0.6 $\mu g \cdot cm^{-2}$	ED_{50} for inhibition of spore germination	Not reported	Lukens and Horsfall, 1967
Stemphylium sarcinaeforme	0.62 $\mu g \cdot cm^{-2}$	ED_{50} for inhibition of spore germination	Laboratory, no soil	Lukens *et al.*, 1965
Monilinia fructicola	<0.1 $\mu g \cdot cm^{-2}$	ED_{50} for inhibition of spore germination	Laboratory, no soil	Lukens *et al.*, 1965
Saccharomyces pastorianus	0.06 $mg \cdot L^{-1}$	ED_{50} for inhibition of spore germination	Laboratory, no soil	Lukens and Horsfall, 1967
Lagenidium callinectes (marine fungus) (zoospores)	2.3 mg $ai \cdot L^{-1}$	LC_{100} for failure of zoospores to encyst or germinate after 30 min. of exposure	Laboratory, no soil	Ruch and Bland, 1979
Alternaria tenuis	1.17 mg $ai \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Ruch and Bland, 1979
Neurospora crassa	0.33–0.42 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Ruch and Bland, 1979

Table B-1. Continued

Species	Dosage	Response	Conditions	Reference
Alternaria tenuis	2.4 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Richmond and Somers, 1963
Aspergillus niger	0.48 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Richmond and Somers, 1963
Botrytis allii	0.99 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Richmond and Somers, 1963
Botrytis fabae	0.69 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Richmond and Somers, 1963
Neurospora crassa	0.36 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Richmond and Somers, 1963
Penicillium expansum	0.15 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Richmond and Somers, 1963
Penicillium italicum	0.06 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Richmond and Somers, 1963
Rhizopuys nigricans	3.6 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratory, no soil	Richmond and Somers, 1963
Ventura inaequalis	0.27 $mg \cdot L^{-1}$	ED_{50} for fungistatic effect	Laboratroy, no soil	Richmond and Somers, 1963
Monilinia fructicola	9.02 $mg \cdot L^{-1}$	100% inhibition of growth	Laboratory, no soil	Rich, 1959
Candida albicans	0.4 $mg \cdot L^{-1}$	27% decrease in RNA guanine synthesis; 36% decrease in RNA adenine synthesis	Laboratory, no soil	Gale *et al.*, 1971
Candida albicans	1.2 $mg \cdot L^{-1}$	34% decrease in RNA guanine synthesis; 42% decrease in RNA adenine synthesis	Laboratory, no soil	Gale *et al.*, 1971
Candida albicans	2 $mg \cdot L^{-1}$	54%-84% decrease in RNA guanine synthesis; 72%–94% decrease in RNA adenine synthesis; 69%–78% decrease in RNA pyrimidine synthesis	Laboratory, no soil	Gale *et al.*, 1971
Candida albicans	4.0 $mg \cdot L^{-1}$	84% decrease in RNA guanine synthesis; 94% decrease in RNA adenine synthesis; 97%–99% decrease in RNA pyrimidine synthesis	Laboratory, no soil	Gale *et al.*, 1971
Candida albicans	6.0 $mg \cdot L^{-1}$	88% decrease in RNA guanine synthesis; 95% decrease in RNA adenine synthesis; 98%–99% decrease in RNA pyrimidine synthesis	Laboratory, no soil	Gale *et al.*, 1971

Table B-1. Continued

Species	Dosage	Response	Conditions	Reference
Saccharomyces pastorianus	12.6 $mg \cdot L^{-1}$	39% decrease in uptake of ^{14}C glucose; 96% decrease in incorporation of ^{14}C glucose	Laboratory, no soil	Montie and Sisler, 1962
Saccharomyces pastorianus	1.0 $mg \cdot L^{-1}$	26% decrease in growth after 1 h	Laboratory, liquid culture	Montie and Sisler, 1962
Saccharomyces pastorianus	3.16 $mg \cdot L^{-1}$	63% decrease in growth after 1 h; 18% decrease in growth afte 5 h	Laboratory, liquid culture	Montie and Sisler, 1962
Saccharomyces pastorianus	10.0 $mg \cdot L^{-1}$	75% decrease in growth after 1 h; 79% decrease in growth after 5 h	Laboratory, liquid culture	Montie and Sisler, 1962
Saccharomyces pastorianus	6.31 $mg \cdot L^{-1}$	44% inhibition of colony development 36–48 h after exposure	Laboratory, plated cells	Montie and Sisler, 1962
Saccharomyces pastorianus	10.0 $mg \cdot L^{-1}$	86% inhibition of colony development 36–48 h after exposure	Laboratory, plated cells	Montie and Sisler, 1962
Saccharomyces pastorianus	7.94 $mg \cdot L^{-1}$	67% inhibition of fermentation; 64% inhibition of aerobic respiration	Laboratory, plated cells	Montie and Sisler, 1962
Saccharomyces pastorianus	10.0 $mg \cdot L^{-1}$	86% inhibition of fermentation; 75% inhibition of aerobic respiration	Laboratory, plated cells	Montie and Sisler, 1962
Saccharomyces pastorianus	12.6 $mg \cdot L^{-1}$	96% inhibition of fermentation; 83% inhibition of aerobic respiration	Laboratory, plated cells	Montie and Sisler, 1962
Yeast hexokinase	5 $mg \cdot L^{-1}$	11% decrease in hexokinase activity	Laboratory, plated cells	Dugger *et al.*, 1959
Yeast hexokinase	100 $mg \cdot L^{-1}$	42% decrease in hexokinase activity	Laboratory, plated cells	Dugger *et al.*, 1959
Yeast hexokinase	1000 $mg \cdot L^{-1}$	53% decrease in hexokinase activity	Laboratory, plated cells	Dugger *et al.*, 1959
Monilinia fructicola (spores)	1.8 $mg \cdot g^{-1}$	ED_{50} for reduction in O_2 uptake by fresh spores	Laboratory, no soil	McCallan *et al.*, 1954
Monilinia fructicola (spores)	160 $mg \cdot g^{-1}$	ED_{50} for reduction in fresh spore germination	Laboratory, no soil	McCallan *et al.*, 1954

Table B-1. Continued

Species	Dosage	Response	Conditions	Reference
Neurospora sitophila (spores)	>1000 $mg \cdot g^{-1}$	ED_{50} for reduction in O_2 uptake and germination	Laboratory, no soil	McCallan *et al.*, 1954
Alternaria oleracea (spores)	170 $mg \cdot g^{-1}$	ED_{50} for reduction in fresh spore O_2 uptake and germination	Laboratory, no soil	McCallan *et al.*, 1954
Aspergillus niger (spores)	>3200 $mg \cdot g^{-1}$	ED_{50} for reduction in O_2 uptake by fresh spores	Laboratory, no soil	McCallan *et al.*, 1954
Aspergillus niger (spores)	>1000 $mg \cdot g^{-1}$	ED_{50} for reduction in fresh spore germination	Laboratory, no soil	McCallan *et al.*, 1954
Neurospora sitophila (spores)	26.4 $mg \cdot g^{-1}$	41% reduction in the dissimilation of amino acids in spores incubated for 16 h	Laboratory, no soil	Owens and Novotny, 1959
Neurospora sitophila (conidia fractions)	2.5 $mg \cdot g^{-1}$	Change in phosphorus content: 30% increase in organic P; 6% decrease in ribose nucleic acid	Laboratory, incubation with glycerol, no soil	Owens and Novotny, 1959
Neurospora sitophila	7.6 $mg \cdot g^{-1}$	Change in phosphorus content: 11% increase in inorganic P; 100% increase in organic P; 29% decrease in lipids; 19% decrease in ribose nucleic acid; 64% decrease in germination	Laboratory, incubation with glycerol, no soil	Owens and Novotny, 1959
Neurospora sitophila	12.7 $mg \cdot g^{-1}$	Change in phosphorus content: 33% increase in inorganic P; 92% increase in organic P; 39% decrease in lipids; 22% decrease in ribose nucleic acid; 100% decrease in germination	Laboratory, incubation with glycerol, no soil	Owens and Novotny, 1959
Neurospora sitophila (conidia)	21.1 $mg \cdot g^{-1}$	Decrease in spore constituents: carbohydrates 48%; lipids 2%; proteins 4%; 59% decrease in CO_2 released from spores	Laboratory, no soil	Owens and Novotny, 1959

Table B-1. Continued

Species	Dosage	Response	Conditions	Reference
Neurospora sitophila (conidia)	21.1 $mg \cdot g^{-1}$	Decrease in spore constituents: carbohydrates 48%; lipids 2%; proteins 4%; 59% decrease in CO_2 released from spores	Laboratory, no soil	Owens and Novotny, 1959
Neurospora sitophila (conidia)	7.2 $mg \cdot g^{-1}$	85% decrease in O_2 consumed by spores per hour per milligram; 75% decrease in CO_2 produced by spores per hour per milligram	Laboratory, glucose added as metabolite, no soil	Owens and Novotny, 1959
Neurospora sitophila (conidia)	14.3 $mg \cdot g^{-1}$	98% decrease in O_2 consumed by spores per hour per milligram; 88% decrease in CO_2 produced by spores per hour per milligram	Laboratory, glucose added as metabolite, soil	Owens and Novotny, 1959
Neurospora sitophila (conidia)	9.3 $mg \cdot g^{-1}$	78% decrease in O_2 consumed by spores per hour per milligram; 51% decrease in CO_2 produced by spores per hour per milligram	Laboratory, acetate added as metabolite; no soil	Owens and Novotny, 1959
Neurospora sitophila (conidia)	18.6 $mg \cdot g^{-1}$	97% decrease in O_2 consumed by spores per hour per milligram; 83% decrease in CO_2 produced by spores per hour per milligram	Laboratory, acetate added as metabolite, no soil	Owens and Novotny, 1959