Canadian Environmental Protection Act

Priority Substances List
Assessment Report

Non-pesticidal Organotin Compounds

Government of Canada | Gouvernement du Canada
Environment Canada | Environnement Canada
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Canada

CANADA'S GREEN PLAN

Canadian Environmental Protection Act

PRIORITY SUBSTANCES LIST
ASSESSMENT REPORT

NON-PESTICIDAL ORGANOTIN COMPOUNDS

Government of Canada
Environment Canada
Health and Welfare Canada

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TABLE OF CONTENTS

LIST OF TABLES

Synopsis

Non- pesticidal organotin compounds (notably mono- and di- methyltin, butyltin, and octyltin compounds) are not manufactured in Canada. They are imported, however, mainly for use as poly(vinyl chloride) (PVC) stabilizers and are also used as industrial catalysts. Data identified for 1984 indicate that approximately 290 tonnes of methyltin compounds, 1020 tonnes of butyltin compounds, and a much smaller quantity of octyltin compounds were imported to Canada. More recent data have not been identified.

Based on limited fate information, non-pesticidal organotin compounds are expected to exist predominantly in the aquatic environment. The mono- and di- alkyltin compounds are water-soluble and are not expected to volatilize from water in significant quantities. They undergo biodegradation and photolysis in water and are not expected to persist for long periods. Mono- and di- methyltin and mono- and di- butyltin have been detected in water and sediment at several locations in Canada. However, the sources of these compounds to the environment are uncertain. The methyltin compounds may have resulted from the natural methylation of tin or from anthropogenic sources. The butyltin compounds were primarily degradation products of the pesticide tributyltin, the antifouling use of which has been regulated in Canada since 1989. Leaching of organotin-stabilized PVC pipe by water could also be a source of organotin entry into the Canadian environment.

The assessment of effects on the environment focused on aquatic biota since they are the most likely to be exposed to non-pesticidal organotin compounds. There is limited information on the toxicity of most of these compounds to organisms in both the freshwater and marine environments. Nonetheless, it was possible to compare estimated effects thresholds to environmental concentrations for the mono- and di- methyltin and mono- and di- butyltin compounds. None of the effects thresholds was exceeded by environmental concentrations found in areas where contamination was suspected, indicating that these compounds are unlikely to cause harmful effects to freshwater or marine biota in Canada. Exposure to mono- and dioctyltin compounds is unlikely since they have not been found in Canada or elsewhere in any environmental medium. Although toxicity data for the octyltin compounds are lacking, it is unlikely that they would cause harmful effects to aquatic biota.

The non-pesticidal organotin compounds that were assessed are not volatile and are not expected to contribute to phenomena such as ozone depletion, global warming, or the formation of ground-level ozone.

Available data are insufficient to serve as a basis for estimation of the exposure of the general population to any of the non-pesticidal organotin compounds. Available data on the toxicity of these compounds in experimental animals and humans are also limited.

Based on these considerations, the Minister of the Environment and the Minister of National Health and Welfare have concluded that non-pesticidal organotin compounds do not constitute a danger in Canada to the environment or to the environment on which human life depends. Therefore, non-pesticidal organotin compounds are not considered to be "toxic" as defined under Paragraphs 11(a) and 11(b) of the *Canadian Environmental Protection Act* (CEPA). However, the Ministers have concluded that it is not possible to assess whether any of the non-pesticidal organotin compounds constitutes a danger in Canada to human life or health as defined under Paragraph 11(c) of CEPA.

1.0 Introduction

The *Canadian Environmental Protection Act* (CEPA) requires the Minister of the Environment and the Minister of National Health and Welfare to prepare and publish a Priority Substances List that identifies substances, including chemicals, groups of chemicals, effluents, and wastes that may be harmful to the environment or constitute a danger to human health. The Act also requires both Ministers to assess these substances and determine whether they are "toxic" as defined under Section 11 of the Act which states:

> "...a substance is toxic if it is entering or may enter the environment in a quantity or concentration or under conditions
>
> (a) having or that may have an immediate or long-term harmful effect on the environment;
>
> (b) constituting or that may constitute a danger to the environment on which human life depends; or
>
> (c) constituting or that may constitute a danger in Canada to human life or health."

Substances that are assessed to be "toxic" according to this Section may be placed on Schedule I of the Act. Consideration can then be given to developing regulations, guidelines, or codes of practice to control any aspect of these substances' life cycle, from the research and development stage through manufacture, use, storage, transport, and ultimate disposal.

The non-pesticidal organotin compounds considered in this assessment are presented in Table 1. They are primarily those of monomethyltin, dimethyltin, monobutyltin, dibutyltin, mono-octyltin and dioctyltin. The assessment of whether non-pesticidal organotin compounds are "toxic" was based on the determination of whether they **enter** or may enter the Canadian environment in concentrations or quantities that could lead to **exposure** of humans or other biota to the extent that adverse **effects** could result.

Data relevant to the assessment of whether non-pesticidal organotin compounds are "toxic" to the environment under CEPA were obtained from original and review articles published up to April, 1992 (data obtained after this date were not considered for inclusion in this assessment). These articles were identified from primary journal searches, as well as searches of the following abstracting services and databases:
Chemical Abstracts, Biological Abstracts, ENVIROLINE, TOXLINE, TOXLIT, U.S. National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances (RTECS), CURRENT CONTENTS, U.S. Environmental Protection Agency Toxic Releases Inventory, and Corpus Information Services. Information was also obtained from the CEPA Domestic Substances List and from Statistics Canada.

To identify the toxicological data relevant to the assessment of effects on human health, literature searches of the following electronic databases were conducted: Hazardous Substances Data Bank (HSDB, U.S. National Library of Medicine), Registry of Toxic Effects of Chemical Substances (RTECS, U.S. National Institute for Occupational Safety and Health), Integrated Risk Information System (IRIS, U.S. Environmental Protection Agency), Chemical Carcinogenesis Research Information System (CCRIS, U.S. National Cancer Institute), TOXLINE (1981 to present; U.S. National Library of Medicine), the TOXLINE backfile (1965 to 1980), TOXLIT (1981 to present), and TOXLIT (U.S. National Library of Medicine) backfile. The EMBASE database (online version of *Excerpta Medica)* was searched (for 1973 to present) using a "hedge" to cover toxicological information.

To identify data relevant to the estimation of exposure of the general population to non-pesticidal organotin compounds, literature searches of the following electronic databases were conducted: Environmental Bibliography (Environmental Studies Institute, California), ENVIROLINE (R.R. Bowker, New York), POLLUTION ABSTRACTS (Cambridge Scientific Abstracts, Maryland), and Food Science and Technology Abstracts (International Food Information Service, England) back to 1980, and the complete files of ELIAS (Environment Canada's Departmental Library Catalogue and collection from Fisheries and Oceans Canada), AQUAREF (Ecosystem Sciences and Evaluation Directorate, Environment Canada), MICROLOG (Micromedia, Canadian Research Index), Cooperative Documents Project databases, University of Guelph (CODOC/GDOC), and Canada Institute for Scientific and Technical Information (CISTIMON). Information on exposure is also included in some of the toxicological sources noted above, especially HSDB, TOXLINE, and EMBASE.

A background report prepared by Nieboer and Bryant (1992) under contract to Health and Welfare Canada was consulted to identify information relevant to the assessment of effects of non-pesticidal organotin compounds on human health. The following external organizations were contacted to identify data relevant to estimation of exposure of the Canadian population to non-pesticidal organotin compounds: the Ontario Ministry of the Environment (Graham, 1992); Alberta Environment (Halina, 1992); Scepter/Canron Inc. (Lister, 1992); and the National Sanitation Foundation International (Kenel, 1992).

Data relevant to the assessment of whether non-pesticidal organotin compounds are "toxic" to human health obtained after completion of these sections of this report *(i.e.,* June 1992) were not considered for inclusion. Nonvalidated studies of Industrial Bio-Test Laboratories Inc. identified during the survey of the scientific literature were noted, but were not used in assessing whether non-pesticidal organotin compounds are "toxic" under CEPA.

Available data are insufficient to serve as a basis for estimating exposure of the general population to any of the non-pesticidal organotin compounds. Therefore, this assessment focuses principally on the environmental effects of these compounds.

Although review articles were consulted where considered appropriate, all original studies that form the basis for the determination of "toxic" under CEPA have been critically evaluated by the following Environment Canada staff (effects on the environment) and Health and Welfare Canada staff (effects on human health):

Environment Canada	Health and Welfare Canada
R.J. Maguire	G. Long
	M.E. Meek
	S. Savard

In this report, a synopsis that will appear in the *Canada Gazette* is presented. In addition, an extended summary of the technical information that is critical to the assessment is presented in Section 2.0. The assessment of whether non-pesticidal organotin compounds are "toxic" under CEPA is presented in Section 3.0. Supporting documentation that discusses the technical information in greater detail has also been prepared and is available upon request.

As part of the review and approvals process established by Environment Canada, the environmental sections of this Assessment Report were reviewed by Dr. C.H. Farr, Atochem North America, King of Prussia, Pennsylvania. The health-related sections of this Assessment Report were approved by the Standards and Guidelines Rulings Committee of the Bureau of Chemical Hazards of Health and Welfare Canada. The final Assessment Report was reviewed and approved by the Environment Canada/Health and Welfare Canada CEPA Management Committee.

Copies of this Assessment Report and the unpublished supporting documentation are available upon request from the:

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2.0 Summary of Information Critical to Assessment of "Toxic"

2.1 Identity, Properties, Production, and Uses

There are few data in the literature on the physical and chemical properties of non-pesticidal organotin compounds. However, many organotin compounds dissociate in water into an organotin cation and a companion anion. In water, these species will likely exist as hydrates or complexes depending on the nature and concentration of other solutes *(e.g.*, chloride ion). Consequently, the aquatic persistence, fate, and toxicity of such compounds may be limited to considerations of just six species, those of mono- and di- methyltin, butyltin, and octyltin.

Non-pesticidal applications of organotin compounds are reviewed elsewhere (Gitlitz and Moran, 1983) and summarized here. Mono-organotin compounds are mainly used in the stabilization of poly(vinyl chloride) (PVC) films during manufacture. Smaller quantities are used for glass coating *(e.g.*, butyltin trichloride). Diorganotin compounds are also used mainly as PVC stabilizers, the most important of which are dimethyltin, dibutyltin, and dioctyltin compounds. Dioctyltin compounds are generally used as additives for PVC food packaging products. Dimethyltin compounds are also used for this purpose in some countries, but not in Canada. Other important industrial uses for diorganotin compounds are as catalysts in producing:

- polyurethane foams;
- esters used as plasticizers, lubricants, and heat-transfer fluids; and
- room-temperature-vulcanized silicone elastomers to produce flexible silicone rubbers.

Smaller quantities are used for glass coating *(e.g.*, dimethyltin dichloride), as anthelmintics for poultry, and as stabilizers for lubricating oils, hydrogen peroxide, and polyolefins.

For Canada, the relative importance of various uses of organotin compounds is not definitely known but it is assumed that the use pattern is similar to that reported for the United States (Wilkinson, 1984). In 1982, the most important non-pesticidal applications in the United States were as PVC stabilizers and as catalysts for polyurethane and silicone elastomers, accounting for 67% and 8% of organotin consumption, respectively. Biocidal uses accounted for 20% of the market, and all other uses (including uses as latex paint preservatives, anthelmintics, and coccidiostats) accounted for 5%.

All organotin compounds now used in Canada are imported. In 1984, an estimated 290 tonnes of methyltin compounds were imported to Canada (CIS, 1985a). None was exported. The estimated demand was 170 tonnes for PVC pipe and 120 tonnes for PVC siding and profiles (moulds). In the same year, 1020 tonnes of butyltin compounds were imported to Canada (CIS, 1985b). None was exported. The estimated demand was 405 tonnes for PVC pipe and 615 tonnes for siding and profiles.

There are two major manufacturers of PVC at four locations in Canada - Sarnia, Niagara Falls, Shawinigan, and Fort Saskatchewan (CIS, 1988). The largest use of PVC is in the manufacture of pipe and fittings, and most PVC pipes and fittings used in Canada are made in Canada (CIS, 1988; Statistics Canada, 1989). Organotin compounds are not used only at the four locations previously mentioned. Many smaller companies buy PVC resin and organotin stabilizers, and prepare organotin-stabilized PVC on site (Shermet, 1992). Although the major use of organotin compounds is as PVC stabilizers, only about 30% of all PVC was stabilized with organotin compounds in 1991 (Miller, 1992).

There is no information on amounts of octyltin compounds imported to Canada, but it is assumed that the amounts are far smaller than amounts of methyltin and butyltin compounds because they are used only as stabilizers in some PVC for food packaging (Health and Welfare Canada, 1986).

2.2 Entry into the Environment

Possible routes of entry of non-pesticidal organotin compounds to the environment are:

(a) atmospheric transport from other countries;

(b) losses during transportation;

(c) losses from the manufacture of organotin-containing materials, and from organotin-catalyzed production of polyurethanes and silicones;

(d) losses from weathering or leaching of organotin-containing materials;

(e) leaching of organotin-stabilized PVC pipe by water;

(f) losses from landfill-disposed, organotin-containing materials;

(g) losses from incineration of organotin-containing materials; and

(h) biological production, in the case of environmental methylation of tin to produce methyltin species.

Although it is reasonable to assume that organotin compounds are entering the Canadian environment from non-pesticidal uses, unequivocal evidence of their presence in effluents or emissions is not available. It is clear that some organotin compounds are entering the environment, but it is not clear if they arise from pesticidal or non-pesticidal uses, or from natural methylation of inorganic tin.

It is likely that the most important route of entry of non-pesticidal organotin compounds to the Canadian environment would be through leaching of PVC pipe by water. This is because the most important non-pesticidal use of organotin compounds is

for PVC stabilization, PVC is used principally in the production of pipe, and continuous contact with water is assured. Available information on non-pesticidal sources of methyltin, butyltin, and octyltin species to the Canadian environment is discussed in the following text.

Methyltin species. In Canada, monthly sampling of sewage treatment plant influents, effluents, and sludges in Montreal, Toronto, Hamilton, Sarnia, and Vancouver over a seven-month period (1990 to 1991) revealed no contamination by methyltin species (detection limit 40 ng Sn/L for influents and effluents and 2 ng Sn/g dry weight for sludge) (Chau *et al.,* 1992). No methyltin species were found in leachate samples collected from five landfills in southern Ontario in 1990 (detection limit 40 ng Sn/L) (Chau *et al.,* 1992).

Methyltin species may also be introduced to the environment through microbial methylation of tin (Chau *et al.,* 1980; Weber and Alberts, 1990). In addition to anthropogenic origins for the methyltin species observed in fish, it is possible that the methyltin species were produced naturally, *i.e.,* through microbial methylation of inorganic tin in the fish gut and/or in micro-organisms that are eventually consumed by fish. It should be noted that the monomethyltin and dimethyltin species found in the environment are not degradation products of trimethyltin compounds introduced to the environment, because trimethyltin compounds are not used industrially or as pesticides.

Butyltin species. The monobutyltin and dibutyltin found in a limited study of influents and effluents from sewage treatment plants in five Canadian cities (Chau *et al.,* 1992) may have been present due to their use as PVC stabilizers or because they were degradation products of the pesticide tributyltin, which was also found in sludge samples collected at the same time. The fact that butyltin species were found in sewage treatment plant effluent indicates incomplete removal during sewage treatment. These data are too few to allow meaningful generalizations to be made or to allow the quantitation of releases of butyltin species to the Canadian environment, but they do demonstrate that releases are occurring as a result of pesticidal organotin use or non-pesticidal organotin use, or both.

It should be noted that for both butyltin and methyltin PVC stabilizers, concentrations of organotin stabilizers leached from PVC pipe in the laboratory decline fairly quickly *(i.e.,* over days to weeks) with continuous leaching by water (Boettner *et al.,* 1981; Wu *et al.,* 1989; Quevauviller *et al.,* 1991), and are likely to do so in PVC pipe used in Canada. The United States Environmental Protection Agency has estimated that organotin concentrations in surface waters resulting from the leaching of PVC-stabilized pipes and tubing would be in the pg Sn/L range (U.S. EPA, 1983). The Canadian Standards Association has set a limit for extractable organotin compounds from potable water pipes and fittings (Allidina, 1992).

No butyltin species were found in leachate samples collected from five landfills in southern Ontario in 1990 (detection limit 40 ng Sn/L) (Chau *et al.,* 1992).

Octyltin species. There is no information available about octyltin species entering the environment. Monthly sampling of sewage treatment plant influents, effluents, and sludges in Montreal, Toronto, Hamilton, Sarnia, and Vancouver over a seven-month period (1990 to 1991) revealed no contamination by octyltin species (detection limit 40 ng Sn/L for influents and effluents and 2 ng Sn/g dry weight for sludge) (Chau *et al.*, 1992). No octyltin species were found in leachate samples collected from five landfills in southern Ontario in 1990 (detection limit 40 ng Sn/L) (Chau *et al.*, 1992).

2.3 Exposure-related Information

2.3.1 *Fate*

Most of the data in the literature on the persistence of organotin compounds refer to the aquatic environment. Data on the persistence of the mono- and di- methyltin, butyltin, and octyltin species in aquatic environments are in some cases fragmentary or nonexistent, but by analogy with the tributyltin species, it is assumed that none of these species would be persistent in aquatic environments. Half-lives are estimated to be less than a few months at 20^0C, and longer at lower temperatures. Relevant information for the methyltin, butyltin, and octyltin species follows.

Methyltin species. Inorganic tin and methyltin species undergo biological or abiotic methylation in aquatic environments, although it does not appear to be an important process in short-term studies (Maguire, 1991). Except for the examination of natural methylation mechanisms, little work has been done on the persistence of methyltin species in aquatic environments and distribution among various environmental compartments.

In comparison with butyltin species, for which there is more information, it is likely that sunlight photolysis and microbial degradation of methyltin species will be important, and that in the absence of methylating organisms, methyltin species in aquatic environments would not be persistent, with half-lives of less than a few months at. 20^0C.

Butyltin species. Tributyltin does not volatilize significantly from water (Maguire *et al.*, 1983), and it is unlikely that the more hydrophilic dibutyltin and monobutyltin would volatilize. Thus these species, and non-pesticidal methyltin and octyltin species, are not expected to contribute to phenomena such as ozone depletion, global warming, or the formation of ground-level ozone.

In 90% water/10% acetonitrile solutions, monobutyltin and dibutyltin were stable for at least 9 days in the dark, but were degraded by light of wavelength 300 nm (Maguire *et al.*, 1983). The half-life of degradation of monobutyltin was 0.4 days, and after 9 days, the concentration of inorganic tin accounted for about 70% of the initial monobutyltin concentration. The half-life of dibutyltin was > 9 days, and monobutyltin and inorganic tin were the only products observed. The butyltin species are less stable at acidic pH values (Burns *et al.*, 1987).

The logarithms of the n-octanol-water partition coefficients (log K_{ow}) for monobutyltin trichloride, dibutyltin dichloride, and tributyltin chloride are 0.09, 0.05, and 2.2, respectively (Tsuda *et al.,* 1988). This indicates that monobutyltin and dibutyltin would not be bound to the organic portion of sediment to the same extent as the more lipophilic tributyltin species. Hinga *et al.,* (1987) determined that the mean sediment-water partition coefficient for dibutyltin was about 100 times smaller than that for tributyltin (values were 450 and 40 000, respectively). Stang and Seligman (1987) noted that there was a wide variation in adsorption coefficients of butyltin species to sediments, which may reflect lack of equilibrium and differences in sediment composition. They determined partition coefficients for different sediments in the following ranges: monobutyltin, 1 700 to 29 000; dibutyltin, 2 100 to 26 000; and tributyltin, 6 200 to 55 000.

The biological availability of sediment-associated dibutyltin and monobutyltin has received little attention. Oligochaetes can take up tributyltin from sediment and can degrade it to inorganic tin (Maguire and Tkacz, 1985), and it is assumed that dibutyltin and monobutyltin in sediment would also be bioavailable and biodegradable.

There are few data on the uptake of monobutyltin and dibutyltin species by aquatic organisms. Tsuda *et al.,* (1988) determined the logarithms of the bioconcentration factors in carp *(Cyprinus carpio)* to be 2.1, 1.0, and 3.5, for monobutyltin, dibutyltin, and tributyltin, respectively, in agreement with their octanol-water partition coefficients. Martin *et al,* (1989) noted a similar pattern for rainbow trout *(Oncorhynchus mykiss).*

The persistence of dibutyltin and monobutyltin species in aquatic ecosystems is likely to depend strongly on ecosystem-specific characteristics, such as temperature and the kinds and concentrations of butyltin-tolerant and -degrading organisms, as is the case with tributyltin (Maguire, 1987). Most work indicates that these chemicals are not persistent in aquatic ecosystems *(i.e.,* overall half-lives from all degradation processes would be less than a few months at 20^0C).

Octyltin species. Only two laboratory-based persistence studies on dioctyltin compounds were identified (Akagi and Sakagami, 1971; Mazayev *et al.,* 1976). Both studies indicated that dioctyltin was not persistent. It is difficult to estimate the persistence of mono-octyltin and dioctyltin in aquatic ecosystems using only these two studies as a basis for prediction. However, compared with the butyltin species, it is expected that the octyltin species would not be persistent in aquatic environments, with half-lives of less than a few months at 20^0C.

2.3.2 *Concentrations in the Environment*

Most of the data on the occurrence of organotin compounds in Canada refer to the aquatic environment. They are the result of one national survey (Maguire *et al.,* 1986) and several regional or local surveys (Maguire *et al.,* 1982; Maguire and Tkacz, 1985; Maguire *et al.,* 1985; Kaye *et al.,* 1986; Harding and Kaye, 1988; Seakem Oceanography Ltd., 1989; Cullen *et al.,* 1990) at a total of about 275 locations. Detection limits vary between the studies depending on the technique and sample size.

The findings for the methyltin, butyltin, and octyltin species in Canada are summarized separately in the following. More detailed discussions of the analytical methods for these species, their environmental occurrence in Canada and other countries, and their persistence and fate are given in the Supporting Document. Analytical methods for organotin compounds do not determine the anionic moiety (Maguire, 1991), and for this reason the species are only referred to as methyltin, butyltin, and octyltin species.

Data on concentrations of non-pesticidal organotin compounds in ambient or indoor air in Canada (or elsewhere) were not identified.

Methyltin species. Monomethyltin and dimethyltin have been found in fresh water, seawater, and sediment in Canada at concentrations similar to those observed in other countries. They were found in about 10% of all water samples analyzed.

For monomethyltin, the highest concentration observed in fresh water in Canada was 1 100 ng Sn/L. The mean and median concentrations derived from original data in all studies (n = 32, n.d. values for an additional 242 samples not included in calculations, detection limit 10 ng Sn/L) were 324 ng Sn/L and 200 ng Sn/L, respectively. Monomethyltin was found in only 2 of 70 seawater samples, at concentrations of 80 and 120 ng Sn/L (n.d. values for an additional 68 samples, detection limit 10 ng Sn/L).

For dimethyltin, the highest concentration observed in fresh water in Canada was 320 ng Sn/L. The mean and median concentrations derived from original data in all studies (n = 27, n.d. values for an additional 242 samples not included in calculations, detection limit 10 ng Sn/L) were 92 ng Sn/L and 40 ng Sn/L, respectively. Dimethyltin was not found in any of 70 seawater samples (detection limit 10 ng Sn/L). In some locations, environmental methylation of tin seems to be the most likely explanation for the presence of methyltin species. In industrial areas, it is possible that there is input of anthropogenically-derived methyltin species, or that anthropogenically-derived tin is biologically methylated.

There are few data on the occurrence of methyltin species in Canadian biota. Monomethyltin and dimethyltin species have been found outside Canada in algae, seaweed, eelgrass, mussels, oysters, limpets, and other marine organisms, generally at concentrations less than 120 ng Sn/g wet weight. The exception is eelgrass, in which monomethyltin has been found at concentrations of up to 2 300 ng Sn/g wet weight François and Weber, 1988).

A potential source of exposure to non-pesticidal organotin compounds in drinking water is migration of stabilizers from PVC pipe, which is used fairly extensively in distribution systems in Canada (Lister, 1992). Despite this, few data have been identified on concentrations of these compounds in water supplies. Available information is restricted to one United States report of concentrations of monomethyltin (range: 0.49 to 8.1 ng Sn/L) and dimethyltin (range: 0.40 to 2.2 ng Sn/L) in a limited number of tap water samples collected in Florida in 1977 (Braman and Tompkins, 1979).

Butyltin species. Most of the data on the environmental occurrence of monobutyltin and dibutyltin in Canada were obtained during surveys for tributyltin before its antifouling uses were regulated under the *Pest Control Products Act* in 1989 (Agriculture Canada, 1989). Although it is reasonable to presume that this regulation has resulted in a substantial decrease in environmental butyltin concentrations, especially in fresh water, no large-scale surveys have been undertaken in Canada since 1989. Concentrations of butyltin species in water and shellfish have decreased after similar regulations were introduced in France (Alzieu, 1991), England (Waite *et al.*, 1991), and the United States (Valkirs *et al.*, 1991; Wade *et al.*, 1991).

Monobutyltin and dibutyltin have been found in fresh water, seawater, and sediment at many locations across Canada, especially in surveys conducted before 1986. Many sampling locations were not randomly selected but were in fact sites where maximum concentrations would be expected. The presence of the monobutyltin and dibutyltin species in, or close to, harbours, marinas, and shipping lanes was attributed to the degradation of the antifouling agent tributyltin. Concentrations were similar to those observed in water in areas of heavy boating or shipping traffic in other parts of the world. The two species were found in about 15% of all water samples analyzed.

For monobutyltin, the highest concentration observed in fresh water in Canada was 5 700 ng Sn/L. The mean and median concentrations derived from original data in all studies (n = 65, n.d. values for an additional 209 samples not included in calculations, detection limit 0.1 to 10 ng Sn/L) were 216 ng Sn/L and 27 ng Sn/L, respectively. In seawater, the highest concentration observed was 170 ng Sn/L. The mean and median concentrations derived from original data in all studies (n = 25, n.d. values for an additional 104 samples not included in calculations, detection limit 0.1 to 10 ng Sn/L) were 18 ng Sn/L and 3 ng Sn/L, respectively.

For dibutyltin, the highest concentration observed in fresh water in Canada was 3 700 ng Sn/L. The mean and median concentrations derived from original data in all studies (n = 65, n.d. values for an additional 209 samples not included in calculations, detection limit 0.1 to 10 ng Sn/L) were 148 ng Sn/L and 25 ng Sn/L, respectively. In seawater, the highest concentration observed was 830 ng Sn/L. The mean and median concentrations derived from original data in all studies (n = 38, n.d. values for an additional 91 samples not included in calculations, detection limit 0.1 to 10 ng Sn/L) were 98 ng Sn/L and 25 ng Sn/L, respectively.

There are few data on the occurrence of butyltin species in Canadian biota. Concentrations of monobutyltin and dibutyltin in fish and molluscs ranged from 7 to 179 ng Sn/g wet weight (Maguire *et al.*, 1986; Cullen *et al.*, 1990; Scott *et al.*, 1991; Wong and Chau, 1992) (detection limits 1 to 10 ng Sn/g wet weight). Such concentrations have been observed in biota in other countries.

A limited number of samples (n = 27) of several marine foodstuffs has been analyzed for tributyltin and its degradation products by Health and Welfare Canada (Forsyth and Cléroux, 1991). Monobutyltin and dibutyltin were not detected [detection

limits < 1.0 ng/g (0.7 ng Sn/g*) and < 1.0 ng/g (0.5 ng 5n/g*) wet weight, respectively, for monobutyltin and dibutyltin] in any samples of fish (cod, haddock, perch, trout) but were present in canned and fresh molluscs [concentrations of monobutyltin ranged from 1.2 ng/g (0.8 ng Sn/g*) wet weight in fresh clams to 5.9 ng/g (4.0 ng Sn/g*) in canned mussels and those for dibutyltin ranged from 3.1 ng/g (1.6 ng 5n/g*) in fresh clams and canned cockles to 46.7 ng/g (23.9 ng Sn/g*) in canned mussels]. The monobutyltin and dibutyltin in these samples likely resulted from the use of the antifouling pesticide tributyltin, applied either to boats or to nets in aquaculture; while certain organotin compounds are still registered in Canada for use on boat hulls, no products are currently registered for aquaculture uses. Analyses have also been conducted for butyltin species in fruit drinks (Forsyth, 1992). A limited number of fruit drinks contained monobutyltin [0.1 to 0.2 ng/mL (70 to 140 ng Sn/L*) in 4 of 42 samples, detection limit 0.6 ng/mL (41 ng Sn/L*)].

Based on a U.S. Department of Agriculture survey, it was reported that 8% of 1 031 samples of turkey liver contained dibutyltin (limit of detection 0.04 mg Sn/kg) which resulted from the use of dibutyltin dilaurate as a coccidiostat and anthelmintic in turkeys (Epstein *et al.,* 1991). It was not detected in companion muscle tissue samples. Data are not available to indicate whether use patterns of this compound for this purpose are similar in Canada to those in the United States.

Octyltin species. The mono-octyltin and dioctyltin species have not been found to date in Canada or elsewhere in biota or any environmental medium. These species (if present above their detection limits of 10 ng Sn/L in water) would have been detected by the analytical methods used in the major Canadian surveys for butyltin and methyltin species (Maguire *et al.,* 1982; 1986), and possibly in other Canadian studies as well.

The leaching of octyltin species from PVC plastic packaging, in which they are used as heat and light stabilizers, is a potential source of octyltin compounds in foodstuffs. Analyses have been conducted for octyltin species in selected Canadian foodstuffs (Forsyth, 1992). Out of 15 samples of edible oils, 5 contained both mono-octyltin [5.5 to 26.3 ng/g (2.8 to 13.5 ng Sn/g*); detection limit 1.0 ng/g (0.5 ng Sn/g*)] and dioctyltin [25.2 to 113.3 ng/g (8.7 to 39.1 ng Sn/g*); detection limit 1.0 ng/g (0.3 ng 5n/g*)] A limited number of fruit drinks contained mono-octyltin [4.5 to 16.3 ng/mL (2300 to 8400 ng 5n/L*) in 5 of 42 samples] and dioctyltin [0.9 to 4.3 ng/mL (300 to 1500 ng Sn/L*) in 3 of 42 samples]; however, detection limits were not specified.

2.4 Effects-related Information

2.4.1 *Experimental Animals and* In Vitro

In a bioassay conducted by the United States National Cancer Institute (1978), dibutyltin diacetate was administered to rats in their diet at dose levels of 0, 3.33, or 6.65 mg Sn/kg (b.w.) for 78 weeks, followed by 26 weeks of observation. There was a

* units converted to remove ambiguity when interpreting analytical data, and to be consistent with units used elsewhere in this report

significant dose-related increase in mortality in male but not in female rats. An accidental loss of tissue samples in females (high dose only) precluded an evaluation of carcinogenicity. There were no significant increases in the incidence of neoplasms in males, and the United States National Cancer Institute concluded that there was "no conclusive evidence" for carcinogenicity in this sex.

Concurrently, mice were administered dibutyltin diacetate in their diet at dose levels of 0, 9.9, or 19.8 mg Sn/kg (b.w.) · day for 78 weeks, followed by 14 weeks of observation (United States National Cancer Institute, 1978). A significant increase in mortality occurred in females. Though there was a small increase in the incidence of hepatocellular adenomas in female mice, it was not statistically significant. The United States National Cancer Institute concluded that there was "no conclusive evidence" for carcinogenicity in mice of either sex.

Mosinger (1979) reported the results of two-year studies in which mixtures of monomethyltin and dimethyltin compounds were administered to Wistar rats in their diet at dose levels of 10 mg/kg (b.w.) · day. The mixtures consisted of 25:75 dimethyltin bis(iso-octyl thioglycolate) [otherwise known as dimethyltin bis(iso-octyl mercaptoacetate) - see Table 1] and monomethyltin tris(iso-octyl thioglycolate) [otherwise known as monomethyltin tris(iso-octyl mercaptoacetate)]; 80:20 monomethyltin bis(iso-octyl thioglycolate) sulphide [otherwise known as monomethyltin bis(iso-octyl mercaptoacetate) sulphide] and dimethyltin (iso-octyl thioglycolate) sulphide [otherwise known as dimethyltin (iso-octyl mercaptoacetate) sulphide]; and 80:20 monomethyltin (mercaptoethyl oleate) sulphide and dimethyltin (mercaptoethyl oleate) sulphide.

On the basis of the results of these studies, it was concluded that none of these mixtures was carcinogenic. However, because of several limitations, including limited group sizes (n = 20 of each sex), administration of a single dose level, inadequate statistical analyses, and examination of a limited range of non-neoplastic endpoints, these studies are considered to be inadequate and contribute little meaningful information for assessing the evidence of carcinogenicity for these compounds.

Studies on the genotoxicity of monomethyltin have not been identified and only one report appears to be available in which dimethyltin did not bind irreversibly to DNA *in vitro* (Barbieri and Silvestri, 1991 *in:* Nieboer and Bryant, 1992). Results have been mixed for the genotoxicity of dibutyltin compounds, which have been more extensively investigated. While not genotoxic in the *Salmonella*/microsome test or in a dominant lethal assay in *Drosophila melanogaster*, a mutagenic response in ovary cells of Chinese hamsters has been reported in a limited study in which there was no positive control or replication (Li *et al.*, 1982). Dibutyltin dichloride has also been reported to be positive in a micronucleus test in mice orally administered 50 mg/kg (b.w.) (Life Science Research Limited, 1991).

2.4.2 *Humans*

No quantitative information was identified on the toxicological effects produced in humans following chronic exposure to non-pesticidal organotin compounds.

2.4.3 *Ecotoxicology*

The toxicity of tin compounds has been studied extensively (Hall and Pinkney, 1985; Snoeij *et al.*, 1987; Cooney and Wuertz, 1989; and references therein). Organotin compounds are more toxic to aquatic biota than inorganic tin compounds. Progressive introduction of organic groups to the tin atom in any $R_nSn^{(4-n)+}$ series produces maximal biological activity against all species when n = 3 *(i.e.,* for the triorganotin compounds). Within the class of triorganotin compounds, however, toxicity varies considerably with the nature of the organic substituents (Davies and Smith, 1980).

The most toxic compounds to insects are the trimethyltin compounds; to mammals, the triethyltin compounds; to Gram-negative bacteria, the tripropyltin compounds; and to Gram-positive bacteria, yeasts, fungi, and fish, the tributyltin compounds. Further increase in the alkyl chain length produces a sharp drop in toxicity. Triphenyltin compounds are particularly toxic to phytoplankton (Wong *et al.,* 1982), while tricyclohexyltin compounds show high acaricidal activity (Davies and Smith, 1980). The variation of the anionic moiety, X, within any particular series of R_3SnX compounds usually has little effect on biological activity (Davies and Smith, 1980; Polster and Halacka, 1971).

In this report, environmental concentrations of the organotin species are compared using the most sensitive aquatic organism, with emphasis being placed on ecologically significant toxicity tests *(e.g.,* LC_{50} values are chosen in preference to tests such as the Microtox assay). Only limited information was identified on the toxicity of most non-pesticidal organotin species to fresh water and marine organisms, and no information was identified on the toxicity of the mono-octyltin species to aquatic organisms. No information was identified on the toxicity of non-pesticidal organotin compounds to wild birds or mammals.

Given that tributyltin is considerably more toxic than monobutyltin or dibutyltin, contamination of mono- or di- butyltin with small amounts of tributyltin in testing solutions can lead to apparent toxicity that in fact results from the presence of the tributyltin contaminant (Wester and Canton, 1987). Toxicity thresholds reported for mono-organotin and diorganotin compounds may therefore reflect the toxicity of triorganotin contaminants and should be considered as upper limits.

For monomethyltin species, the water flea, *Daphnia magna,* is the most sensitive freshwater organism in acute toxicity tests. The 48-h LC_{50} for this organism was 0.46 mg Sn/L (Steinhauser *et al.,* 1985). The most sensitive marine organism tested is the diatom *Skeletonema costatum.* The 72-h EC_{50} for growth for this diatom was 0.04 mg Sn/L (Walsh *et al.,* 1985). No data were identified for the chronic toxicity of

monomethyltin to aquatic organisms or for the toxicity of monomethyltin in sediment to benthic organisms.

For dimethyltin species, *Daphnia magna* is the most sensitive freshwater organism in acute toxicity tests. The 48-h LC_{50} for this organism was 0.03 mg Sn/L (Steinhauser *et al.*, 1985). For marine organisms, a "worst case" was chosen for the most sensitive organism, the diatom *Skeletonema costatum.* The 72-h EC_{50} for growth for this diatom was assumed to be 0.27 mg Sn/L (it was reported as >0.27 mg Sn/L - Walsh *et al.*, 1985). No data were identified for the chronic toxicity of dimethyltin to aquatic organisms or for the toxicity of dimethyltin in sediment to benthic organisms.

For monobutyltin species, the red killifish *(Oryzias latipes)* is the most sensitive freshwater organism in acute toxicity tests. The 48-h LC_{50} for this fish was 16 mg Sn/L (Nagase *et al.*, 1991). The only data identified for marine organisms are for yeasts, and the most sensitive marine yeasts are *Aureobasidium pullulans, Candida albicans,* and *Sporobolomyces alborubescens,* with 48-h IC_{50} values for growth of 2.1 mg Sn/L (Cooney *et al.*, 1989). No data were identified for the chronic toxicity of monobutyltin to aquatic organisms or for the toxicity of monobutyltin in sediment to benthic organisms.

For dibutyltin species, the mosquito larva *(Culex pipiens)* is the most sensitive freshwater organism in acute toxicity tests. The 24-h LC_{50} for this larva was 0.1 mg Sn/L (Gras and Rioux, 1965). The most sensitive marine organism tested is the diatom, *Skeletonema costatum.* The 72-h EC_{50} for growth for this diatom was 0.01 mg Sn/L (Walsh *et al.*, 1985). There are no data on the toxicity of dibutyltin in sediment to benthic organisms. There are some data on the chronic toxicity of dibutyltin to clams and fish. Exposure of freshwater clams *(Anodonta anatina)* to 0.015 mg Sn/L dibutyltin dichloride for seven months (weekly static renewal) caused decreases in weight and carbohydrate stores, but no mortality (Holwerda and Herwig, 1986). The no-observed-effect- concentration (NOEC) for histopathological effects in guppies *(Poecilia reticulata)* was <0.125 mg Sn/L for exposure of three months (Wester and Canton, 1987). De Vries *et al.* (1991) investigated the comparative toxicity of various organotin compounds in early life stages of rainbow trout *(Oncorhynchus mykiss).* Beginning with yolk sac fry, trout were continuously exposed for 110 days. Dibutyltin dichloride, with a no-lethal-effect-level of 0.019 mg Sn/L for trout yolk sac fry, was about 1000 times less toxic than tributyltin chloride. The lowest-observed-effect-concentration (LOEC) for mortality was 0.095 mg Sn/L. At the end of the exposure period, resistance to infection was examined by an intraperitoneal challenge with *Aeromonas hydrophila,* a bacterium pathogenic to fish. Resistance to the bacterial challenge was decreased with dibutyltin dichloride at about 0.1 mg Sn/L. This might be indicative of a suppressed immune function or generally diminished fish health. No data were identified for the toxicity of dibutyltin in sediment to benthic organisms.

No data were identified for the toxicity of mono-octyltin to aquatic or benthic organisms.

For dioctyltin species, *Daphnia magna* is the most sensitive freshwater organism in acute toxicity tests. The 48-h LC_{50} for this species was 0.001 mg Sn/L (Steinhauser *et al.*, 1985). No data were identified for the toxicity of dioctyltin to marine organisms or for the toxicity of dioctyltin in sediment to benthic organisms.

3.0 Assessment of "Toxic" under CEPA

3.1 CEPA 11(a) Environment

This environmental assessment considers the six major groups of non-pesticidal organotin compounds that could enter the Canadian environment from the products currently in commerce: mono- and di- methyltin, butyltin, and octyltin species. The quantities of non-pesticidal organotin compounds entering the Canadian environment are not known for any of several possible routes of entry.

Based on limited information on fate, the mono- and di- alkyltin compounds are hydrophilic compounds that do not partition strongly to sediment or tissue. It is also unlikely that they will volatilize to the atmosphere in significant quantities. Most of the available data indicate that mono- and di- alkyltin compounds are not persistent in water with half-lives expected to range from a few days to less than a few months at 20^0C. Because the aquatic environment is the most important repository for organotin compounds, this assessment will focus on the most sensitive aquatic biota exposed to organotin compounds.

There is limited information on the toxicity of most non-pesticidal organotin compounds to freshwater and marine biota. In this assessment, effects thresholds for the most sensitive aquatic biota were estimated by dividing the lowest-observed-effect-level in toxicity tests by various factors that account for the limited data available (Table 2). Emphasis was placed on ecologically relevant test results *(e.g.,* mortality rather than Microtox endpoints). The estimated effects thresholds (EET) were then compared to the mean environmental concentrations (EC) observed in Canada for the freshwater and marine environments. If the EET/EC ratio was $\leq$ 1 for a given compound, then the potential exists for that compound to cause harmful effects to aquatic biota.

The environmental concentrations listed in Table 2 are assumed to overestimate the true mean concentrations in Canada because most sampling was conducted at sites where maximum concentrations would be expected (*e.g.,* harbours and marinas for the butyltin compounds), and the detection frequencies at these sites were low, ranging from 0 to 29%. Non-detections were not included in the calculations of the mean environmental concentrations.

Comparison of the estimated effects thresholds to environmental concentrations for the mono- and di- methyltin and mono- and di- butyltin compounds indicates that the EET/EC ratios ranged from 5 to >1 350 in the freshwater and marine environments (Table 2). Therefore, these compounds are unlikely to cause harmful effects to freshwater or marine biota in Canada. The mono- and di- octyltin compounds have not been found (to date) in Canada or elsewhere in any environmental medium (Table 2). Although toxicity data are lacking for the octyltin compounds, it is unlikely that they could cause harmful effects to aquatic biota because these biota are not exposed to the octyltin compounds.

No data were identified on the toxicity of non-pesticidal organotin compounds to wildlife. Due to the low toxicity of the non-pesticidal organotin compounds to aquatic invertebrates and fish, adverse effects on aquatic-based wildlife due to decreased availability of prey are considered unlikely.

Therefore, on the basis of the available data, non-pesticidal organotin compounds are not considered to be "toxic" as defined under Paragraph 11(a) of the *Canadian Environmental Protection Act.*

3.2 CEPA 11(b) Environment on Which Human Life Depends

The non-pesticidal organotin compounds discussed in this assessment are not appreciably volatile and are not expected to contribute to phenomena such as ozone depletion, global warming, or the formation of ground-level ozone. They are not suspected of being associated with other known direct effects on the environment on which human life depends.

Therefore, on the basis of available data, non-pesticidal organotin compounds are not considered to be "toxic" as defined under Paragraph 11(b) of the *Canadian Environmental Protection Act.*

3.3 CEPA 11(c) Human Life or Health

Population Exposure. Available data on the exposure of the general population to non-pesticidal organotin compounds are extremely limited. Information on concentrations in air has not been identified and data on concentrations in drinking water are limited to only a few samples from Florida. A potential source of non-pesticidal organotin compounds in drinking water, however, is migration of stabilizers from PVC pipe, which is used fairly extensively in distribution systems in Canada.

Available data on concentrations in food are limited to small surveys of individual foodstuffs designed principally for development of analytical methods (Forsyth and Cléroux, 1991; Forsyth *et al.*, 1992). Information relevant to estimation of population exposure is restricted to concentrations of monobutyltin and dibutyltin in a limited number of marine species (Forsyth and Cléroux, 1991), monobutyltin in fruit drinks (Forsyth, 1992), and mono-octyltin and dioctyltin in edible oils and fruit drinks (Forsyth, 1992).

In addition, available models and data on sources and physical/chemical properties are inadequate to allow prediction of which medium, *e.g.*, food, air, or drinking water, is potentially the most important source of exposure of the general population to the non-pesticidal organotin compounds.

Available data are considered insufficient, therefore, to qualitatively or quantitatively estimate exposure of the general population in Canada to any of the non-pesticidal organotin compounds.

Effects. The initial step in evaluating whether any of the non-pesticidal organotin compounds considered here are "toxic" as defined under Paragraph 11(c) of CEPA is an assessment of the weight of evidence for carcinogenicity (an effect for which it is generally believed that there is no threshold), based on the scheme developed by the Bureau of Chemical Hazards in the derivation of the "Guidelines for Canadian Drinking Water Quality" (Health and Welfare Canada, 1989). Only one carcinogenicity bioassay has been reported for any of the compounds *(i.e.,* dibutyltin diacetate) considered for this assessment. In this study, there was no evidence of carcinogenicity in male rats and mice and no convincing evidence in female mice exposed to dibutyltin diacetate. Results in female rats were inadequate for the assessment (United States National Cancer Institute, 1978). The results have been mixed from a limited number of investigations concerning the genotoxicity of dibutyltin compounds. Owing to the limitations of the carcinogenesis bioassay (in particular, the accidental loss of tissue samples for high dose females), dibutyltin compounds have been classified in Group V ("inadequate data for evaluation") of the classification scheme for carcinogenicity developed for use in the derivation of the "Guidelines for Canadian Drinking Water Quality" (Health and Welfare Canada, 1989).

Additional data on the carcinogenicity of any of the non-pesticidal organotin compounds considered in this assessment are restricted to inadequate studies on mixtures of monomethyltin and dimethyltin compounds (Mosinger, 1979).

Therefore, all non-pesticidal organotin compounds considered in this assessment have been classified in Group V ("inadequate data for evaluation") of the classification scheme for carcinogenicity developed for use in the derivation of the "Guidelines for Canadian Drinking Water Quality" (Health and Welfare Canada, 1989).

For compounds classified in Group V of this scheme, tolerable daily intakes (TDIs) are developed based on division of relevant effect levels by appropriate uncertainty factors. Based on a preliminary review of available information, it appears that adequate data on subchronic repeated dose toxicity in experimental animals to serve as a basis for development of a TDI are only available for monomethyltin, dimethyltin, and dibutyltin compounds. Though subchronic studies on dioctyltin compounds are available, they appear, on the basis of preliminary review, to be limited in terms of number of dose levels, duration of exposure, group sizes and possibly, the range of endpoints examined.

To assess whether compounds classified in Group V are "toxic" under CEPA, tolerable daily intakes are compared to estimates of population exposure. However, available data are considered insufficient to qualitatively or quantitatively estimate exposure of the general population in Canada to any of the non-pesticidal organotin compounds considered here. Therefore, it is not possible to evaluate whether current concentrations of any of these compounds present in the environment constitute a danger in Canada to human life or health.

Therefore, on the basis of available data, it is not possible to assess whether any of the non-pesticidal organotin compounds considered here are "toxic", as defined under Paragraph 11(c) of the *Canadian Environmental Protection Act.*

3.4 Conclusion

On the basis of available data, non-pesticidal organotin compounds are not considered to be "toxic" as defined under Paragraphs 11(a) and 11(b) of the *Canadian Environmental Protection Act.* It has been concluded that available data are insufficient to assess whether non-pesticidal organotin compounds are "toxic", as defined under Paragraph 11(c) of the *Canadian Environmental Protection Act*

4.0 Recommendations for Research and Evaluation

1. It is recommended that additional data be generated on the physical/chemical properties of non-pesticidal organotin compounds, in order to characterize partitioning in the general environment more fully. This research is considered to be of high priority.

2. It is recommended that concentrations of organotin compounds be determined in drinking water supplies distributed through PVC pipe in which they are used as stabilizers. Additional data are also needed on concentrations of organotin compounds in all foodstuffs, particularly those used as stabilizers in PVC packaging and those used as veterinary drugs in poultry. This research is considered to be of high priority.

3. Acquisition of additional data on sources and concentrations of non-pesticidal organotin compounds in the Canadian environment is also recommended, including monitoring of:

    - effluents from the largest PVC-fabricating plants in Canada;
    - sewage treatment plant influents, effluents, and sludges;
    - landfill leachates; and
    - fresh water, seawater, sediment, and aquatic biota.

    This research is considered to be of medium priority.

If warranted, based on consideration of additional data on sources and concentrations acquired as recommended, the following studies may also be desirable:

- subchronic toxicity studies on monobutyltin, mono- octyltin, and dioctyltin compounds;
- carcinogenicity bioassays on monomethyltin, dimethyltin, and dibutyltin compounds in which non-neoplastic endpoints, such as organ weight changes and biochemical and hematological effects, are also examined in two species of experimental animals.

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Table 1 Non-pesticidal Organotin Compounds in Canadian Commerce from 1984 to 19861

Chemical Name	CAS No.
Monomethyltin compounds	
methyltin trichloride[2]	993-16-8
methyltin sulphide	33397-79-4
methyltin tris(laurylmercaptide)	52165-03-4
methyltin tris(iso-octy1 mercaptopropionate)	53040-42-9
methyltin tris(iso-octy1 mercaptoacetate)[2]	54849-38-6
methyltin tris(2-ethylhexyl mercaptoacetate)	57583-34-3
methyltin tris(octanoyloxyethylmercaptide)	57813-62-4
methyltin tris(2-mercaptoethyl oleate)	59118-79-5
methyltin tris(lauroyloxyethylmercaptide)	67859-62-5
methyltin tris(tetradecanoyloxyethylmercaptide)	68928--38-1
methyltin tris([2]-linoleoyloxyethylmercaptide)	68928-40-5
methyltin tris(hexadecanoyloxyethylmercaptide)	68928-41-6
methyltin tris(decanoyloxyethylmercaptide)	68928-50-7
Dimethyltin compounds	
dimethyltin dichloride[2]	753-73-1
dimethyltin sulphide	13269-74-4
dimethyltin bis(iso-octyl mercaptoacetate)[2]	26636-01-1
dimethyltin bis(iso-octy1 mercaptopropionate)	42378-34-7
1,1 ,3,3-tetramethyl 1 ,[3]-bis(oleoyloxy)distannoxane	43136-18-1
dimethyltin bis(laurylmercaptide)	51287-84-4
dimethyltin bis(2-ethy1hexyl mercaptopropionate)	57057-50-8
dimethyltin bis(2-ethy1hexyl mercaptoacetate)	57583-35-4
dimethyltin bis(octanoyloxyethylmercaptide)	57813-60-2
dimethyltin bis([2]-oleoyloxyethylmercaptide)	67859-63-6
dimethyltin bis([2]-linoleoyloxyethylmercaptide)	67859-64-7
dimethyltin bis(decyloyloxyethylmercaptide)	67874-41-3
dimethyltin bis(lauroyloxyethylmercaptide)	68928-42-7
dimethyltin bis(tetradecanoyloxyethylmercaptide)	68928-48-3
dimethyltin bis(neodecanoate)	68928-76-7
Mono-n-butyltin compounds	
n-butyltin trichloride[2]	1118-46-3
n-butylstannoic acid	2273-43-0
n-butylchlorotin dihydroxide	13355-96-9
n-butyltin sulphide	15666-29-2
n-butyltin tris(2-ethy1hexanoate)	23850-94-4
n-butyltin tris(iso-octyl mercaptoacetate)	25852-70-4
n-butylthiostannonic acid	26410-42-4
[(n-buty1thioxostanny1)thio]-acetate, 2-ethy1hexy1	26821-65-8
n-butyltin tris(2-ethy1hexyl mercaptoacetate)	26864-37-9
n-butyltin tris(iso-octyl mercaptopropionate)	36118-60-2
n-butyltin tris(octanoyloxyethylmercaptide)	59118-80-8

Table 1 Non-pesticidal Organotin Compounds in Canadian Commerce from 1984 to 1986[1] (Cont.)

Chemical Name	CAS No.
n-butyltin tris(2-oleoyloxyethylmercaptide)	67361-76-6
n-butyltin tris(decanoyloxyethylmercaptide)	67874-51-5
n-butyltin tris(tetradecanoyloxyethylmercaptide)	68928-34-7
n-butyltin tris(2-linoleoyloxyethylmercaptide)	68928-37-0
n-butyltin tris(hexadecanoyloxyethylmercaptide)	68928-39-2
n-butyltin tris(lauroyloxyethylmercaptide)	68928-52-9
n-butyltris[(β-hydroxyethy1)thio]tin	70729-71-4
Di-n-butyltin compounds	
di-n-butyltin dilaurate[2]	77-58-7
di-n-butyltin maleate	78-04-6
di-n-butyltin mercaptopropionate	78-06-8
di-n-butyltin dichloride[2]	683-18-1
di-n-butyltin oxide[2]	818-08-6
di-n-butyltin diacetate[2]	1067-33-0
di-n-butyltin bis(laurylmercaptide)	1185-81-5
di-n-butyltin bis(2-ethylhexanoate)	2781-10-4
tetra-n-butyl-1,3-bis(lauryloxy) distannoxane	3669-02-1
di-n-butyltin sulphide	4253-22-9
di-n-butyltin distearate	5847-55-2
di-n-butyltin bis(2-ethylhexyl mercaptoacetate)	10584-98-2
di-n-butyltin bis(oleate)	13323-62-1
di-n-butyl-n-butoxytin chloride	14254-22-9
di-n-butyltin bis(methyl maleate)	15546-l 1-9
di-n-butyltin bis(2-ethylhexyl maleate)	15546-12-0
di-n-butyltin bis(n-butyl maleate)	15546-16-4
di-n-butyltin bis(acetylacetonate)	22673-19-4
di-n-butyltin bis(iso-octyl maJeate)[2]	25168-21-2
di-n-butyltin bis(neodecanoate)	25168-22-3
di-n-butyltin bis(iso-octyl mercaptoacetate)[2]	25168-24-5
di-n-butyltin bis(iso-octyl mercaptopropionate)	26761-46-6
di-n-butyltin bis(2-mercaptoethyl laurate)	28570-24-3
di-n-butyltin bis(lauryl maleate)	33466-31-8
di-n-butyltin bis(2-ethylhexyl mercaptopropionate)	53202-61-2
di-n-butyltin bis(tetradecyl maleate)	60659-60-1
di-n-butyltin bis(stearyl maleate)	61813-52-3
di-n-butyltin bis(2-oleoyloxyethylmercaptide)	67361-77-7
di-n-butyltin bis(2-linoleoyloxyethylmercaptide)	67859-61-4
di-n-butyltin bis(tetradecanoyloxyethylmercaptide)	67859-65-8
di-n-butyltin [N-(carboxymethyl)-N-(2-hydroxyethyl)] glycinate	68239-46-3
di-n-butyltin bis(hexadecanoyloxyethylmercaptide)	68298-42-0
di-n-butyltin bis(pentadecyl maleate)	68299-23-0
di-n-butyltin bis(decanoyloxyethylmercaptide)	68928-47-2
di-n-butyltin bis(isotridecyl mercaptopropionate)	84896-44-6

Table 1 Non-pesticidal Organotin Compounds in Canadian Commerce from 1984 to 1986[1] (Cont.)

Chemical Name	CAS No.
Th-n-butyltin compounds	
tri-n-butyltin hydride[3]	688-73-3
stannane, [(2-octyl- 1 ,4-dioxo- 1 ,4-butanediyl) bis(oxy)] bis(tributyl-[3]	67701-37-5
Mono-n-octyltin compounds	
n-octyltin tris(iso-octyl mercaptoacetate)	26401-86-5
n-octyltin tris(2-ethylhexyl mercaptoacetate)[2]	27107-89-7
Di-n-octyltin compounds	
di-n-octyltin dilaurate	3648-18-8
di-n-octyltin bis(2-ethylhexyl maleate)	10039-33-5
di-n-octyltin bis(2-ethylhexyl mercaptoacetate)[2]	15571-58-1
di-n-octyltin maleate[2]	16091-18-2
di-n-octyltin bis(laurylmercaptide)	22205-30-7
di-n-octyltin bis(iso-octyl mercaptoacetate)[2]	26401-97-8
di-n-octyltin bis(neodecanoate)	68299-15-0
Other organotin compounds	
tetraphenyltin[4]	595-90-4
propanoic acid, 3,3'-[bis[[2-(iso-octyloxy)-2-oxoctyl]thio] stannylene]bis-, dibutyl ester[5]	63397-60-4

1 Environment Canada (1992). CAS No. means Chemical Abstracts Service Registry number. In the text all alkyl groups are n-alkyl groups unless specified otherwise. Structures of these chemicals are given in the Supporting Document.

2 The assessment of effects of non-pesticidal organotin compounds on human health was limited to these compounds, for which at least some data on mammalian toxicity were identified, in addition to the following closely related compounds: monomethyltin (mercaptoethyl oleate) sulphide, dimethyltin dibromide, dioctyltin dichloride, dioctyltin oxide, and dioctyltin bis(mercaptopropionate).

3 Not included in this assessment because it was reported in very small amounts and the major use of tributyltin is as an antifouling pesticide, the environmental fate and effects of which have been extensively reviewed, and its anti fouling use regulated in Canada under the *Pest Control Products Act* (Agriculture Canada, 1989).

4 Not included in this assessment because it was reported in a very small amount, and because there are no reports of its environmental occurrence, fate and effects in Canada or elsewhere. Moreover, the company which reported it has not imported it since 1986 (Scollard, 1992). It should be noted that the initial product of metabolism of tetraphenyltin is triphenyltin, a pesticide whose toxicity and environmental chemistry have been extensively reviewed, but which is no longer used in Canada. There is no information on the environmental occurrence, persistence, and effects of triphenyltin in Canada.

5 Not included because it was reported in a very small amount, and there is no information on the uses, analysis, fate, occurrence or toxicity of this unusual alkoxyalkyltin compound.

Table 2 Comparisons Between Estimated Effects Thresholds for Environmental Biota and Environmental Concentrations of Non-pesticidal Organotin Compounds

Organotin Compound	Endpoint for Most Sensitive Environmental Organism	Factors Applied	Estimated Effects Threshold (EET)	Environmental Concentrations (EC)	EET/EC
Monomethyltin (Freshwater)	48-hour LC_{50} for *Daphnia magna* 460 µg Sn/L (Steinhauser *et al*., 1985)	20^a x 2^c	11.5 µg Sn/L	0.324 µg Sn/L (mean across Canada were detected; detection frequency = 12%	35
Monomethyltin (Marine)	72-hour EC_{50} for growth in the diatom *Skeletonema costatum* 40 µg Sn/L (Walsh *et al*., 1985)	20^a x 2^c	1.0 µg Sn/L	0.1 µg Sn/L (mean in seawater where detected; detection frequency = 3%	10
Dimethyltin (Freshwater)	48-hour LC_{50} for *Daphnia magna* 30 µg Sn/L (Steinhauser *et al*., 1985)	20^a	1.5 µg Sn/L	0.092 µg Sn/L (mean across Canada where detected; detection frequency = 10%	16
Dimethyltin (Marine)	72-hour EC_{50} for growth in the diatom *Skeletonema costatum* > 270 µg Sn/L (Walsh *et al*, 1985)	20^a	> 13.5 µg Sn/L	<0.01 µg Sn/L (no detection observed in seawater, n = 70; detection limit = 0.01 µg Sn/L)	> 1350
Monobutyltin (Freshwater)	48-hour LC_{50} for the red killifish, *Oryzias latipes* 16 000 µg Sn/L (Nagase *et al*., 1991)	20^a x 4^c	200 µg Sn/L	0.216 µg Sn/L (mean across Canada were detected; detection frequency = 24%)	926

Table 2 Comparisons Between Estimated Effects Thresholds for Environmental Biota and Environmental Concentrations of Non-pesticidal Organotin Compounds (Cont.)

Organotin Compound	Endpoint for Most Sensitive Environmental Organism	Factors Applied	Estimated Effects Threshold (EET)	Environmental Concentrations (EC)	EET/EC
Monobutyltin (Marine)	48-hour IC_{50} for growth inhibition in selected marine yeasts 2100 µg Sn/L (Cooney *et al*., 1989)	20^a x 10^c	10.5 µg Sn/L	0.018 µg Sn/L (mean in seawater where detected; detection frequency = 19%	583
Dibutyltin (Freshwater)	110 d LOEL for mortality in rainbow trout, *Oncorhynchus mykiss* 95 µg Sn/L (de Vries *et al*., 1991)	10^b	9.5 µg Sn/L	0.148 µg Sn/L (mean across Canada where detected; detection frequency = 24%	64
Dibutyltin (Marine)	72-hour EC_{50} for growth in the diatom Skeletonema costatum 10 µg Sn/L (Walsh *et al*., 1985)	20^a	0.5 µg Sn/L	0.098 µg Sn/L (mean in seawater where detected; detection frequency = 29%	5
Mono-octyltin (Freshwater)	No toxicity data available	N/A	N/A	<0.01 µg Sn/L (no detections observed across Canada, n = 274; detection limit = 0.01 µg Sn/L)	N/A
Mono-octyltin (Marine)	No toxicity data available	N/A	N/A	<0.01 µg Sn/L (no detections observed in seawater, n = 70; detection limit = 0.01 µg Sn/L)	N/A

Table 2 Comparisons Between Estimated Effects Thresholds for Environmental Biota and Environmental Concentrations of Non-pesticidal Organotin Compounds (Cont.)

Organotin Compound	Endpoint for Most Sensitive Environmental Organism	Factors Applied	Estimated Effects Threshold (EET)	Environmental Concentrations (EC)	EET/EC
Dioctyltin (Freshwater)	48-hour LC50 for *Daphnia magna* 1.0 µg Sn/L (Steinhauser *et al*., 1985)	$20^a \times 10^c$	0.005 µg Sn/L	<0.01 µg Sn/L (no detections observed across Canada, n = 274; detection limit = 0.01 µg Sn/L)	>0.5
Dioctyltin (Marine)	No toxicity data available	N/A	N/A	<0.01 µg Sn/L (no detections observed in seawater, n = 70; detection limit = 0.01 µg Sn/L)	N/A

a to convert from acute LC50, IC50, or EC50 to chronic NOEL and to account for the extrapolation of lab results to the field and differences in species sensitivity
b to convert from chronic Lowest-Observed-Effect-Level (LOEL) to a NOEL and to account for difference between laboratory and field conditions and species sensitivity
c to compensate for limited toxicity data or use of non-standard toxicity tests