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EXPLORATORY REPORT TIN AND TIN COMPOUNDS

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SUMMARY

This report contains general information on both inorganic and organic tin compounds concerning existing standards, emissions, exposure levels and effect levels. The document is to be considered as a discussion paper with respect to the steps either to be taken for further risk evaluation or to be initiated to fill knowledge gaps identified.

In the Netherlands tin is produced from concentrate and secondary production (about 3,500 tonnes Sn per year), whereas about 5,000 tonnes Sn per year is applied. Of this amount about 4,200 tonnes is applied as inorganic tin (most for the manufacturing of tin-plate) and about 800 tonnes Sn is applied as organotin compounds (most in pesticides, PVC-stabilizer and anti-fouling paints).

Available information indicates that the risk of inorganic tin compounds to humans is very small, if present at all. The risks of inorganic tin compounds to aquatic ecosystems are considered to be small and are likely to be restricted to surface waters in the vicinity of industrial sites. Although little information on exposure to and effects of inorganic tin in soil organisms, current and future emissions to soil indicate a low priority to fill in these gaps in knowledge.

As to organotin compounds no quantitative data on the inhalatory exposure to humans are available. However, it is assumed that environmental concentrations unlikely present a risk to the general population. The risk of organotin compounds in food to humans is unknown, mainly because no quantitative data on exposure are available.
Available data indicate that triphenyl and tributyl organotin compounds present a risk to aquatic ecosystems in the Netherlands. Information on the toxicity to sediment and soil organisms as well as on the occurrence in soil are lacking, hampering a sound risk evaluation.

It is recommended [a] to determine the concentrations of organotins in food, [b] to execute the ban on triphenyl tin acetate and triphenyl hydroxide as soon as possible and [c] to stimulate alternative methods for anti-fouling paints. Further it is advocated to initiate a study into the degradation rates of organotins in sediment and soil in order to determine the maximum allowable emission per year.

1. INTRODUCTION

This scoping report is part of the preparation for drawing up the integrated criteria document tin and tin compounds. The objective of this report is to bring the knowledge of the participants in the scoping meeting to the same level, and to put forward points for discussion and decision-making as to further risk evaluation or activities to be initiated to fill knowledge gaps.

It should be emphasized that the present report does not aim to be exhaustive: the actual standards and recommendations, the sources and exposure levels in the Netherlands are merely outlined, whereas on the other hand the principal effects and indicative (no) effect levels are described. Therefore all proposed toxicological limit values should be considered as provisional.

2. ACTUAL STANDARDS AND GUIDELINES

Table 2.1 gives an overview of the actual standards and guidelines in force in the Netherlands (Contaminantenboekje, 1991; VROM, 1991) for tin and tin compounds. Furthermore tin is considered a "black-list" substance.

Table 2.1 Actual standards and guidelines for tin in the environment in the Netherlands

Environmental compartment/ type of standard	Concentration	Reference
SOIL AND GROUNDWATER		
* soil		
reference value		
(A value; multifunctional)	20 $mg.kg^{-1}$ dw	MPV (1989-1992)
B value; suspicion of risk	50 $mg.kg^{-1}$ dw	MPV (1989-1992)
C value; unacceptable risk	300 $mg.kg^{-1}$ dw	MPV (1989-1992)
* groundwater		
reference value (A value)	10 $\mu g.l^{-1}$	MPV (1989-1992)
B value	30 $\mu g.l^{-1}$	MPV (1989-1992)
C value	150 $\mu g.l^{-1}$	MPV (1989-1992)
SURFACE WATER AND SEDIMENT		
* sediment		
reference value	20 $mg.kg^{-1}$ dw	MPV (1989-1992)
AIR		
* indoor air (work space)		
MAC inorganic tin compounds (except for SnH_2)	2 $mg.m^{-3}$	MAC (1989)
MAC organotin compounds	0.1 $mg.m^{-3}$	MAC (1989)
MAC tin oxide	2 $mg.m^{-3}$	MAC (1989)
FOOD AND DRINKING WATER		
* food		
Tolerable Daily Intake (TDI) (inorganic tin compounds)	2 $mg.bw.day^{-1}$	Contaminantenboekje (1991); JECFA (1988)
Canned food	150 ppm	Contaminantenboekje (1991); JECFA (1988)
OTHER		
* chemical waste		
tin compounds	5,000 $mg.kg^{-1}$ dw	BACA (1991)

dw : dry weight
bw : body weight

Many organotin compounds, especially tributyltin compounds and triphenyltin compounds, are used as pesticides. Standards and guidelines are available for tributyltin oxide (see table 2.2) and triphenyltin compounds (see table 2.3). The use of other tributyltin compounds as pesticides is prohibited in the Netherlands, therefore standards and guidelines are not further specified.

Tributyltin oxide is accepted as a pesticide in the Netherlands, but it is considered as a "black-list" substance. Furthermore tributyltin oxide is considered a "attention substance".

Table 2.2 Actual standards and guidelines for tributyltin oxide in the environment in the Netherlands

Environmental compartment/ type of standard	Concentration	Reference
SOIL AND GROUNDWATER		
* soil		
reference value		
(A value; multifunctional)	0.0001 $mg.kg^{-1}$ dw	MPV (1989-1992)
B value; suspicion of risk	1 $mg.kg^{-1}$ dw	MPV (1989-1992)
C value; unacceptable risk	10 $mg.kg^{-1}$ dw	MPV (1989-1992)
* groundwater		
reference value (A value)	0.0001 $\mu g.l^{-1}$	MPV (1989-1992)
B value	0.5 $\mu g.l^{-1}$	MPV (1989-1992)
C value	2 $\mu g.l^{-1}$	MPV (1989-1992)
SURFACE WATER AND SEDIMENT		
* surface water		
basic value	0.0001 $\mu g.l^{-1}$	VROM (1991)
ceiling value	0.01 $\mu g.l^{-1}$	VROM (1991)
for drinking water preparation	0.1 $\mu g.l^{-1}$	Staatsblad (1983)
* sediment		
reference value	0.0001 $mg.kg^{-1}$ dw	MPV (1989-1992)
ceiling value	0.0015 $mg.kg^{-1}$ dw	MPV (1989-1992)
FOOD AND DRINKING WATER		
* drinking water	0.1 $\mu g.l^{-1}$ value not to be exceeded (Cat I)	WLB (1984)
OTHER		
* chemical waste		
organotin compounds	50 $mg.kg^{-1}$ dw	BACA (1991)

dw : dry weight

Triphenyltin hydroxide is accepted as a pesticide in the Netherlands, triphenyltin acetate is only permitted in combination with other pesticides (Maneb, Metalaxyl).
Triphenyltin acetate, triphenyltin chloride and triphenyltin hydroxide are considered "black-list" substances. Furthermore triphenyltin acetate, triphenyltin chloride and triphenyltin hydroxide are considered "attention substances".

Table 2.3 Actual standards and guidelines for triphenyltin compounds in the environment in the Netherlands

Environmental compartment/ type of standard		Concentration	Reference
SOIL AND GROUNDWATER			
*	soil		
	B value (individual)	1 $mg.kg^{-1}$ dw	MPV (1989-1992)
	B value (total pesticides)	2 $mg.kg^{-1}$ dw	MPV (1989-1992)
	C value (individual)	10 $mg.kg^{-1}$ dw	MPV (1989-1992)
	C value (total pesticides)	20 $mg.kg^{-1}$ dw	MPV (1989-1992)
*	groundwater		
	reference value (A value)	below detection limit	MPV (1989-1992)
	B value (individual)	0.5 $\mu g.l^{-1}$	MPV (1989-1992)
	B value (total pesticides)	1 $\mu g.l^{-1}$	MPV (1989-1992)
	C value (individual)	2 $\mu g.l^{-1}$	MPV (1989-1992)
	C value (total pesticides)	5 $\mu g.l^{-1}$	MPV (1989-1992)
SURFACE WATER AND SEDIMENTS			
*	surface water		
	ceiling value	0.01 $\mu g.l^{-1}$	VROM (1991)
	for drinking water preparation (individual)	0.1 $\mu g.l^{-1}$	Staatsblad (1983)
	for drinking water preparation (total)	0.5 $\mu g.l^{-1}$	Staatsblad (1983)
*	sediment		
	ceiling value	0.001 $mg.kg^{-1}$ dw	MPV (1989-1992)
FOOD AND DRINKING WATER			
*	drinking water		
	pesticides total	0.5 $\mu g.l^{-1}$	WLB (1984)
	pesticides individual	0.1 $\mu g.l^{-1}$ value not to be exceeded (Cat I)	WLB (1984)
OTHER			
*	chemical waste		
	organotin compounds	50 $mg.kg^{-1}$ dw	BACA (1991)

dw : dry weight

3. APPLICATIONS, SOURCES AND EMISSIONS

3.1 PRODUCTION

Tin exists in three states: Sn (elemental), Sn^{2+} (Sn(II) or stannous compounds) and Sn^{4+} (Sn(IV) or stannic compounds). It can form a variety of both inorganic and organometallic compounds. Essentially all organometallic tin compounds are of the Sn^{4+} type (IPCS, 1980). Physical/chemical characteristics of tin and some tin compounds are given in Appendix A.
The chief ore of tin is cassiterite or tinstone, SnO_2. Tin is produced by extraction with sodium carbonate or by electrolytic refining. In the Netherlands, primary production of tin takes place from imported tin concentrate. Estimated data on the production of tin in Europe and world-wide are shown in table 3.1.

Table 3.1 Production of tin (in tonnes Sn per year)

Country/continent	Production	References
Netherlands: production from ore	0	
Netherlands: production from concentrate (1988)	3,463	MEZ (1989)
Netherlands: secondary production (1991, estimate)	120	Meijer (1992)
Most important primary producers:		
Malaysia (1988)	48,000	MEZ (1989)
Brazil (1988)	41,000	MEZ (1989)
Indonesia (1988)	28,000	MEZ (1989)
China (1988)	25,000	MEZ (1989)
Most important secondary producers:		
France (1975)	10,000	IPCS (1980)
World: total production of tin (1975)	236,000	IPCS (1980)
primary tin (1975)	216,000	IPCS (1980)
secondary tin (1975)	20,000	IPCS (1980)
World: total production of tin (1988)	224,000	MEZ (1989)
World: production of concentrate or primary tin (1988)	196,000	MEZ (1989)

0 : no production takes place

Tin-plate consist for 0.3% of tin (in 1989). Production waste of tin applying industries is collected (cutting and clipping waste), and tin is recovered from this waste. Only one tin recovery plant is currently (1992) operating in the Netherlands, with a handling capacity of 50,000 tonnes tin-plate per year. The estimated amount of 40,000 tonnes handled tin-plate per year (1991) leads to an amount of recovered tin of 120 tonnes per year (Meijer, 1992).
Waste tin-plate is also collected from household waste (tin-plate is 2.1% of all unsorted collected household waste) in the Netherlands, with 46% sorted out by magnets in 1988 (Ecotech, 1989; Novem, 1991). Tin is not (yet) recovered from this tin-plate waste (it is directly used in steel production), but the possibility is studied in a pilot plant at the VAM in Wijster (Novem, 1991).

3.2 APPLICATIONS

3.2.1 Metallic tin

Metallic tin is mainly used by industries producing tin-plate, solder, babbitt, brasses and bronzes, pewter, printer's alloy (type metal), and tin chemicals. Table 3.2 gives an overview of some applications in the Netherlands in processes and products. It should be noted that the amount of tin applied in the Netherlands exceeds the amount produced from concentrate and from secondary production (see table 3.1). This may be explained from the import of metallic tin.

Table 3.2 Applications of tin in the Netherlands in 1988 (in tonnes Sn)

Application	Amount	Reference
Total applied in the Netherlands	5,000	MEZ (1989), Oosterhuis et al. (1992)
Tin-plate	2,250	Oosterhuis et al. (1992)
Solder and tin containing alloys	-	
Inorganic tin chemicals (see also table 3.4)	-	
Organotin chemicals (see also tables 3.3 and 3.5)	800*	Crijns (1992)

* : calculated
\- : no data available

Total world production of tin-plate is 13.10^6 tonnes per year (Recycling, 1986). It is the largest single use of tin, about one third of the world tin production is used for the manufacturing of tin-plate (Recycling, 1986). In addition to its use in food and beverage packaging, tin-plate is used extensively in aerosol containers (IPCS, 1980).

Roughly 30% of the world tin production is used for the manufacturing of solder (Recycling, 1986). Tin-lead solder contains from 2% tin for container-seaming up to 63% for electrical connections. In lead-free solder alloys, tin is alloyed with antimony, silver, zinc or indium. The largest quantities of solder are used in car radiators, air conditioners, heat exchangers, plumbing and sheet metal joining, container seaming, generating equipment, electronic equipment, and computers (IPCS, 1980).

Copper-tin alloys are called bronze or speculum (Van der Klis, 1989). The major applications are in marine and railway engineering. Metals used for casting or lining bearing shells are classed as white bearing alloys, but are better known as babbitt. Babbitt alloys are used in bearings in marine propulsion, rail and road transportation, compressors, motors, generators, and fans. Type metals are lead-based alloys containing 3-13% tin that are widely used in the printing trade. Pewter, which contains 90-95% tin, is used in the production of a wide variety of household articles.
Special alloys using tin include dental amalgams, which are mainly silver-tin-mercury alloys, alpha-type titanium alloys that are used in aircraft, and zirconium alloys used in nuclear reactors (IPCS, 1980).

A large amount of the manufactured tin chemicals consists of organotin compounds. Table 3.3 gives an overview of the production of organotin compounds in the Netherlands and world-wide. Organotin compounds are manufactured at one location in the Netherlands. According to the data presented, the Netherlands is an important producer of tributyltin compounds, of which most are exported.

According IPCS (1980), in 1976 only 0.8% of the global metallic tin consumption was used for the production of organotin compounds, but more recent production data (CBS, 1987; MEZ, 1989) indicate that this percentage is much higher.

Table 3.3 Production of organotin compounds in the Netherlands and world-wide (in tonnes organotin per year: 1985-1988)

Country/continent	Production	References
Netherlands		
Total production and import[1+2)]	4,000	CBS (1987)
of which tri organotin compounds	2,700	CBS (1987)
of which triphenyltin compounds	300[3)]	Crijns (1992)
of which tributyltin compounds	1,300[4)]	Evers et al. (1993 in press)
of which di/mono organotin compounds	1,400	CBS (1987)
Export[2)]	3,300	CBS (1987)
Application	850	CBS (1987)
Production of butyl- and phenyltin compounds (di, tri and tetra)	2,500	Crijns (1992)
EC		
Production of butyl- and phenyltin compounds	23,000	Crijns (1992)
World		
Total production	40,000	CBS (1987)
Production of tributyltin compounds	4,500	IPCS (1990)

1) production and import data are accumulated to conceal individual data, total production capacity (including intermediaries) is approximately 5,000 tonnes.yr^{-1}
2) data include the import of products, except for PVC plastics
3) total production, including 150 tonnes.yr^{-1} intermediates, so actual amount sold will be 150 tonnes.yr^{-1}
4) production data might also include intermediates (not mentioned by Evers et al. (1993 in press))
5) sales data

3.2.2 Inorganic tin compounds

Applications of inorganic tin and its compounds are shown in table 3.4. No data on the use in the Netherlands are available.

Table 3.4 Applications of inorganic tin and its compounds (IPCS, 1980)

Compound	Application
Tin(IV) chloride	Mordant in dyeing of silk, preparation of other inorganic and organic tins, manufacture of blueprint and other sensitized papers
Tin(IV) oxide	Ceramic glaze opacifier, ceramic pigments
Tin(IV) hydride	Gas-plate tin on metal, ceramics
Tin(II) acetate	Catalyst
Tin(II) chloride	Electrotinning of steel strip, tin coating of sensitized paper, antisludge agent for oils, stabilizer of perfumes in soaps, additive for drilling mud, electroplating, catalyst in organic reactions
Tin(II) fluoroborate	Tin-plating baths
Tin(II) fluoride	Toothpaste and dental preparations
Tin(II) 2-ethylhexoate	Catalyst for polyurethane foam production and incurring silicone oil formulations
Tin(II) oxalate	Catalyst for coal hydrogenation, catalyst for acid type esterification, transesterification or polyesterification
Tin(II) oxide	Manufacture of gold-tin and copper-tin ruby glass
Tin(II) sulphate	Immersion plating of steel wire, electrotinning strip, with copper sulphate for lacquer finishes
Tin(II) tartrate	Dyeing and printing of textiles
Na-stannate	Alkaline electroplating tin baths, stabilization of chemicals
Na-pentafluorostannite	Dentifrice formulations

3.2.3 Organotin compounds

The applications of organotin compounds in the Netherlands are given in table 3.5.

In the Netherlands most of the organotin compounds are used as fungicides for agricultural and horticultural purposes (see also table 3.7). Only 35% (in 1985) is used as a stabilizer in plastics (PVC). This is in contrast with the global situation, where 76% (in 1981) of all manufactured organotin compounds are used as PVC-stabilizer (CBS, 1987). Table 3.5 shows a decreasing amount of fungicides applied in 1990 compared with 1988, based on sales figures. More than 90% of all fungicides are applied in potato farming (Crijns, 1992).

Table 3.5 Applications of organotin compounds in the Netherlands (in tonnes organotin compounds per year)

Application	Amount	Year	Reference
Pesticides (total)[1]	390-420	1985	CBS (1987)
* fungicides	200	1974/1975	CBS (1987)
(TPTA, TPTH)	370-400	1985	CBS (1987)
	360	1988	Crijns (1992)
	240	1989	Crijns (1992)
	230	1990	Crijns (1992)
* miticides, acaricides	20	1985	CBS (1987)
(azocyclotin, cyhexatin,	10	1988	Crijns (1992)
fenbutatinoxide)	12	1989	Crijns (1992)
	9	1990	Crijns (1992)
PVC-stabilizer	200-370	1985	CBS (1987)
Anti-fouling paints	95	1985	CBS (1987)
* professional ships	90	1985	CBS (1987)
* fishing nets and cages	-	-	IPCS (1990)
* yachting	7	1985	CBS (1987)
	prohibited[2]	1992	Crijns (1992)
Wood preservatives	15-20	1985	CBS (1987)
Catalytic purposes	5-10	1985	CBS (1987)

- : no application in the Netherlands reported
? : no application data available
1) : because of the biocidal properties of organotin compounds, they are used as: agricultural fungicides, general biocides, bactericides and biostats, slimicides, helminthicides, nematocides, herbicides, rodent repellents, molluscicides, ovicides, antifoulants, miticides and insect chemosterilants (IPCS, 1980). See also table 3.7 for the organotin compounds used in the Netherlands as pesticides
2) : prohibited for all ships <25m length, it is expected however that because of large supplies (both at store and users) application has taken place

Less important applications, not further mentioned, are rubber-stabilizers, anti-corrosion in transformers, capacitors and cables, anti-oxidant textile oils, glass coating, fibre-glass treatment and disinfectant for textile.

Tables 3.6 and 3.7 show some applications of organotin compounds per group of organotin compounds.

Table 3.6 Applications of organotin compounds (CBS, 1987; Crijns, 1992; IPCS 1990)

Compound	Application
Tetra organotin compounds	Intermediates for the manufacturing of mono/di/tri organotin compounds
Tri organotin compounds	Fungicides, miticides, anti-fouling paints and wood preservatives
tributyltin compounds	Anti-fouling paints and coatings, agricultural fungicides, molluscicides
triphenyltin compounds	Agricultural fungicides, anti-fouling paints, paint fungicide (conservation agent)
Di organotin compounds	Stabilizing agents in chlorinated plastics and catalyst for poly urethane production
Mono organotin compounds	Stabilizing agents in chlorinated plastics, almost always in combination with di organotin compounds
Estertin compounds	Stabilizing agents in chlorinated plastics

Table 3.7 Applications of organotin compounds as pesticides permitted in the Netherlands (Gewasbeschermingsgids, 1991)

Compound	Application
Tricyclohexyl-1,2,4-triazole-1-yltin	Fruit growing (outside); tree and plant growing (outside) against "spint"
Tricyclohexyltin hydroxide	Fruit growing (outside); berry plants (after harvest or before blooming); selected vegetables; tree growing against "spint"
Bis[tris(2-phenyl-2,2-dimethyl-ethyl)tin]oxide	Fruit growing (outside); berry plants (after harvest or before blooming); selected vegetables; tree growing against "spint"
Triphenyltin acetate[1)]	Potatoes against Phytophtora; Celeriac (outside) against Septoria appiicola; Gladiolus against Botrytis
Triphenyltin hydroxide[2)]	Potatoes against Phytophtora; Apple and pear trees against Nectria galligena (after harvest); Celeriac (outside) against Septoria appiicola
Tributyltin oxide	Wound treatment for trees

1): only permitted in combination with other pesticides
2): also in combination with other pesticides

3.3 EMISSIONS AND WASTE STREAMS

3.3.1 Emissions

Tin and tin compounds are emitted to air and water in the Netherlands. Table 3.8 gives an overview of the tin containing emissions in the Netherlands.

Table 3.8 Emissions of tin and tin compounds in the Netherlands in tonnes Sn per year (most recent data, 1985-1991)

Source	Air	Water	Soil	Reference
Pesticides				
Organotin used as pesticide	?	<2.8[1)]	<140[1)]	CBS (1987)
of which triphenyltin	?	1.1		Crijns (1992)
Organotin from anti-fouling paint		5-12[2)]		Evers et al. (1993 in press)
Industry				
Total industrial tin emission	1.6	18-24		Du Mortier, 1992; ER (pers. comm. 1992b and 1993); Crijns (1993)
of which organotin		0.15-6.7		ER (pers. comm. 1993); Crijns (1992)
Others				
Tin in waste water from dentists		0.9		CUWVO (1990)
Organotin compounds from other sources[3)]		3[4)]		CBS (1987)

? no data are available on organotin pesticide emissions during application to air
1) Approximately 140 tonnes Sn per year are applied as organotin pesticides. A small amount (<2%) of the organotin will end up in surface water; the rest will end up in soil, groundwater or as a residue on crops
2) estimated emission to the Dutch surface waters (fresh and salt)
3) like wood preservatives or catalysts
4) both water and soil

Table 3.8 shows that the use of pesticides (more accurately: fungicides and acaricides, see table 3.7) in agriculture is one of the major sources of organotin compounds in the Netherlands. Organotin compounds are mainly used in potato farming (against *Phytophtora* infestation) and in fruit growing (against "spint"). All organotin compounds applied are sprayed onto the crops or trees to be treated (Gewasbeschermingsgids, 1991).

The organotin compounds strongly bind to the soil, they scarcely leach out. Most of the organotin compounds that end up in water around the area of use, originate from diverted spraying (mainly from airplanes) or equipment cleaning. Locally, for example in potato growing areas during the growing season, this may be a major source of emissions to water (Annema, 1988; Crijns, 1992).

In surface water the use of anti-fouling paint on ships is a major source of organotin compounds contamination. In the Netherlands, the yearly consumption (1985) of these paints was approximately 100 tonnes (in organotin), which corresponds with approximately 35 tonnes Sn per year (CBS, 1987). One may assume that the amount used equals the amount emitted into the environment, primarily into the surface water. It is estimated the 80% of this emission takes place at sea (not specifically in the Dutch coastal waters) (CBS, 1987).
Based on Evers et al. (1993 in press) it is estimated that in 1989 approximately 5-12 tonnes organotin (as Sn) were emitted into the Dutch surface waters from anti-fouling paint on sailing or anchored ships. Point-source emissions of organotin compounds take place at shipyards and other places where the paint is applied or old paint removed. These emissions are estimated at 0.15 tonnes organotin (as Sn) per year (Evers et al., 1993 in press).

Tables 3.9 and 3.10 present an overview of the registered industrial emissions of tin and tin compounds in the Netherlands to air and water.

Table 3.9 Industrial emissions and emissions of waste incinerations of tin and tin compounds to air and water in the Netherlands (kg Sn.yr^{-1}) (Crijns, 1992; Du Mortier, 1992; Emission Registration, pers. comm. 1992b; Emission Registration, pers. comm. 1993; Evers et al., 1993; Matthijsen and Scheffer, 1992)

Source	Emission to air (1985-1991)	Emission to water (1985-1991)
Chemical industry	0	10,00-16,000
Metal and metallurgical industry	900	55
Anodizing industry		6,000[1]
Metal products industry	50	65
Shipbuilding industry	10	150[2]
Electrotechnical industry		2,000
Public utilities	30	
Waste incinerators	240[3]	50
Total	*1,200*	*18,000-24,000*

1) Du Mortier (1992)
2) Evers et al. (1993, in press)
3) Matthijsen and Scheffer (1992)

Table 3.10 Industrial Emissions of tin and tin compounds to air and water in the Netherlands (kg Sn.year^{-1}) (Emission Registration 1992a and pers. comm. 1993; Crijns 1992; Du Mortier, 1992;)

Compound	Emission to Air (1985-1991)	Emission to Water (1985-1991)
Tin and Tin(II)-compounds		100
Tin(II)-ion		2,000
Tin(IV)-compounds		55
Tin(IV)-ion		9,300
Tin oxide	62	
Inorganic Tin-compounds	1,000	
$SnCl_4$	500	
Organotin compounds	1	6,700[1]
		150[2]
Tin and tin compounds		6,000[3]
Total	1,600	18,000-24,000

1) organotin compounds "not specified" and triphenyltin salts (Emission Registration, pers. comm. 1993); probably these data are outdated, because of improved waste water treatment, and replaced by the data from Crijns (1992)
2) emission in 1990 (Crijns, 1992)
3) emission from anodizing industry in 1990 (Du Mortier, 1992)

Organotin compounds emitted to the water are mainly emitted by the basic chemical industries in the province of Zeeland, originating from the organotin producing facility there. Crijns (1992) estimated that the emission from that facility in 1985 was 6,800 kg. In 1990 the emission has been reduced to 150 kg as a result of improved physical/ chemical waste water treatment. Other tin emission data are more difficult to compare, probably because of differences in registration. However, there seems to be a decrease in tin emissions to both air and water during the past 5 years.

Tin in waste water from dentists and dental laboratories originates from the tin present in amalgam used for fillings (CUWVO, 1990). Total emissions to water are 1,300 kg per year. Of this amount 400 kg is filtered, and 900 kg ends up in the sewage system. Based on data on the retention of mercury, it is estimated that of this amount probably 100 kg ends up in surface water and 800 kg in sewage sludge. Filtered amalgam is reused or ends up in domestic waste.

A small but possibly lasting emission of organotin compounds into the indoor air could arise from wood treated with organotin based wood preservatives. This emission is expected to be fairly small, since the vapour pressure of the tributyltin compounds used is low, and because they are reported to form polycarbonate like networks in the wood matrix (Annema, 1988).

No data are available on foreign emissions transported by border-crossing rivers such as the River Rhine. These emissions have not been measured yet and are difficult to estimate. Also no data are available on transfrontier emissions by air.

3.3.2 Waste streams

Table 3.11 gives an overview of the tin containing waste streams in the Netherlands.

Table 3.11 Waste streams of tin and tin compounds in the Netherlands in tonnes Sn per year (most recent data, 1984-1986)

Source	Landfill	Incineration	Total	Reference
Tin-plate in household waste	153[1)]		153	Ecotech (1989)
Organotin used in PVC	60	35	95	CBS (1987)
Other inorganic tin in household waste[2)]	?	?	65	Ansems (1986)
Other organotin compounds in waste[3)]	?	?	10	CBS (1987)

1) 46% of tin-plate in household waste is recycled, tin-plate contains 0.3% Sn (see text)
2) estimated amount of tin in discarded household electronics (TVs, VCRs, radios, etc.)
3) like wood preservatives or catalysts

Tin-plate contains about 0.3% Sn. The total amount of tin-plate in the Dutch household waste is 94,500 tonnes per year. Of this, 46% is recycled (used as scrap metal in the steel production, no tin recovery). The remaining 51,000 tonnes per year tin-plate or 153 tonnes Sn per year may be assumed to end up in landfills, for all Dutch waste incinerators are equipped with magnetic metal separators (Ecotech, 1989). A pilot study has been performed to test the recovery of tin from tin-plate from household waste (Novem, 1991). In the landfills, tin-plate will rapidly corrode to tin(IV)oxide, which is unlikely to leach out in the percolate for it is insoluble in water (Lide, 1990). In laboratory leaching tests, no tin could be detected in the percolate from tin containing (0.13%) aluminium production waste (Van de Beek et al., 1987).

Also the major part of the organotin containing PVC waste will end up in landfills (see table 3.8). Organotin compounds will leach out off PVC (Annema, 1988), but are unlikely to end up in the percolate, for organotin compounds bind strongly to organic matter (Annema, 1988). Tin emissions caused by PVC waste incineration is included in the Emission Registration (see table 3.9).

Tin compounds from other sources (like inorganic tin from solder, organotin from wood preservatives) also end up in landfills. They are unlikely to end up in the percolate from these landfills because of their strong bond to organic matter. No data on the amount of tin present in the percolate from actual landfills is available.

Sludge from waste water treatment facilities is still often used in agriculture. The organotin compounds present in this sludge present another form of diffuse emission. In a Swiss study (Fent et al., 1989 (cited in IPCS, 1990)) it was estimated that in Switzerland the amount of organotin ending up in the soil was 900 kg per year. It is estimated that a similar amount of inorganic tin will end up in the soil, resulting from waste water from dentists and dental laboratories.

3.4 TRENDS

Production (primary and secondary)

No data on trends in the primary production of tin are available, but production has been fairly stable in the past (see table 3.1). In the Netherlands production of tin from concentrate has decreased during the past years, this because of the competition for the concentrate (MEZ, 1989).
In the past, the amount of tin recovered from secondary sources has been rather constant. However, it has been estimated that, in the future, 30% of the total demand for tin will be met by secondary recovery (IPCS, 1980). Waste tin-plate is collected from household waste (tin-plate is 2.1% of all unsorted collected household waste) in the Netherlands, with 46% sorted out by magnets in 1988. This precentage is expected to increase to 70% in 1995; 100% should be achievable by 2000 (Ecotech, 1989; Novem, 1991). If all the tin from tin-plate in Dutch household waste is recovered, the total secondary tin production in the Netherlands would be approximately 400 tonnes per year (including the tin already recovered from tin-plate production waste), or roughly 10% of the total tin production (from concentrate and secondary) in the Netherlands.

Applications

Advancing technology is expected to decrease the amount of tin needed to produce tin-plate. New developments that will decrease the amount of tin are the use of thinner tin layers, and the introduction of the all steel softdrink can (Ecotech, 1989).

The trend in solders is to use less lead, solders with a lower lead content or lead-free solder. This might lead to an increase in the demand for tin (Recycling, 1986).

The amount of organotin compounds applied in potato farming has been growing steadily up between 1980 and 1985 (Annema, 1988), however between 1988 and 1990 there has been a sharp decrease in sales figures (see also table 3.5). In the application in fruit growing, the trend is to use more tin-free pesticides and integrated pest control, leading to a decrease in organotin compounds applied in this area (Annema, 1988). The Dutch National Plan on Crop Protection includes objectives on reduction of use and emissions of pesticides. The Dutch government has issued a guideline in which is stated that the use of fungicides in agriculture should be reduced by 15% in 1995 and by 25% by 2000 compared to 1990.

Recently introduced legislation in the Netherlands, Europe and also in the U.S. prohibits the use of organotin containing anti-fouling paint for ships with a length of < 25 m. Legislation is also being introduced to reduce the amount of organotin compounds in anti-fouling paints as well as to control the rate at which the paints emits the organotin. Research is being done on alternatives for organotin containing anti-fouling paints, but it is not expected that those alternatives will replace organotin in the near future (Annema, 1988; Evers et al., 1993 in press).

The application of organotin compounds as PVC stabilizers strongly depends on the development of the PVC market. There is strong environmental pressure against the use of PVC, which might lead to a decrease in PVC production and thus a decrease in the use of organotin as PVC stabilizer. Compared to other PVC stabilizers (like cadmium and

lead compounds), however, the application of organotin compounds are expected to increase. Unless the demand for PVC decreases sharply, it is expected that the use of organotin compounds as PVC stabilizers will be growing in the foreseeable future.

Emissions and waste streams

The Dutch government has issued a guideline in which is stated that emissions of fungicides in agriculture should be reduced by 15% in 1995 and by 25% by 2000 compared to 1990. This reduction may take place by the use of more efficient methods of applying, better care by the users (less spillage) and by a general reduction in the use, for example by growing more pest resistant races. During the past 5 years, an decrease in the use of fungicides seems to have taken place (see table 3.5).

Emissions of organotin compounds from anti-fouling paint are sure to decrease, now that the use of it is banned for small ships (<25m). In the long term the emissions are expected to decrease even further, as alternatives become available. Alzieu (1989: cited in IPCS, 1990) reported that in France the tributyltin concentrations in water decreased after legislative restrictions in the use of tributyltin compounds in anti-fouling paint came into effect. In the Netherlands, however, this effect has not been found, probably because of the transport of these compounds from sediment to surface water (Evers et al., 1993 in press).

Although industrial tin emission data are difficult to compare, probably because of differences in registration, there seems to be a decrease in tin emissions to both air and water during the past 5 years. A decrease in organotin emissions from organotin production is caused by better effluent treatment.

The amount of tin waste originating from tin-plate containing household waste is expected to decrease, as more tin-plate will be recycled and the tin content of tin-plate decreases (Ecotech, 1989).

Based on the agreement with dentists on the use of amalgam separators, the emission of inoganic tin to surface water and agricultural soil will decrease.

4 OCCURRENCE AND CONCENTRATIONS

4.1 SOIL AND GROUNDWATER

4.1.1 Soil

De Bruijn and Denneman (1992) calculated the natural background concentration of tin in the soil in the Netherlands, based on samples taken by Edelman and RIVM. They determined a reference line, which shows the tin concentration depending on the clay content of the soil: Sn = 4 + 0.6xL, in which Sn represents the tin concentration in the soil in $mg.kg^{-1}$ and L represents the clay content of the soil in percent. The reference value of standard soil (containing 25% clay and 10% organic matter) is 19 $mg.kg^{-1}$ (7 $mg.kg^{-1}$ on poor soils and 34 $mg.kg^{-1}$ on rich soils).

Bowen (1966: cited in IPCS, 1980) reported levels of tin in soil in the USA ranging from about 2 to 200 $mg.kg^{-1}$, the metal being strongly adsorbed by the humus. Tin concentrations of 30 to 300 $mg.kg^{-1}$ have been reported in peat from Finland by Gordon (1952: cited in IPCS, 1980). Several other authors (IPCS, 1980) reported tin levels in soils of several countries varying between 75 and $>1{,}000$ $mg.kg^{-1}$.

In the soil, organotin compounds will degrade eventually to inorganic Sn(IV) compounds. Tri-organotin will first be converted to di-organotin, then via mono-organotin to inorganic tin (Harrison, 1989). This conversion is a combination of chemical, biochemical and photochemical processes, with a reported half-time of 140 days (Barnes et al., 1973 and Odeymi, 1980: both cited in Annema, 1988).
Organotins strongly bind to the soil. In 16-week leaching tests (Blunden, 1984: cited in Annema, 1988) no significant leaching of tri-organotin compounds was found from various soil types.
Inorganic tin is also not expected to be very mobile, for common forms of inorganic tin, like SnO_2 or $Sn(OH)_4$, are very insoluble minerals. Gerritse et al. (1982: cited in Bockting et al., 1992) showed that tin is a relatively immobile metal in sand and loam soils. Inorganic tin can be converted to organotin compounds by means of biomethylation. This has been shown in laboratory experiments, but no quantitative data are available (IPCS, 1980; Annema, 1988).

4.1.2 Groundwater

Stuyfzand (1991) has measured the occurrence of tin in groundwater in the Netherlands. For groundwater with $pH \geq 6.2$ the tin content was ≤ 2 $\mu g.l^{-1}$, in groundwater with $pH < 6.2$ the tin content was <30 $\mu g.l^{-1}$. It should be noted that for more than 95% of the measurements, the tin content was below the detection limit. No increased tin levels were found in areas where organotin pesticides are used (Crijns, 1992).

4.2 SURFACE WATER AND SEDIMENT

4.2.1 Surface water

Organotin concentrations in surface water in the Netherlands are shown in table 4.1.

Table 4.1 Organotin concentrations in surface water in the Netherlands (μg Sn.l^{-1}) (Annema, 1988; Crijns, 1992; Evers et al., 1993 in press)

Location	Substance	Average	max.	year	note
Fresh water					
Rhine (Lobith)	triphenyltin	0	-	1991	n=1
Meuse (Eijsden)	triphenyltin	0.006[1)]	-	1991	n=1
Scheldt	triphenyltin	0	-	1991	n=1
Other					
waterbodies	total tin	0.002 - 2.8	4	1989-90	
	organotin	0.002 - 0.26	2	1987-90	
	tributyltin	0.44	0.93	1991	in yachting harbours
	triphenyltin	0.01 - 0.26	1.14	1991	0 in 78% of samples
Salt water					
Wester Scheldt	tributyltin	0 - 0.24[2)]	-	1988	
	monobutyltin	0 - 0.5 [3)]	-	1988	
Other					
waterbodies	tributyltin	0 - 1.6	-	1988-89	in harbours
	tributyltin	0 - 0.009	-	1988	other locations

0 not present, or below detection limit
- no (significant) data available
1) could also be 0.002 μg.l^{-1}, conflicting data in same reference
2) could also be 0 - 0.6 μg.l^{-1}
3) could also be 0 - 0.95 μg.l^{-1}

Quentin (1988) reports tin concentrations in rivers of 0.006-0.04 μg.l^{-1}. According to De Bruijn and Denneman (1992), the average background concentration of tin in surface waters is 0.002 μg Sn.l^{-1} (90-percentile 0.1 μg.l^{-1}).
It has been reported by Vinogradov (1953) and Mason (1966) (both cited in IPCS, 1980) that tin is present in sea water in amounts of about 3 μg.l^{-1}. Van der Sloot (1979: cited in Stuyfzand, 1991) reports a tin content of 0.6 μg.l^{-1} for the ocean. Hunt and Wilkinson (1990) report that the tin concentration in open sea is $<$0.08 μg Sn.l^{-1}.
In England, France and Canada maximum tributyltin concentrations varying between 0.15 and 3 μg Sn.l^{-1} have been found in fresh and sea water systems (Annema, 1988).

In many different studies (IPCS, 1990) it was shown that the highest organotin concentrations in water were found in yachting areas. The concentrations varied throughout the year, the highest concentrations found when most of the boats were painted.
In fresh and salt water, there is a positive relationship between the size of the yachting harbour, the number of yachts and the measured tributyltin concentrations. In salt water, highest tributyltin concentrations are found in yachting harbours with stagnant water,

lower concentrations in harbours with tidal movements, and lowest concentrations in harbours used for professional shipping (Evers et al., 1993 in press).
In France significantly lower tributyltin concentrations were found after prohibiting the use of anti-fouling paints for ships smaller than 25 m. In the Netherlands, however, this effect has not been found, probably because of the transport of these compounds from sediment to surface water (Evers et al., 1993 in press).
In a number of the measurements the maximum value exceeds the ceiling value for the compound (0.001 $\mu g.kg^{-1}$ dw, see chapter 2).

In surface water, organotin compounds degrade poorly (Annema, 1988), reported half-times for biochemical degradation range from 75 to well over 220 days (Crijns, 1992), in sediment degradation is expected to be even slower (Annema, 1988). Photochemical degradation is much faster, with reported half-times of 7.5-18 days (Crijns, 1992). Data on degradation rates are difficult to compare, for most half-times reported are the time required to half the amount of one compound regardless the formation of degradation products, while sometimes the time reported is the time needed to totally (to inorganic tin) degrade the component. Many factors influence the degradation (salinity, pH, amount of light, presence of micro-organisms etc.).
Organotin compounds will absorb strongly in the sediment and to suspended particles (10-95%) (Annema, 1988; IPCS, 1980; IPCS, 1990). In the surface water itself, the concentration in the surface microlayer is reported to be up to 10 times higher than the concentration in the bulk water (Annema, 1988).

After adaptation, activated sludge waste water treatment has shown to break down 87% of the tributyltin oxide to inorganic tin (Annema, 1988).
A Swiss waste water treatment facility was able to remove down 98% of the butyltin compounds fed to it in the waste water. This plant was equipped with a final filtration step. It was estimated that the removal would have been 87% without this last filtration (IPCS, 1990). The total butyltin content of the effluent was 0.011 $\mu g.l^{-1}$.

4.2.2 Sediment

Organotin concentrations in the sediments in the Netherlands are shown in table 4.2.

In England, Canada, Switzerland and the USA, tributyltin concentrations varying between 0 and 11,000 μg $Sn.kg^{-1}$ dw have been found in fresh and saline sediments (IPCS, 1990).
In general highest concentrations have been measured near ship yards and yachting harbours. In a number of the measurements the maximum value exceeds the ceiling value for the compound (see Chapter 2).

Table 4.2 Organotin concentrations in the sediment ($\mu g\ Sn.kg^{-1}$) (Annema, 1988; CBS, 1987; Crijns, 1992; Evers et al., 1993 in press; IPCS, 1990)

Location	Substance	Average or range	Max.	Year	
Netherlands:					
fresh water sediment:					
Rhine	butyltin	<3 - 300 dw	-	1988	
along potato fields	triphenyltin	5,560 dw[1)]	81,500 dw[1)]	1988[2)]	
		370 dw[1)]	1,300 dw[1)]	1989[2)]	
various locations	tributyltin	0 - 6,000 dw	-	1987[4)]	
	triphenyltin	0 - 320 dw	-	1986-91[5)]	
West-Overijssel	cyhexatin		410 dw	1990	n=?
saline water sediment:					
Eems Dollard	tributyltin	<25 ww	-	-	
	triphenyltin	68 - 91 ww[3)]	-	1985	
Wester Scheldt	tributyltin	<3 - 500 dw	-	1988	
Easter Scheldt	tributyltin	100 dw	-	1988	n=1
	triphenyltin	10 dw	-	1986	n=1

0 below detection limit
- no (significant) data available
dw dry weight
ww wet weight
1) manipulated value: "calculated to reference soil (10% solid substance)"
2) could also be 1989 and 1990 resp., conflicting data in same reference
3) concentrations measured at different depths in the sediment, the higher values for the deeper samples
4) 0 in 79% of the samples
5) 0 in 42% of the samples

Organotin compounds degrade slowly to inorganic tin in the sediment (Annema, 1988). Degradation products of organotin compounds, inorganic Sn(IV) compounds (Harrison, 1989) are likely to end up in the sediment, for they are insoluble in water (IPCS, 1980; Lide, 1990).

Biomethylation of inorganic and organic tin in sediment and in river sludge to mono-, di- and trimethyltin has been reported (Annema, 1988). Biomethylation of inorganic Sn(IV) and of methyltin compounds has also been shown in laboratory tests using micro-organisms from marine sediments (Harrison, 1989). Quentin (1988) reports methyltin concentrations in sea- and freshwater of 0.01-8.5 $\mu g.l^{-1}$.

4.3 AIR

4.3.1 Air (outdoor)

No data on the occurrence of tin in the outdoor air in the Netherlands are available.

The background level of tin is about 0.01 $\mu g.m^{-3}$ (WHO, 1993 in press). Tin concentrations from 0.003 to 0.3 $\mu g.m^{-3}$ were found in 60.6% of 754 samples tested from 22 cities in the USA. More than 50% of samples from 3 urban and 3 rural sites were below detectable level. The highest concentration of tin (0.8 $\mu g.m^{-3}$) was found in a sample from a Boston, USA, industrial site. In Heidelberg, Germany, tin concentrations of 0.096-0.167 $\mu g.m^{-3}$ were reported (IPCS, 1980).

Tin emission concentrations of 10-640 $\mu g.m^{-3}$ were reported from electric furnaces at certain plants in Japan, in 1972. At a distance of 700 metres, the atmospheric tin concentrations still ranged from 3.8 to 4.4 $\mu g.m^{-3}$ (IPCS, 1980).

Tin is present in dust suspended above cities (Vollenbroek, 1989). In the air above Arnhem 86 μg dust.m^{-3} was measured. This dust contained 233 mg Sn.kg^{-1}, corresponding to at least 0.02 μg Sn.m^{-3}. It should be noted that this is probably the highest amount measured.
Otson (1989) reports 0.004 $\mu g.m^{-3}$ in the air (airborne suspended particles) in Toronto, Canada, and did not detect any tin in the air in Whitehorse, Canada (a small rural town).

4.3.2 Rainwater

No data on the occurrence of tin in the rainwater are available.

4.3.3 Air (indoor)

No data on the occurrence of tin in the indoor air in the Netherlands are available.

Otson (1989) measured 0.001-0.002 $\mu g.m^{-3}$ in the indoor air (airborne suspended particles) in Canadian houses (in Toronto and Whitehorse). No clearcut relationship has been found between indoor and outdoor tin concentrations. No data are available on the influence of smoking on the tin concentrations in indoor air.

4.4 FOOD AND DRINKING WATER

4.4.1 Food

Table 4.3 gives an overview of average tin concentrations in some foodstuffs and beverages in the Netherlands, as well as the total daily intake of tin as obtained from a duplicate meal study.

Table 4.3 Tin concentrations in food and beverages in the Netherlands

Food	Concentration[1] (mg.kg^{-1}, median or range)	Reference
24h diet[2]	<0.21 (<0.09-9.81) mg.pers^{-1}.d^{-1}	Vaessen and Van Ooik, 1988
Potatoes	0.0-0.2	Contaminantenboekje, 1991
Vegetables	<0.07	Contaminantenboekje, 1991
	<0.07	Vaessen et al., 1981
Union (fresh and in glass)	<0.21	Contaminantenboekje, 1991
	0.4-2.2 dw	Vaessen et al., 1981
Beans	<0.09	Contaminantenboekje, 1991
Fruit	<1.5	Contaminantenboekje, 1991
	<0.25-2.23 dw (<0.02-1.52 ww)	Vaessen et al., 1985
Meat	0-0.3	Contaminantenboekje, 1991
Kidney	0-0.3	Contaminantenboekje, 1991
Liver	0-0.3	Contaminantenboekje, 1991
Fishery products	0.1-0.35	Contaminantenboekje, 1991
Coffee milk	0-130	Contaminantenboekje, 1991
Fruit (canned)	0-210	Contaminantenboekje, 1991
Mushrooms (canned)	42-350	Contaminantenboekje, 1991
	594-4,900 dw (42-406 ww)	Vaessen et al., 1981
Mushrooms (glass)	0.12-0.15	Vaessen et al., 1981
Asparagus (canned)	0-380	Contaminantenboekje, 1991
Soup (canned)	-110	Contaminantenboekje, 1991

1) on wet weight basis, unless otherwise specified
2) daily intake, not concentration
< indicates that (some of the) the measured value(s) was/were below the detection limit
ww wet weight
dw dry weight

The total daily intake (<0.21 mg.day^{-1}) of tin as determined from the duplicate meals, is well below the maximum daily intake standard as set by the WHO/FAO (140 mg.day^{-1}) (Vaessen and Van Ooik, 1988).
Only in certain canned foods (mainly mushrooms and asparagus) is the tin content higher than standards set by the Dutch "Warenwet". Tin could in many cases not be detected in fresh fruit or vegetables.
Most of the tin in the daily diet originates from food in tin-cans (Vaessen and Van Ooik, 1988).

Tin-cans are not the only source of tin contamination of food. Also the organotins used as stabilizers in PVC can leach out off the plastic into the food or water stored in plastic containers or bottles. This amount is very small though, an increase of up to 0.07 mg Sn.kg^{-1} (0.433 mg organotin compound.kg^{-1}) in tin content of beverages stored in PVC after 2 months of storage at 30°C is reported by Carr (1969: cited in IPCS, 1980), depending on the type of beverage stored. Crompton (1979) reports that beer stored in PVC bottles contained 1.7 μg Sn per litre after 8 weeks storage at 20 °C. Woggon (1974) reported <0.15 ppm organotin PVC stabilizer (di-n-octyltindithioglycolacid-2-ethylhexylester) had migrated from the PVC plates into distilled water and into a 3% acetic acid solution after 10 days at 45°C, 0.5 mg.kg^{-1} of the organotin stabilizer had

migrated into a 50% ethanol solution after 10 days at 45°C.

Because of accumulation in aquatic food, the tin found in fishery products possibly is organic tin. Furthermore, organic tin could be present as pesticide residues on foodstuffs. No reliable data on concentrations organotin in foodstuffs are available, but in the Netherlands, organotin (tricyclohexyltin) residues have been found on apples and pears (IPCS, 1980). It has also been reported that the concentrations of tricyclohexyltin in treated products under glasshouse conditions, including cucumbers, tomatoes, and bell peppers are unlikely to exceed 0.5 $mg.kg^{-1}$ (IPCS, 1980). Triphenyltin residues in various foodstuffs, such as potatoes, carrots, and sugar beet, rarely exceed 0.1 $mg.kg^{-1}$ and can be considerably reduced by washing (WHO, 1993 in press).

4.4.2 Drinking water

In 32 out of 175 samples taken in 1964 of municipal water in the USA tin was detected, concentration ranged from 0.8-30 $\mu g.l^{-1}$ (mean range 1.1-2.2 $\mu g.l^{-1}$ (IPCS, 1980; WHO, 1993 in press).

No data on the occurrence of tin or organotin compounds in drinking water in the Netherlands are available. According to KIWA (pers. comm. 1993) tin concentrations are much lower than 1 $\mu g.l^{-1}$.
Tin concentrations in drinking water are expected to be very low, since the Sn level in groundwater is low (see Stuyfzand, 1991). Furthermore most of the organotin compounds in water strongly absorb to the suspended particles (which are not present in drinking water), and inorganic tin compounds are hardly soluble in water.
Organotin may leach out off PVC pipe used for the transportation of the water. This may be a source of tin in drinking water. Boettner (1982: cited in Hunt and Wilkinson, 1990) reported initial organotin concentrations of 35 $\mu g.l^{-1}$ in water flowing through PVC pipe. Concentrations dimethyltin in drinking water flowing through PVC pipe at a temperature of 37°C decreased from 45 $\mu g.l^{-1}$ on the first day at a rate of 3.0-0.25 $\mu g.l^{-1}.day^{-1}$ during the next 3 weeks. Drinking water flowing through pipe made of chlorinated PVC at 72°C had a dimethyltin concentration of 2.6 $\mu g.l^{-1}$ on the first day, the decrease over the next 3 weeks was 1.0-0.03 $\mu g.l^{-1}.day^{-1}$ (Boettner, 1981: cited in Annema, 1988).

4.5 HUMAN EXPOSURE LEVELS

Intake of tin originates from food and beverages, drinking water and from the air.
Food and beverages are the main source of tin intake. Intake by means of the daily diet ranged from <0.09-9.81 $mg.person^{-1}.day^{-1}$ in a duplicate meal study conducted by Vaessen and Van Ooik (1988), the median value being <0.21 $mg.person^{-1}.day^{-1}$. In some extreme cases (large amounts of tin-food with a high Sn content) the daily intake of tin could be >38 $mg.day^{-1}$ (IPCS, 1980).

The intake of tin from the air is strongly influenced by the area of residence (urban or rural) but is small compared to the intake by means of food, and is also hard to estimate accurately for lack of reliable data. Schroeder (1964, cited in: IPCS, 1980) estimated the daily tin intake from the air to be 0.003 $mg.day^{-1}$. No data are available on the influence of smoking on human tin intake.

Because very few data are available on the organotin contents of food and beverages, drinking water and air, no human exposure levels for these tin compounds can be estimated. Because of accumulation in aquatic food, the tin found in fishery products possibly is organic tin. Furthermore, organic tin could be present as pesticide residues on foodstuffs.

5. EFFECTS

5.1. HUMAN TOXICITY

According to ECETOC (1991), effects on humans of tin and tin compounds have been evaluated by the following institutes:

* ACGIH (American Conference of Governmental Industrial Hygienists) (1986);
* ATSDR SARA (Agency for Toxic Substances and Disease Registry, Superfund Amendments and Reauthorization Act) (1990);
* BUA (Beratergremium für Umweltrelevante Altstoffe der Gesellschaft Deutscher Chemiker) (1988);
* EEC (Commission of the European Communities) (listed);
* MAK (Senatskommission zur Prüfung Gesundheidschädlicher Arbeitsstoffe der Deutschen Forschungsgemeinschaft) (1988);
* NIOSH (National Institute for Occupational Safety and Health (1976);
* WHO IPCS/EHC (World Health Organization, International Programme on Chemical Safety, Environmental Health Criteria (1980 and 1990).

This chapter is mainly based on Evers et al. (1993 in press), IPCS (1980), JECFA (1989), IPCS (1990), JMPR (1990), JMPR (1992) and WHO (1993, in press). Tributyltin is abbreviated as TBT, tributyltin oxide as TBTO, triphenyltin (fentin) as TPT, triphenyltin hydroxide as TPTH and triphenyltin acetate as TPTA.
Data on inorganic tin compounds are calculated to metallic tin, organotin compounds are not converted.

Although tin is present in small amounts in most animal and human tissues, it is uncertain whether it is an essential element for mammals. However, there is evidence that tin is essential for the normal growth of rats. Both inorganic and organotin compounds in concentrations similar to those present in feeds were found to stimulate growth rate in rats maintained on purified amino acid diets (Schwarz et al., 1970, 1971 and 1973 (cited in IPCS, 1980)). The authors concluded that tin, as an essential element, could have a function at the active site of some metal-depending enzymes; however, this has still to be confirmed. No evidence exists however that tin is essential for other species including man (IPCS, 1980).

5.1.1 Chemobiokinetics and metabolism

Although a substantial amount of literature exists on the absorption, distribution, excretion, and storage of tin, the results of much of the early research are questionable in the light of modern analytical techniques (IPCS, 1980).
The behaviour of organotin compounds depends both on their chemical structure and on speciation (IPCS, 1990).

Absorption

* **Inhalation**

No quantitative data are available on inhalatory absorption of inorganic tin and organotin compounds.

* **Ingestion**

Ingested inorganic tin is poorly absorbed. Most studies indicate that less than 5% is absorbed from the gastrointestinal tract, although values as high as 20% have been reported. The influence of oxidation state and anion complement on the rate of gastrointestinal absorbtion was found to be important (IPCS, 1980).

In general, organotin compounds are more readily absorbed from the gut than inorganic tin compounds. Absorption varies with tin compound and species. As a rule, tin compounds with a short alkyl chain are more readily absorbed (IPCS, 1980). 20-50% of TBT is absorbed, depending on the vehicle used (IPCS, 1990), and approximately 40% of TPTH (JMPR, 1992).

* **Skin absorption**

No data are available on skin absorption of inorganic tin compounds.

Trialkyltin compounds are usually well absorbed. TBT is absorbed at approximately 10%) (IPCS, 1990). Dermal absorption of TPT ranging between 34-38% has been found, of which more than 95% was bound to the skin (JMPR, 1992).

Distribution

Absorbed inorganic tin leaves the vascular system rapidly. The highest concentrations are found in the lung, kidney, liver and bone. Penetration of the blood-brain and placental barriers appears to be very slight in experimental animals. With the exception of the lungs, inorganic tin does not accumulate in organs with increasing age.

Following intravenous administration to mice and rats, the highest concentrations of dibutyl- and diethyltin were found in liver and kidney tissue. The distribution of TBT in organisms is usually rapid. In a number of species (rat, mouse, rabbit and guinea-pig), it is found preferentially in the liver and kidneys and, to a lesser extent, in the spleen, fat, lungs, brain and muscle (IPCS, 1990). TBT can be transferred across the blood-brain barrier and from the placenta to the fetus (IPCS, 1990).

In humans, comparatively high (inorganic) tin concentrations were found in lung, kidney, liver, bone, and also in lymph nodes. Fairly high levels were reported in the teeth (IPCS, 1980). The lungs seem to accumulate tin with advancing age. Tin was rarely detected in tissues of still born infants, indicating that inorganic tin does not readily cross the placental barrier.

Biotransformation

Inorganic tin is unlikely to be oxidized or reduced during absorption and systemic transport (IPCS, 1980).

Many organotin compounds are transformed, to some extent, in the tissues. The dealkylation and dearylation of tetra-, tri-, and disubstituted organotin compounds seem to occur in the liver, but the dealkylation of diethyltin compounds appears to take place both in the gut and in tissues of other organs (IPCS, 1980).

TBT metabolism in mammals is rapid; dealkylated metabolites are detectable in blood within 3 hours of TBT administration (IPCS, 1990).

Elimination

The major route of excretion of absorbed inorganic tin is the kidney although a small fraction (< 15%) is excreted into the bile. Relatively large differences in half-lives have been found, ranging from 10 to 120 days, depending on the route and the site of application (IPCS, 1980).

The mode of excretion of organotin compounds largely depends on the type of the compound. The route of excretion for many compounds is not known. Biological half-lives of different organotin compounds varies and many compounds disappear slowly from the organs (IPCS, 1980).

Excretion of TBT is via the bile rather than the urine (IPCS, 1990). An initial rapid elimination, followed by a slower phase in the faeces, has been reported. The rate of TBT loss differs with different tissues, and estimates for biological half-lives in mammals range from 23 to about 30 days (IPCS, 1990). TPT is also excreted mainly via the bile. Faecal elimination was found to be biphasic with half-lives of 9 and 50-60 hours (JMPR, 1992).

5.1.2 Toxicity

Acute toxicity (single and short-term exposure): animal data

Inorganic tin compounds

No data on lethal concentrations in case of acute or short-term inhalatory exposure are available.

Few data are available in case of oral exposure. Acute oral LD_{50} values range from 220 to 3,200 mg compound.kg^{-1} body weight (150 to 1,950 mg Sn.kg^{-1} bw) in rats (see also table 5.1) (IPCS, 1980). Rabbits are less sensitive (WHO, 1993 in press). It must be noted, however, that tin hydride is not taken into account in this report. Many of the reported effects are localized because of irritant properties of inorganic tin compounds. Vomiting and diarrhoea are typical signs that follow oral intake of foods with a high tin content (IPCS, 1980).

Compared with most organotin derivatives, inorganic tin and its salts are not highly toxic, mainly because of their poor absorption and rapid tissue turnover.

Systemic effects of inorganic tin compounds after oral or parenteral administration include effects on the liver, kidneys, bloodforming organs and central nervous system (IPCS, 1980).

Organotin compounds

LC_{50}s (4 hour exposure) for the rat vary between 44 and 69 mg TPT.m^{-3} in case of inhalatory exposure (JMPR, 1992).

Few data are available on acute inhalatory exposure to TBT. "Nose only" inhalation LC_{50} (4 hours) for the rat is 77 mg TBTO.m^{-3} (65 mg.m^{-3} when only inhalable particles are considered). TBT vapour/air mixtures produce no observable toxic effects, even at saturation. However, TBT is very hazardous as an inhaled aerosol, producing lung irritation and oedema (IPCS, 1990).

Data on lethal doses of organotin compounds in case of oral exposure are given in table 5.1.

Trimethyl and triethyl compounds are more toxic than the higher homologues of the trialkyltin group. The oral toxicity diminishes progressively from tripropyltin to trioctyltin compounds. This is probably because of poorer absorption of higher trialkyltin compounds from the gastrointestinal tract (IPCS, 1980).

Table 5.1 Acute toxicity studies: LD_{50}s found for several inorganic and organotin compounds (using various test animals) after oral exposure (IPCS, 1980; IPCS, 1990; JMPR, 1992 and WHO, 1993 in press)

Compound/group Animals tested	LD_{50} (*mg compound.kg^{-1} bw*)	LD_{50} (*mg Sn.kg^{-1} bw*)
Inorganic tin		
Tin (II) chloride dihydrate		
Rat	700 - 3,200	425 - 1,950
Sodium pentafluorostannite		
Rat	220 -580	150 - 390
Organotin		
Mono-organotin compounds		
Mouse/Rat	1,400 - >6,000	
Di-organotin compounds		
Various animals tested	35 - 8,500	
Tri-organotin compounds		
Various animals tested	4 - 10,000	
* **TPT compounds**		
Various animals tested	21 - 491	
* **TBT compounds**		
Mouse/rat	10 - 234	
Tetra-organotin compounds		
Various animals tested	7 - 6,000	

Distinction should be made between the acute effects of di-, tri-, and tetrasubstituted organotin compounds. The principal toxicological difference is that some trisubstituted compounds have a specific effect on the central nervous system producing cerebral oedema, whereas disubstituted compounds do not produce this effect but are potent irritants that can induce an inflammatory reaction in the bile duct. Toxicologically, the tetrasubstituted compounds resemble trisubstituted compounds, which are, generally, more toxic than the mono- and disubstituted derivatives (IPCS, 1980).

Acute effects of TPT include anorexia, emesis, tremor and diarrhoea followed by drowsiness and ataxia (JMPR, 1992).

Effects of acute exposure to TBT compounds may include alterations in blood lipid levels, the endocrine system, liver and spleen, and transient deficits in brain development. The toxicological significance of these effects, reported after high single doses of the compound, is questionable and the cause of death remains unknown (IPCS, 1990).

Several studies have demonstrated that the trialkyl derivatives of tin, and notably TBT compounds, are inhibitors of oxidative phosphorylation in mitochondria and are, therefore, responsible for inhibiting energy transfer (IPCS, 1990).

Acute toxicity (single and short-term exposure): human data

Inorganic tin compounds

No data on acute toxicity in case of inhalation of inorganic tin compounds are available.
Acute symptoms that have been reported following ingestion of food with a high tin content include nausea, vomiting, diarrhoea, stomach cramps, fatigue, and headache. The lowest concentration of tin reported in association with such outbreaks was about 150-250 mg $Sn.l^{-1}$ in canned tomato, orange and apple juice.
Five human volunteers did not experience any symptoms from the ingestion of fruit juice containing concentrations of 500-730 mg $Sn.l^{-1}$ (corresponding to 1.7-3.6 mg $Sn.kg^{-1}$ body weight) but all had gastrointestinal disturbances at a level of 1,370 mg $Sn.l^{-1}$ (corresponding to 4.4-6.7 mg $Sn.kg^{-1}$ body weight). Only one case of nausea recurred when the trial was repeated 1 month later. The apparent development of tolerance in this study remains to be explained.
Similar cases have been reported after consumption of canned herring, salmon, fruit salad, apricots, peaches, cherries, asparagus, pumpkin and rhubarb, with lowest toxicity concentrations reported of 250 mg $Sn.l^{-1}$ (IPCS, 1980; JECFA, 1989).

Organotin compounds

Inhalation of vapours of methyltin compounds have been reported to cause psychological disturbances, difficulties in breathing, headaches and stomachaches (Evers et al., 1993 in press). Acute intoxication caused by the inhalation of triphenyltin compounds has been reported in some instances (farmers treating plants). The first symptoms started from a few minutes to about 2 hours after the first exposure. Symptoms were flushes, nausea, shortness of breath, general malaise, severe headache, epigastric pains and loss of consciousness. All subjects recovered completely (IPCS, 1980).
In four cases of acute poisoning due to exposure to organotin vapours (not identified), patients were reported to have suffered from such symptoms as vertigo, headaches, nausea, vomiting, and visual disturbances. Clinically, stasis of the papilla was found and all patients displayed pathological findings on the electroencephalograms. These were reversible in 7-25 days, and all cases recovered clinically.

There have been no reported cases of poisoning from ingestion of TBTO and other TBT salts (IPCS, 1990). No data are available on acute oral administration of other organotin compounds.

Dibutyl- and tributyltin compounds produced skin irritation in workers 1-8 hours after contact (IPCS, 1980). Experimental application to the skin of volunteers showed that some compounds (e.g., dibutyltin dichloride and tributyltin chloride) produced this effect, whereas others such as dibutyltin maleate and tetrabutyltin did not. Di- and tributyltin compounds cause eye irritation after brief contact. A 20% solution of triphenyltin acetate produced irritation of the skin and the mucous membranes of the upper respiratory tract while tricyclohexyltin hydroxide was reported not to cause skin irritation at a concentration of 0.01 $mg.kg^{-1}$ body weight (IPCS, 1980).
TBTO is a skin and eye irritant and severe dermatitis has been reported after direct contact with the skin. The potential problem is made worse by the lack of an immediate response to the skin (IPCS, 1990).

Subacute and (sub)chronic toxicity (long-term exposure): animal data

Exposure by inhalation

An overview of NO(A)ECs and LO(A)ECs of tin and tin compounds (inorganic and organic) is given in table 5.2.

Table 5.2 Subacute, subchronic and chronic inhalatory toxicity NO(A)ECs and LO(A)ECs (mg compound.m^{-3}) of tin and tin compounds (IPCS, 1980 and 1990)

Test species	Exposure time	NO(A)EC	LO(A)EC	Effects	References
inorganic tin compounds					
tin (IV) chloride					
Guinea pig	several months, 10 minutes.d^{-1}		3,000 (1,350)*	Transient irritation of the nose and eyes.	Pedley (1927)
organotin compounds					
triphenyltin hydroxide (aerosol)					
Rat	13w, 5d.w^{-1}, 6h.d^{-1}	0.05	0.5	"Nose only"-inhalation study. Affection of haematological and biochemical parameters.	JMPR (1992)
tributyltin oxide (aerosol)					
Rat	4-5w, 5d.w^{-1}, 4h.d^{-1}		2.8	"Nose only"-inhalation study. Mortality, apathy, respiratory distress, reduced consumption and body weight gain,inflammatory reactions respiratory tract,lymphocytic effects.	Schweinfurth (1985)
tributyltin oxide (vapour)					
Rat	4-5w, 5d.w^{-1}, 4h.d^{-1}	0.16		"Nose only"-inhalation study. No observable effects.	Schweinfurth (1985)
tributyltin chloride					
Rat	4m		4	Minor irritation of the eye and nose, initial increase in relative liver weight, reduction over whole period, fat drops in liver, diffuse oedema of the brain, inflammatory changes in respiratory tract.	Gohlke et al. (1969)

( ) : NO(A)ECs and LO(A)ECs are presented in mg $Sn.m^{-3}$ in case of inorganic tin compounds
* : Data are calculated to mg $Sn.m^{-3}$

Inorganic tin compounds

For inorganic tin compounds few data are available. Exposure of guinea pigs by inhalation to tin(IV) chloride (3 $mg.l^{-1}$ (1,350 mg $Sn.m^{-3}$) for 10 minutes, daily, for "several months") produced only transient irritation of the nose and eyes (Pedley, 1927 (cited in IPCS, 1980)).

Organotin compounds

A "nose only" inhalation study (lasting 4-5 weeks) by Schweinfurth (1985: cited in IPCS, 1990) with rats exposed for 4 $h.d^{-1}$ (5 days per week; 21 to 24 exposure periods) to an aerosol of TBTO (2.8 $mg.m^{-3}$) produced mortality (50% of males and 60% of the females), apathy, and respiratory distress. Food consumption and body weight gain were reduced. There were inflammatory reactions within the respiratory tract and lymphotoxic effects (depletion of lymphocytes in the thymic cortex, atrophy of the thymus, and lymph nodes). Inhalation of TBTO vapour/air mixtures produced no observable effect. A concentration of 0.16 $mg.m^{-3}$ in the inhalation chamber, which corresponds to the equilibrium vapour pressure of TBTO at room temperature, was considered to be the NOEC for rats (Schweinfurth, 1985 (cited in IPCS, 1990)).

Groups of SPF-Wistar rats were exposed to 0.05, 0.5 and 2.0 mg TPTH.m^{-3} for 6 $h.d^{-1}$, 5$d.w^{-1}$ for 13 weeks (nose only exposure). Analytical concentrations were 0.014, 0.338 and 1.997 $mg.m^{-3}$ and MMAD 3 μm. Observations were made for clinical signs, body weight, food consumption, ophthalmoscopy, haematology and clinical chemistry, organ weights, macroscopy and histopathology. WBC, glucose, total bilirubin were decreased in females at 0.5 and 2.0 $mg.m^{-3}$. Calcium and phosphorus level were decreased in all exposed female groups. Calcium level decreased also in males at 0.5 and 2.0 $mg.m^{-3}$. IgG increased in females at the high exposure level and IgM increased in males at 0.5 and 2.0 $mg.m^{-3}$. Macroscopic examinations showed lesions in the form of multiple red foci in the lungs of most dead rats. At 2.0 $mg.m^{-3}$ the upper and lower air passages, in particular the lungs, were severely affected. The NOAEL in this study is, according to the author, 0.05 $mg.m^{-3}$ (JMPR, 1992).

Oral exposure

Inorganic tin compounds

An overview of NO(A)ELs and LO(A)ELs of inorganic tin compounds is given in table 5.3. Long-term studies with lowest NO(A)ELs or LO(A)ELs are described below.

Mice and rats (Schroeder and Balassa, 1961; Schroeder et al., 1968 (cited in IPCS, 1980)) given tin(II) chloride in drinking water at a concentration of 5 mg $Sn.l^{-1}$ (0.5 $mg.kg^{-1}$ $bw.day^{-1}$) grew normally throughout their life. The life span of mice of both sexes and of male rats was not affected but that of female rats was shorter and there was an increased incidence of fatty degeneration of the liver. Vacuolar changes in the renal tubules were apparent in rats of both sexes.

In a 115-week study, Cpb-WU rats were exposed to 0, 200, 400, or 800 mg $tin.kg^{-1}$ food (0, 10, 20, or 40 mg $Sn.kg^{-1}$ $bw.day^{-1}$) as stannous chloride. Anaemia was observed in weeks 4 and 13 at each dose level, but not during the 2nd year of the study. At autopsy, the only effect noted was a slight increase in the relative spleen weight at 400 and 800 $mg.kg^{-1}$, but no histopathological changes were observed. A slightly increased tin content in the bones was seen at the highest dose level only. The NOAEL in this study was 400 $mg.kg^{-1}$ food, equivalent to 20 $mg.kg^{-1}$ body weight per day (Sinkeldam et al., 1981 (cited in JECFA, 1982a)).

Table 5.3 Subacute, subchronic and chronic oral toxicity NO(A)ELs and LO(A)ELs (mg compound.kg^{-1} body weight.day^{-1} or mg Sn.kg^{-1} bw.day^{-1} (between parenthesis)) of inorganic tin compounds (IPCS, 1980; JECFA, 1982a and WHO, 1993 in press)

Test species	Exposure time	NO(A)EL	LO(A)EL	Effects	References
tin chloride					
Mouse	lifetime (dw)	(0.5)#		No effects on liver, tubules growth and lifespan.	Schroeder et al. (1961 and 1968)
Rat	115w (d)	(20)	(40)	Slight increase in relative spleen weight, no histopathol. changes	Sinkeldam et al. (1981)
Rat	lifetime (dw)		(0.5)#	Increased incidence of fatty degeneration in the liver (only in females, not in males), vacuolar changes in the renal tubulus in both sexes, reduced lifespan in females.	Schroeder et al. (1961 and 1968)
tin (II) chloride/sulphate/oxalate/orthophosphate/tartrate					
Rat	4-13w (d)	300# (150)*		Hepatic alterations, reduced food intake and retarded growth, slight anaemia and atrophy in the pancreas.	De Groot et al. (1973)
sodium pentafluorostannite					
Rat	15-30d (o)	20 (13)*	100 (65)*	A dose-related decrease in haemoglobin concentration, and dose-related retardation of growth.	Conine et al. (1975 and 1976); Yum et al. (1976)
sodium pentachlorostannate					
Mouse	lifetime (dw)	(500)#		No adverse effects.	Walters and Roe (1965)
Rat	1y (d)	1,000# (400)*		No pathological changes in gastro-intestinal tract, kidneys or liver.	Roe et al. (1965)
tin (II) oleate					
Mouse	lifetime (d)	(700)#		No adverse effects.	Walters and Roe (1965)
tin (II) ethyl hexoate					
Rat	1y (d)	500# (150)*		No pathological changes in gastro-intestinal tract, kidneys or liver.	Roe et al. (1965)

( ) : NO(A)ECs and LO(A)ECs are presented in mg Sn.kg^{-1} body weight.day^{-1}
* : Data are calculated to mg Sn.kg^{-1} body weight.day^{-1}
\# : Data are calculated from mg.kg^{-1} diet or mg.kg^{-1} drinking water to mg.kg^{-1} body weight.day^{-1}
(dw): drinking water (d): diet (o): other

Table 5.4 Subacute, subchronic and chronic oral toxicity NO(A)ELs and LO(A)ELs (in mg compound.kg^{-1} body weight.day^{-1}) of organotin compounds (IPCS, 1980; IPCS, 1990; JMPR, 1992 and WHO, 1993 in press)

Test species	Exposure time	NO(A)EL	LO(A)EL	Effects	References
dibutyltin dichloride					
Rat	6w (d)		2.5	Dose related decrease in weight of thymus and related lymphoid organs and immunological effects.	Seinen et al. (1976)
Rat	54d (d)	1	2.5	Reduced growth and food intake, changes in bile duct.	Barnes and Stoner (1958)
Rat	3m (d)	2	4	Mild anaemia, growth retardation, decreased food intake.	Gaunt et al. (1968)
dioctyltin dichloride					
Rat	6w (d)		2.5	Dose related decrease in weight of thymus and related lymphoid organs and immunological effects. Same, but less pronounced effects in diethyl dichloride and dipropyl dichloride.	Seinen et al. (1976)
Rat	8-12w (d)		3.75	Reduction in thymus weight and immunocompetence.	Miller et al. (1985)
dioctyltin-S,S'-bis(isoctylmercapoacetate)					
Rat	12m (d)		10	Increase kidney weight in females.	Nikonorow et al. (1973)
triethyltin hydroxide					
Rat	4w (d)		1#	Weakness in the hind legs, and developing of resistance. Specific lesion of CNS.	Stoner et al. (1955) Magee et al. (1957)
Rat	90d (d)		0.25	Affected growth, interstitial oedema in the CNS.	Verschuuren et al. (1966)
triethyltin sulphate					
Rat	4m (dw)		0.5#	Paralysis of posterior limbs, severe cerebral oedema, spongiosis in adults.	Suzuki (1971)
tricyclohexyltin hydroxide (cyhexatin)					
Rat	90d (d)		2.5#	Slight reduction in weight gain in females.	Shirasu (1970)
Rat	90d (d)		40#	Intra- en extrahepatic cholangitis, mild kidney effects.	Shirasu (1970)
Rat	2-generation (d)	0.1	0.5	Decreased weight gain in females.	JMPR (1992)
Rat	2y (d)	3		No effects stated.	JMPR (1971)
Rabbit	14d (d)		0.75	Dose related decrease in body weight (teratogenicity study).	JMPR (1992)
Dog	2y (d)	0.75	12	Loss of weight, decreased food intake.	JMPR (1971)
triphenyltin acetate					
Guinea pig	12w (d)		0.3#	Decrease in lympho/leucocytes and histological changes in lymphatic tissues in females.	JMPR (1971)
G. pig	4m (d)		0.6#	Haematological effects.	JMPR (1971)
G. pig	104d (d)		0.9#	Reduced immunoresponse in females.	JMPR (1971)
G. pig	2y (d)		0.3#	Dose-related inhibition of growth rate.	JMPR (1971)
triphenyltin chloride					
Rat	2w (d)		0.75#	Reduction relative thymus weight.	Snoeij et al. (1985)

: Data are calculated from mg.kg^{-1} diet or mg.kg^{-1} drinking water to mg.kg^{-1} body weight.day^{-1}
(dw): drinking water (d): diet (o): other

Species	Exposure time	NO(A)EL	LO(A)EL	Effects	References
triphenyltin hydroxide					
Mouse	3m (d)	3.4	17	Affection of haematological parameters, increased liver weight, decrease in relative weight of ovaries, adrenals, kidneys, heart and brain in females.	Suter and Horst(1986)
Mouse	80w (d)	1	4	Decreased body weight gain, increased liver weight at 16 $mg.kg^{-1}$ $bw.day^{-1}$.	JMPR (1992)
Rat	3-4w (d)		1.25'	*Suppression of cell-mediated immunity.*	Vos et al (84)
Rat	30d (d)	1.8	3.6	Decreased food consumption in females, increase in thrombocyte count in females.	Leist et al. (1982)
Rat	12w (d)		5'	Reduction in haemoglobin and leucocytes.	JMPR (1971)
Rat	13w (d)	0.3	1.5	Affected biochemical parameters.	Suter and Horst (86)
Rat	104w (d)		0.3	Dose-related increase in mortality in females, decreased serum immunoglobulins.	JMPR (1992)
Rat	2y (d)	0.1		Effects not stated.	JMPR (1971)
Rabbit	13d (o)	0.1	0.3	Dose-related decrease in mean body weight gain and food consumption.	JMPR (1992)
Dog	8w (d)		0.6'	Decrease in leucocytes number.	JMPR (1971)
Dog	52w (d)	0.21	0.63	Affected biochemical parameters, increased relative liver weight in females.	Sachsse et al. (1987)
tributyltin oxide					
Mouse	3m	4	16	Histological changes in liver, dose-related increased liver weight.	Biodynamics (1989)
Rat	4w (d)		0.5	Reduced eosinophil counts (males), erythrocyte rosettes.	Krajnc et al. (1984)
		0.5	2	Dose-related increased (ALAT) alanine amino transferase activity, increased monocytes count (males), reduced absolute and relative thymus weight (females)	
		2	8	Reduced weight gain (males), slight *atrophy of hepacytes*, reduced blood haemoglobin and haemocrit (females), reduced mean erythrocytes volume, effects on immunoglobulin levels.	
Rat	4w (d)	0.6	6	Decrease in absolute and relative thymus weight (males), decrease in thymus cell counts (males), reduction in thymus cortex (males).	Schering (1989)
Rat	5w (d)	0.6	6	Changed immunological response.	Schering (89)
Rat	6w (d)	0.5	2	Significant reductions in haemocrit, affected insulin level.	Krajnc et al. (1984)
Rat	6w (d)		2	Changed immunological response.	Vos et al (84)
Rat	26w (o)		3	Dose-related reduction in absolute and relative thymus weight, increased pituitary weight.	Funahashi (1980)
		3	6	Reduced body weight, slight decrease in spleen weight, increased relative pituitary and relative adrenal weight.	
Rat	2y (d)	0.025	0.25	Changed immunological response. decrease in splenic iron content.	Wester et al. (1990)
tributyltin chloride					
Rat	2w (d)		15'	Marked reductions in body weight and brain weight associated with reduced food intake (25%).	Snoeij et al. (1984)

Organotin compounds

An overview of NO(A)ELs and LO(A)ELs of organotin compounds is given in table 5.4.

The toxicity of organotin compounds is essentially determined by the number and nature of the organic substituents. In general, the toxicity to mammals decreases from tri- to monoorganotins. The tetraorganotin compounds resemble the triorganotins in their toxicity, but effects are often less and delayed. This has been explained by a conversion of tetra- into triorganotin compounds in the liver, or in the mucosa of the intestinal tract.
Target organs and organ systems of organotin compounds are the central nervous system (CNS), the liver and bile duct, the immune system and the skin.
There are few toxicological data on mono-alkyltins but they appear to be the least significant of the organotins with respect to potential health effects.
A number of dialkyltins have a marked selective effect on the immune system of rats. Dioctyltin and dibutyltin compounds in particular induce a dose-related decrease in the weight of the thymus, spleen and lymph nodes. The reduction in lymphoid organ weights is associated with depletion of lymphocytes from the thymus and thymus-dependent areas of spleen and lymph nodes. The thymic cortex is depleted of lymphocytes without signs of overt cell destruction. Several immune function studies have shown that the cell-mediated immune responses and T-cell dependent humoral activity are suppressed.
Recent studies have revealed that some di- and triorganotin compounds share immunotoxic properties, which are found to be the most sensitive criteria of their toxicity. For instance, the characteristic toxic effect of TBTO is on the immune system, due to effects on the thymus, the cell-mediated function is impaired. The mechanism of action is unknown, but may involve the metabolic conversion to dibutyltin compounds. Non specific resistance is also affected.
There are also some indications that trimethyltin and triethyltin may have immunotoxic potential but this is probably overshadowed by their potent neurotoxicity. Trimethyltin induces pathological changes in the limbic brain, and in particular the hippocampus, of rodents. Triethyltin induces interstitial oedema of the white matter of the brain and spinal cord leading to myelinopathy. These compounds also appear to be neurotoxic in man, the reversibility of the effects being dependent on the severity of the damage that was induced (Hunt and Wilkinson, 1990; IPCS, 1990; Penninks, 1985; Snoeij, 1987).

Long-term studies with lowest NO(A)ELs or LO(A)ELs for TPT and TBT are described below.

Groups of 70 rats/sex received a diet containing 0, 5, 20 or 80 mg.kg^{-1} TPTH for 104 weeks. Observations were made for clinical signs, food consumption, body weight, ophthalmoscopy, haematology, clinical chemistry, urinalysis, organ weights, macroscopy and histopathology. Mortality was increased (dose-related) in all treated females. The survival was 75%, 51%, 36% and 23%, respectively. Preceding mortality clinical signs were ruffled fur, reduced activity, ataxia, stiff gait and hunched posture. The following changes in immunoglobulin levels were observed only after 50 weeks: IgG1 levels were reduced for all treated females and 80 mg.kg^{-1} males, IgG2a levels were reduced for all treated females and IgG2c levels were reduced for all treated males. Effects on other parameters were found at higher dose levels. Because mortality was increased in females and immunoglobulins were decreased at the lowest dose level of 5 mg TPTH.kg^{-1} diet (equal to 0.3 (males) and 0.4 (females) mg TPTH.kg^{-1} bw.day^{-1} no NO(A)EL could be established (JMPR, 1992).
Lowest NOAEL was found in a embryotoxicity study on rabbits (22 rats, 13 d, TPTH by

gavage). Observations were made for clinical signs, body weight and food consumption. A dose-related decrease in mean body weight gain and food consumption was observed at 0.3 and 0.9 mg TPTH.kg^{-1} bw.day^{-1}. The NO(A)EL for maternal toxicity was 0.1 mg TPTH.kg^{-1} bw.day^{-1}.

Wester et al. (1990: cited in IPCS (1990)) carried out a 106-week toxicity and carcinogenicity study with groups of 50 weanling Wistar rats of each sex. An additional group of 10 rats was used for an interim sacrifice after 1 year. TBTO was fed at 0, 0.5, 5, or 50 mg TBTO.kg^{-1} diet (equivalent to 0, 0.025, 0.25, or 2.5 mg TBTO.kg^{-1} body weight). Increased food consumption occurred in all treated males (not clearly dose-related), and there was increased water consumption in males at 5 and 50 mg.kg^{-1}. Haematological changes were noted mainly at the high-dose level. Serum IgM and IgA levels increased, while the IgG level decreased (females). No effect was observed on circulating concentrations of T_4, free T_4, TSH, LH, FSH, or insulin; only the free T_4:T_4 ratio was decreased. Organ weight changes consisted of increased liver, kidney, adrenal, and pituitary weights and decreased thyroid weight. Non-neoplastic histological alterations consisted of a decrease in cell height of the thyroid follicles (at 50 mg.kg^{-1} diet after 1 and 2 years), decrease in splenic iron content (at 5 and 50 mg.kg^{-1} after 1 year only), and vacuolation of kidney proximal tubular epithelium and nephrosis (at 50 mg.kg^{-1} after 2 years only).
General effects on the lymphoid organs were not recorded, though several specific tests of immune function were performed. A dose-related decrease in resistance to *T. spiralis* infection was seen at 5 and 16 months, achieving statistical significance ($p < 0.05$) at 5 and 50 mg.kg^{-1} diet. It would appear that 0.5 mg.kg^{-1} diet (0.025 mg.kg^{-1} bw.day^{-1}) is the NOEL. In all other studies a concentration of 5 mg TBTO.kg^{-1} per day in the diet (equivalent to 0.5 mg.kg^{-1} bw.day^{-1}, based on the short term studies) was the NOEL with respect to general, as well as specific, effects on the immune system (IPCS, 1990).

Subacute and (sub)chronic toxicity (long-term exposure): human data

Exposure by inhalation
Inorganic tin compounds
Inhalation of elemental tin does not produce any effects in man, whereas extended occupational exposure to tin (IV) oxide dust and fumes (no quantitative datat) can produce a benign pneumoconiosis termed stannosis. This condition develops after at least 3-5 years of exposure and is characterized by small dense shadows in the pulmonary X-ray picture without impairment of pulmonary function. No cases of pneumoconiosis were observed in 10 years, after the dust concentration had been reduced to 10 mg.m^{-3}. An important feature of stannosis is that fibrosis of the lung does not develop, providing that other agents such as silica are not present (IPCS, 1980).

Organotin compounds
Paint sprayers (female), working with a latex paint with TBTO fungicide, experienced at first irritation of the nasal mucosa and the conjunctivae. The exposure continued for another fortnight during which the symptoms and signs became more severe, and included bleeding from the nose and mucous discharges. Examination revealed rhinitis with distinct hyperaemia and haemorrhages of the nasal septum. The workers reported that the symptoms were less severe during weekends. When the addition of the fungicidal solution to the paint was abandoned, the symptoms disappeared. In another study, identical

symptoms were found. Measurements performed later indicated that the concentrations of tin in the breathing were below 0.05 $mg.m^{-3}$ air (IPCS, 1980).

A worker engaged in the manufacture of butyltin compounds was reported to suffer from a reduced sense of smell. It was first observed after an exposure period of 16 months, and a further deterioration of the olfactory sense was established during the following 8 months. The state persisted without any noted improvement for 2 years. Other reported symptoms were headaches in the occipital region, nasal haemorrhages, lassitude and a feeling of stiffness in the shoulders (IPCS, 1990).
Seventy percent of the workers in a rubber factory using TBTO in the vulcanization process reported irritation of the upper respiratory tract (and eyes). About 20% also experienced lower chest symptoms (irritation, tightness, and pain), but in all cases pulmonary function was unaffected. The extent of the exposure was not recorded (IPCS, 1990).

Oral exposure
Inorganic tin compounds
Packaged military rations were fed to 9 young male adult volunteers for successive 24-day periods. The average (inorganic) tin content of a control fresh diet was 13 $mg.kg^{-1}$ (in dry solids), while C-rations stored at 1°C contained an average concentration of 33 $mg.kg^{-1}$, and rations stored at 37°C contained an average 204 $mg.kg^{-1}$. All the tin ingested was accounted for by faecal elimination. No toxic effects were noted (Calloway and McMullen, 1966 (cited in IPCS, 1980)). In other comparable studies, also no toxic effects were found (IPCS, 1980).

Organotin compounds
The hazard associated with the use of organotin compounds was unmasked by an episode of intoxication in 1954 involving over 200 cases, 100 of which were fatal. The cause was the ingestion of an oral preparation containing diethyltin diiodide at 15 mg per capsule. It was suggested, however, that ethyltin triiodide, triethyltin iodide, and tetraethyltin were present as impurities. Predominant symptoms and signs included severe headaches, nausea and vomiting, visual and psychological disturbances and sometimes loss of consciousness. At autopsies and decompressive surgery, cerebral oedema of the white matter was found. In many cases, symptoms lasted for at least 4 years; follow-up information on the subjects involved is not available. The lethal dose of the preparation was in some instances only 25 capsules taken during one week. Ingestion of 3 capsules was enough to cause intoxication in a 9-year-old child. It must be noted, however, that (based on the number of capsules sold) the majority of the capsules were consumed without any known adverse effects (IPCS, 1980).

Reproductive (and developmental) toxicity: animal data

Exposure by inhalation
No animal data on reproductive (and developmental) toxicity of tin and tin compounds in case of inhalatory exposure are available.

Oral exposure
Inorganic tin compounds
Testicular degeneration was observed in rats administered 10 mg stannous chloride per kg in the feed for 13 weeks (WHO, 1993 in press). Stannous chloride with casein in an

aqueous medium at dose levels of 0, 200, 400 or 800 mg.kg^{-1} feed did not affect the reproductive performance of rats, although a transient anaemia was observed in the offspring prior to weaning (WHO, 1993 in press).
The teratogenic potential of inorganic tin was evaluated in mice, rats and golden hamsters. Stannous chloride was orally administered in doses of 0, 0.5, 2.3, 11 or 50 mg.kg^{-1} body weight for 10 consecutive days (day 6 though day 15 of gestation) (0, 0.3, 1.4, 6.8 and 32 mg Sn.kg^{-1} bw.day^{-1}) in pregnant mice and rats and for 5 consecutive days (days 6 through 10) in pregnant hamsters. No teratogenic or fetotoxic effects were found (JECFA, 1982a).
Theuer et al. (1971: cited in IPCS, 1980) gave groups of pregnant rats sodium pentafluorostannite, sodium pentachlorostannite, and tin (II) fluoride corresponding to tin levels in the diet of 125, 250, and 500 mg.kg^{-1} (corresponding to approximately 12.5, 25 and 50 mg Sn.kg^{-1} bw.day^{-1} (calculated)). The rats were killed on day 20 of gestation. No effects were seen in the fetuses; tin concentrations in the fetuses were about 1 mg.kg^{-1} compared with approximately 0.65 mg.kg^{-1} in control fetuses, indicating a rather low transplacental transfer.
There was no evidence of embryotoxicity of inorganic tin compounds in animals.

Organotin compounds
Most embryotoxicity and teratogenicity studies have been performed on tricyclohexyltin hydroxide, triphenyltin compounds and tributyltin oxide.

Most data on tricyclohexyltin hydroxide (cyhexatin) concern reproductive and developmental toxicity.
The results of rabbit oral teratogenicity studies available to JMPR (1990, meeting in 1989) were discrepant. In two cases, the results were negative with respect to all parameters measured, with NOAELs of 3 and 1 mg.kg^{-1} bw.day^{-1} (top doses). In a third study, the NOAEL was 0.5 mg.kg^{-1} bw.day^{-1} based on increased post-implantation losses. Hydrocephalus was observed in 8 pups from 4 litters at 3 mg.kg^{-1} bw.day^{-1} (which could have been induced by infection). This study is of uncertain validity because of possible non-homogeneity in the test material. An other study in which maternal toxicity, increased abortions hydrocephalus were found at the lowest test level (0.75 mg.kg^{-1} bw.day^{-1}) again the possibility of infection could not be ruled out (JMPR, 1990).
A recent rabbit teratology study (0, 0.75, 1.5 and 3 mg.kg^{-1} bw.d^{-1} for days 6-19 of gestation) indicated maternal toxicity, pre- and post-implantation losses, fetotoxicity, and reduction in litter size which severity appeared to be related to the product particle size, a smaller particle size resulting in increased toxicity. A high incidence of unilateral or bilateral folded retinas (exceeding the control range) was noted at the lowest dose tested (0.75 mg.kg^{-1} bw.day^{-1}); the significance of this finding was uncertain. An increase in the occurrence of dilation of the third and/or lateral ventricle of the brain and increased incidences of 12/13 ribs and thickened ribs were noted at 3.0 mg.kg^{-1} bw.day^{-1}. There was no evidence of hydrocephaly at 0.75 mg.kg^{-1} bw.day^{-1} (JMPR, 1992). The LOAEL was therefore 0.75 mg.kg^{-1} bw.day^{-1}.
Two reproductive studies of tricyclohexyltin hydroxide in rats were available. The first study utilized doses of 0, 0.1, 0.5 or 6 mg.kg^{-1} bw.day^{-1} in a two-generation study with 1 or 2 litter per generation. Reproductive parameters were unaffected, except for reduced post-natal pup weight gain at 6 mg.kg^{-1} bw.day^{-1}. There was no evidence of induced abnormal development of pups *in utero*. The NOAEL in this study was 0.1 mg.kg^{-1} bw.day^{-1}, with decreased weight gain occurring in females at 0.5 mg.kg^{-1} bw.day^{-1}. The second study, utilizing dietary concentrations of 0, 10, 30 or 100 mg.kg^{-1}, which

incorporated a teratology component, indicated a NOAEL of 10 mg.kg^{-1} diet, equivalent to 0.5 mg.kg^{-1} bw.day^{-1}. Decreased body-weight gain in pups during lactation and reduced pup survival in F_0-F_{1a} offspring were observed at 1.5 mg.kg^{-1} bw.day^{-1}. There was no evidence of compound-induced developmental abnormalities (JMPR, 1992).

In several teratogenicity studies with rats, hamsters, and rabbits, TPTA or TPTH caused maternal and embryotoxicity, but irreversible structural effects were not observed. In rabbits, the most sensitive species, the NOAEL for embryo/fetotoxicity was 0.3 mg TPTH.kg^{-1} bw.day^{-1} in a study that utilized doses of 0, 0.1, 0.3 or 0.9 mg TPTH.kg^{-1} bw.day^{-1}. In this study the NOAEL for maternal toxicity was 0.1 mg TPTH.kg^{-1} bw.day^{-1} (see also table 5.4).

The potential embryotoxicity of TBTO has been evaluated in three mammalian species (mouse, rat, and rabbit) after oral dosing of the mother. The main malformation noted in rat and mouse fetuses was cleft palate, but this occurred at dosages overtly toxic to the mothers. These results are not considered to be indicative of teratogenic effects of TBTO at doses below those producing maternal toxicity. The lowest NOAEL, with regard to embryotoxicity and fetotoxicity for all three species, was 1.0 mg TBTO.kg^{-1} body weight per day (IPCS, 1990).

Reproductive (and developmental) toxicity: human data
No human data on reproductive (and developmental) toxicity of tin and tin compounds in case of inhalatory, oral or dermal exposure are available.

5.1.3 Genotoxicity and carcinogenicity

Genotoxicity

Inorganic tin compounds
Stannous chloride was found not to be mutagenic in a rec-assay in *Bacillus subtilus* (WHO, 1993 in press). Other data on genotoxicity of inorganic tin are not available (IPCS, 1980).

Organotin compounds
Diorganotin compounds:
Dioctyltin dichloride (DOTC) gave negative results in the Ames test and for induction of unscheduled DNA synthesis in primary cultures of rat hepatocytes. Westendorf et al. (1986) reported that DOTC clearly exhibited genotoxic activities in V79 Chinese hamster cells. It was also reported that covalent interactions of DOTC to purify DNA of V79 cells occurred. Sagelsdorff et al. (1990), however, found no evidence for a covalent binding of DOTC in *in vitro* and *in vivo* tests (after administration by oral gavage to male and female rats). According to the authors the study gave no indication for genotoxic activity mediated by DNA binding. They also referred to a number of confidential negative short-term tests for mutagenicity. No evidence of mutagenicity was found for dibutyltin diacetate in the Ames test. Dibutyltin dichloride and dioctyltin dichloride have been reported to be positive in mammalian cell mutation assays *in vitro* in the absence of metabolic activation, and dibutyltin sulphide increased the incidence of chromosomal aberrations in rat bone marrow cells *in vivo* (WHO, 1993 in press).

Triorganotin compounds:
Trimethyltin may have spindle inhibiting properties. Human lymphocyte cultures treated with trimethyltin *in vitro* exhibited a reduction in average chromosome length (WHO, 1993 in press).
No data on genotoxicity were available on tricyclohexyltin hydroxide.
A variety of genotoxicity tests have been performed with TPT. Most tests were negative. TPTH and TPTA were negative in the Ames test, but positive results were obtained with TPTH in two mouse lymphoma mutation tests and in two chromosome aberration tests in human lymphocytes *in vitro*. A number *in vivo* studies for chromosome aberrations (a micronucleus test in mice, a cytogenetic test in chinese hamsters and dominant lethal assays in mice and rats) were negative. Therefore, it is concluded that TPT has genotoxic properties *in vitro*, which are not expressed or detectable in current *in vivo* assays (JMPR, 1992).
A variety of genotoxicity tests have been performed with TBTO. TBTO has negative results in bacterial and yeast mutagenicity tests. In mammalian cells *in vitro*, TBTO gave negative results for induction of point mutations and sister chromatid exchange, but chromosomal aberrations were induced in Chinese hamster ovary cells in the presence of S9. Mice given oral doses failed to show an increased incidence of micronuclei in bone marrow polychromatic erythrocytes (WHO, 1993 in press). IPCS (1990) states that negative results were obtained in the vast majority of studies, and there is no convincing evidence that TBTO has any mutagenic potential.

Carcinogenicity

Animal data
Exposure by inhalation
No animal data on carcinogenicity of inorganic tin and organotin compounds in case of inhalatory exposure are available.

Oral application: Inorganic tin compounds
Few reports are available concerning the carcinogenicity of inorganic tin compounds.
In an oral carcinogenicity study with $B6C3F_1$ mice (dose levels 0, 1,000, or 2,000 mg stannous chloride.kg^{-1} food) (0, 90 or 180 mg Sn.kg^{-1} bw.day^{-1}), a dose related significant increase in the incidence of hepatocellular adenomas and/or carcinomas in female mice was found. However, the highest incidence was within the historical range for female $B6C3F_1$ mice. In a study with F-344 rats receiving 0, 1,000, or 2,000 mg stannous chloride.kg^{-1} diet (0, 30 or 60 mg Sn.kg^{-1} bw.day^{-1}) for 105 weeks, no increased tumour incidences were observed (DHHS, 1982 (cited in WHO, 1993 in press)).
Walters and Roe (1965: cited in IPCS (1980)) administered either sodium chlorostannate at 1 or 5 g Sn.l^{-1} (100 or 500 mg Sn.kg bw.day^{-1}) in drinking water or tin (II) oleate at 5 g Sn.kg^{-1} of diet (700 mg Sn.kg^{-1} bw.day^{-1}) to mice, for up to one year; the survivors were then killed. The incidence of lymphomas, hepatomas, or pulmonary adenomas did not increase with any of the regimens.
Roe et al. (1965: cited in IPCS (1980) reported 3 malignant tumours in 30 August rats that survived for 1 year or more on a diet containing sodium chlorostannate at a concentration of 20 g.kg^{-1} (400 mg Sn.kg^{-1} bw.day^{-1}), whereas a control group of 33 rats did not exhibit any case of malignant tumours. The difference was not statistically significant. No tumours were seen in another group of 27 rats surviving on a diet containing tin (II) 2-ethylhexoate at a concentration of 5-10 g.kg^{-1} (75-150 mg Sn.kg^{-1} bw.day^{-1}).

Administration of tin (II) chloride to rats and mice at 5 mg $Sn.l^{-1}$ (0.5 $mg.kg^{-1}$ $bw.day^{-1}$) in drinking water throughout their life-time did not produce any increase in the incidence of tumours compared with a control group consisting of an equal number of animals (Kanisawa and Schroeder, 1969 (cited in IPCS, 1980)).
There is no evidence for carcinogenicity of inorganic tin compounds after oral application to animals.

Oral application: Organotin compounds

Diorganotin compounds:
F-344 rats and $B6C3F_1$ mice were fed diets containing dibutyltin diacetate at 66.5 or 133 $mg.kg^{-1}$ $bw.day^{-1}$ (rats) and 76 or 152 $mg.kg^{-1}$ $bw.day^{-1}$ (mice) for 78 weeks. Non-significant increased incidences of hepatocellular adenomas in female mice and both hepatocellular adenomas and carcinomas in male mice were noted (NCI, 1979 (cited in WHO, 1993 in press)).
Rats were administered a mixture of octyltin trichloride and dioctyltin dichloride in the diet at doses equivalent to approximately 0.26, 0.74, 2.3, or 6 $mg.kg^{-1}$ $bw.day^{-1}$ for 2 years. A highly significant increased frequency of primary tumours of the thymus, especially thymic lymphomas, was noted in females in the highest dose group. The females also showed an increased incidence of generalized malignant lymphomas, as did the males in the two upper dose groups, although there seemed to be an unusually low incidence of such tumours in the control groups. In animals treated at the lower dose levels, no increase in the incidence of primary thymic tumours or generalized malignant lymphomas was observed. The thymic tumours have been ascribed to dioctyltin compounds (US EPA, 1988 (cited in WHO, 1993 in press)).

Triorganotin compounds:
Two carcinogenicity studies on tricyclohexyltin hydroxide are available. In a 2-year study on rats receiving tricyclohexyltin hydroxide at concentrations up to 12 $mg.kg^{-1}$ body weight per day the pattern of tumour incidence throughout both the control and test groups appeared to be random and did not suggest a dose-response relationship. In a 2 year study on dogs no indication of carcinogenicity was found (JMPR, 1971 (cited in IPCS, 1980)).

In an 18-month study, mice were given an oral dose (stomach tube) of 0.46 $mg.kg^{-1}$ body weight per day of triphenyltin acetate between the ages of 7 and 28 days. There was no statistically significant increase in tumours compared with a control group (Innes et al., 1969 (cited in IPCS, 1980)).

According to JMPR (1992), two long-term/carcinogenicity studies in mice and one in rats were performed for TPTH in which there was no evidence of carcinogenicity. However, there were indications in each of these studies that the doses received by the animals may have been lower than intended owing to instability of the test compound in the diet.
In a third long-term/carcinogenicity study (80 weeks) in mice at dietary concentrations of TPTH of 0, 5, 20 or 80 $mg.kg^{-1}$, increased liver and decreased kidney weights, increased nodular hyperplasia and hepatocellular adenoma and carcinoma occurred at 80 $mg.kg^{-1}$ diet only. Based on decreased body-weight gain at 20 $mg.kg^{-1}$ diet, a NOAEL has been established at 5 $mg.kg^{-1}$, equal to 1 $mg.kg^{-1}$ $bw.day^{-1}$ (JMPR, 1992).

In a second long-term/carcinogenicity study in rats (104 weeks) (with dietary concentrations of TPTH of 0, 5, 20 or 80 $mg.kg^{-1}$), an increase in mortality was observed at all dose levels. The incidence of pituitary adenomas was increased in females at 20 and 80 $mg.kg^{-1}$ diet. At 80 $mg.kg^{-1}$ diet, the incidence of Leydig cell tumours was increased. These changes were accompanied by non-neoplastic lesions in the pituitary and the testes (JMPR, 1992). Because of the increased mortality and decreased serum immunoglobulins, a NOAEL could not be established at the lowest concentration of 5 $mg.kg^{-1}$, equal to 0.3 and 0.4 $mg.kg^{-1}$ $bw.day^{-1}$ for males and females respectively (see also table 5.4).

Wester et al. (1990: cited in IPCS (1990)) reported the result of a 106-week study on carcinogenicity in Wistar rats at dietary TBTO doses of 0, 0.5, 5, and 50 $mg.kg^{-1}$ (equivalent to 0, 0.025, 0.25, or 2.5 mg $TBTO.kg^{-1}$ body weight per day). At the highest dose level, general toxicological effects were present. The incidence of benign tumours of the pituitary (mainly prolactinomas) was elevated at 0.025 and 2.5 $mg.kg^{-1}$ $bw.day^{-1}$, but not at 0.25 $mg.kg^{-1}$ $bw.day^{-1}$, for both sexes. At 2.5 $mg.kg^{-1}$ $bw.day^{-1}$, a significant increase was noted in the incidence of adrenal medullary tumours (pheochromocytomas) in both sexes and of parathyroid adenomas in male animals, while the incidence of adrenal cortical tumours was significantly decreased at 0.025 and 0.25 $mg.kg^{-1}$ $bw.day^{-1}$ in males only. Isolated occurrence of pancreatic carcinoma was found in treated female rats. These were not considered to be compound related since there was no dose dependency and the incidence rates were low.
The pituitary tumours found at 0.025 $mg.kg^{-1}$ body weight per day are considered as having no biological significance since there was no dose-response relationship. These tumours types appear usually at high and variable background incidences. The significance is, therefore, questionable. A second study on mice is in progress (IPCS, 1990)

Skin application
No animal data on carcinogenicity of tin and tin compounds in case of skin application are available.

Human data
No human data on carcinogenicity of inorganic tin and organotin compounds in case of inhalatory, oral and dermal exposure are available.

5.2 ECOTOXICITY

Tin is considered to be an essential element; tin deficiency caused growth deformities in mammals (see section 5.1).
Because of the higher toxicity of some organo-metallic tin compounds compared with inorganic forms of tin, a distinction is made between these two groups.

5.2.1 Transformation, bioaccumulation and biomagnification

Transformation
Crustaceans, molluscs, fish and mammals are able to biodegrade organotin compounds. Tributyltin compounds are degraded to di- and monobutyltin compounds and finally to inorganic tin. The biodegradation rate, however, is low (Annema, 1988). This counts especially for oysters and mussels, which results in higher bioconcentration factors in these species (Lee, 1991).

Bioaccumulation
Organotin compounds are found to bioaccumulate. Bioconcentration factors (BCF: ratio of the Sn-concentration in the organism in $mg.kg^{-1}$ (wet weight) divided by the Sn-concentration in water in $mg.l^{-1}$) are presented in table 5.5.

Table 5.5 Bioconcentration factors (BCFs) as derived from laboratory and field observations in different taxonomic groups

Compound	BCF	Species	Reference
Inorganic tin	-	-	-
Trimethyltin	0.5-90	Algae	Annema, 1988
Tributyltin	100-30,000	Bacteria	IPCS, 1990
	5,500-30,000	Algae	IPCS, 1990
	1,000-63,000	Molluscs	Evers et al., 1993 in press
	100-2,600	Fish	Evers et al., 1993 in press
Triphenyltin	400->3,000	Fish	Crijns et al., 1992

- : no data available

Bioconcentration factors of organotin compounds vary considerably and are often much higher than predicted from the K_{ow}. These differences in observed values may be due to differences in species metabolism and elimination rates, to differences in exposure concentrations, but also to the fact that processes other than partition play a role as well, such as a bond of tin compounds to proteins like S, O or N containing ligands.

The highest bioconcentration factors have been found in the field. This may reflect the long period of time required for reaching a steady state equilibrium (Laughlin, 1990) but may also indicate the influence of food: contamination via food was more important than via the water for marine molluscs (IPCS, 1990).

Biomagnification

Bioaccumulation in mussels and crabs was greater if TBT contaminated phytoplankton and artemia was used as a food organism, respectively (Laughlin, 1988). Contamination via food was more important than via the water (IPCS, 1990). There is, however, no evidence for biomagnification.

5.2.2 Toxicity to aquatic organisms

Inorganic tin

Tin can have two oxidation states: Sn(II) and Sn(IV). In the environment probably only Sn(IV) occurs (Bockting et al., 1992).

The acute and chronic toxicity of inorganic tin to freshwater organisms are presented in table 5.6.

Table 5.6 Acute and chronic toxicity data on inorganic tin (II) and tin (IV) in $\mu g\ Sn.l^{-1}$ derived from laboratory experiments with various freshwater species (after Wong, 1980; Biesinger, 1972; Kapur and Yadav, 1982; Walsh, 1985, 1987; Martin and Holdich, 1986: quoted in Van de Plassche et al., 1992)

Species	Life stage	Exposure time	Criterion		Result ($\mu g.l^{-1}$)	
Acute toxicity						
Green algae						
S. quadricauda		4 h	prim.prod.	(EC50)	23,000	**
A. falcatus		4 h	prim.prod.	(EC50)	8,800	*
		4 h	prim.prod.	(EC50)	5,500	**
Crustaceans						
D. magna	<24 h	48 h	immobility	(EC50)	42,000	*
C. pseudogracilis		96 h	mortality	(LC50)	50,100	*
Chronic toxicity						
Green algae						
A. falcatus		8 d	growth	(EC50)	7,500	*
		8 d	growth	(EC50)	900	**
S. costatum ***		3 d	growth	(EC50)	290	*
T. pseudodonata ***		3 d	growth	(EC50)	320	*
Crustaceans						
D. magna	<24 h	21 d	reproduction	(NOEC)	180	*
Fish						
C. carpio	eggs	?	hatchability	(NOEC)	7,800	*

* tin (II): chemical form $SnCl_2$
** tin (IV): chemical form $SnCl_4$
*** marine species

Toxicity data are scarce: acute toxicity data are available for algae and crustaceans only, whereas chronic toxicity data also includes information on the effects on a fish species. Although tin (IV) seems to be more toxic than tin (II) the data are too limited to draw conclusions. Crustaceans show to be more susceptible to inorganic tin than algae or fish. The data are too scarce to draw conclusions about differences in sensitivity between freshwater and marine species, although toxicity data on saltwater algae are lower than those derived from freshwater algae.

No data are available on sediment toxicity.

Triphenyl tin

Acute and chronic toxicity of triphenyl tin to freshwater and marine organisms are presented in table 5.7.

Table 5.7 Lowest acute and chronic toxicity data on triphenyltin compounds in μg TPT compound.l^{-1} derived from laboratory experiments with various aquatic species (after Steinhäuser, 1985; Woolins, 1984; Meinema, 1986; Eureco, 1990; Tooby, 1975; Walsh, 1985; Harada, 1984; AQUIRE, 1991; Wong, 1982; De Vries, 1991; Hall, 1985: quoted in Crijns et al., 1992)

Species	Compound	Exposure time	Criterion		Result (μg.l^{-1})
Acute toxicity					
Bacteria					
Ph. phosphoreum	TPTC	30 min	luminescence	(EC50)	16
Algae					
S. costatum *	TPTH	3 d	growth	(EC50)	0.6
Crustaceans					
G. fasciatus	TPTH	4 d	mortality	(LC50)	66
D. magna	TPTH	2 d	immobility	(EC50)	11
N. spinides *	TPTF	4 d	mortality	(LC50)	8
Molluscs					
B. nasutus	TPTA	1 d	mortality	(LC50)	12
B. glabarata	TPTA	1 d	mortality	(LC50)	660
Worms					
T. tubifex	TPTC	2 d	mortality	(LC50)	70
Insects					
Ch. riparius	TPTA	?	mortality	(LC50)	50
Fish					
S. gairdneri	TPTH	4 d	mortality	(LC50)	15
A. alburnus *	TPTF	4 d	mortality	(LC50)	400
(Sub)chronic toxicity					
Green algae					
A. falcatus	TPTC	8 d	growth	(EC50)	2
S. quadricauda	TPTC	8 d	growth	(EC50)	40
Molluscs					
L. stagnalis	TPTH	9 d	mortality	(LC100)	10
Fish					
O. mykiss	TPTC	120 d	growth	(NOEC)	0.05

* : marine species

Most data refer to acute toxicity and are expressed as L(E)C50-values. The acute toxicity ranges from 0.6 to 660 μg TPT.l^{-1} and is between 10 and 100 μg TPT.l^{-1} for most species. The only available chronic NOEC-value has been determined for fish (0.05 μg TPT.l^{-1}). All data are derived from Crijns et al. (1992); it should be noted that these authors included the lowest EC50 and NOEC reported only.
The data are too scarce to draw conclusions about differences in sensitivity between freshwater and marine species: triphenyl tin shows to be more toxic to marine algae than to freshwater algae, but the opposite is found for fish.

No data are available on sediment toxicity.

Tributyl tin

Toxicity data of tributyl tin to aquatic organisms derived from laboratory studies are presented in table 5.8. Most data concern marine species.

Table 5.8 Lowest long-term toxicity data on tributyltin compounds in μg TBT compound.l^{-1} derived from laboratory experiments with various aquatic species (Dooley, 1987; Walsh, 1985; Matthijsen, 1989; Bushong, 1990; Davidson, 1986; Beaumont, 1984; Valkirs, 1987; Roberts, 1987; Walsh, 1986; Vries, 1991: quoted in Evers et al., 1993 in press. His and Robert, 1985; Gibbs et al., 1987: quoted in IPCS, 1990)

Species	Exposure time	Criterion		Result (μg.l^{-1})
Bacteria				
P. phosphoreum *	0.01 d	growth	(EC50)	0.02
Algae				
S. costatum *	3 d	growth	(EC50)	0.36
Crustaceans				
D. magna	20 d	repro, growth	(NOEC)	0.56
A. tonsa *	6 d	?	(NOEC)	0.10[1)]
A. sculpta *	63 d	repro	(NOEC)	0.09
Molluscs				
L. stagnalis	33 d	growth	(NOEC)	0.32
C. gigas *		growth	(NOEC)	0.02
M. edulis *	15 d	mortality	(LC50)	0.10
C. virginica *	66 d	growth	(NOEC)	0.04
M. mercenaria *	5 d	growth	(EC)	0.05
N. lapillas *	360 d	imposex	(NOEC)	0.001
Worms				
N. arenaceodentata *	70 d	repro, growth, mortality?	(NOEC)	0.05
Fish				
S. gairdneri	110 d	mortality	(NOEC)	0.04

* : marine species
1) : derived from 11% . NOEC of 0.012 (Bushong, 1990 (cited in Evers et al., 1993 in press))

The data presented only refer to long-term toxicity since sufficient data are available. A large body of data exists on the effects of TBT on marine molluscs. Recently special attention is paid to the induction of imposex: the development of male characteristics in

female gastropods. The females develop a penis and ultimately become infertile. Imposex has been observed at exposure levels as low as of 0.0025 μg TBT.l^{-1}, probably extrapolated, because very close to detection limit (Stickle, 1990, quoted in Evers et al., 1993 in press). According to IPCS (1990) the NOEC for the development of imposex is less than 0.001 μg TBT.l^{-1}, based on observations of Bryan et al. (1986) and Gibbs et al. (1988) on *Nucella lapillas*.

In table 5.9 results of available microcosm studies are presented. These studies indicate that the most sensitive species are affected by TBT at concentrations greater than 0.05 μg TBT.l^{-1}.

Table 5.9 Effects of tributyl tin in freshwater and marine microcosm (after IPCS, 1990 (references 1-5) and Evers et al., 1993 in press (reference 6))

Exposure time	Concentration (μg TBT.l^{-1})	Number of species	Effects	Reference
14 days	0.12; 1.2 *	6	More than 50% mortality at the lowest dose level	1
4-20 days	0.49; 0.2	6	No mortality was observed	2
60 days	0.01-2.52	30	Mortality and reduced number of species, species diversity, settlement and condition. The NOEC = 0.05 μg TBT.l^{-1}	3
60-90 days	3.5-35	2	Partial or total mortality was observed (snail and fish)	4
4 months	0.06-0.17; 1-3	7	Mortality and reduced number of species, species diversity and growth inhibition was observed at 0.06-0.17 μg TBT.l^{-1}	5
?	4.7	>5	Changes in species composition, 100% mortality of daphnids	6

* g.m^{-2} of marsh

1: Jordan (1985) 2: Salzar and Salazar (1985) 3: Henderson (1986)
4: Cardarelli (1973) 5: Beamont et al. (1987) 6: Miniero (1991)

There are many field observations on effects of tributyl tin on marine species, mainly bivalves (during the last decade) and gastropods (during the last two decades) (IPCS, 1990). Tributyl tin has been associated with mortality and failure of settlement of bivalve larvae, reduced growth, shell thickening, and other malformations in oysters (*C. gigas*), imposex in mud snails and imposex concurrent with population decline in dogwhelks (*N. lapillas*). Generally field and laboratory results are in good agreement.
Imposex has been observed in *N. lappilus* in Dutch surface waters as well. In Zealand waters imposex occurred after 7 weeks, the highest prevalence was found in yacht yards. Natural populations show imposex to such a degree that they are will become extinct (Mertens and Van Zwol, quoted in Evers et al., 1993 in press).

Data are available on sediment toxicity. Various experiments indicate that there is a number of species that are exposed to TBT directly from the sediment, such as oligochaetes and molluscs (Evers et al., 1993 in press). According to Langston (1991, quoted in Evers et al., 1993 in press) a concentration of 0.3 mg $Sn.kg^{-1}$ sediment may result into the extinction of mussel populations.
Mathiessen and Thain (1989; quoted in IPCS, 1990) studied the recolonization by marine species in the field of TBT contaminated sediment. They found effects of TBT on the burrowing activity of the polychaete *A. marina* at all concentration levels tested (0.1 - 100 $mg.kg^{-1}$), but they did not find effects on other species, including molluscs. The authors pointed out that unaffected groups were associated with the surface sediment (which is removed by the water movement and replaced by uncontaminated sediment), whereas the affected species are exposed to deeper layers of TBT contaminated sediment.

5.2.3 Terrestrial species

Inorganic tin
Available data are limited to effects on soil enzyme activities (acid phosphatase, alkaline phosphatase, arylsulphatase, ureas) and microbe-mediated processes (soil respiration, N-mineralization, nitrification). In most cases the NOEC are likely more than 100 mg $Sn.kg^{-1}$ (standard soil). NOEC value in the range of 10 - 100 mg $Sn.kg^{-1}$ may be found for urease inhibition, whereas for inhibition of soil respiration the NOEC could be less than 10 mg $Sn.kg^{-1}$. However, in most studies only one or two test concentrations were used and no distinct concentration-effect relationship could be distinguished. Therefore these data were found to be not suitable for deriving a maximum acceptable concentration (Van de Plassche et al., 1992).

Triphenyl tin
No toxicity data on soil organisms are available.

Tributyl tin
Data on the toxicity of tributyl tin to terrestrial organisms is limited.
TBT-compounds are toxic to insects exposed either topically or via feeding on treated wood. The LD50 of TBTO for *Anobium punctatum* was 0.254 $kg.m^{-3}$ wood (Baker and Taylor, 1967; quoted in IPCS, 1990). Treatment of wood with TBTO at 1.9 $kg.m^{-3}$ resulted into a high mortality in bee colonies over winter (Kalnins and Detroy, 1984; quoted in IPCS, 1990).
In a terrestrial microcosm study no effects were observed among 7 endogenous soil species after exposure to 167 mg $TBTO.cm^{-3}$ of soil for 77 days (Gile et al., 1982; quoted in IPCS, 1990).

5.3 TOXICOLOGICAL LIMIT VALUES

5.3.1 Humans

There is evidence that tin is essential for the normal growth of rats. It was suggested that tin could have a function on the active site of some metal-depending enzymes. No evidence exists, however, that tin is essential for other species including man.

Inorganic tin compounds

Oral exposure:
Metallic tin and inorganic tin compounds are of low toxicity to animal species. This is mainly due to its low absorption, low tissue accumulation, and rapid excretion primarily in the faeces. There was no evidence for embryotoxicity or carcinogenicity of inorganic tin compounds in animals. There were no data on genotoxicity, except for one negative rec-assay in bacteria.
In humans nausea, vomiting, diarrhoea, stomach cramps, fatigue and headache were observed following the consumption of canned products with tin concentrations as low as 150-250 mg $Sn.kg^{-1}$. In contrast, no toxic effects were noted in other cases involving foodstuffs containing up to 700 mg $Sn.kg^{-1}$ for periods of 6-30 days. There is no evidence of adverse effects in humans associated with chronic exposure (exposure levels not mentioned) to tin.

The "Joint FAO/WHO Expert Committee on Food additives" stated in 1982 that the threshold concentration for effects in humans is about 200 mg $Sn.kg^{-1}$ in the food. On the basis of this information a provisional maximum tolerable daily intake of 2 $mg.kg^{-1}$ bw (includes food additive use of stannous chloride) was established. In 1989 the committee converted this TDI into a PTWI of 14 mg $Sn.kg^{-1}$ bw and emphasized that this value was applicable to chronic tin exposure. No further details of the derivation of the TDI or PTWI were given by the JECFA.

Inhalatory exposure:
The available data are far too limited to derive a toxicological limit value for inhalatory exposure.

Organotin compounds

The toxicity of the organotin compounds is essentially determined by the number and nature of the organic substituents. In general, the toxicity to mammals decreases from tri- to mono-organotin compounds. The tetraorganotin compounds resemble the triorganotins in their toxicity, but effects are often less and delayed. This has been explained by a conversion of tetra- into triorganotin compounds.

A number of the dialkyltin have a marked selective effect on the immune system, especially on t-lymphocytes of rats. Dioctyltin and dibutyltin compounds in particular induce a dose-related decrease in the weights of thymus, spleen and lymph nodes. Immunotoxic and renal effects have also been described for some of the trialkyltins in rats. Thymus weight reduction was found in rats fed tributyltin and triphenyltin. Trimethyltin and triethyltin may also be immunotoxic but this is overshadowed by their potent neurotoxicity in rodents. These compounds also appear neurotoxic in man. Several of the organotins have been shown to be hepatotoxic in rodents, the bile duct being particularly affected. Changes in the liver occurred at or above dose levels that resulted in immunodeficiency. Genotoxicity and carcinogenicity data are only available for a small number of the organotins. Although the majority of the tests have been proved negative the overall picture is unclear as some individual compounds have given positive or equivocal results in one test or another. Reproductive toxicity is mainly of concern for tributyltin oxide.

For all organotin compounds data on effects after inhalatory exposure were considered to be too limited to derive (tentative) toxicological limit values. For two organotin compounds (TBTO and triphenyltin hydroxide) a NOAEL for inhalatory exposure was available.

With respect to oral exposure (tentative) toxicological limit values were established for dioctyltin dichloride (DOTC), tributyltin oxide (TBTO), triphenyltin compounds (fentin, TPT) and tricyclohexyltin compounds (cyhexatin). These compounds will be discussed in more detail. With respect to the other organotin compounds data were considered to be too limited to derive (tentative) toxicological limit values. For dibutyltin dichloride a NOAEL for oral exposure was available.

Di-n-butyltin dichloride - oral exposure

Oral exposure to di-n-butyltin dichloride caused reduction of food intake, depressed growth and mild anaemia at the highest dose group in a 90-day study in rats. The NOAEL was 2 $mg.kg^{-1}$ $bw.day^{-1}$. An oral 56-d study in rats resulted in a NOEL of 1 $mg.kg^{-1}$ $bw.day^{-1}$. No data on embryotoxicity or teratogenicity were available. Di-n-butyltin dichloride was positive in a mammalian cell mutation test *in vitro*.

Dioctyltin dichloride (DOTC) - oral exposure

The characteristic toxic effect of DOTC in experimental animals is the induction of thymus atrophy and immunocompetence. Lymphocyte depletion was observed in the thymus and the thymus-dependent areas of the spleen and lymph nodes. No data on embryotoxicity or teratogenicity were available.

In a long-term oral carcinogenicity study with rats given a mixture of DOTC and mono-n-octyltin trichloride, a significantly increased incidence of thymic lymphomas was seen in females in the highest dose group (6 $mg.kg^{-1}$ $bw.day^{-1}$). There was also a significant increased incidence of generalized malignant lymphomas in males and females in the higher dose groups, although there seemed to be an unusually low incidence of such tumours in the control group. In the lower dose groups no increased tumour incidences were observed, the NOAEL being 0.74 $mg.kg^{-1}$ $bw.day^{-1}$.

With respect to genotoxicity some data were available. DOTC was negative in the Ames test and in a test for induction of unscheduled DNA synthesis. One study reported covalent binding of DOTC to purified DNA and induction of mutations in V79 Chinese hamster cells *in vitro* (Westendorf et al., 1986). In another study there was no evidence for covalent binding to DNA both *in vitro* and *in vivo*. The authors also referred to several confidential negative mutagenicity and genotoxicity tests (Sagelsdorff et al., 1990). This is supported by a variety of confidential **negative** *in vitro* and *in vivo* genotoxicity tests evaluated by the RIVM within the framework of the EEC Scientific Committee on Food, working group packaging materials. It is concluded that there is no evidence for the genotoxicity of DOTC.

On the basis of the 2-year study with rats a tentative toxicological limit value is derived. Assuming that the effects observed can be attributed to DOTC and taking into account that the concentration of DOTC in the mixture tested amounted to 32.5% the NOAEL for DOTC is 32.5/100 x 0.74 = 0.24 $mg.kg^{-1}$ $bw.day^{-1}$. Applying a safety factor of 100 results in a tentative toxicological limit value of 2.4 μg $DOTC.kg^{-1}$ $bw.day^{-1}$.

Tributyltin oxide (TBTO) - oral exposure

The characteristic toxic effect of TBTO in experimental animals is on the immune system; due to effects on the thymus, the cell-mediated function is impaired. The mechanism of action is unknown, but may involve the metabolic conversion to dibutyltin compounds. Non-specific resistance is also affected. Immunotoxic effects have been shown in several animal species at overtly toxic doses. Only rats exhibit signs of effects on the immune system without other overt signs of toxicity and is clearly the most sensitive species. The NOAEL in short-term rat studies was 5 $mg.kg^{-1}$ diet (corresponding to 0.6 $mg.kg^{-1}$ $bw.day^{-1}$).

In humans, there have been no reported cases of poisoning from ingestion of TBTO or other TBT salts.

From a variety of tests there is no evidence for genotoxicity of TBTO. There is also no evidence for carcinogenicity of TBTO. Teratogenic and embryotoxic effects only occur at doses causing maternal toxicity.

In a chronic drinking water study with rats the NOAEL for immunotoxic effects (suppression of the resistance to the nematode *Trichinella spiralis*) was 0.5 mg $TBTO.kg^{-1}$ diet per day (corresponding to 0.025 mg $TBTO.kg^{-1}$ $bw.day^{-1}$). In the Drinking water guidelines (WHO, in press) a safety factor of 100 was applied to this NOAEL, resulting in a TDI of 0.3 μg $TBTO.kg^{-1}$ bw.

This TDI is accepted by the RIVM. It must be noted that with the present knowledge, the effects on host resistance are probably of most relevance in assessing the potential hazard

to man, but that there is insufficient experience in these test systems to fully assess their significance (IPCS, 1990).

Tributyltin oxide (TBTO) - inhalatory exposure
Data on effects of inhalatory exposure to TBTO are limited to one 4-5 week study in rats, with exposure for 5 days a week for 4 hours a day. In this study a NOAEC of 0.16 mg TBTO.m^{-3} was found.

Triphenyltin (Fentin) (TPT) - oral exposure
Toxic effects of fentin in experimental animals include a decrease in food consumption and body weight, increased liver weights, changes in haematological and biochemical parameters as well as effects on the immune system. There was no evidence for teratogenicity. Embryotoxicity only occurred at doses which caused maternal toxicity.
Three carcinogenicity studies (two in mice and one in rats) did not provide any evidence for carcinogenicity. In another study in mice an increased incidence of nodular hyperplasia in the liver and hepatocellular adenoma and carcinoma occurred only at the highest dose group (80 mg.kg^{-1} bw.day^{-1}). At this dose level toxic effects in the liver occurred (marked increased weight, nodules, foci and nodular lesions). In a second study in rats an increased incidence of pituitary adenomas (females) and Leydig cell tumours (males) was observed at the higher dose groups. These tumours were accompanied by non-neoplastic lesions in the pituitary and the testes.
A variety of genotoxicity tests were conducted with fentin. Most *in vitro* tests were negative. However, two mouse lymphoma tests and two chromosome aberrations tests in human lymphocytes *in vitro* were positive. Since a number of different *in vivo* tests (micronucleus test in mice, a cytogenetic test in Chinese hamsters and dominant lethal tests in rats and mice) were negative, it is concluded that fentin has genotoxic properties *in vitro* which are not expressed or detectable in current *in vivo* assays.
Because of the fact that the genotoxic properties of fentin are not expressed or detectable in current tests *in vivo* and the observed correlation between toxicity and carcinogenicity of fentin it is concluded that it is justified to use a threshold extrapolation for risk assessment. The toxicological limit value will be based on animal data.

In 1970 the "Joint FAO/WHO Meeting on Pesticide Residues" (JMPR) established an ADI of 0-0.0005 mg fentin.kg^{-1} bw, based upon a NOAEL of 0.1 mg.kg^{-1} bw.day^{-1} in an earlier long-term study in rats (critical effect decrease in white blood cells). This ADI was applicable to TPTH, TPTA en TPTCl and to the sum of the compounds. In a more recent long-term study in rats (1989) increased mortality was observed at the lowest dose (0.3 mg.kg^{-1} bw.day^{-1}). Applying a safety factor of 500 to this LOAEL would result in approximately the same ADI as previously established. Therefore, in 1991, the previous ADI of 0-0.0005 mg fentin.kg^{-1} bw was retained by the JMPR. This ADI is supported by NOAELs derived from other recent studies. This ADI is accepted by the RIVM.

Triphenyltin hydroxide - inhalatory exposure
Data on effects of inhalatory exposure to TPTH are limited to one 13 week study in rats, with exposure for 5 days a week for 6 hours a day. In this study a NOAEC of 0.05 mg TPTH.m^{-3} was found.

Tricyclohexyltin hydroxide (cyhexatin) - oral exposure
Most data on cyhexatin concern reproductive and developmental toxicity. In two- and three-generation reproduction studies with rats there was no evidence for compound

related reproductive or developmental abnormalities, except for a reduced post-natal weight gain and an increased pup mortality at or above 1.5 mg.kg^{-1} bw.day^{-1}. The NOAELs were 0.1 and 0.5 mg.kg^{-1} bw.day^{-1}, respectively.
In 1989 the JMPR concluded that the results of rabbit oral teratogenicity studies were discrepant. Two studies were negative with respect to all parameters studied, with NOAELs of 3 and 1 mg.kg^{-1} bw.day^{-1}. A third study was considered to be of uncertain validity. In 1991, the results of a recent rabbit study became available (0, 0.75, 1.5 and 3 mg.kg^{-1} bw.day^{-1}). There were increased incidences of 12/13 ribs and thickened ribs at the highest dose group and increased incidences of unilateral or bilateral folded retinas at all dose levels. Therefore, 0.75 mg.kg^{-1} bw.day^{-1} was the LOAEL.
In a 2-year oral study with rats cyhexatin caused reduced body weight gain and increased (relative) spleen and liver weights in mammals as well as mild kidney effects (not specified). There were no indications of carcinogenicity of cyhexatin. In an oral (dated) 2-year study with dogs a NOAEL of 0.75 mg.kg^{-1} bw.day^{-1} was found.
No data on genotoxicity were available.

The JMPR derived in 1970 a "temporary acceptable daily intake for man" of 0-0.0075 mg cyhexatin.kg^{-1} bw, on the basis of a NOAEL of 0.75 mg.kg^{-1} bw.day^{-1} from a long-term study in dogs. In 1989 the ADI was rounded up to 0-0.008 mg.kg^{-1} bw.
In 1991, on the basis of the recent two-generation study in rats, resulting in a NOAEL of 0.1 mg cyhexatin.kg^{-1} bw.day^{-1}, and applying a safety factor of 100, the ADI was adjusted to 0-0.001 mg.kg^{-1} bw. The committee recommended that cyhexatin should be reviewed again in 1994. The adjusted ADI is accepted by the RIVM.

5.3.2 Ecosystems

Inorganic tin

Surface water
Recently Van de Plassche et al. (1992) calculated indicative maximum tolerable concentrations (MTR) for inorganic tin for fresh and marine water separately. This resulted into indicative MTRs of 18 μg Sn.l^{-1} (applying a safety factor of 10 to the lowest chronic NOEC) and 0.29 μg Sn.l^{-1} (applying a safety factor of 1,000 to the lowest acute EC50) for fresh and marine water, respectively. Based on the available data they suggested to use the indicative MTR of 18 μg Sn.l^{-1} for both types of waters. There is no reason to deviate from the proposal of Van de Plassche et al. (1992).
The geometric mean background concentration (total Sn) was found to be 0.002 μg Sn.l^{-1}. Usually the geometric mean is considered indicative for the negligible risk concentration. For tin, however, De Bruin and Denneman (1992) proposed to use the 90% percentile value, being more representative for the background value in the Netherlands: 0.10 μg Sn.l^{-1}. There is no reason to deviate from this proposal.

Sediment
No sediment toxicity data are available. Therefore the maximum tolerable concentration of inorganic tin is derived from that determined for surface water according to the equilibrium partitioning method. The geometric mean of available partition coefficients (log Kp=6.09) as reported by Bockting et al. (1992) is 1,230,000 l.kg^{-1}. This Kp is derived on total metal contents of particulate matter, assuming that Kp's for sediment are roughly 1.5 times lower than for particulate matter. Application of this coefficient to a

maximum acceptable concentration of 18 μg Sn.l^{-1} yields a rather high tentative maximum tolerable risk level in sediment of 22,000 mg Sn.kg^{-1}.
Based on the 90% percentile as the background concentration of tin in surface water (0.10 μg Sn.l^{-1}), the background concentration in sediment is approximately 120 mg Sn.kg^{-1}, applying the before-mentioned partition coefficient.

Soil
The Kp for soil is not available. According to Bockting et al. (1992) adsorption of tin is assumed to be comparable with lead, resulting in a Kp = 1,900 l.kg^{-1}. It should be noted that this value is much lower than the one used for sediment. Application of this coefficient to a maximum acceptable concentration of 18 μg Sn.l^{-1} yields a maximum tentative tolerable level in soil of 34 mg Sn.kg^{-1}.

Triphenyl tin

Surface water
There are no sufficient data (less than four chronic toxicity data) for refined effects assessment. According to the RIVM guidance document (Slooff, 1992) the lowest acute L(E)C50-value for acute toxicity for minimal algae, crustaceans and fish is divided by a factor 100 and, subsequently, is compared to the lowest chronic NOEC-value divided by a factor 10: the lowest one is selected.
For freshwater these values are 0.11 and 0.005 μg TPT.l^{-1}, respectively, resulting in an indicative MTR for fresh water of 0.005 μg TPT.l^{-1}. For marine water only acute data on algae, crustaceans and fish are available, resulting in an indicative MTR of 0.006 μg TPT.l^{-1}. Based on these data 0.005 μg TPT.l^{-1} is tentatively proposed as MTR.

Sediment
No sediment toxicity data are available. Therefore the maximum tolerable concentration of triphenyl tin may be derived from that determined for surface water according to the equilibrium partitioning method. Kp-values determined in the laboratory are in the range of 1,000, whereas higher values are found in the field (Tas, pers. comm.). As stated by Soderquist (1979) sorption of triphenyl tin is complex and depends on organic carbon content, clay content, pH, salt concentration. Quantitative information, however, is lacking and therefore no MTR is derived for sediment.

Soil
Due to lack of information no MTR for soil could be determined.

Tributyl tin

Surface water
Since there are no distinct differences in susceptibility to tributyl tin between freshwater and marine species, a maximum tolerable concentration is derived for both aquatic environments based on the total data set. The short-term toxicity data on bacteria and algae were used as well, taking into account 50% of the reported EC50-values. This results into a data set of 13 values, enabling a refined effects assessment according to Aldenberg and Slob (1991). Applying this extrapolation method a maximum acceptable concentration of 0.003 μg TBT.l^{-1} is calculated.

Sediment

Due to lack of information no MTR for soil could be determined (see also triphenyl tin; however Kp-values are generally higher for tributyl tin). Based on recolonization experiments it can be derived that the MTR should be below 0.1 mg TBT.kg^{-1}.

Soil

Data on the toxicity to terrestrial species are related to the use of TBT as a wood preservative. The only information on effects on soil organisms is available for a one dose tested microcosm and therefore not useful to derive a MTR.

6. EVALUATION

Tin is a naturally occuring element. It has shown to be essential for rats; but no evidence exists, however, that tin is essential for other species.

6.1 RISKS TO HUMANS

6.1.1 Inorganic tin compounds

There is no evidence that inorganic tin produces carcinogenic effects. No data were available on genotoxicity except for one negative rec-assay.

The "Joint FAO/WHO Expert Committee on Food additives" (JECFA) derived in 1989 a provisional tolerable weekly intake (PTWI) of 14 mg $Sn.kg^{-1}$ bw, applicable to chronic tin exposure. It should be noted, however, that this PTWI is derived from acute human data, lacking a sound underpinning. From animal toxicity data (table 5.3) the reliable lower NO(A)ELs range from 150-500 mg $Sn.kg^{-1}$ bw per day. Applying a safety factor of 100, this corresponds with an indicative toxicological limit value of about 10-35 mg $Sn.kg^{-1}$ per week. These figures are in line with the PTWI derived by JECFA.
Inorganic tin naturally occurs in food in drinking water, but most of the tin in the daily diet originates from food in tin-cans. The total daily intake of both inorganic and organic tin as determined from duplicate meals in the Netherlands is less than 0.21 mg per person per day (range <0.09-9.81). Based on a body weight of 70 kg, this corresponds to about 0.02 mg $Sn.kg^{-1}$ bw per week (range of about <0.01-1.0).
Taking into account the factorial difference of 700 (140->1400) the risk of inorganic tin in food and drinking water for humans is considered very small, if present at all.

For inhalatory exposure the available toxicity data are considered to be too limited to derive a toxicological limit value. One 66-year old paper reports that exposure of guinea pigs to 1.35×10^6 μg $Sn.m^{-3}$ for 10 minutes per day for several months produced only transient irritation of the nose and eyes.
No reliable data on the occurrence of tin in the outdoor air in the Netherlands are available, but data from other countries suggest a range of 0.001 - 0.01 $\mu g.m^{-3}$, whereas locally in the vicinity of industry levels of about 0.1 $\mu g.m^{-3}$ may be expected.
Although data are very scarce, the available information indicates that the risk resulting from tin in air for humans is very small.

6.1.2. Organic tin compounds

Genotoxicity and carcinogenicity data are only available for a small number of organotins. Although the majority of the genotoxicity and carcinogenicity tests provided negative results, the overall picture is unclear as some individual compounds have given positive or equivocal results in one test or another.

The oral toxicity of organotin compounds varies with the number and nature of the organic substituents. The lowest (available) NO(A)ELs for the various organotin

compounds range from 0.025 $mg.kg^{-1}$ $bw.day^{-1}$ for tributyltinoxide to 0.24 $mg.kg^{-1}$ $bw.day^{-1}$ for dioctyltin dichloride. For some compounds tentative toxicological limit values, TDIs or ADIs have been derived. In order to estimate the risks these data are converted to tolerable daily intake of Sn, based on conversion factors presented in appendix C: dioctyltin dichloride: 0.00067 $mg.kg^{-1}$ bw; tributyltinoxide: 0.00012 $mg.kg^{-1}$ bw; triphenyltin: 0.00015 $mg.kg^{-1}$ bw; tricyclohexaylin hydroxide: 0.0003 $mg.kg^{-1}$ bw).
Information on human oral exposure to organic tin compounds, however, is lacking. The percentage of organic tin compounds in the total daily intake of tin is probably very low and orginates from leaching from plastic, pesticide residues and accumulation in aquatic food. Based on a body weight of 70 kg, the total daily intake of both inorganic and organic tin compounds is about 0.003 mg $Sn.kg^{-1}$ bw (range of about <0.0015-0.15). Hence, there may be some risks if the organic tin compounds in the total diet is about 1%.

Inhalation toxicity data are restricted to a NOAEC of 0.16 $mg.m^{-3}$ for tributyltin oxide (4-5 wk study in rats, 5 d/wk, 4 h/d) and a NOAEC of 0.05 $mg.m^{-3}$ for triphenyltin hydroxide (13 wk study in rats, 5 d/wk, 6 h/d).
No data on the occurrence of organotin compounds in outdoor or indoor air in the Netherlands are available. Background concentrations probably are very low, but locally during application as anti-fouling materials or fungicides the environmental organotin concentrations may temporarely reach levels that present a risk for humans.

6.2 RISKS TO ECOSYSTEMS

6.2.1 Inorganic tin compounds

For both fresh and marine surface waters an indicative maximum tolerable risk concentration of 18 μg $Sn.l^{-1}$ was derived, whereas a background value of 0.10 μg $Sn.l^{-1}$ was considered to be the negligible risk concentration (section 5.3.2).
No data are available on the occurrence of inorganic tin in Dutch surface water, except for some measurements on background concentrations. No data are available on foreign emissions transported by large border-crossing rivers like the river Rhine and Meuse. From chapter 3 it can be derived that approximately 12-19 tonnes inorganic tin was emitted into Dutch surface waters. Therefore a certain risks for aquatic species cannot be excluded in surface waters in the vicinity of chemical, anodizing and electrotechnical industrial sites.

For sediment an indicative maximum tolerable risk concentration and a negligible risk concentration of 22,000 mg $Sn.kg^{-1}$ and 120 mg $Sn.kg^{-1}$ was derived, respectively (section 5.3.2). Although data on the occurrence of inorganic tin in Dutch surface waters are lacking, the relatively high MTR makes it highly probable that the risks involved are small, if present at all.

For soil a very tentative maximum tolerable risk level of 34 mg $Sn.kg^{-1}$ was derived, based on a much lower Kp value as applied for the sediment. The background levels in the Netherlands range from 7 $mg.kg^{-1}$ for poor soils to 34 $mg.kg^{-1}$ for rich soils.The

available data are insufficient to determine the risks related to elevated inorganic tin concentrations resulting from application of waste water sludge in agriculture.

6.2.2 Organotin compounds

There is sufficient information available to derive (indicative) maximum tolerable concentrations for triphenyl tin and tributyl tin.

Triphenyl tin

Surface water
For triphenyl tin an indicative MTR of 0.005 μg TPT.l^{-1} was proposed for both fresh and marine water, roughly corresponding to 0.0015 μg Sn.l^{-1}. This is a factor 2 below the currrent standard (ceiling value) of 0.01 μg TPT.l^{-1}.
Data on the occurrence of triphenyl tin are restricted to single measurements in fresh water bodies in the Netherlands (table 4.1) and vary from 0 (not prsent or below detection limit[0.001]) to 0.002 μg Sn.l^{-1} in large water bodies. Higher concentrations have been reported to occur in other fresh water bodies (average of values above the detection limit: 0.01-0.26 μg Sn.l^{-1}), though in most samples (78%) the concentrations were found below the detection limit.
Based on these very limited data the risks cannot be quantified. However, the data indicate that the indicative maximum tolerable risk concentration is in the range of the environmental triphenyl tin concentrations. It is therefore very probably that in areas where triphenyl tin is used as agricultural fungicide or anti-fouling paint the aquatic organisms are at risk.

Sediment and soil
Due to a lack of both toxicity data and Kp-values, no MTRs could be derived for sediment or soil. Also no data on the occurrence in soil are available. Concentrations in fresh water sediment range from below detection limit (42% of the samples) to some hundreds or thousands of μg.kg^{-1} dw, much lower concentrations being found in saline water sediments (less than 100 μg.kg^{-1} ww). The risks involved are unknown.

Tributyl tin

Surface water
Toxicity data were sufficient to allow the refined effects assessment, resulting in a maximum tolerable risk level of 0.003 μg TBT.l^{-1}, roughly corresponding to 0.001 μg Sn.l^{-1}. The current standard for TBTO is 0.01 and 0.0001 μg.l^{-1}, ceiling and basic value respectively.
Most data on the occurrence of tributyl tin are based on measurements in yachting harbours. Highest concentrations are found in large yachting harbours with stagnant water. In fresh water harbours the average concentration was 0.441 μg Sn.l^{-1}, in salt water the concentration ranged from below detection level to 1.6 μg Sn.l^{-1}. At other locations the concentration are usually below 0.01 μg Sn.l^{-1}, although levels up to 0.24 μg Sn.l^{-1} have been recorded (Wester Scheldt).
The exposure data are too limited to allow a sound risk evaluation. The concentrations

found in yachting harbours, however, will impose a risk to aquatic species: at the average concentration found in these harbours the percentage of species that is fully protected is 4% only.

Sediment and soil

Information is insufficient to derive a MTR for sediment or soil. Toxicity data indicate that the MTR should be below 100 μg TBT.kg^{-1} in sediment. TBT concentrations in sediments in the Netherlands range from below detection limit (79% of the samples) to some hundreds or thousands of μg.kg^{-1} dw. The risks involved are unknown.

7. RECOMMENDATIONS

Inorganic tin

Available information indicates that the risk of inorganic tin compounds to humans is very small, if present at all. The risks of inorganic tin compounds to aquatic ecosystems are considered to be small and are likely restricted to surface waters in the vicinity of industrial sites. There is uncertainty about the risks of inorganic tin to soil organisms: a very tentative MTR is close to background levels in soil. More information may be required on both the toxicity of inorganic tin to soil organisms and exposure levels, especially in areas where waste water sludge is used. However, the priority for such an effort seems to be low. The yearly amount of inorganic tin in sludge may be estimated at 2,500 kg (dentists: 800 kg, households: 700 kg [daily intake 0.2 mg Sn, 100% excreted via urine and faeces, retention of 70%] and a category "others" of 1,000 kg based on data available for other metals). So far approximately 25% of the sludge is applied in agriculture, corresponding to an amount of 600 kg Sn. Such an application results into a annual increase in Sn concentration of 0.2 $\mu g.kg^{-1}$ in the upper 10 cm of the soil. Assuming no leaching out of tin to deeper parts of the soil compartment, it will take almost a 1000 years to increase the background concentration with 1%. Taking into account the "resolution on other organic manure" (which will lead to a reduction of sludge application in agriculture, to be in force late 1993) and the convenant with dentists (wich is in force since february 1992), the actual Sn load will be even lower in the near future. Based on these considerations there is no need for more information on both the exposure and the toxicity of inorganic tin.

Organic tin

As to organic tin compounds no quantitative data on the inhalatory exposure of humans are available. However, taking into account the estimated emission into air (table 3.10) and the available NOEC-values (section 6.1.2) the environmental concentrations do not present a risk to the general population. The risk of organic tin compounds in food to humans is unknown, chiefly since no data on exposure are available. It is estimated that there may be some risk if the organic tin compounds in the total diet is about 1%. Therefore more information is needed on the occurrence of organic tin in foodstuffs.

Available data indicate that organotin compounds (triphenyl and tributyl) present a risk to aquatic ecosystems in the Netherlands. The exposure data refer to the period 1988-1991. It is the objective to reduce the use and emissions of pesticides: the use of fungicides in agriculture should be reduced by 15% in 1995 and by 25% by 2000 compared to 1990. Both triphenyltin acetate and triphenyltin hydroxide are listed as pesticides to be banned. However, so far no measures were not taken and it is only recently (April 1993) that the amendment on the Pesticides Act was approved by the parliament. It is advocated to execute the ban on these compounds as soon as possible.

No information is available on the toxicity to sediment and soil organisms, whereas data on occurrence is only available for sediment. Most of the organotins in sediment orginate from anti-fouling paints and wood preservatives, whereas those in the soil mainly result

from application in potato farming. In surface waters there may be a risk of exceeding the MTR in harbours. Therefore research into alternative methods for anti-fouling paints should be stimulated.
In the soil and sediment the organotin compounds are slowly degraded to inorganic tin. It is suggested to initiate a study into the degradation rates of organotins in sediment and soil in order to determine the maximum allowable emission per year.

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APPENDIX A: PHYSICAL/CHEMICAL CHARACTERISTICS OF TIN AND SOME TIN COMPOUNDS

Tin (Lide, 1990)

chemical formula:	Sn
atomic number:	50
atomic weight:	118.7
physical form:	solid
colour:	white (ß-tin) or gray (α-tin)
melting point:	232°C
boiling point:	2,270°C
α -> ß transition temperature:	13.2°C (α at t < 13.2°C)
density:	7.31 (white, ß-tin)
	5.75 (gray, α-tin)
solubility:	insoluble in water
	soluble in strong acids like HCl, H_2SO_4
CAS registry number:	7440-31-5

Tributyltin oxide (WHO, 1990)

chemical formula:	$C_{24}H_{54}OSn_2$
molecular weight:	596
physical form:	liquid
refractive index (20°C):	1.4880-1.4895
melting point:	<-45°C
boiling point (130 Pa):	173°C
relative density (20°C):	1.17-1.18
vapour pressure (20°C):	0.001 Pa
solubility:	<1 - >100 mg.l^{-1} in water (may be related to reactions with normal constituents of water)
	very soluble in a number of organic solvents (like ethanol, ether, etc.)
log P_{ow} (distilled water):	3.19 - 3.84
(sea water):	3.54
CAS registry number:	56-35-9

Triphenyltin acetate (Crijns, 1992)

chemical formula:	$(C_6H_5)_3SnOOCCH_3$
molecular weight:	409.60
physical form:	white crystals
melting point:	124°C
boiling point:	>350°C (decomposes >230°C)
density (20°C):	1.84 kg.l^{-1}
vapour pressure (25°C):	0.176 mPa
solubility in fresh water:	1-20 mg.l^{-1}
in saline water:	<1 mg.l^{-1}
Henry's law constant:	$5.20.10^{-1}$ Pa.m^3.mol^{-1}
log P_{ow}:	-
CAS registry number:	900-95-8

Triphenyltin hydroxide (Crijns, 1992)

chemical formula:		$(C_6H_5)_3SnOH$
molecular weight:		367.02
physical form:		white amorphous powder
melting point:		124°C
boiling point:		>350°C (decomposes >230°C)
density (20°C):		1.4 $kg.l^{-1}$
vapour pressure (25°C):		0.047 mPa
solubility	in fresh water:	1-8 $mg.l^{-1}$
	in saline water:	<1 $mg.l^{-1}$
Henry's law constant[1]:		$1.24.10^{-3}$ $Pa.m^3.mol^{-1}$; $0.12.10^{-1}$ $Pa.m^3.mol^{-1}$
log P_{ow}:		3.3 ; 3.8
CAS registry number:		76-87-9

1) reference gives 2 different values

APPENDIX B: LIST OF SOME INORGANIC TIN COMPOUNDS AND ORGANOTIN COMPOUNDS

Metallic tin and inorganic tin compounds

Tin metal
Tin(IV) chloride
Tin(IV) oxide
Tin(IV) hydride
Tin(II) acetate
Tin(II) chloride
Tin(II) fluoroborate
Tin(II) fluoride
Tin(II) 2-ethylhexoate
Tin(II) oxolate
Tin(II) oxide
Tin(II) sulphate
Tin(II) tartrate
Sodium stannate
Sodium pentafluorostannite

Organotin compounds

Monosubstituted compounds

Ethyltin trichloride
Ethyltin triiodide
Butyltin trichloride
Butylstannoic acid
Butylthiostannoic acid
Butyltin-S,S',S''-tris (isooctylmercaptoacetate)
Butyltin-S,S',S''-tris (2-ethylhexylmercaptoacetate)
Butyltin sulphide
Octyltin trichloride
Octyltin tris(2-ethyl hexylmercaptoacetate)
Cyclohexylstannoic acid

Disubstituted compounds

Dimethyltin dichloride
Dimethyltin S,S'-bis (isooctyl mercaptoacetate)
Diethyltin dichloride
Diethyltin diiodide
Diethyltin dioctanoate
Dipropyltin dichloride
Diisopropyltin dichloride
Dibutyltin dichloride
Dibutyltin oxide
Dibutyltin diacetate
Dibutyltin dilaurate
Dibutyltin maleate
Dibutyltin sulphide
Dibutyltin di (2-ethylhexoate)
Dibutyltin dioctanoate
Dibutyltin di (nonylmaleate)
Dibutyltin ß-mercapto propanoate
Dibutyltin bis(lauryl mercaptide)
Dibutyltin "lauratemaleate"
Dibutyltin S,S'-bis (isooctylthioglycolate)
Dibutyltin S,S'-bis(2-ethylhexylmercaptoacetate)
Dipentyltin dichloride
Dioctyltin dichloride
Dicotyltin acetate
Dioctyltin dilaurate
Dioctyltin maleate
Dioctyltin dibutylmaleate
Dioctyltin-S,S'-(ethylene glycol-bis-mercaptoacetate)
Dioctyltin-S,S'-bis(isooctylmercaptoacetate)
Dioctyltin mercaptoacetate
Dioctyltin ß-mercapto propanoate
Dioctyltin-S,S'-bis (butyl mercaptoacetate)
Dioctyltin-S,S'-bis(2-ethylhexylmercaptoacetate)
Dioctyltin-S,S'-bis (laurylmercaptoacetate)
Dioctyltin bis(2-ethylhexylmaleate)
Dioctyltin bis(dodecyl mercaptide)
Dioctyltin-S,S'-(1,4-butanediol-bis-mercapto acetate)
Dioctyltin di(1,2-propyleneglycolmaleate)
Dioctyltin bis(isobutyl maleate)
Diphenyltin dichloride
Dicyclohexyltin oxide
Didodecyltin dibromide
Dioctadecyltin dibromide

Trisubstituted compounds

Triethyltin bromide
Triethyltin chloride
Triethyltin iodide
Triethyltin sulphate
Triethyltin hydroxide
Triethylstannylmethyl (1-propynyl) formal
Triethylstannylphenyl acetylene
1-Triethylsiloxi-1-propyne
2-Trichloro-1-(butine-1'-oxide)-1-(triethyl stannyloxy)ethane
Trivinyltin chloride
Tributyltin chloride
Tributyltin fluoride
Bis(tributyltin) oxide
Tributyltin acetate
Tributyltin linoleate
Tributyltin benzoate
Tributyltin salicylate
Tributyltin methacrylate
Tributyltin laurate
Tributyltin oleate
Trihexyltin acetate
Tricyclohexyltin hydroxide (Cyhexatin)
Trioctyltin chloride
Triphenyltin chloride
Triphenyltin hydroxide
Triphenyltin acetate
p-Bromophenoxy triethyltin

Tetrasubstituted compounds

Tetramethyltin
Tetraethyltin
Tetrabutyltin
Tetraisobutyltin
Tetraphenyltin
Tetraoctyltin
Stannous octanoate
Tin (II) cyclopentadienyl

APPENDIX C: LIST OF CONVERSION FACTORS FOR SOME TIN COMPOUNDS

Inorganic tin compounds

Compound	Conversion factor (tin compound ==> tin)
Sodiumchlorostannate	0.3721
Sodiumpentachlorostannite	0.5424
Sodiumpentafluorostannite	0.6680
Sodiumtincitrate	0.3588
Tin(II) 2-ethyl-hexoate	0.2930
Tin(II)chloride	0.6260
Tin(II)chloridedihydrate	0.6159
Tin(II)citrate	0.4849
Tin(II)fluoride	0.7575
Tin(II)oleate	0.1741
Tin(II)orthophosphate	0.6521
Tin(II)oxalate	0.5742
Tin(II)oxide	0.8812
Tin(II)sulphate	0.5527
Tin(II)sulphide	0.7873
Tin(II)tartrate	0.4449
Tin(IV)chloride	0.4556
Tin(IV)oxalate	0.4027
Tin(IV)oxide	0.7876
Tin(IV)sulphate	0.3819

Organotin compounds

Compound	Conversion factor (tin compound ==> tin)
Butylstanoicacid (Bu-SnO_2·H2O)	0.5256
Butylthiostanoicacid (Bu-S-SnO_2·H2O)	0.4602
Butyltintrichloride	0.5098
Butyltin(2-Et-HexSAc)$_3$	0.1510
Dibutyltindiacetate	0.3381
Dibutyltindichloride	0.3906
Dibutyltindilaurate	0.1879
Dibutyltinoxide	0.4768
Dibutyltin(2-Et-HexSAc)$_2$	0.1856
Didodecyltindibromide	0.1923
Diethyltindichloride	0.4791
Diethyltindiiodide	0.2756
Dimethyltindichloride	0.5403
Dioctadecyltindibromide	0.1511
Dioctyltindichloride	0.2853
Dioctyltin(2-Et-HexSAc)$_2$	0.1578
Dioctyltin(BuSAc)$_2$	0.1856
Dioctyltin(dodecylS)$_2$	0.1587
Dipentyltindichloride	0.3576
Dipropyltindichloride	0.4304
Octyltintrichloride	0.3509
Octyltin(2-Et-HexSAc)$_3$	0.1410
Tetrabutyltin	0.3419
Tetraethyltin	0.5052
Tetramethyltin	0.6637
Tetraoctyltin	0.2076
Tetrapentyltin	0.2943
Tetrapropyltin	0.4078
Tributyltinabietate	0.2006
Tributyltinacetate	0.3400
Tributyltinbenzoate	0.2887
Tributyltinchloride	0.3646
Tributyltinfluoride	0.3841
Tributyltinlaurate	0.2425
Tributyltinlinoleate	0.2084
Tributyltinnaphthenate	0.2573
Tributyltinoleate	0.2077
Tricyclohexyltinhydroxide	0.3081
Tricyclohexyl-1,2,4-triazole-1-yl-tin	0.2715
Triethyltinacetate	0.3951
Triethyltinchloride	0.4918
Triethyltinhydroxide	0.5325
Triethyltinsulphate	0.3931
Trihexyltinacetate	0.2739
Trioctyltinchloride	0.2403
Triphenyltinacetate	0.2901
Triphenyltinhydroxide	0.3234
(Tributyltin)$_2$-oxide	0.3982
(Tributyltin)$_2$-sulphide	0.3878
(Triethyltin)$_2$-sulphide	0.5349
[(2-Phenyl-2,2-dimethyl-ethyl)$_3$-tin]$_2$-oxide	0.2255