June 2023

The SCOEL recommendation document covers the following substances:

Substance name	EC number	CAS RN
Tin	231-141-8	7440-31-5
Tin dichloride	231-868-0	7772-99-8
Tin difluoride	231-999-3	7783-47-3
Tin diiodide	233-667-3	10294-70-9
Ditin pyrophosphate	239-635-5	15578-26-4
Tin sulphate	231-302-2	7488-55-3
Tin tetrabromide	232-184-5	7789-67-5
Tin tetrachloride	231-588-9	7646-78-8
Tin tetraiodide	232-208-4	7790-47-8
Tin dioxide	242-159-0	18282-10-5

This text is not part of the official SCOEL Recommendation and is provided to give additional helpful information to the reader as regards chemicals addressed by the SCOEL Recommendation. The list is non-exhaustive and is presented for information purposes only.

Social Europe

Recommendation from the Scientific Committee on Occupational Exposure Limits for tin and inorganic tin compounds

SCOEL/SUM/97
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European Commission

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8-hour TWA	
Tin and tin dioxide:	insufficient data
Other inorganic tin compounds:	insufficient data
STEL (15 min):	-
Additional classification:	-

Substance identification (pure tin)

Tin	Sn
CAS number:	7440-31-5
Atomic mass:	118.71
Melting point:	231.9°C
Boiling point:	2602°C
Density:	7.27 (white, above 13°C), 5.77 (grey, below 13°C) g/cm^3
Solubility in water:	Insoluble

Pure tin is a silver-white, shiny metal with the atomic symbol Sn and belongs to the carbon group (group IVA). Tin occurs naturally as several stable isotopes with mass numbers ranging from 112 to 124. Some important inorganic tin (II) and (IV) compounds are identified in Tables 1 and 2, respectively.

This summary document is based on a joint NEG-DECOS criteria document (Westrum and Thomassen, 2002). SnH_4 (stannane), a basic compound for the manufacture of a large number of organotin compounds, is not included.

1. Occurrence/use

Tin is found throughout the Earth's crust at a few parts per million. It is mined chiefly as cassiterite (SnO_2). Other tin ores are complex sulphides, stannite (Cu_2FeSnS_4) and teallite ($PbZnSnS_2$). Metallic tin is obtained by smelting tin ore. The ore is mixed with salt and roasted at about 600°C, washed in water and then mixed with anthracite as a reducing agent and smelted at about 1 500°C. After refining, the tin is cast into bars.

Annual world production of tin has been quite stable at about 210-230 kilotonnes for decades, including 15-20 kilotonnes of secondary metal recovered from scrap waste or detinning.

Table 1. Chemical identification of some inorganic tin (II) compounds

Chemical name	**Tin dichloride**	**Tin difluoride**	**Tin diiodide**	**Tin pyrophosphate**	**Tin sulphate**
Synonym	Stannous chloride	Stannous fluoride	Stannous iodide	Stannous pyrophosphate	Stannous sulphate
Chemical formula	$SnCl_2$	SnF_2	SnI_2	$Sn_2P_2O_7$	$SnSO_4$
Molecular weight	189.6	156.7	372.5	411.4	214.8
CAS number	7772-99-8	7783-47-3	10294-70-9	15578-26-4	7488-55-3
Melting point (°C)	247	213	320	Decomposes at 400°C	Decomposes at 378°C
Boiling point (°C)	623	850	714	-	-
Density (g/cm^3)	3.90	4.57	5.28	4.01	4.15
Solubility in water	Soluble	Soluble	Slightly soluble	Insoluble	Decomposes

Because of its resistance to corrosion, tin is used as a protective coating for other metals. Another important property of tin is its ability to form alloys with other metals. Thus, tin metal is commonly used in tin plating, manufacture of food cans, and solder and alloy production. Dental amalgams contain varying proportions of tin (12-30%).

$SnCl_4$ is used as a dehydrating agent in organic synthesis, a stabiliser for plastics and a chemical intermediate for other tin compounds. $SnCl_2$ serves as a reducing agent in manufacturing ceramics, glass and inks. SnF_2 has been used as a prophylactic agent in preventive dentistry for decades. Sn (II) is important in nuclear medicine and has almost ideal redox properties for the reduction of the visualising label technetium-99m.

Table 2. Chemical identification of some inorganic tin (IV) compounds

Chemical name	Tin tetrabromide	Tin tetrachloride	Tin tetraiodide	Tin dioxide
Synonym	Stannic bromide	Stannic chloride	Stannic iodide	Stannic oxide
Chemical formula	$SnBr_4$	$SnCl_4$	SnI_4	SnO_2
Molecular weight	438.3	260.5	626.3	150.7
CAS number	7789-67-5	7646-78-8	7790-47-8	18282-10-5
Melting point (°C)	31	-33	143	1630
Boiling point (°C)	205	114	364.5	**subl.** 1800-1900
Density (g/cm^3)	3.34	2.23	4.46	6.85
Solubility in water	Highly soluble	Decomposes	Soluble	Insoluble

Occupational exposure

Most of the operations associated with the extraction of tin ore are wet processes. However, tin dust and oxide fumes may escape during bagging of concentrate, in ore rooms and during smelting operations (mixing-plant and furnace tapping), as well as during the periodic cleaning of bag filters used to remove particulate matter from smelter furnace flue gas. Tin reclamation also involves possible exposure to tin dusts and fumes.

Tin production may also involve exposure to silica, lead and arsenic in the mining of the sulphide ores of tin, and to bismuth and antimony as well in the roasting and melting process. Similarly, the preparation and use of tin alloys and solders entail exposure to these heavy metals. Tin mining may further involve exposure to radon, thorium and uranium.

No systematic data on occupational exposure levels in tin production or processing are available.

Tin levels (expressed as mg dust per m^3) measured in the workroom air from tin melting works were: check sampling shed 2.22, dracco (filters for furnace gases) 1.10, smelting furnace man 1.55, refining furnace man 0.82, orehouse skipman 0.34, plumber 0.12, electrician 0.05 and engineer 0.02. The methods of sampling and analysis were not described.

An environmental survey to determine the type of exposure in a Chilean tin foundry showed air concentrations of tin between 8.6 and 14.9 mg/m^3.

According to the Norwegian occupational exposure database EXPO (data since 1984), 420 air out of 3 407 filter samples (8-h personal monitoring) analysed for tin contained levels above the detection limit (0.002 mg/m^3). Branches and job functions with tin levels higher than 0.05 mg/m^3 were: railway repair/termite welding, electronic production/surface coating, defence activities/spraying, metal coating/surface coating, and metal casting/cleaning. The highest recorded exposure level (8-h TWA) was 5.7 mg/m^3.

Studies in the UK showed mean concentrations of tin in diet of 1-2 mg/kg or about 3 mg/d. The primary sources of tin are said to be canned goods.

Toxicokinetics

No valid data on inhalation uptake of inorganic tin are available.

The oral uptake of tin is generally low and below a few per cent of the dose. The absorption apparently occurs by passive diffusion and depends on the solubility of the tin compound. About 50% of the dose was absorbed in man when 0.11 mg Sn/d was ingested with the diet. From a test diet containing an additional 50 mg Sn/d as $SnCl_2$, only 3% was absorbed.

No data on the absorption of tin from dermal exposure are available. On the basis of the physical properties and the low oral uptake of tin, dermal absorption can be assumed to be negligible.

It has been suggested that the differences in the relative affinity of the kidneys and liver for Sn(II) and Sn(IV) indicate valence stability and that tin is not rapidly oxidised or reduced during absorption and systemic transportation.

Inorganic tin distributes mainly to bone but also to the lung, liver and kidney. Some data indicate that tin may have a higher affinity to the thymus than to other organs. Animal data suggest that inorganic tin does not easily pass the blood-brain barrier. Absorbed tin is mainly excreted via the kidneys. The biological half-life has been estimated to be 10-20 d for tin (II) in rat liver and kidney. In rat bone the half-life of tin (II) and tin (IV) is reportedly about 20-100 d.

2. Health significance

Acute toxicity

No clinical or experimental reports on acute effects through inhalation of inorganic tin have been identified. Metal fume fever caused by inorganic tin has not been documented (Blanc and Boushey, 1993). Symptoms such as wheezing, cough, chest pain and dyspnoea on exertion reported in workers handling $SnCl_4$ were probably due to elevated levels of hydrogen chloride formed by the combination of $SnCl_4$ and water in the presence of heat (Levy *et al.*, 1985).

There is one early case report of a worker who over 10 years had been exposed once or twice daily for 10-15 min to fumes of $SnCl_4$ and hydrochloric acid (Pedley, 1927). The fumes were stated to be unbearably irritating to the uninitiated. The most prominent symptoms were throat pain, heaviness in the stomach, and feeling of chilliness in the chest. The same report gives a short note on an experiment with guinea pigs exposed by inhalation to 3 000 mg/m^3 $SnCl_4$ (corresponding to 1 370 mg Sn/m^3), 10 min/day for «several months». The only reported finding was that transient irritation of the eyes and nose developed. However, no further details were given on the animal study (Pedley, 1927).

The acute oral toxicity of tin compounds appears to be low in animals. The oral 24-h LD_{50} for $NaSn_2F_5$ in rats and mice is about 0.4 g Sn/kg bw. The oral LD_{50} for $SnCl_2$ in rats is about 1.7 g Sn/kg bw. Major toxic symptoms after treatment with $NaSn_2F_5$ or $SnCl_2$ were diarrhoea, ataxia, general depression, fore- and hindleg weakness advancing to flaccid paralysis prior to death. Further data from this experiment suggest that both F and Sn contribute to the toxicity of $NaSn_2F_5$ (Conine *et al.*, 1975).

Intratracheally injected $SnCl_2$ in saline (single dose, ~ 6µg Sn/animal or ~0.24 mg Sn/kg bw) followed by infection with aerosolised Streptococcus caused 36% increased mortality in mice. Similar effects were seen with *e.g.* fly ashes, carbon, bentonite and a number of metal oxides. According to the authors, in the case of soluble metals inhalation exposure yields similar results (Hatch *et al.,* 1985). Changes in the immune response were seen in mice following higher intraperitoneal doses of $SnCl_2$ (5 mg/kg bw) and $SnCl_4$ (3.5 mg/kg bw) (Dimitrov *et al*., 1981).

Many case reports deal with an acute gastrointestinal illness following the intake of canned fruit or fruit juice, mostly as a result of the detinning of unlacquered cans by corrosion. Estimated doses ingested are 30-200 mg during a short period of time. Symptoms most frequently reported are nausea, abdominal cramps, vomiting and diarrhoea. In an experimental study, nausea, vomiting and diarrhoea followed the drinking of tin-containing fruit juices by human volunteers only at single tin doses of about 330 mg and above. No effects were observed after the ingestion of about 130 mg, or 1.7-2.6 mg Sn/kg. The authors stated that there was no evidence from their experiments that toxicity was due to the absorption of tin and that the most likely cause was local irritation of the mucous membranes of the alimentary tract (Benoy *et al.*, 1971). Mucosal irritation by $SnCl_2$ and $SnCl_4$ is well documented in animals.

Occupational exposure to tin dioxide

There are several case reports of workers exposed to tin dioxide dust and fumes for 3 years or more in tin melting works, scrap metal recovery plants and hearth tinning. The only positive finding is the chest X-rays presenting a non-fibrosing pneumoconiosis called stannosis. In general, no information on exposure levels is available.

Robertson and co-workers examined the employees, including pensioners, from a tin melting works and described chest X-ray changes in 121 out of 215 workers. The changes were widespread, tiny, dense shadows or softer, larger, more nodular opacities. Typical changes were found in workers handling raw ore, smelting furnace house workers and refinery furnace men. The length of employment was 3-50 years. None of the men had any clinical symptoms or signs referable to pneumoconiosis. None of the films suggested fibrosis or significant emphysema. Lung function studies showed no disability, whatever the radiographic category. The population at the tin melting works had lower mortality (131 deaths) than expected when compared to the male population in the UK (expected 166) in the period 1921-55 (Robertson and Whitaker, 1955; Robertson, 1960; Robertson, 1964).

Hlebnikova made a survey over a number of years of workers exposed to condensation aerosols formed during the melting of tin and consisting mainly of SnO_2. Total silica concentration in the aerosols did not exceed 3%. Total dust concentration in air varied between 3 and 70 mg/m^3. Workers developed pneumoconiosis after 6 to 8 years of employment. No cases of pneumoconiosis were observed 10 years after the dust concentration had been reduced to 10 mg/m^3. No further details are given (Hlebnikova, 1957, cited by WHO (1980)).

In a Belgian case-control study (n=272), a significantly increased risk of chronic renal failure was found for occupational exposure to tin (OR 3.72, 95%CI 1.22-11.3). Exposures were scored independently by three industrial hygienists (Nuyts *et al.*, 1995).

Animal studies with tin and tin dioxide

Rats were experimentally exposed to 50 mg of metallic tin dust in saline from a tin melting works by a single intratracheal administration. Four months later X-ray photographs

showed widespread tiny densities throughout the rat lungs similar to those seen previously in tin-exposed workers. Histologically, there was no fibrous response of any kind in rats monitored for one year (Robertson, 1960).

Gross and histological examination of various tissues from three species of animals showed no fibrosis, neoplasia or other adverse effects following intravenous administration of SnO_2 or Sn particles at high doses. The species, doses and times from dosing to examination were: rats, 250-1000 mg SnO_2/kg, 200-800 mg Sn/kg, 4-26 months; rabbits 250 mg SnO_2/kg, 200 mg Sn/kg, 6-26 months; and dogs, similar doses to rabbits, 4-5 years (Fischer and Zimmerman, 1969).

Silica containing 97% crystalline SiO_2 (50 mg), SnO_2 (50 mg) or a mixture of SnO_2-SiO_2 (25 mg each in 1 ml saline) dust was instilled intratracheally in rats. The *in vivo* cytotoxicity, interleukin-1 release from rat pulmonary cells, and fibrogenic effects up to 30 d after dosing correlated well with the free SiO_2 content in the dusts, whereas SnO_2 was suppressive to the effect of SiO_2 (Wang *et al.*, 1994). These animal studies suggest that SiO_2 rather than SnO_2 cause the pulmonary inflammatory responses.

Effects on mineral status

Eight adult males were given mixed diets containing 0.11 mg Sn/d (control diet) and 50 mg Sn/d (test diet, 50 mg of additional Sn as $SnCl_2$ in fruit juice) for 20 days each in a crossover design. There was no effect on the faecal and urinary excretion rates of copper, iron, manganese, magnesium and calcium. Zinc and selenium excretion rates were moderately changed. Hematocrit and serum ferritin were not affected (Greger *et al.*, 1982; Johnson *et al.*, 1982; Johnson and Greger, 1982).

In humans, $SnCl_2$ (36 mg Sn) given with $^{65}ZnCl_2$ solutions (0.5, 4 and 6 mg Zn, respectively) or turkey test meals (4 mg Zn) inhibited zinc absorption, measured by whole-body counting of the retention of ^{65}Zn after 7 to 10 days. According to the authors, the doses required to inhibit Zn absorption under the conditions in this study were well in excess of those ordinarily found in the diet (Valberg *et al.*, 1984). Solomons *et al.* (1983) were unable to demonstrate inhibition of zinc uptake in human volunteers ingesting 25, 50 or 100 mg Sn as $SnCl_2$.

$SnCl_2$ (2 mg Sn/kg bw/d) given orally to rabbits for 1 month decreased zinc and copper concentration in bone marrow and increased iron concentrations in liver and kidneys (Zareba and Chmielnicka, 1989). In contrast, Beynen *et al.* (1992) found that iron status in rabbits was decreased by dietary tin concentrations above 100 mg Sn/kg diet but unaffected at lower doses.

A 28-day study with $SnCl_2$ in Wistar rats showed that Fe, Cu and Zn concentrations in tissues and plasma were unaffected at 1 mg, slightly decreased at 10 mg and markedly decreased at 50 mg Sn/kg feed (Pekelharing *et al.*, 1994). The two higher doses correspond to about 0.7 and 3.5 mg Sn/kg bw/d, respectively.

Oral doses of $SnCl_2$ (0.3, 1.0 and 3.0 mg Sn/kg bw) were given twice daily to Wistar rats for 90 days. The high dose caused significant decreases in the femur weight, serum and femoral calcium, serum lactic dehydrogenase and alkaline phosphatase activities, liver succinate dehydrogenase activity and femoral acid phosphatase activity. The medium dose produced significant reduction in liver succinate dehydrogenase activity, femoral calcium and femoral acid phosphatase activity. At the low dose, a slight non-significant decrease in calcium in the femoral epiphysis was observed. These results suggest a NOEL below 0.6 mg/kg/d (Yamaguchi *et al.*, 1980).

Male Wistar rats given an oral dose of 1.0 mg Sn/kg at 12-h intervals for 28 days had an increased tin content of the femoral diaphysis and epiphysis. Other effects were decreased calcium content in bone and decreased acid and alkaline phosphatase activities in the femoral epiphysis (Yamaguchi *et al.*, 1981).

Effects on skin

Patch tests with metallic tin in 73 nickel-sensitive patients revealed 6 positive allergic and 4 doubtful reactions. The low frequency of doubtful reactions made it unlikely that metallic tin is irritant (Menné *et al.*, 1987). Patch testing with $SnCl_2$ 1% in vaseline and a tin disc suggested that some patients are sensitised to tin. A low frequency of doubtful reactions suggested that metallic tin and $SnCl_2$ at 1% were non-irritating. However, irritant reactions were frequent in patients tested with $SnCl_2$ 10% and 5% in vaseline (Olivarius *et al.*, 1993). In 199 patients with suspected allergic reactions to metals, 13 had positive patch tests with 2% $SnCl_2$ in vaseline (26 metals tested) (Rammelsberg and Pevny, 1986). One out of 50 craftsmen in the ceramics industry had a positive reaction by patch testing with 2.5 % elementary tin in vaseline (Gaddoni *et al.*, 1993). A worker producing metal patterns for body parts on trucks, exposed to airborne dust from an alloy which used to contain tin, had dermatitis around the eyes, forehead and wrists. He had a positive patch test to tin. The case is reported as an occupational allergic dermatitis due to tin (Nielsen and Skov, 1998).

Larsson *et al.* (1990) determined non-irritant levels of $SnCl_2$ and $SnCl_4$ on skin (5% in alcohol) and on oral mucosa (3% and 0.05% in alcohol) in Sprague-Dawley rats. Lesions of allergic contact type could not be found in the rat oral mucosa.

Mutagenicity and carcinogenicity

In vitro studies have shown that $SnCl_2$ causes DNA-damage in human white blood cells, in Chinese hamster ovarian cells and in E. coli. DNA-damage in human lymphocytes has been shown after treatment with $SnCl_4$.

Some reports from China are concerned with the health of tin miners. There were 1 724 lung cancer cases registered at the Yunnan Tin Corporation in the period 1954 to 1986, of which 90% had a history of working underground. Assumed contributing factors included diet, arsenic, radon and tobacco. Tin is not considered a carcinogenic factor in these studies (Qiao *et al.*, 1989; Taylor *et al.*, 1989; Forman *et al.*, 1992; Qiao *et al.*, 1997).

Increased mortality from lung cancer was also present among Cornish tin miners. There was a clear relation between exposure to radon and death from lung cancer (Fox *et al.*, 1981; Hodgson and Jones, 1990).

Tin given to rats in drinking water (5 mg/l, corresponding to about 0.4 mg/kg bw/d, lifelong exposure) resulted in increased incidence of degenerative changes in liver and kidneys and reduced longevity in females. No tumorigenic or carcinogenic effects were observed (Schroeder *et al.*, 1968).

$SnCl_2$ given orally in feed for 105 weeks (1 000 or 2 000 mg/kg feed, corresponding to 20-182 and 35-348 mg Sn/kg bw/d, respectively) was judged by the NTP not to be carcinogenic in Fischer 344 rats or B6C3F1 mice, although C-cell tumours of the thyroid gland (significantly increased in low dose male rats only) may have been associated with the administration of the chemical (NTP, 1982).

No data are available on reproductive and developmental effects.

Recommendations

Deposition of tin particles in the lungs of tin ore miners induces "stannosis", with no indication of fibrosis. No apparent disability beyond the chest X-ray opacities is found. However, the scientific literature consists of case reports in humans, with poor exposure assessment and old methods of examination. Reports on effects concerning micropathology and cell toxicity in the respiratory system are scarce. One case-control study reports increased risk of chronic renal failure for occupational exposure to tin, but no quantitative exposure data were given. Overall, although there is little or no evidence that inhaled tin and tin dioxide, which are poorly soluble in water, pose any significant health risk at the exposure levels found in the work environment. The available data are insufficient to derive a health-based OEL value.

With respect to more soluble tin compounds, the only inhalation study found in the literature dates back to 1927. It reports transient irritation of the nose and eyes in guinea pigs exposed daily to 3 000 mg/m^3 $SnCl_4$ for 10 min. The report is very brief and no NOAEL can be identified. Intratracheally injected $SnCl_2$, caused increased bacterial infectivity in mice at a single dose corresponding to about 0.24 mg Sn/kg. Oral toxicity studies in laboratory animals suggest systemic effects on liver and kidneys at 0.4 and on mineral status at 0.6-0.7 mg Sn/kg/d. As a worst-case scenario, asuming 100% absorption, a body weight of 70 kg and a pulmonary ventilation of 10 m^3 during an 8-h working day, these doses (0.24-0.7 mg Sn/kg) would correspond to ambient air levels of 1.7-5 mg Sn/m^3.

Due to lack of data on tin salts other than $SnCl_2$ and $SnCl_4$ and since it cannot be excluded that soluble tin salts may cause irritation at lower air levels than those that would be expected to result in systemic effects, the scientific data are considered insufficient to derive a health-based OEL value.

The available reports on metallic tin and $SnCl_2$ are not sufficient to classify tin as a carcinogen or a contact allergen.

There are no data suggesting a need for skin notation for tin or inorganic tin compounds.

Organotin compounds are not considered in this document.

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