Tin(IV) oxide, tin(II) oxide

(CAS No: 18282-10-5, 21651-19-4)

Health-based Reassessment of Administrative Occupational Exposure Limits

Committee on Updating of Occupational Exposure Limits,
a committee of the Health Council of the Netherlands

No. 2000/15OSH/091, The Hague, 22 October 2003

Preferred citation:
Health Council of the Netherlands: Committee on Updating of Occupational Exposure Limits. Tin(IV) oxide, tin(II) oxide; Health-based Reassessment of Administrative Occupational Exposure Limits. The Hague: Health Council of the Netherlands, 2003; 2000/15OSH/091.

1 Introduction

The present document contains the assessment of the health hazard of tin oxide by the Committee on Updating of Occupational Exposure Limits, a committee of the Health Council of the Netherlands. The first draft of this document was prepared by AAE Wibowo, Ph.D., and MM Verberk, Ph.D. (Coronel Institute, Academic Medical Centre, Amsterdam, the Netherlands).

The evaluation of the toxicity of tin oxide has been based on the reviews published by the American Conference of Governmental Industrial Hygienists (ACG96) and the World Health Organization (WHO80) and in 'The handbook on the toxicology of metals' (Mag86). Where relevant, the original publications were reviewed and evaluated as will be indicated in the text. In addition, in May 1998, literature was searched in the databases Medline, Embase, and Chemical Abstracts starting from 1996, 1998, and 1970, respectively. HSELINE, CISDOC, MHIDAS, and NIOSHTIC (covering the period 1985/1987 until 1998) as well as Poltox (Toxline, Cambridge Scient. Abstr., FSTA; covering the period 1990 until 1995), databases available from CD-ROM, were also consulted. The following key words were used: tin oxide, stannic oxide, stannous oxide, 18282-10-5, and 21651-19-4. The final literature search was carried out in Toxline and Medline in January 2003.

In April 2003, the President of the Health Council released a draft of the document for public review. The committee received no comments.

2 Identity

name	:	tin (IV) oxide	tin (II) oxide
synonyms	:	tin dioxide; stannic oxide; stannic anhydride; stannic dioxide; white tin oxide	tin monoxide; tin protoxide; stannous oxide
molecular formula	:	SnO_2	SnO
structural formula	:	-	-
CAS number	:	18282-10-5	21651-19-4

3 Physical and chemical properties

	tin(IV) oxide	tin(II) oxide
molecular weight	150.70	134.70
boiling point	1800-1900ºC (sublimes)	-
melting point	1630ºC	at 60 kPa: 1080ºC (decomposes)
flash point	not available	not available
vapour pressure	not available	not available
solubility	insoluble	insoluble
log $P_{octanol/water}$	1.29 (estimated)	1.29 (estimated)
conversion factors	not applicable	not applicable

Data from Wea87; http://esc.syrres.com.

Tin(IV) and tin(II) oxide are white or slightly grey and brown to black powders, respectively. Tin(IV) oxide occurs naturally as the mineral cassiterite. Commercial grades of tin(IV) oxide are also known as polishing powder, putty powder, or tin ash (Bud89).

4 Uses

Tin(IV) oxide is used for polishing glass and metals, in manufacturing milk-coloured, ruby, and alabaster glass, enamels, pottery, and putty, as a mordant in printing and dyeing fabrics, and in fingernail polishes. Tin(II) oxide is used as a reducing agent and in the preparation of stannous salts (Bud89).

5 Biotransformation and kinetics

There is very little data on the absorption of inhaled tin oxide dust particles, but tin levels in the lungs are reported to be elevated by exposure to tin oxide (Rob61). Inorganic tin is very poorly absorbed through the digestive tract; it is estimated to be less than 5% (WHO80). WHO estimated the mean total daily intake of tin by man to range from 0.2 to 17 mg. A diet consisting of fresh foods probably provided about 1 to 4 mg/day. The likely daily intake from water was

estimated to be less than 30 g/day, and the daily amount entering the body from air, less than 1 g (WHO80).

The absorption through the digestive tract depends on the oxidation state of tin: tin (II) is more readily absorbed than tin (IV). The anion complement may also influence the rate of absorption (WHO80). When absorbed through the digestive tract, tin leaves the vascular system rapidly. Baselt and Cravey reported that the average concentration of tin in the blood of normal subjects is 0.14 mg/L, that it resides primarily in the erythrocytes, and that it is believed to originate from the diet (Bas89). According to the WHO, bone is the main site of deposition, and the highest concentrations of tin are found in the lungs, kidneys, liver, and bone (levels not reported) (WHO80). Schroeder et al. reported that tin was found in almost every tissue they analysed in American adults, the intestinal tract containing the highest concentrations. Median concentrations (in dry ash) found were 18 mg/kg in the oesophagus, 16 mg/kg in the stomach, 28 mg/kg in the duodenum, 36 mg/kg in the jejunum, 79 mg/kg in the ileum, 130 mg/kg in the caecum, 45 mg/kg in the sigmoid colon, and 57 mg/kg in the rectum. In lungs, adrenals, and liver, median concentrations were 37, 23, and 23 mg/kg, respectively. Tin was seldom detected in the brain or muscle (Sch64). Magos stated that the biological half-life of inorganic tin in bone is about 400 days, both in man and in animals. Penetration of the blood-brain barrier and placenta appears to be very slight. With the exception of the lungs, inorganic tin does not accumulate in the soft organs with increasing age (Mag86).

Inorganic tin is mainly excreted in the urine. The fraction excreted with the bile varies with the type of compound and is probably below 15% (WHO80). The tin concentrations in the urine of Americans have been estimated to average 0.023 mg/L, with a range from 0-0.040 mg/L (Bas89).

The committee concludes that respirable tin oxide dusts is very poorly absorbed through the lungs, most probably due to its insolubility in water.

6 Effects and mechanism of action

Human data

There is no epidemiological data available. Various case-reports on effects due to occupational exposure to tin oxide have been published. Long-term occupational exposure by inhalation to tin oxide aerosols results in accumulation of the compound in the lung tissues, since it is poorly absorbed. This means that the lungs are the target organ.

The clinical picture in the lungs has been called benign pneumoconiosis (Cut49, Dun50, Pen48, Rob49, Rob61, Spen54, Zor66). The diagnosis was mostly based on chest X-rays. In some cases, the patients were asymptomatic with normal lung functions (Cut49, Zor66). Dundon and Hughes described a case of a 72-year-old man who complained of constant lower abdominal pain during 2 weeks, nocturia, and a rapid loss of body weight. Occupational history of the patient showed that he had been working for 18 years near a de-tinning furnace, in which tin oxide was recovered from tin scrap by a calcining process. He did not have any symptoms of respiratory disease during his working life. Nine years after his last exposure, he was admitted to a hospital because of epigastric pain and vomiting. A chest roentgenogram showed a 'peculiar widespread mottling of both lung fields by discrete shadows'. There were no respiratory complains. The vital capacity of the lungs was 85% of normal value (Dun50).

Robertson et al. examined the lungs of 7 deceased subjects who had been employed in the tin-melting industry using chemical and X-ray-diffraction analysis and X-ray-emission spectroscopy. They found evidence of pneumoconiosis with nodular opacities, each about 3 to 5 mm in size, in 6 of the subjects. Macroscopic examination of the lung tissues showed black or dark dust foci, 2 to 5 mm in size and distributed uniformly. No silicotic or fibrous nodules were seen. Microscopically, the dust foci consisted of dense aggregates of dust-laden macrophages surrounding the respiratory bronchioles. Sometimes, the dust deposition was more extensive and it surrounded the entire length of the terminal non-respiratory bronchiole. A few dust-laden cells were seen lying free in the alveoli. Chemical analysis and X-ray-diffraction analysis showed that the lungs contained tin oxide. X-ray-emission microanalysis identified the tin in the lung phagocyte (Rob61).

More recently, Sluis-Cremer et al. argued whether this form of abnormality could be called pneumoconiosis at all, since no tissue reaction of any kind was found. They reported 2 cases in which both subjects showed moderately profused small nodules on radiographic examination of the chest. One subject had been employed in a tin mine for 26 years. He was free of symptoms and credited as clinically normal. The other subject had been employed in a de-tinning plant for 15 years with minor complaints of productive cough, and basal crackles were present on auscultation. The forced expired vital capacity (FVC) of the lungs was 90% and the one-second forced expiratory volume (FEV_1) 96% of predicted values. Lung biopsy of this patient showed focal aggregations of macrophages containing blackish dust particles in some of the air spaces and in the perivascular and peribronchial connective tissue. There was a slight increase of

perivascular and peribronchial connective tissue, but the dust deposits in the air spaces were not associated with reticulin or collagen deposition. Electron microscopy analysis displayed tin to be present in the dust. Exposure levels were not reported (Slu89).

Animal data

The committee did not find experimental animal data on the potential irritation or sensitisation of the tin oxides.

Schafer and Bowles reported an oral LD_{50} of 775 mg/kg bw for tin(II) oxide in deer mice (Sch85).

De Groot et al. claimed that there were marked differences in the toxicity of various oxides and salts of tin in rats when given by oral administration. In their experiments, groups of rats (Wistar; n=10/sex/group) were fed diets containing 0, 0.03, 0.10, 0.30, or 1.00% of various tin salts or oxides for periods of either 4 or 13 weeks. Animals received tin(II) and tin(IV) oxide for 13 and 4 weeks, respectively. Effects on behaviour, mortality, body weights, food consumption, blood, urine, and biochemical parameters, and organ weights were examined and gross and microscopic examinations were performed. No adverse effects were noted at any dose of both tin oxides, as well as stannous sulphide and oleate. However, severe growth retardation, decreased food efficiency, slight anaemia, and slight histological changes in the liver were observed with 0.3% or more of stannous chloride, orthophosphate, sulphate, oxalate, and tartrate. The authors concluded that the differences in response to different tin compounds suggested that insoluble tin compounds are relatively harmless substances whereas cationic tin compounds soluble in water or dilute acid may be toxic at dietary levels above 0.1% (Gro73). From these studies, the committee concludes that dietary exposure to levels of tin(II) and tin(IV) oxide up to 1% for 13 and 4 weeks, respectively, did not induce any effect in rats. From data on initial body weight, body weight gain, and total food intake of animals exposed to tin(IV) oxide in the 4-week experiment with tin(IV) oxide and from data on body weight and food intake of control animals during the several time points in the 13-week study with tin(II) oxide*, the committee estimates that the daily intakes may have been about 700-850 mg tin per kg bw.

The committee did not find data on long-term exposure by inhalation or on carcinogenicity, mutagenicity/genotoxicity, and reproduction toxicity.

---

* For the 13-week study, only data on animals exposed to tin(II) chloride and control animals were presented.

7 Existing guidelines

The current administrative occupational exposure limit (MAC) for tin oxide in the Netherlands is 2 mg/m^3, 8-hour TWA .

Existing occupational exposure limits for tin oxide in some European countries and in the USA are summarised in the annex.

8 Assessment of health hazard

The tin oxides are poorly absorbed from the respiratory and intestinal tract resulting in little systemic toxicity.

Numerous cases of workers occupationally exposed to tin oxide dusts, aerosols, or fumes have shown that these can produce a benign pneumoconiosis, called stannosis, which is characterised by small dense shadows in the pulmonary X-ray picture. However, no fibrosis or impairment of lung function is observed. The committee did not find human data on exposure-response relationships.

With respect to experimental animal data on tin oxides, the committee only found an oral study in which rats were given tin(II) and tin(IV) oxide in their diets for 13 and 4 weeks, respectively. At the maximum dose level tested of 1%, estimated to be about 700-850 mg tin/kg bw/day, no effects were observed.

The committee considers the toxicological database on tin (IV) and tin (II) oxide too poor to justify recommendation of a health-based occupational exposure limit.

The committee concludes that there is insufficient information to comment on the level of the present MAC-value.

References

ACG96 American Conference of Governmental Industrial Hygienists (ACGIH). Tin. In: TLVs® and other occupational exposure values - 1996. [CD-ROM]. Cincinnati OH, USA: ACGIH®, Inc, 1996.

ACG03a American Conference of Governmental Industrial Hygienists (ACGIH). Guide to occupational exposure values - 2003. Cincinnati OH, USA: ACGIH®, Inc, 2003: 130.

ACG03b American Conference of Governmental Industrial Hygienists (ACGIH). 2003 TLVs® and BEIs® based on the documentation of the Threshold Limit Values for chemical substances and physical agents & Biological Exposure Indices. Cincinnati OH, USA: ACGIH®, Inc, 2003: 56.

Arb02 Arbejdstilsynet. Grænseværdier for stoffer og materialer. Copenhagen, Denmark: Arbejdstilsynet, 2002: 38 (At-vejledning C.0.1).

Bas89 Baselt RC, Cravey RH. Tin. In: Disposition of toxic drugs and chemicals in man. 3rd ed. Chicago IL, USA: Year Book Medical Publ, Inc, 1989.

Bud89 Budavari S, O'Neil MJ, Smith A, et al. The Merck index. An encyclopedia of chemicals, drugs, and biologicals. 11th ed. Rahway NJ, USA: Merck & Co, Inc, 1989: 1384, 1385.

Cut49 Cutter HC, Faller WW, Stocklen JB, et al. Benign pneumoconiosis in a tin oxide recovery plant. J Ind Hyg Toxicol 1949; 31: 139-41.

DFG02 Deutsche Forschungsgemeinschaft (DFG): Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area. List of MAK and BAT values 2002. Maximum concentrations and biological tolerance values at the workplace. Weinheim, FRG: Wiley-VCH, 2002: 104 (rep no 38).

Dun50 Dundon CC, Hughes JP. Stannic oxide pneumoconiosis. Am J Roentgenol Radium Ther Nucl Med 1950; 63: 797-812.

EC03 European Commission: Directorate General of Employment and Social Affairs. Occupational exposure limits (OELs). http://europe.eu.int/comm/employment_social/h&s/areas/oels_en.htm.

Gro73 De Groot AP, Feron VJ, Til HP. Short-term toxicity studies on some salts and oxides of tin in rats. Food Cosmet Toxicol 1973; 11: 19-30.

HSE02 Health and Safety Executive (HSE). EH40/2002. Occupational Exposure Limits 2002. Sudbury (Suffolk), England: HSE Books, 2002: 27.

Mag86 Magos L. Tin. In: Friberg F, Nordberg GF, Vouk VB, eds. Handbook on the toxicology of metals. 2nd ed. Amsterdam, the Netherlands: Elsevier, 1986: 568-93 (Vol II).

Pen48 Pendergrass EP, Pryde AW. Benign pneumoconiosis due to tin oxide. J Ind Hyg Toxicol 1948; 30: 119-23.

Rob49 Robertson AJ, Whitaker PH. Radiological changes in pneumoconiosis due to tin oxide. J Fac Radiol 1949; 6: 224-33.

Rob61 Robertson AJ, Rivers D, Nagelschmidt G, et al. Benign pneumoconiosis due to tin oxide. Lancet 1961; i: 1089-93.

Sch85 Schafer EW Jr, Bowles WA Jr. Acute oral toxicity and repellency of 933 chemicals to house and deer mice. Arch Environ Contam Toxicol 1985; 14: 111-29.

Sch64 Schroeder HA, Balassa JJ, Tipton IH. Abnormal trace metals in man: tin. J Chronic Dis 1964; 17: 483-502.

Slu89 Sluis-Cremer GK, Thomas RG, Goldstein B, et al. Stannosis. A report of 2 cases. S Afr Med J 1989; 75: 124-6.

Spe54 Spencer GE, Wycoff WC. Benign tin oxide pneumoconiosis. AMA Arch Ind Hyg Occup Med 1954; 10: 295-7.

Swe00 Swedish National Board of Occupational Safety and Health. Occupational exposure limit values and measures against air contaminants. Solna, Sweden: National Board of Occupational Safety and Health, 200; Ordinance AFS 2000:3.

SZW03 Ministerie van Sociale Zaken en Werkgelegenheid (SZW). Nationale MAC-lijst 2003. The Hague, the Netherlands: Sdu, Servicecentrum Uitgevers, 2003: 41.

TRG00 TRGS 900. Grenzwerte in der Luft am Arbeitsplatz; Technische Regeln für Gefahrstoffe. BArbBl 2001; 2.

Wea87 Weast RC, ed. CRC handbook of chemistry and physics. 68th ed. Boca Raton FL, USA: CRC Press, Inc, 1987: B-140.

WHO80 World Health Organization: International Programme on Chemical Safety (WHO/IPCS). Tin and organotin compounds. A preliminary review. Geneva, Switzerland: WHO, 1980; Environmental Health Criteria 15.

Zor66 Zorn O. Barytose und Zinnoxidelunge. Zentralbl Arbeitsmed 1966; 16: 133-5.

Annex

Occupational exposure limits for tin oxides in various countries.

country - organisation	occupational exposure limit[a]		time-weighted average	type of exposure limit	note[b]	reference[c]
	ppm	mg/m^3				
the Netherlands						
- Ministry of Social Affairs and Employment	-	2[d]	8 h	administrative		SZW03
Germany						
- AGS	-	2[e, f]	8 h			TRG00
	-	8[e, f]	15 min			
- DFG MAK-Kommission	-	-[g]				DFG02
Great Britain						
- HSE	-	2[e]	8 h	OES		HSE02
	-	4[e]	10 min			
Sweden	-	-				Swe00
Denmark	-	2[e]	8 h			Arb02
USA						
- ACGIH	-	2[h]	8 h	TLV		ACG03b
- OSHA	-	-				ACG03a
- NIOSH	-	2	10 h	REL		ACG03a
European Union						
- SCOEL	-	-				EC03

[a] As tin.
[b] S = skin notation, which means that skin absorption may contribute considerably to body burden; sens = substance can cause sensitisation.
[c] Reference to the most recent official publication of occupational exposure limits.
[d] Holds for tin (IV) oxide.
[e] Holds for inorganic tin compounds (except, in most cases, tin hydride (SnH_4)).
[f] Inhalable dust.
[g] Listed among substances for which studies of the effects in man or experimental animals had yielded insufficient information for the establishment of MAK values.
[h] Holds for oxides and inorganic compounds, except tin hydride (SnH_4).