Antimony sulfide (Sb2S3): Human health tier II assessment

21 April 2016

CAS Number: 1345-04-6

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:www.nicnas.gov.au

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	diantimony trisulfide antimony orange antimony vermilion antimonous sulfide CI pigment red 107
Structural Formula	
Molecular Formula	S3Sb2
Molecular Weight (g/mol)	339.72
Appearance and Odour (where available)	Black crystalline solid or amorphous red-orange powder
SMILES	S{2-}1.[Sb]{3+}(.S{2-}).S{2-}.[Sb]{3+}.1

Import, Manufacture and Use

Australian

The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was between 100–1000 tonnes.

No specific Australian use, import, or manufacturing information has been identified.

International

The following international uses have been identified through: the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; Galleria Chemica; the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments (National Toxicology Program (NTP) and the International Agency for Research on Cancer (IARC)).

The chemical has reported commercial uses, including:

- as a photoconductor in video camera tubes for CCTV and film service (widely used in the medical field);
- as a solid lubricant for friction linings and brake pads;
- for manufacturing matches or Bengal lights;
- as a filler in resinoid bonding abrasives;
- as a red or yellow pigment for manufacturing ruby glass;
- as an enamel compound; and
- as a constituent of camouflage paints, smoke or marine markers, visual fire control and signalling.

The chemical has reported site-limited uses, including:

- as a primer for military ammunition;
- for producing high-quality pyrotechnics such as electrically ignited detonators;
- as a starting material for producing antimony and other antimony compounds such as antimony oxide and chloride; and
- the colloidal form is used in laboratory and clinical imaging (bone marrow imaging, lymphoedema assessment, and scintigraphic mapping).

The chemical has reported non-industrial use (alone or in combination with other products) as an oral homeopathic remedy (NTP, 2002).

Restrictions

Australian

The chemical is not specifically listed in the *Poisons Standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP). However, the chemical is covered by the Schedule 6 listing for 'antimony compounds' in the SUSMP (SUSMP, 2016).

Schedule 6:

'ANTIMONY COMPOUNDS **except**:

(a) when included in Schedule 4;

(b) antimony chloride in polishes;

(c) antimony titanate pigments in paint; or

(d) in paints or tinters containing 5 per cent or less of antimony calculated on the non-volatile content of the paint or tinter.'

Schedule 6 chemicals are described as 'Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label'. Schedule 6 chemicals are labelled with 'Poison' (SUSMP, 2016).

Antimony or antimony compounds other than antimony titanate pigments (>5 %, calculated as a percentage of the element present in the non-volatile content of the paint), are also listed in Section Seven - Appendix I (Paint or tinters) of SUSMP under the First Group, with restrictions for manufacture, sale, supply or use/applications (SUSMP, 2016).

International

The chemical is listed on the following (Galleria Chemica):

- EU Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain;
- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient ‘Hotlist’);
- The Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex II Part 1: List of substances which must not form part of the composition of cosmetic products;
- China List of substances banned for use in cosmetics; and
- Philippines ingredients restricted for use in cosmetics—List of substances which must not form part of the composition of cosmetic products.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

Exposure Standards

Australian

The chemical has an exposure standard of 0.5 mg/m^3 time weighted average (TWA).

International

The following exposure standards are identified (Galleria Chemica):

TWA of:

- 0.5 mg/m^3 in Canada, China, Denmark, France, Ireland, Netherlands, Norway, Singapore, South Africa, Spain, United Kingdom and the USA;
- 0.1 mg/m^3 in Japan;
- 0.3–2 mg/m^3 in Russia; and
- 1 ppm in Sweden.

Short-term exposure limit (STEL) of:

- 0.75–1.50 mg/m^3 in Canada (Saskatchewan and Yukon) and the USA (Washington); and
- 2 mg/m^3 in Hungary.

Health Hazard Information

Based on the chemical having similar bioaccessibility, valency and poor water solubility to antimony oxide (NTP, 2005; Skeaff et al., 2012), toxicological data from the extensively studied antimony oxide are used in this assessment, where appropriate.

Toxicokinetics

The chemical can be absorbed via inhalation and ingestion, although absorption from the gastrointestinal tract in humans is reported to be poor (IPCS, 1998). A reference value of 1 % for gastrointestinal absorption has been recommended for antimony in humans (ATSDR, 1992).

Case studies in smelter workers have shown that the inhaled chemical can be deposited and retained in the lungs for several years. Following absorption, antimony accumulates in the vascular system of organs and tissues, mainly in the liver and kidneys (NTP, 2002). Occupational exposure to antimony trisulfide via inhalation at concentrations >3 mg/m^3 (0.003 mg/L) resulted in urinary excretion of 0.8–9.6 mg/L antimony (IARC, 1989).

Female dogs which inhaled antimony trisulfide dust from a smelter (particle size <2μm) at a concentration of 5.5 mg/m^3 (0.0055 mg/L) for 10 weeks, excreted 16–18 mg/L antimony in the urine (IARC, 1989).

In an in vitro bioaccessbility study conducted in accordance with the OECD Series on Testing and Assessment: Guidance Document Number 29, the solubility of antimony trisulfide particles (median values of 5.8–26.2 μm) was tested in five different simulated human body fluids with pH ranging from 1.6 to 7.4, for two and 24 hours. The particles dissolved at least partially in all fluids, with the degree of dissolution dependent on the duration and solution composition. The particles dissolved to the highest extent (8.5 %) at pH 7.4 after 24 hours. There was no clear trend in pH dependence for solubility (REACH).

Acute Toxicity

Oral

The chemical has low acute toxicity in rats following oral exposure.

In an acute oral study conducted according to the OECD Test Guideline (TG) 401, the median lethal dose (LD50) in rats was greater than 2000 mg/kg bw (REACH).

Dermal

The chemical has low acute dermal toxicity in rats.

The dermal LD50 in rats was >2000 mg/kg bw (REACH).

Inhalation

The chemical has low acute toxicity in rats following inhalation exposure.

The median lethal concentration (LC50) in rats was >5.04 mg/L after four hours' exposure (REACH). The particle diameter for the chemical was reported to be 4.6 µm. Black foci in the lungs of rats was observed; however, there were no deaths or other treatment-related symptoms of toxicity over an observation period of 15 days (NTP, 2002).

Observation in humans

In a case study, a 24-year old woman ingested antimony sulfide (dose not stated) and was hospitalised one hour later. Although clinical examination results were normal, the patient complained of epigastralgia (pain in the epigastric region), dysphagia (swallowing difficulties) and a metallic taste in the mouth. Immediate treatment was given and continued for five days. The patient did not show signs of intoxication, and routine biological tests remained in the normal range. The patient was discharged after six days (IPCS, 1998; NTP, 2002).

Cases of accidental antimony poisoning were reported following the use of old porcelain houseware. It was suggested that acidic beverages caused the release of antimony from these houseware, at concentrations sufficient to cause toxicity (NTP, 2002).

In a radiological investigation, six workers exposed to antimony smelter fumes (average airborne concentration of 10–12 mg/m^3) for two to 12 hours displayed evidence of pneumonitis. Lung inflammation was perihilar (surrounding the hilum of the lung) with no peripheral parenchymal damage observed. The symptoms were reduced following removal from exposure and treatment with penicillin aerosols (IPCS, 1998).

Corrosion / Irritation

Skin Irritation

No animal data are available.

Eye Irritation

The chemical is not considered an eye irritant.

In an eye irritation study (OECD TG 405), New Zealand White rabbits (two males and one female) were exposed to the pure chemical (dose not specified). Observations were made at one, 24, 48 and 72 hours, and seven and 14 days following administration. The mean scores for ocular lesions at 24, 48 and 72 hours were calculated to be 0.22 for corneal opacity, 0 for iris lesion, 1.44 for redness of the conjunctivae and 1 for chemosis. Most of the effects were fully reversible within 14 days and the chemical was not considered to be an eye irritant (REACH).

Observation in humans

There are some reports indicating antimony compounds can cause skin, eye and respiratory irritation in humans. However, the information available is not sufficient to warrant hazard classification.

Exposure to antimony compounds in occupational settings was reported to cause skin, eye and respiratory irritation. The major source of antimony sulfide exposure is as antimony sulfide ore (stibnite), which may contain arsenic and antimony oxide (from ore smelting) (IARC, 1989; NTP, 2002).

Transient skin eruptions, known as 'antimony spots' were common among workers exposed to antimony and antimony salts. These eruptions mainly occurred following exposure to heat and when sweating occurred (NTP, 2002).

The dust and fumes of antimony sulfide were reported to irritate the respiratory tract and mucous membranes, causing symptoms such as laryngitis, pharyngitis, rhinitis and bronchitis. However, available case reports are of combined exposure with antimony oxide, which is produced from the antimony sulfide ore smelting process (IPCS, 1998). One inhalation study mentioned that respiratory irritation was not observed in workers exposed to antimony sulfide only, for eight months to two years; however, other toxicity effects were present (see **Repeat Dose Toxicity – Observation in humans**) (ATDSR, 1992).

Sensitisation

Skin Sensitisation

No data for the chemical are available.

Antimony oxide is not a skin sensitiser in animal studies (NICNAS).

Repeated Dose Toxicity

Oral

No data are available.

Dermal

No data are available.

Inhalation

Cardiotoxicity was observed in animal studies, in addition to lung and liver effects. Heart dilation with degenerative changes in the myocardium (heart failure) was observed in rats and rabbits at 3.1 mg/m^3–5.6 mg/m^3, but not in dogs at 5.6 mg/m^3. Based on these effects in animals, the chemical may cause severe effects following repeated inhalation exposure. However, the current information in both humans and animals is inconsistent, and do not warrant hazard classification.

Read-across data from the oxides has been used for antimony sulfide based on bioelution and bioaccessibility testing (NTP, 2005; Skeaff et al., 2012). As hazard classification for antimony oxide for repeated dose inhalation toxicity is being considered under a Tier III assessment (NICNAS), this report will be amended if necessary following the antimony oxide Tier III assessment, if required.

In a repeated dose inhalation toxicity study (non-guideline), rats (n = 10) and rabbits (n = 11) were exposed to antimony sulfide dust at 3.1 and 5.6 mg/m^3, respectively, seven hours/day, five days/week for six weeks. Dogs (n = 4/dose) were exposed to antimony sulfide dust at 5.3 mg/m^3 for seven weeks and to 5.6 mg/m^3 for 10 weeks. Electrocardiographic (ECG) changes in the heart were observed (time not stated) with elevations in the RS-T segment and flattening of T-waves in all animals. The primary

pathologic observation was heart dilation with degenerative changes in the myocardium (hyperaemia and swelling of myocardial fibres), in all animals. Lung effects (focal haemorrhage and congestion) were secondary to heart failure. Parenchymal degeneration in the liver was also observed in rabbits. These cardiotoxic changes were observed in dogs exposed at the higher dose only (5.6 mg/m^3), but were less severe compared with rats and rabbits (IARC, 1989; ATSDR, 1992; NTP, 2002).

In another repeated dose inhalation toxicity study (non-guideline), Sprague Dawley (SD) rats (n = 10/sex) were exposed to antimony sulfide ore at 1700 mg/m^3 for one hour, one to six months every two months, up to 12 months. The animals developed acute transitory pneumonitis following the first exposure. Although inflammation in the lungs of the animals was not prominent at the end of exposure, dust-laden phagocytic cell deposits within the alveolar septae were observed during periodic sacrifice (NTP, 2002).

In a combined chronic carcinogenicity study (see **Carcinogenicity**), Wistar rats (n = 90/sex/group) were exposed to antimony ore concentrate (consisting of 46 % antimony primarily as antimony sulfide, <4 % titanium, 0.5 % aluminium, 0.2 % tin, 0.3 % lead and iron, and 0.08 % arsenic) at 0 or 36–40 mg/m^3, seven hours/day, five days/week for 52 weeks. Lung lesions (interstitial fibrosis, alveolar cell hyperplasia and metaplasia) were observed in both sexes (time not stated), but were reported to be less severe in males. It was noted that arsenic was also present together with antimony in the lungs (IARC, 1989; NTP, 2002).

Observation in humans

Repeated exposure to antimony compounds is reported to cause symptoms such as headache, joint or muscular pain, vertigo, coughing, loss of sleep and appetite. Gastrointestinal disorders following exposure to antimony sulfide occurred more rapidly than as observed for arsenic, resulting in prominent abdominal pain, diarrhoea, vomiting and ulcers (ATSDR, 1992; NTP, 2002).

Similar to reports in animals (see **Repeat dose toxicity – inhalation**), increased mortality and morbidity from heart disease were reported among workers in the abrasives industry. In a case study, 124 workers were exposed to antimony sulfide at airborne concentrations of 0.6–5.5 mg/m^3 for eight months to two years. Six sudden deaths occurred and another two deaths occurred from chronic heart disease; four of which were under the age of 45. Among the 75 workers examined, ECG changes (primarily T-wave) were seen in 37 workers. Following cessation of exposure, no more deaths due to heart disease and no abnormal increase in heart effects were reported. However, ECG changes persisted in 12 out of 56 workers who were re-examined (IARC, 1989, NTP, 2002). After further evaluation, it was noted that an estimation of coronary heart risk following antimony exposure was not possible due to a cohort age effect not taken into consideration and the lack of a control group (NTP, 2002).

Other occupational case studies reported for antimony sulfide involved combined exposure to other antimony compounds (in particular, antimony oxide) and arsenic.

A case study in a Newcastle factory found that the number of deaths due to heart disease among male antimony workers was lower than or equal to expected values in the region. However, the lungs of 44 out of 262 men displayed 'simple pneumoconiosis'. The duration of employment could be associated with the antimony content in the lungs (NTP, 2002).

In another case study in an antimony smelter in Texas (1937–1971), no statistically significant increases in deaths due to heart disease were observed in a total amount of 1014 workers. There were 28 workers employed between one to 15 years at the Texas smelter who were examined using X-rays, and pneumoconiosis was confirmed in three out of five suspected cases. The estimated airborne concentrations of antimony in 1966 were ~0.081–75 mg/m^3, and ~0.05–6.2 mg/m^3 between 1975–1976 (NTP, 2002).

However, another study (documented in 1948) reported that workers exposed to airborne concentrations of around 30–37 mg/m^3 antimony did not show adverse lung effects on X-ray examination, and no antimony was detected in the blood or urine (NTP, 2002).

Genotoxicity

No data are available for the chemical.

Antimony compounds are stated to have no mutagenic properties (NTP, 2002).

Carcinogenicity

Only one study in rats is available. Based on the limited data, the chemical is not considered to be carcinogenic.

The IARC has classified the chemical as '*Not classifiable as to its carcinogenicity to humans*' (Group 3), based on inadequate evidence for carcinogenicity in humans, and limited evidence for carcinogenicity in animal testing (IARC, 1989).

In a chronic carcinogenicity study, Wistar rats (n = 90/sex/group) were exposed to antimony ore concentrate by inhalation at 0 or 36–40 mg/m^3, seven hours/day, five days/week for 52 weeks. Animals (n = 15/sex) were euthanised at six, nine and 12 months, and the remaining animals at 18–20 weeks following cessation of exposure. No significant difference in survival rates was observed between the treated and control groups. The first tumour was observed in the lungs of one female that died at 41 weeks following commencement of exposure. Treatment-related lung tumours (bronchoalveolar carcinomas, squamous cell carcinomas and scirrhous cell carcinomas) were observed in 17/68 females that survived after observation of the first lung tumour. Male rats did not develop lung tumours, although a higher concentration of antimony was found in their lungs compared with females (IARC, 1989; NTP, 2002).

Some case studies have indicated a possible association between occupational antimony exposure and an increased risk of lung cancer. However, no conclusions can be drawn due to concomitant exposure to other chemicals such as antimony oxide and arsenic, which are carcinogens (NTP, 2002).

Reproductive and Developmental Toxicity

No data for the chemical are available.

Studies for antimony oxide do not indicate specific reproductive or developmental toxicity (NICNAS).

Risk Characterisation

Critical Health Effects

The chemical may cause harmful effects following repeated exposure to high concentrations by inhalation.

Public Risk Characterisation

Given the uses identified for the chemical, it is unlikely that the public will be exposed. Hence, the public risk from this chemical is not considered to be unreasonable.

Occupational Risk Characterisation

The primary source of exposure to antimony sulfide is as antimony sulfide ore (stibnite), which may contain arsenic and other antimony compounds such as antimony oxide,

Given the critical systemic long-term health effect for antimony compounds in general, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise inhalation exposure are implemented. The chemical should be appropriately labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine the appropriate controls.

Based on the available data, the hazard classification in the Hazardous Substances Information System (HSIS) (Safe Work Australia) is considered appropriate.

NICNAS Recommendation

Current risk management measures are considered adequate to protect public and workers' health and safety, provided that all requirements are met under workplace health and safety, and poisons legislation as adopted by the relevant state or territory.

The chemical is not recommended for classification under the current approved criteria and adopted GHS. The current exposure standard is considered adequate to protect workers from inhalation hazard.

Further assessment of this chemical may be necessary if new hazard data become available on antimony oxide that could be relevant to this chemical.

Regulatory Control

Advice for industry

Control measures

Control measures to minimise the risk from inhalation exposure to the chemical should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical are is used. Examples of control measures that could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemicals from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;

- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemical has not been undertaken as part of this assessment.

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