IPCSINTOX Databank

UKPID MONOGRAPH

ANTIMONY TRIOXIDE

WN Harrison PhD CChem MRSC
SM Bradberry BSc MB MRCP
ST Beer BSc
JA Vale MD FRCP FRCPE FRCPG FFOM

National Poisons Information Service
(Birmingham Centre),
West Midlands Poisons Unit,
City Hospital NHS Trust,
Dudley Road,
Birmingham
B18 7QH

This monograph has been produced by staff of a National Poisons Information Service Centre in the United Kingdom. The work was commissioned and funded by the UK Departments of Health, and was designed as a source of detailed information for use by poisons information centres.

Peer review group: Directors of the UK National Poisons Information Service.

ANTIMONY TRIOXIDE

Toxbase summary

Type of product

Used mainly as a fire retardant in plastics, rubbers, textiles, paper and paints.

Toxicity

Acute antimony trioxide poisoning is rare. Exposure may occur in industry. Fatal dose not known.

Features

Topical

- Irritant to the skin and eyes.
- "Antimony spots" (papules and pustules around sweat and sebaceous glands) may develop after repeated exposure, particularly in warm conditions.

Ingestion

Moderate ingestions:

- Features usually occur within two hours with nausea,

vomiting, abdominal pain and diarrhoea. A garlic odour on the breath has been described following ingestion of other antimony salts.

Substantial ingestions:

- Severe vomiting and diarrhoea (which may contain blood) and haemorrhagic gastritis may ensue. Myocardial depression, vasodilation and fluid loss may cause shock with hypotension, electrolyte disturbances and acute renal failure. Cerebral oedema, coma and convulsions are possible. A fatality occurred following ingestion of a soluble antimony trioxide derivative (Miller, 1982).

Inhalation

- Irritant to the respiratory tract and mucous membranes causing conjunctivitis, laryngitis, pharyngitis, tracheitis, rhinitis bronchitis and rarely non-cardiogenic pulmonary oedema.
- There may be radiological evidence of pneumonitis.
- Chronic occupational inhalation may cause pneumoconiosis with cough, wheeze and diffuse, punctate opacities in the middle and lower zones.

Management

Dermal

1. If possible the patient should remove soiled clothing and wash him/herself.
2. Wash contaminated hair and skin with soap and copious amounts of water.
3. Pay special attention to skin folds, fingernails and ears.
4. A physician may need to examine the area if irritation or pain persists after washing.

Ocular

1. Immediately irrigate the affected eye thoroughly with tepid water or 0.9% saline for at least 10-15 minutes.
2. Any particles lodged in the conjunctival recesses should be removed.
3. Continue irrigation with saline infusion using drip tubing.
4. Repeated instillation of local anaesthetic may reduce discomfort and help more thorough decontamination.
5. Corneal damage may be detected by instillation of fluorescein.
6. Patients with corneal damage and those whose symptoms do not resolve rapidly should be referred for ophthalmological assessment.

Ingestion

Minor ingestions (very mild or no symptoms):

1. Gastrointestinal decontamination is unnecessary.
2. Symptomatic and supportive measures only.

Moderate/substantial ingestions:

1. Gastric lavage should be considered only if the patient presents within one hour; its value is unproven.
2. Symptomatic and supportive measures as dictated by the patient's condition.
3. Monitor the ECG, biochemical and haematological profiles.
4. Collect urine and blood for antimony concentration measurements to confirm diagnosis although these assays are not widely

available. Check with NPIS.
5. Chelation therapy with dimercaprol, DMSA or DMPS may be considered; seek specialist advice from an NPIS physician.

Inhalation

Acute exposure
1. Remove from exposure.
2. Secure cardiorespiratory stability.
3. Perform a chest X-ray in symptomatic patients.
4. Treat symptomatically.

5. If significant respiratory symptoms occur investigate for systemic toxicity: ECG, biochemical and haematological profiles and blood and urine samples for antimony concentration determination.

Chronic exposure
1. Investigate as for other causes of pneumoconiosis.
2. Obtain blood and urine for antimony concentration measurements to confirm diagnosis. However, these assays are not widely available. Check with NPIS.
3. Consider the possibility of systemic toxicity.

References

Bailly R, Lauwerys R, Buchet JP, Mahieu P, Konings J.
Experimental and human studies on antimony metabolism: their relevance for the biological monitoring of workers exposed to inorganic antimony.
Br J Ind Med 1991;48: 93-7.

Lauwers LF, Roelants A, Rosseel M, Heyndrickx B, Baute L.
Oral antimony intoxications in man.
Crit Care Med 1990; 18: 324-6.

Miller JM.
Poisoning by antimony: a case report.
South Med J 1982; 75: 592.

Renes LE.
Antimony poisoning in industry.
Arch Ind Hyg Occup Med 1953; 7: 99-108.

Werrin M.
Chemical food poisoning.
Q Bull Assoc Food Drug Offic 1963; 27: 38-45.

White Jr GP, Mathias CGT, Davin JS.
Dermatitis in workers exposed to antimony in a melting process.
J Occup Med 1993; 35: 392-5.

Winship KA.
Toxicity of antimony and its compounds.
Adverse Drug React Acute Poisoning Rev 1987; 2: 67-90.

Substance name

Antimony trioxide

Origin of substance

Minerals such as senarmontite and valentinite.
(MERCK, 1996)

Manufactured by roasting antimony trisulphide ores.
(IARC, 1989)

Synonyms

Diantimony trioxide
Antimony white
Exitelite
Flowers of antimony
Valentinite (DOSE, 1992)

Chemical group

A compound of antimony, a group VA element

Reference numbers

CAS	1309-64-4	(DOSE, 1992)
	1327-33-9	(CSDS, 1989)
RTECS	CC 5650000	(CSDS, 1989)
UN	1549	(CSDS, 1989)
HAZCHEM	NIF	

Physicochemical properties

Chemical structure
Sb_2O_3 (DOSE, 1992)

Molecular weight
291.5 (DOSE, 1992)

Physical state at room temperature
Crystalline solid (CSDS, 1989)

Colour
White (CSDS, 1989)

Odour
None (CHRIS, 1997)

Viscosity
NA

pH
NIF

Solubility
Slightly soluble in water. Soluble in potassium hydroxide, hydrochloric acid. Insoluble in organic solvents.
(CSDS, 1989)

Autoignition temperature
NA

Chemical interactions
Reacts with organic acids, alcohols, glycols, alpha-hydroxy acids, o-dihydric phenols, sugar alcohols and other polyhydroxy compounds. (OHM/TADS, 1997)

Major products of combustion
When heated to combustion emits toxic antimony fumes.
(HAZARDTEXT, 1997)

Explosive limits

NIF

Flammability
Ignites on heating in air. (CSDS, 1989)

Boiling point
1550°C (DOSE, 1992)

Density
5.2 (DOSE, 1992)

Vapour pressure
NA

Relative vapour density
NA

Flash Point
NA

Reactivity
Reacts explosively with chlorinated rubber.
Forms explosive mixtures with perchloric acid when hot.
(CSDS, 1989)

Uses

Fire retardant in plastics, rubbers, textiles, paper and paints.
(IARC, 1989)
In enamels and glass.
Tartar emetic (antimony potassium tartrate) manufacture.
(DOSE, 1992)

Hazard/risk classification

Index no. 051-005-00-X
Risk phases
Carc. Cat. 3; R40 - Possible risk of irreversible effects.
Xn; R40 - Harmful, possible risk of irreversible effects.
S(2-)22-36 - Keep out of reach of children. Do not breathe dust.
Wear suitable protective clothing.
EEC No. 215-175-0 (CHIP2, 1994)

INTRODUCTION

Antimony trioxide is a trivalent antimony compound which occurs naturally as the ores valentinite and senarmontite. It is produced commercially by the vapour phase reaction of antimony trisulphide and oxygen.

It is used as a fire retardant and as an additive in enamel and glass manufacture.

EPIDEMIOLOGY

The main route of antimony trioxide exposure is occupational inhalation of dusts. Workers have been exposed in the production of antimony trioxide (Renes, 1953; Schnorr et al, 1995) and from its formation as a by-product of metal smelting (Gerhardsson et al, 1982; Potkonjak and Pavlovich, 1983; Jones, 1994; Schnorr et al, 1995).

Other occupational exposure has occurred in the manufacture of ceramics (Motolese et al, 1993), batteries (Kentner et al, 1995) and alloys (White et al, 1993).

Accidental intoxication has been reported after leaching of antimony from agate and ceramic containers into acidic beverages (Dunn, 1928; Monier-Williams, 1934; Werrin, 1963).

It has been suggested that stibine generated from microbial growth on cot mattresses containing antimony trioxide is a contributing factor in sudden infant death syndrome (SIDS). Although there is some evidence consistent with undue antimony exposure in infants who died of SIDS (Taylor, 1996) other evidence disagrees such that it is most unlikely stibine is the only cause of the syndrome (de Wolff, 1995). A provisional comment by the Chair of the UK Expert Group on Cot Death Theories states so far "there is no evidence of risk to babies" from cot mattress PVC (Bradbury, 1997).

Historically, intoxication has resulted from the medical use of antimony trioxide and potassium tartrate as tartar emetic in the treatment of a variety of conditions including malaise, fever, whooping cough and syphilis (Miller, 1982).

MECHANISM OF TOXICITY

The mechanism of toxicity of antimony compounds is unclear but may involve disruption of thiol proteins via binding to sulphydryl groups (de Wolff, 1995).

TOXICOKINETICS

Absorption

Antimony trioxide may be absorbed by inhalation and ingestion, though gastrointestinal absorption in man is poor.

Distribution

Absorbed trivalent antimony readily enters red blood cells and accumulates primarily in the spleen, liver and bone (IPCS, 1996).

Lauwers et al (1990) estimated that the total body antimony pool in a patient who died following accidental antimony potassium tartrate ingestion was only five per cent of the ingested dose with high antimony concentrations in the liver, gall bladder and gastrointestinal mucosa. This is consistent with antimony undergoing enterohepatic circulation (see below).

Excretion

Antimony compounds are eliminated mainly in the urine, with small amounts appearing in faeces via bile after conjugation with glutathione. A significant amount of antimony excreted in bile undergoes enterohepatic circulation (Bailly et al, 1991). Some 6-24 months after parenteral antimony therapy, Mansour et al (1967) reported increased urine antimony concentrations (range 5.8-145.3 µg/L) compared to untreated controls (range 2.9-9.1 µg/L).

Gerhardsson et al (1982) reported significantly ($p<0.001$) higher antimony concentrations in the lung tissue of 40 deceased smelter and refinery workers who had been exposed to antimony for some 30 years, compared to 11 unexposed controls. The time from last exposure to death varied from 0-23 years. The antimony concentration in liver and kidney was not significantly different between the two groups, suggesting that following occupational inhalation antimony may be retained in the lung for several years without significant systemic distribution.

Kentner et al (1995) estimated a renal elimination half-life of four days following occupational inhalation of antimony trioxide and stibine in 21 employees of a starter battery manufacturing plant.

CLINICAL FEATURES: ACUTE EXPOSURE

Dermal exposure

Antimony trioxide is an irritant although antimony dermatitis typically occurs during chronic occupational exposure.

Ocular exposure

Antimony trioxide is an eye irritant. Conjunctivitis and blurred vision were reported in workers exposed to antimony trioxide fumes in a smelter plant (Renes, 1953).

Ingestion

Acute poisoning is rare. Antimony trioxide is poorly soluble and is not readily absorbed from the gastrointestinal tract. Cases have been reported only when the compound has been leached into acidic beverages or has been converted into a more soluble form.

Gastrointestinal toxicity

Ingestion of a substantial quantity of antimony trioxide may result in nausea, vomiting and diarrhoea.

Over fifty people were "very sick" and treated in hospital after drinking lemonade contaminated with 13 mg/L antimony. Antimony trioxide had leached from an enamel container in which the drinks were stored overnight. All the patients recovered completely within several days (Dunn, 1928).

Similarly, antimony trioxide leached from enamel or ceramic glaze into acidic beverages caused "a burning sensation in the stomach", colic, nausea, vomiting and "collapse" (Monier-Williams, 1934).

One hundred and fifty children developed nausea, vomiting, abdominal pain and diarrhoea some 15 minutes after drinking antimony-contaminated lemonade. The lemonade had a pH of 2.5-3.1 and leached an estimated 30 mg/L antimony into solution from an agate pot in which it was stored for 20-22 hours. Most of the affected children recovered within a few hours, the remainder recovering within a few days (Werrin, 1963).

In 1982 Miller recounted the case of the author Oliver Goldsmith who died after ingesting a mixture of antimony trioxide and potassium tartrate. The estimated dose was 132-198 mg antimony. He succumbed after 18 hours severe vomiting and diarrhoea.

Cardiovascular and peripheral vascular toxicity

Electrocardiographic abnormalities are associated typically with chronic antimony exposure although have not been associated with exposure to antimony trioxide alone.

Following acute antimony ingestion two patients had "moderate bradyrhythmic dysfunctions" at presentation (Lauwers et al, 1990). Phlebitis occurred in four patients who accidentally ingested antimony potassium tartrate (Lauwers et al, 1990).

Inhalation

Pulmonary toxicity

Dusts and fumes of antimony trioxide are irritant to the respiratory tract and mucous membranes and inhalation causes laryngitis (ranging

from hoarseness to aphonia), pharyngitis, tracheitis, rhinitis, epistaxis, and bronchitis (Renes, 1953). Metal fume fever has been described (Anonymous, 1984) though less frequently than following exposure to zinc oxide.

Radiological evidence of pneumonitis was found in six workers exposed to antimony smelter fumes for 2-12 hours. Inflammatory changes were characteristically peri-hilar with no evidence of peripheral parenchymal damage. Symptoms were alleviated by removal from exposure (and treatment with penicillin aerosols). The average airborne antimony concentration was 10-12 mg/m^3 with a maximum measured breathing zone concentration of 70.7 mg/m^3 (Renes, 1953).

Gastrointestinal toxicity

Workers heavily exposed (not specified) to antimony trioxide in a smelter plant developed "gastritis", abdominal pain, diarrhoea and vomiting. Urine antimony concentrations ranged from a trace up to an "exceptionally high" 600 mg/L (Renes, 1953).

Neurotoxicity

"Neuritis", dizziness and headache were reported amongst workers exposed to antimony trioxide fumes at an antimony smelting plant (Renes, 1953).

Nephrotoxicity

Albuminuria was reported in a "severely ill" worker with a urine antimony concentration of 600 mg/L after exposure to antimony trioxide in a smelting plant (Renes, 1953). Removal from exposure and symptomatic treatment for several days "provided relief".

CLINICAL FEATURES: CHRONIC EXPOSURE

The major source of antimony trioxide exposure is as a by-product in the smelting of metal ores (which may also contain arsenic) and in industries such as ceramic, glass and alloy manufacture. Inhalation and dermal contact are the most common routes of exposure.

Dermal exposure

Dermatitis following contact with antimony trioxide is well described (Oliver, 1933; McCallum, 1989). Typical lesions arise on the arms, legs and in the flexures, sparing the face, hands and feet (Renes, 1953; McCallum, 1989).

Papules and pustules predominate around sweat and sebaceous glands with areas of eczema and lichenification (Paschoud, 1962). These so-called "antimony spots" occur mainly in the summer (McCallum, 1989).

Skin lesions developed in 23 men employed at an antimony trioxide production plant. Most of those affected were furnace workers with lesions typically appearing within two weeks of exposure. Itching, erythematous papules and pustular eruptions were characteristic, usually on dust laden sweaty areas of skin. The lesions usually

resolved over two weeks in individuals removed to cooler parts of the factory. Histological examination showed epidermal cellular necrosis associated with an acute dermal inflammatory reaction. Antimony trioxide patch testing was negative whilst injection of methacholine into the affected areas caused enlargement of the lesions. The author concluded that antimony trioxide dust initiated an irritant reaction when it penetrated sweat ducts (Stevenson, 1965).

White et al (1993) described three cases of occupational antimony dermatitis following several months exposure to antimony dust and antimony trioxide fumes. Two of these patients also experienced frequent nose bleeds. Both problems resolved when exposure ceased. In one patient patch testing for antimony was negative and in another the urine antimony concentration was 53.2 µg/L ("normal" < 1.0 µg/L).

Positive patch testing to antimony trioxide has been noted in enamellers and decorators in the ceramics industry (Motolese et al, 1993).

Ocular exposure

Antimony trioxide is an irritant. Conjunctivitis was reported in 14 of 51 workers exposed to antimony trioxide dust in a smelting plant (Potkonjak and Pavlovich, 1983).

Inhalation

Pulmonary toxicity

Chronic occupational antimony trioxide exposure may cause "antimony pneumoconiosis" (Cooper et al, 1968; McCallum, 1989). Typical radiological findings include diffuse, dense, punctate, non-confluent opacities predominately in the middle and lower lung fields, sometimes associated with pleural adhesions (Potkonjak and Pavlovich, 1983).

These changes developed after at least ten years working in an antimony smelting plant where the dust contained nearly 90 per cent antimony trioxide with some antimony pentoxide and small amounts (up to five per cent) of silica (Potkonjak and Pavlovich, 1983). Cough (in 31 of 51 subjects) and exertional breathlessness (in 26 cases) were the symptoms most frequently reported with wheeze, chest pain or generalized weakness in a minority. Nine workers had obstructive lung function defects with a combined restrictive/obstructive picture in five cases but no isolated restrictive defects or radiological evidence of diffuse fibrosis.

Pneumoconiosis was reported also in workers at an antimony oxide production plant. Lung biopsies from two affected individuals revealed antimony concentrations of 600-3000 µg/g (Le Bouffant et al, 1987)

Perforation of the nasal septum has been described in antimony workers but these cases probably have involved concomitant arsenic exposure (McCallum, 1989). There were no cases of nasal septum perforation in 51 workers employed at an antimony smelter for 9-31 years (mean 17.9 years) (Potkonjak and Pavlovich, 1983).

Cardiovascular toxicity

Although ECG changes have been reported in patients treated with antimony drugs there are no reports following exposure to antimony trioxide alone.

In the Czechoslovakian literature Klucik and Ulrich (1960) reported subjective cardiac complaints and ECG changes (not specified in

English abstract) in 14 workers occupationally exposed to antimony trioxide dust. However, significant antimony trisulphide exposure also occurred.

Brieger et al (1954) attributed ECG T-wave changes and sudden deaths to antimony-induced cardiotoxicity following occupational exposure to antimony trisulphide although the reliability of this study has been criticized (McCallum, 1989).

An epidemiological study of workers at an antimony processing plant showed no excess deaths from ischaemic heart disease in workers exposed to antimony trioxide compared with other employees at the same site (McCallum, 1989).

Ingestion

Chronic ingestion is not a recognized toxicological hazard.

MANAGEMENT

Dermal exposure

Ensure adequate self protection before attempting treatment. If possible the patient should remove any contaminated clothing him/herself. Affected areas of skin should be washed with copious quantities of water. Pay special attention to skin folds, fingernails and ears. The most effective treatment for irritant antimony dermatitis is removal from exposure.

Ocular exposure

Irrigate immediately with lukewarm water or preferably saline for at least 10-15 minutes. A local anaesthetic may be indicated for pain relief and to overcome blepharospasm. The use of fluorescein allows detection of corneal damage. Specialist ophthalmological advice should

be sought if any significant abnormality is detected on examination and in those whose symptoms do not resolve rapidly.

Ingestion

Following substantial ingestion of an antimony compound gastric lavage may be considered if presentation is within the first hour. There are no data to confirm that charcoal adsorbs antimony but the administration to a co-operative patient of 50 g activated charcoal within the first hour following a suspected substantial ingestion is reasonable. Other symptomatic and supportive measures should be dictated by the patient's condition. An ECG should be performed and biochemical and haematological profiles monitored. Blood and urine antimony concentrations are not widely available but may be of interest retrospectively to confirm systemic uptake.

Inhalation

Removal from exposure and measures to secure cardiorespiratory stability are the priority following acute inhalation of antimony compounds. An ECG should be performed. Respiratory symptoms in those with possible chronic antimony toxicity should be investigated as for other cases of pneumoconiosis. Urine antimony concentrations may be useful to monitor the initial extent of and subsequent reduction in exposure but these assays are not widely available.

Antidotes

Dimercaprol (British anti-lewisite, BAL) (Thompson and Whittaker, 1947; Braun et al, 1946), dimercaptosuccinic acid (DMSA, Succimer) (Basinger and Jones, 1981) and dimercaptopropane sulphonate (DMPS, Unithiol) (Basinger and Jones, 1981; Hruby and Donner, 1987) have antidotal activity in experimental systemic antimony poisoning (see below). These findings have not been confirmed in controlled studies in man.

Dimercaprol

In vitro studies

Using the pyruvate oxidase system of pigeon brains as a test model, dimercaprol in a molar ratio of 6:1 dimercaprol: antimony was able to protect the enzyme system from inhibition by several antimony salts (Thompson and Whittaker, 1947).

Animal studies

The LD_{50} of intramuscular antimony tartrate administered to rabbits was raised from 90 mg Sb/kg in controls to 160 mg Sb/kg in animals treated with intramuscular dimercaprol (30 mg/kg one hour after intoxication followed by 15 mg/kg at six, 24 and 48 hours) (Braun et al, 1946). A total of 45 controls received 50-200 mg/kg antimony tartrate with 56 treated animals receiving 125-200 mg/kg.

Clinical studies

Four adults with antimony poisoning following the inadvertent consumption of antimony potassium tartrate were treated with intramuscular dimercaprol 200-600 mg daily. Three patients made an uneventful recovery but the fourth, who had a history of cardiorespiratory disease, died on day three. There were no pre-chelation antimony excretion data but in two survivors maximum antimony urine concentrations of 1000 µg/L and 1500 µg/L were reported some 36 and 72 hours after poisoning respectively. Urine volumes were not stated (Lauwers et al, 1990).

Bailly et al (1991) reported a 24 year-old woman who made an uneventful recovery after ingesting an undetermined amount of antimony trisulphide. She was treated with dimercaprol 200 mg tds for five days but there was no evidence of enhanced urinary antimony elimination with therapy.

DMSA

Animal studies

DMSA was given intraperitoneally to mice at a molar ratio of 10:1 DMSA: antimony twenty minutes after administration of potassium antimonyl tartrate (120 mg/kg; twice the LD_{50}). The survival ratio was 28/30 (Basinger and Jones, 1981).

Clinical studies

There are no human data.

DMPS

Animal studies

DMPS has been shown to be an effective chelating agent in mice following intraperitoneal administration of potassium antimonyl tartrate (120 mg/kg; twice the LD_{50}). The survival rate was 19/30

when intraperitoneal DMPS was given twenty minutes after intoxication at a molar ratio of 10:1 DMPS: antimony. However, DMSA was significantly more effective under these conditions (see above) (Basinger and Jones, 1981).

Clinical studies

There are no human data.

Antidotes: Conclusions and recommendations

1. Clinical data regarding antimony chelation are scarce.

2. Dimercaprol effectively chelates antimony but has been superseded by the less toxic thiol antidotes DMPS and DMSA.

3. In limited animal studies DMSA is a more effective antimony chelator than DMPS.

4. Parenteral or oral DMSA therapy may be considered in antimony trioxide poisoning. The discussion of individual cases with an NPIS physician is recommended.

MEDICAL SURVEILLANCE

Improved occupational health measures have reduced industrial airborne antimony concentrations significantly but monitoring of ambient air antimony concentrations remains important in some industries (Bailly et al, 1991; Kentner et al, 1995).

Routine examination of the skin for "antimony spots" and chest radiography for evidence of pneumoconiosis may also be useful. The potential risk of pulmonary carcinogenicity should be remembered (see below).

Although Bailly et al (1991) found that urine antimony excretion among workers exposed to airborne antimony pentoxide and sodium antimoniate correlated to the intensity of exposure, a recent publication from the European Commission concluded "no indicator of effect is available" for biological monitoring of antimony (Apostoli et al, 1994).

Normal concentrations in biological fluids

"Normal" serum and urine antimony concentrations are quoted as approximately 3 µg/L and 0.8 µg/L respectively (Poisindex, 1997).

OCCUPATIONAL DATA

Maximum exposure limit

Long-term exposure limit (8 hour TWA reference period) 0.5 mg/m^3 (Health and Safety Executive, 1997).

OTHER TOXICOLOGICAL DATA

Carcinogenicity

There is some evidence that occupational antimony exposure is associated with an increased risk of lung cancer although frequent concomitant exposure to arsenic and other heavy metals precludes a definitive conclusion about its carcinogenic potential (Gerhardsson et al, 1982; McCallum, 1989; Gerhardsson and Nordberg, 1993; Jones 1994; Schnorr et al, 1995).

Antimony also has been implicated in the aetiology of bladder tumours in patients with schistosomiasis who have been treated with antimony compounds (Winship, 1987).

The International Agency for Research on Cancer has concluded antimony trioxide is "possibly carcinogenic to humans" (IARC, 1989).

Reprotoxicity

In the Russian literature women occupational exposed to antimony aerosols were reported to have a higher incidence of spontaneous abortion, premature births and menstrual disorders. Antimony was present in the blood, urine, placentae, amniotic fluid and breast milk of these women but further details were not available in the English abstract (Belyaeva, 1967).

Genotoxicity

Bacillus subtilis: produced gene conversion and mitotic recombination (DOSE, 1992).

Fish toxicity

LC_{50} (96 hr) bluegill sunfish, fathead minnow 530-833 mg/L (DOSE, 1992).

EEC Directive on Drinking Water Quality 80/778/EEC

Maximum admissible concentration 10 µg/L, as antimony (DOSE, 1992).

WHO Guidelines for Drinking Water Quality

Provisional guideline value 5 µg/L, as antimony (WHO, 1993).

AUTHORS

WN Harrison PhD CChem MRSC
SM Bradberry BSc MB MRCP
ST Beer BSc
JA Vale MD FRCP FRCPE FRCPG FFOM

National Poisons Information Service (Birmingham Centre),
West Midlands Poisons Unit,
City Hospital NHS Trust,
Dudley Road,
Birmingham
B18 7QH
UK

This monograph was produced by the staff of the Birmingham Centre of the National Poisons Information Service in the United Kingdom. The work was commissioned and funded by the UK Departments of Health, and was designed as a source of detailed information for use by poisons information centres.

Date of last revision
28/1/98

REFERENCES

Apostoli P, Porru S, Alessio L.
Antimony. In: Alessio L, Berlin A. Roi R, van der Venne MT, eds. Biological indicators for the assessment of human exposure to industrial chemicals.

Luxembourg: Office for Official Publications of the European Communities, 1994.

Anonymous.
Metals and the lung.
Lancet 1984; 2: 903-4.

Bailly R, Lauwerys R, Buchet JP, Mahieu P, Konings J.
Experimental and human studies on antimony metabolism: their relevance for the biological monitoring of workers exposed to inorganic antimony.
Br J Ind Med 1991; 48: 93-7.

Basinger MA, Jones MM.
Structural requirements for chelate antidotal efficacy in acute antimony(III) intoxication.
Res Commun Chem Pathol Pharmacol 1981; 32: 355-63.

Belyaeva AP.
[The effect produced by antimony on the generative function.]
Gig Tr Prof Zabol 1967; 11: 32-7.

Bradbury J.
Cot death debate becomes heated after publication of new research.
Lancet 1997; 349: 625.

Braun HA, Lusky LM, Calvery HO.
The efficacy of 2,3-dimercaptopropanol (BAL) in the therapy of poisoning by compounds of antimony, bismuth, chromium, mercury and nickel.
J Pharmacol Exp Ther 1946; 87: 119-25.

Brieger H, Semisch CW, Stasney J, Piatnek DA.
Industrial antimony poisoning.
Ind Med Surg 1954; 23: 521-3.

CHIP2/Chemicals (Hazard Information and Packaging for Supply). Regulations 1994.
Health and Safety Commission.
Sudbury: Heath and Safety Executive, 1994.

CHRIS/Chemical Hazard Response Information System.
In: Tomes plus. Environmental Health and Safety Series 1. Vol 33.
United States Coast Guard, 1997.

Cooper DA, Pendergrass EP, Vorwald AJ, Maycock RL, Brieger H.
Pneumoconiosis among workers in an antimony industry.
Am J Roentgenol 1968; 103: 495-508.

CSDS/Chemical Safety Data Sheets. Vol 2.
Cambridge: Royal Society of Chemistry, 1989.

DOSE/Dictionary of substances and their effects. Vol 1.
Cambridge: Royal Society of Chemistry, 1992.

de Wolff FA.
Antimony and health. Incriminating stibine in the sudden infant death syndrome is difficult on current evidence.
Br Med J 1995; 310: 1216-7.

Dunn JT.
A curious case of antimony poisoning.
Analyst 1928; 53: 532-3.

Gerhardsson L, Brune D, Nordberg GF, Wester PO.
Antimony in lung, liver and kidney tissue from deceased smelter workers.
Scand J Work Environ Health 1982; 8: 201-8.

Gerhardsson L, Nordberg GF.
Lung cancer in smelter workers - interactions of metals as indicated by tissue levels.
Scand J Work Environ Health 1993; 19: 90-4.

Hazardtext.
In: Tomes plus. Environmental Health and Safety Series 1. Vol 33.
Colorado: Micromedex, Inc., 1997.

Health and Safety Executive.
EH40/97. Occupational exposure limits 1997.
Sudbury: Health and Safety Executive, 1997.

Hruby K, Donner A.
2,3-dimercapto-1-propanesulphonate in heavy metal poisoning.
Med Toxicol 1987; 2: 317-23.

IARC/International Agency for Research on Cancer.
Antimony trioxide and antimony trisulfide.
IARC Monographs on the Evaluation of Carcinogenic Risk of Humans 1989; 47; 291-305.

IPCS/International Programme on Chemical Safety.
Guidelines for drinking-water quality. 2nd ed. Vol 2. Health criteria and other supporting information.
Geneva: World Health Organization, 1996.

Jones RD.
Survey of antimony workers: mortality 1961-1992.
Occup Environ Med 1994; 51: 772-6.

Kentner M, Leinemann M, Schaller KH, Weltle D, Lehnert G.
External and internal antimony exposure in starter battery production.
Int Arch Occup Environ Health 1995; 67: 119-23.

Klucik I, Ulrich L.
[Electrocardiographic examinations of workers in an antimony metallurgical plant].
Pracovni Lekarstvi 1960; 5: 236-43.

Lauwers LF, Roelants A, Rosseel M, Heyndrickx B, Baute L.
Oral antimony intoxications in man.
Crit Care Med 1990; 18: 324-6.

Le Bouffant L, Giroulle H, Guerrin F, Normand C, Cornu L, Tichoux G.
Pneumoconiose a l'antimoine. Etude anatomo-physique.
[Pneumoconiosis due to antimony]
Inst Natl Sante Rech Med 1987; 155: 447-55.

Mansour MM, Rassoul AAA, Schulert AR.
Anti-bilharzial antimony drugs.
Nature 1967; 214: 819-20.

McCallum RI.
The industrial toxicology of antimony. The Ernestine Henry lecture 1987.
J R Coll Physicians Lond 1989; 23: 28-32.

MERCK/The Merck Index.

Antimony trioxide. In: Budavari S, ed. An encyclopedia of chemicals, drugs, and biologicals. 12th ed.
New Jersey: Merck and Co., Inc., 1996; 120.

Miller JM.
Poisoning by antimony: a case report.
South Med J 1982; 75: 592.

Monier-Williams GW.
Reports on public health and medical subjects. No 73. Antimony in enamelled hollow-ware.
London: HMSO, 1934.

Motolese A, Truzzi M, Giannini A, Seidenari S.
Contact dermatitis and contact sensitization among enamellers and decorators in the ceramics industry.
Contact Dermatitis 1993; 28: 59-62.

OHM/TADS-Oil and Hazardous Materials/Technical Assistance Data System.
In: Tomes plus. Environmental Health and Safety Series 1. Vol 33.
United States Environmental Protection Agency, 1997.

Oliver T.
The health of antimony oxide workers.
Br Med J 1933; 1: 1094-5.

Paschoud J-M.
Deux cas d'eczéma de contact lichénoid rappelant des très près lichen plan.
[Two cases of lichenoid contact eczema which closely resemble lichen planus]
Dermatologica 1963; 127: 99-107.

Poisindex.
In: Micromedex International Healthcare Series. Vol 92.
Colorado: Micromedex, Inc., 1997.

Potkonjak V, Pavlovich M.
Antimoniosis: a particular form of pneumoconiosis. I. Etiology, clinical and X-ray findings.
Int Arch Occup Environ Health 1983; 51: 199-207.

Renes LE.
Antimony poisoning in industry.
Arch Ind Hyg Occup Med 1953; 7: 99-108.

Schnorr TM, Steenland K, Thun MJ, Rinsky RA.
Mortality in a cohort of antimony smelter workers.
Am J Ind Med 1995; 27: 759-70.

Stevenson CJ.
Antimony spots.
Trans St. Johns Hospital Dermatol Soc 1965; 51: 40-5.

Taylor A.
Antimony, cot mattress, and SIDS.
Lancet 1996; 347: 616.

Thompson RHS, Whittaker VP.
Antidotal activity of British Anti-Lewisite against compounds of antimony, gold and mercury.
Biochem J 1947; 41: 342-6.

Werrin M.

Chemical food poisoning.
Q Bull Assoc Food Drug Offic 1963; 27: 38-45.

White Jr GP, Mathias CGT, Davin JS.
Dermatitis in workers exposed to antimony in a melting process.
J Occup Med 1993; 35: 392-5.

WHO/World Health Organization.
Guidelines for drinking-water quality. 2nd ed. Vol 1. Recommendations.
Geneva: World Health Organization, 1993.

Winship KA.
Toxicity of antimony and its compounds.
Adverse Drug React Acute Poisoning Rev 1987; 2: 67-90.

See Also:
Antimony trioxide (ICSC)