TOXICITY SUMMARY FOR
ANTIMONY

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Prepared by

Robert A. Young, Ph.D., D.A.B.T.
Chemical Hazard Evaluation and Communication Group
Biomedical and Environmental Information Analysis Section
Health and Safety Research Division
Oak Ridge National Laboratory*
Oak Ridge, Tennessee

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EXECUTIVE SUMMARY

Antimony (Sb) is a naturally occurring metal that is used in various manufacturing processes. It exists in valence states of 3 and 5 (Budavari, 1989; ATSDR, 1990). Antimony is a common urban air pollutant (Beliles, 1979). Exposure to antimony may be via inhalation, oral and dermal routes (ATSDR, 1990).

Antimony is sparingly absorbed following ingestion or inhalation (Felicetti et al., 1974a; Gerber et al., 1982; ATSDR, 1990). Both gastrointestinal and pulmonary absorption are a function of compound solubility. Antimony is transported in the blood, its distribution varying among species and dependent on its valence state (Felicetti et al., 1974b). Antimony is not metabolized but may bind to macromolecules and react covalently with sulfhydryl and phosphate groups (ATSDR, 1990). Excretion of antimony is primarily via the urine and feces, and is also dependent upon valence state (Cooper et al., 1968; Ludersdorf et al., 1987; ATSDR, 1990).

Acute oral exposure of humans and animals to high doses of antimony or antimony-containing compounds (antimonials) may cause gastrointestinal disorders (vomiting, diarrhea), respiratory difficulties, and death at extremely high doses (Bradley and Frederick, 1941; Beliles, 1979; ATSDR, 1990). Subchronic and chronic oral exposure may affect hematologic parameters (ATSDR, 1990). Long-term exposure to high doses of antimony or antimonials has been shown to adversely affect longevity in animals (Schroeder et al., 1970). Limited data suggest that prenatal and postnatal exposure of rats to antimony interferes with vasomotor responses (Marmo et al., 1987; Rossi et al., 1987).

Acute inhalation exposure of humans may cause gastrointestinal disorders (probably due to ingestion of airborne antimony) (ATSDR, 1990). Exposure of animals to high concentrations of antimony and antimonials (especially stibine gas) may result in pulmonary edema and death (Price et al., 1979). Long-term occupational exposure of humans has resulted in electrocardiac disorders, respiratory disorders, and possibly increased mortality (Renes, 1953; Breiger et al., 1954). Antimony levels for these occupational exposure evaluations ranged from 2.2 to 11.98 mg Sb/m^3. Based on limited data, occupational exposure of women to metallic antimony and several antimonials has reportedly caused alterations in the menstrual cycle and an increased incidence of spontaneous abortions (Belyaeva, 1967). Reproductive dysfunction has been demonstrated in rats exposed to antimony trioxide (Belyaeva, 1967).

No data were available indicating that dermal exposure of humans to antimony or its compounds results in adverse effects. However dermal application of high doses of antimony oxide (1,584 mg Sb/kg) resulted in the death of rabbits within one day (IBTL, 1972). Eye irritation due to exposure to stibine gas and several antimony oxides has been reported for humans (Stevenson, 1965; Potkonjak and Pavlovich, 1983).

The U. S. EPA (U.S. EPA, 1991, 1992) has calculated subchronic and chronic oral reference doses (RfDs) of 4E-4 mg/kg/day based on decreased longevity and alteration of blood chemistry in rats chronically exposed to potassium antimony tartrate in the drinking water (5 ppm equivalent to 0.35 mg Sb/kg/day). An uncertainty factor of 1,000 was applied: 10 for extrapolation from a lowest-observed-adverse-effect-level (LOAEL) to a no-observed-adverse-effect-level (NOAEL), 10 for extrapolation from animal data, and 10 for protection of sensitive populations.

The primary target organ for acute oral exposure to antimony appears to be the gastrointestinal tract (irritation, diarrhea, vomiting) and targets for long-term exposure are the blood (hematological disorders) and liver (mild hepatotoxicity) (ATSDR, 1990). Inhalation exposure to antimony affects the respiratory tract (pneumoconiosis, restrictive airway disorders), with secondary targets being the cardiovascular system (altered blood pressure and electrocardiograms) and kidneys (histological changes) (Renes, 1953; Breiger et al., 1954). Only limited evidence exists for reproductive disorders due to antimony exposure (Belyaeva, 1967).

Although some data indicate that long-term exposure of rats to antimony trioxide and trisulfide increased the incidence of lung tumors (Wong et al., 1979; Watt, 1980; Groth et al., 1986; Bio/dynamics, 1989), the U.S. EPA has not evaluated antimony or antimonials for carcinogenicity and a Weight-of-Evidence classification is currently unavailable.

1. INTRODUCTION

Antimony (CAS No. 7440-36-0) is a naturally occurring metalloid element (displaying both metallic and nonmetallic properties) existing in valence states of 3 and 5 (Budavari, 1989; ATSDR, 1990). Metallic antimony and a few trivalent antimony compounds are the most significant regarding exposure potential and toxicity (ATSDR, 1990). Antimony (Sb) is used in metallurgical processes, paints and enamels, various textiles, rubber, and fire retardation (antimony trioxide). Antimony is a common urban air pollutant, occurring at an average concentration of 0.001 $\mu g/m^3$ (Beliles, 1979). Exposure to antimony may occur via inhalation and by ingestion of contaminated food. Some antimonials such as potassium antimony tartrate have been used medicinally as parasiticides (Beliles, 1979).

2. METABOLISM AND DISPOSITION

2.1. ABSORPTION

Antimony is only slowly absorbed from the gastrointestinal tract. Based on animal data, gastrointestinal absorption of antimony was estimated to be 2 to 7% (Felicetti et al., 1974a; Gerber et al., 1982). The specific chemical form will determine the absorption efficiency of ingested antimony. Antimony has been detected in the blood of occupationally exposed individuals, but it is uncertain if this was due solely to pulmonary absorption or also to ingestion following mucociliary transport from the upper respiratory tract (ATSDR, 1990). Alveolar deposition and subsequent absorption of small particle-size antimony or antimony compounds is likely, with absorption also being a function of compound solubility. Sunagawa (1981) and Ainsworth (1988) provided data showing a lack of a dose-response relationship for absorption, and that antimony levels in lungs and livers of rodents reached a plateau, thereby suggesting that antimony absorption by tissues may be a saturable process. Studies with rabbits exposed to antimony trioxide or pentoxide provided evidence for dermal absorption of these forms of antimony (IBTL, 1972; Myers et al., 1978).

2.2. DISTRIBUTION

Antimony is transported in the blood, its partitioning between erythrocytes and plasma being a function of the valence state of the element. Exposure to trivalent antimony resulted in higher antimony levels in erythrocytes than in the plasma while exposure to pentavalent antimony resulted in a reversal of this partitioning (Felicetti et al., 1974b). However, the distribution of parenterally administered antimony is highly variable among species, and can not be accounted for solely by valence state (Felicetti et al., 1974b; Beliles, 1979). Antimony has been detected in the liver and spleen (more so for pentavalent forms), and the thyroid gland (trivalent forms) (Beliles, 1979). Felicetti et al. (1974a,b) also reported accumulation of antimony in the skeletal system and in fur.

2.3. METABOLISM

Like other metallic elements, antimony is not subject to catabolism. Its metabolic transformations are limited to binding with macromolecules, and covalent interactions with sulfhydryl groups and phosphates. There is no definitive data regarding the interconversion of

valence states of antimony (ATSDR, 1990).

2.4. EXCRETION

Animal and human data are available indicating both urinary and fecal excretion of antimony (ATSDR, 1990).   Workers exposed to antimony trioxide had increased urinary levels of antimony (Cooper et al., 1968; Ludersdorf et al., 1987).   Urinary excretion of antimony was documented for workers exposed to antimony fumes (Renes, 1953; Breiger et al., 1954).   Fecal excretion of antimony following ingestion of antimonials represents primarily ustate of antimony may affect the excretory route.   Parenteral administration experiments with animals and humans have indicated that trivalent antimony is excreted primarily in the urine while pentavalent antimony is excreted in the feces (Felicetti et al., 1974b; Edel et al., 1983).   Whole-body clearance of trivalent antimony tartrate is biphasic with 90% of the body burden being excreted within 24 hours, and the slower phase having a half-life of 16 days (Felicetti et al., 1974b).   Urinary antimony levels in a human subject remained above normal (1 µg/g creatinine) for one week following a single oral dose (amount unknown) of antimony trisulfate (Bailly et al., 1991).   This same study also documented biliary excretion and enterohepatic circulation of parenterally administered antimony in rats.

3. NONCARCINOGENIC HEALTH EFFECTS

3.1. ORAL EXPOSURES

3.1.1. Acute Toxicity

3.1.1.1.   Human

Acute poisoning has occurred as the result of accidental or suicidal ingestion of antimonials (Beliles, 1979) with death ensuing within several hours.   Symptoms of severe antimony poisoning include vomiting, watery diarrhea, collapse, irregular respiration, and hypothermia.   A single dose of potassium antimony tartrate (equivalent to 0.53 mg Sb/kg) produced vomiting (Dunn, 1928).

3.1.1.2.   Animal

Toxic effects ranging from gastrointestinal disorders to death have been documented for animals following acute oral exposure to antimonials.   Bradley and Frederick (1941) reported that a single dose (300 mg Sb/kg) of the organic antimonial, potassium antimony tartrate, induced myocardial infarction and death in rats.   However, several studies using inorganic antimonials (metallic antimony, antimony oxide, or antimony trioxide) reported that doses as high as 27,410 mg Sb/kg were not fatal to rats (ATSDR, 1990).

3.1.2. Subchronic Toxicity

3.1.2.1.   Human

No information was available regarding the toxic effects of subchronic oral exposure of

humans to antimony or antimonials.

3.1.2.2.   Animal

Several studies have described the systemic subchronic toxicity of antimony and antimonials in animals and are summarized in ATSDR (1990) and U.S. EPA (1980).   Subchronic (24-week) exposure of rats to metallic antimony at doses of 500 to 1,000 mg/kg/day decreased plasma protein levels, hemoglobin levels, and hematocrit (Sungawa, 1981; Hiraoka, 1986).   Sungawa (1981) also reported mild hepatotoxicity in rats receiving 418 mg Sb/kg/day (as antimony trioxide) or 500 mg Sb/kg/day (as metallic antimony).   A decrease in red blood cell count was reported by Sungawa (1981) for rats given 418 mg Sb/kg/day (as antimony trioxide) for 24 weeks, and an increase reported by Smyth and Thompson (1945) in rats treated with same compound at a dose of 894 mg Sb/kg/day for 30 days.   Fleming (1982) reported that dogs receiving antimony trioxide (6,644 mg Sb/kg/day) for 32 days exhibited severe weight loss, vomiting, and muscle weakness and dyskinesia of the hind limbs.   A lower dose (84 mg Sb/kg/day) produced severe diarrhea.   Using data from Fleming (1982), ATSDR (1990) reported a no-observed-adverse-effect-level (NOAEL) of 501 mg Sb/kg for rats receiving the compound for 20 days.   Exposure of CD-1 mice to potassium antimony tartrate in the drinking water (5 ppm for 540 days) significantly reduced the lifespan of both males and females (Kanisawa and Schroeder, 1969).

3.1.3. Chronic Toxicity

3.1.3.1.   Human

No information was available regarding the chronic toxicity of antimony in humans.

3.1.3.1.   Animal

Schroeder et al. (1970) showed an increase in serum cholesterol and a decrease in fasting glucose levels for rats receiving a lifetime exposure to potassium antimony tartrate (746 mg Sb/kg) in drinking water.   However, the biological significance of these findings is not certain (ATSDR, 1990).   This same study also provided data showing that long-term exposure of rats to potassium antimony tartrate in the drinking water (0.262 mg Sb/kg/day equivalent to 2 ppm in drinking water) resulted in decreased lifespan.

3.1.4. Developmental and Reproductive Toxicity

3.1.4.1.   Human

No information was available regarding the developmental or reproductive toxicity of antimony in humans.

3.1.4.1.   Animal

The effect of prenatal and postnatal exposure of rats (gestation day one to postnatal day 60) to antimony trichloride indicated that drinking water levels as low as 0.1 mg% produced alterations in vasomotor responses (Marmo et al., 1987; Rossi et al., 1987).   The same effect was observed for

exposure throughout lactation to postnatal day 60. Although the antimony trichloride exposure affected vasomotor responses during the postnatal developmental period, the design of these studies did not allow for definitive conclusions regarding the prenatal developmental toxicity of the compound.

3.1.5. Reference Dose

3.1.5.1. Subchronic

ORAL RfD$_s$:	4E-4 mg/kg/day (metallic antimony)
	9E-4 mg/kg/day (potassium antimony tartrate)
	4E-4 mg/kg/day (antimony tetroxide, antimony trioxide)
	5E-4 mg/kg/day (antimony pentoxide)
UNCERTAINTY FACTOR:	1000
NOAEL:	None

3.1.5.2. Chronic

ORAL RfD$_c$:	4E-4 mg/kg/day (metallic antimony)
	9E-4 mg/kg/day (potassium antimony tartrate)
	4E-4 mg/kg/day (antimony tetroxide, antimony trioxide)
	5E-4 mg/kg/day (antimony pentoxide)
UNCERTAINTY FACTOR:	1000
MODIFYING FACTOR:	1
NOAEL:	None
LOAEL:	0.35 mg/kg/day
CONFIDENCE:	
Study:	Low
Data base:	Low
RfD:	Low
VERIFICATION DATE:	11/06/85
PRINCIPAL STUDY:	Schroeder et al., 1970

COMMENTS: Based on decreased longevity and altered blood chemistry values in rats chronically exposed to 5 ppm potassium antimony tartrate in drinking water. An uncertainty factor of 1,000 was applied to account for extrapolation from animal data (10), use of a LOAEL (10), and for protection of sensitive populations (10). The values for all of the compounds were calculated based on elemental equivalent weight of antimony (U.S. EPA, 1991, 1992).

3.2. INHALATION EXPOSURES

3.2.1. Acute Toxicity

3.2.1.1. Human

Inhalation of antimony dust by factory workers produced gastrointestinal irritation, probably the result of antimony dust transported via the mucociliary escalator (ATSDR, 1990). However, no information regarding the acute inhalation toxicity of antimony was available.

3.2.1.2. Animal

Exposure of rats and guinea pigs to stibine gas (SbH_3) at a concentration of 1,395 mg Sb/m^3 for 30 minutes resulted in pulmonary edema and death (Price et al., 1979). At 799 mg Sb/m^3, the guinea pigs and rats exhibited only renal tubular dilation.

3.2.2. Subchronic Toxicity

3.2.2.1. Human

Several occupational exposure studies have been conducted that evaluated the effects of subchronic exposure of smelter workers to antimony fumes. Renes (1953) reported that < 5 month exposures to antimony fumes at concentrations ranging from 4.69 to 11.82 mg/m^3 produced various conditions (rhinitis, dermatitis, laryngitis, bronchitis, pneumonitis, conjunctivitis, and septal perforations) the severity of which increased with increasing duration of exposure. These workers, however, were also exposed to low concentrations of arsenic (0.39 to 1.10 mg/m^3). The effects of occupational exposure to antimony trioxide (0.58 to 5.5 mg/m^3) for periods of 8 months to 2 years were reported by Breiger et al. (1954). Eight of the employees died during this period but only one death had occurred in the 16 years prior to the use of antimony. Additionally, abnormal electrocardiograms wstudy are compromised by concurrent exposure to phenol formaldehyde resin. However, the investigators noted that no additional deaths occurred and the incidence of electrocardiac anomalies decreased following cessation of antimony use.

3.2.2.2. Animal

Gross et al. (1952) reported that inhalation exposure of rats to antimony trioxide for up to 14 months resulted in lipoid pneumonia. Myocardial damage and alteration of electrocardiograms were observed for rats and rabbits exposed for six weeks to antimony trisulfide dust at concentrations of 2.20 and 4.02 mg Sb/m^3, respectively (Brieger et al., 1954). This same study also noted similar effects in dogs exposed for 10 weeks to antimony trisulfide (3.98 mg Sb/m^3). Thirteen-week exposure of rats to antimony trioxide (0.92 mg Sb/m^3) resulted in nonreversible proliferation of alveolar macrophages (Bio/dynamics, 1989). Several studies reviewed in ATSDR (1990) reported interstitial fibrosis in rats exposed for 12 to 14.5 months to antimony trisulfide or antimony trioxide at concentrations ranging from 1.6 to 83.6 mg Sb/m^3.

3.2.3. Chronic Toxicity

3.2.3.1. Human

Occupational exposure to antimony trioxide and/or antimony pentoxide dust (9 to 31 years) resulted in respiratory effects including pneumoconiosis, chronic bronchitis, chronic emphysema, pleural adhesions, and obstructive pulmonary effects (Cooper et al., 1968; Potkonjak and Pavlovich, 1983).   The pneumoconiosis was characterized by chronic coughing, wheezing, and upper airway irritation.   Shorter-term occupational exposures (Renes, 1953; Brieger et al., 1954) are described in Section 3.2.2.1.

3.2.3.2. Animal

No information regarding the chronic toxicity of antimony or antimony-containing compounds was available, although animal studies employing long-term exposures (12 to 14.5 months) examined the effects of several antimony-containing compounds (see Section 3.2.2.2).

3.2.4. Developmental and Reproductive Toxicity

3.2.4.1. Humans

In a report lacking in detail, Belyaeva (1967) noted an increased incidence of spontaneous abortions and disturbances in the menstrual cycle of women occupationally exposed to airborne metallic antimony, antimony trioxide, and antimony pentasulfide.

3.2.4.2. Animal

Belyaeva (1967) reported a decrease in the number of offspring for rats exposed to antimony trioxide (209 mg Sb/m^3) prior to mating and throughout gestation.   Exposure to this concentration prior to mating resulted in a 67% failure in conception.   Metaplasia in the uterus and disturbances in ovum maturation were detected in the animals that did not conceive.

3.2.5. Reference Concentration

Reference concentrations are currently unavailable.

3.3. OTHER ROUTES OF EXPOSURE

3.3.1. Acute Toxicity

3.3.1.1. Human

No dermatologic or systemic effects were noted for human subjects having induction patches containing antimony trioxide in place for 24 hours (IBTL, 1972).

3.3.1.2. Animal

Dermal application of antimony oxide (1,584 mg Sb/kg) resulted in the death of rabbits within one day (IBTL, 1972) and dermal application of antimony trioxide (6,686 Sb mg/kg) was also fatal to rabbits within one day (Myers et al., 1978).

Ocular application of antimony oxide or antimony thioantimonate (79 to 100 mg Sb) to rabbits resulted in eye irritation (Horton et al., 1986; IBTL, 1972; WRL, 1979) but similar application of antimony trioxide (34.5 to 83.6 mg Sb) did not produce irritation (Gross et al., 1955; Myers et al., 1978).

Exposure of rats and guinea pigs to stibine gas (Price et al., 1979) or antimony trioxide (Bio/dynamics, 1985; 1989) caused eye irritation. It is likely that this effect was due to direct contact to airborne compounds rather than an inhalation-mediated toxicity.

3.3.2. Subchronic Toxicity

3.3.2.1. Human

Occupational exposure to airborne antimony or antimony trioxide has resulted in dermatitis (epidermal cellular necrosis with inflammatory cellular reactions) and ocular irritation (ocular conjunctivitis), the effects being more pronounced in higher temperature environments (Stevenson, 1965; Potkonjak and Pavlovich, 1983).

3.3.2.2. Animal

Thirteen-week dermal exposure of rabbits to a 5% solution of a mixture of antimony trisulfide and antimony pentasulfide did not produce significant signs of toxicity (Horton et al., 1986).

3.3.3. Chronic Toxicity

No human or animal data were available regarding the chronic toxicity of antimony by other exposure routes.

3.3.4. Developmental Toxicity

No human or animal data were available regarding the developmental/reproductive toxicity of antimony by other exposure routes.

3.4. TARGET ORGANS/CRITICAL EFFECTS

3.4.1. Oral Exposures

3.4.1.1. Primary Target(s)

1. Gastrointestinal tract: The gastrointestinal tract appears to be the primary target for acute and long-term oral exposure to antimony and antimonials. Effects are characterized by irritation resulting in vomiting and diarrhea. These effects have been documented for both humans and animals.

3.4.1.2. Other Target(s)

1. Cardiovascular system: Data are available suggesting that oral exposure to antimony or antimonials may affect the cardiovascular system resulting in a decreased hypotensive response.

2. Blood: Animal data are available showing mild hematologic effects although the biological significance of these effects is uncertain.

3. Liver: Limited data in animals have shown mild hepatotoxicity following oral exposure to antimony and antimonials.

4. Developmental toxicity is suggested by animal data showing that prenatal/postnatal exposure to antimony may affect cardiovascular functions.

3.4.2. Inhalation Exposures

3.4.2.1. Primary Target(s)

1. Respiratory tract: The respiratory tract is the primary target for toxicity of inhaled antimony and antimonials following acute, subchronic, and chronic exposure. Both human and animal data have demonstrated various forms of restrictive airway diseases including pneumoconiosis, bronchitis, emphysema, pulmonary edema, and varying degrees of irritation and inflammation.

3.4.2.2. Other Target(s)

1. Cardiovascular system: Cardiovascular effects (alterations in blood pressure and electrocardiograms) have been documented in humans exposed to antimony and antimonials. Similar effects have also been verified using several animals species.

2. Kidney: Renal toxicity (histological changes) has been observed for animals exposed to stibine gas and various antimony oxides and sulfides.

3. Reproductive tract: Limited data are available indicating adverse effects on the reproductive system of women occupationally exposed to antimony. One study in rats has also

shown reproductive/developmental toxicity of antimony.

4. CARCINOGENICITY

4.1. ORAL EXPOSURE

4.1.1. Human

No information was available regarding the carcinogenicity of antimony in humans.

4.1.2. Animal

No definitive data were available regarding the carcinogenicity of antimony in animals. Although evidence for an increased incidence of cancer was not observed for rats and mice fed potassium antimony tartrate (0.262 and 0.35 mg Sb/kg/day, respectively), the maximum tolerated dose was not used (Schroeder, 1968; Kanisawa and Schroeder, 1969; Schroeder, 1970).

4.2. Inhalation Exposure

4.2.1. Human

Evidence of increased incidence of cancer was not detected in workers exposed to antimony oxide for 9 to 31 years (Potkonjak and Pavlovich, 1983).

4.2.2. Animal

Lung tumor incidence was increased for rats exposed for one year to 4.2 or 36 mg Sb/m^3 as antimony trioxide (Wong et al., 1979; Watt, 1980; Groth et al., 1986) or 17.48 mg Sb/m^3 as antimony trisulfide (Wong et al., 1979; Groth et al., 1986). An increased incidence of lung tumors was also reported by Bio/dynamics (1989) for rats exposed to antimony trioxide (3.76 mg Sb/m^3) and by Watt (1983) for pigs exposed to the same compound at a concentration of 4.2 mg Sb/m^3.

4.3. OTHER ROUTES OF EXPOSURE

No data were available regarding the potential carcinogenicity of antimony and antimonials by other routes of exposure.

4.4. EPA WEIGHT-OF-EVIDENCE CLASSIFICATION

The U.S. EPA has not evaluated antimony regarding its potential carcinogenicity and, therefore, a Weight-of-Evidence classification has not been assigned (U.S. EPA, 1991).

4.5. CARCINOGENICITY SLOPE FACTORS

None have been calculated.

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