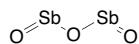


Antimony Trioxide

CAS No. 1309-64-4

Reasonably anticipated to be a human carcinogen

First listed in the *Fifteenth Report on Carcinogens* (2021)



Carcinogenicity

Antimony trioxide is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in experimental animals and supporting evidence from mechanistic studies. The data available from studies in humans are inadequate to evaluate the relationship between human cancer and exposure specifically to antimony trioxide or antimony in general.

Cancer Studies in Experimental Animals

Antimony trioxide administered by inhalation caused lung tumors in rats and mice of both sexes and tumors at several other tissue sites in female rats and in mice of both sexes. No cancer studies in experimental animals exposed to antimony trioxide by other routes were identified. The conclusion of carcinogenicity was based on three studies in three different strains or stocks of rats and one study in mice. NTP studies (2017) examined all organs and tissues in Wistar Han rats and B6C3F1/N mice of both sexes, and three other studies examined primarily the lung in Wistar rats (Groth *et al.* 1986) and Fischer 344 rats of both sexes (Newton *et al.* 1994) and female CDF rats (Watt 1983). The NTP studies were the most informative, based on the study design and detailed report, and the other studies also were adequate to inform conclusions on carcinogenicity after critical evaluation of potential bias.

In the lung, exposure of female rats to antimony trioxide significantly increased the incidences of benign lung tumors (alveolar/bronchiolar adenoma) (Groth *et al.* 1986, NTP 2017), which can progress to malignancy, and incidences of malignant lung tumors (scirrhous carcinoma and/or squamous-cell carcinoma) (Watt 1983, Groth *et al.* 1986). In male rats, the combined incidences of benign lung tumors (alveolar/bronchiolar adenoma) and malignant lung tumors (alveolar/bronchiolar carcinoma) were not significantly increased, but both exceeded the historical control ranges for all past studies (NTP 2017). Considered together with a positive dose-related trend in tumor incidence and increased incidences of lung tumors in female rats and in mice of both sexes (as described below), the increase in combined incidences of benign and malignant lung tumors in male rats was deemed to be exposure-related (NTP 2017). Another study in male and female rats (Newton *et al.* 1994) found no increase in lung-tumor incidence, possibly because the highest tested concentration was too low (as indicated by the absence of changes in survival or body weight in the high-dose groups); this was the only study that reported no increase in tumors.

Exposure of mice to antimony trioxide caused statistically significant increases in the incidences of benign lung tumors (alveolar/bronchiolar adenoma) in females, malignant lung tumors (alveolar/bronchiolar carcinoma) in both sexes, and benign and malignant lung tumors combined (alveolar/bronchiolar adenoma and carcinoma) in both sexes (NTP 2017). These increases were significant at all three tested concentrations, including the low concentration, at which lung clearance overload (in which the lung's capacity to clear the inhaled particles is overwhelmed) did not occur. In rats, lung clearance overload due to high concentrations of inert particles can result in increased lung cancer. In the case of antimony trioxide, evidence suggesting toxicity (i.e., that the antimony trioxide particles

were not inert) and increased benign and malignant lung tumors combined at the low concentration (at which lung clearance overload did not occur) (NTP 2017) showed that the observed lung cancer in rats was not due to overload. The incidences of malignant and combined benign and malignant lung tumors in male mice also were dose-related.

At other tissue sites, antimony trioxide exposure significantly increased the incidences of malignant lymphoma (cancer of the white blood cells) in female mice; skin tumors (benign fibrous histiocytoma alone and combined with malignant fibrosarcoma) in male mice; benign tumors of the adrenal gland (pheochromocytoma) in male and female rats; and combined benign and malignant adrenal-gland tumors (pheochromocytoma) in female rats (NTP 2017). The occurrences of adrenal-gland pheochromocytoma might have been secondary to hypoxia.

Tissue site	Rat		Mouse	
	Malignant	Combined	Malignant	Combined
Lung	↑F	*M	↑M, ↑F	↑M, ↑F
Adrenal gland	—	↑F	—	—
Skin	—	—	—	↑M
Lymphatic system	—	—	↑F	—

Combined = combined incidence of benign and malignant tumors.

↑ = Significant increase; F = in females; M = in males.

* = Considered to be evidence of antimony trioxide carcinogenicity based on multiple factors, although the increase in incidence was not statistically significant (NTP 2017).

— = No exposure-related increase in tumors.

Mechanisms of Carcinogenesis

Antimony trioxide induces several biological effects associated with carcinogenicity that are also observed with other carcinogenic metals; however, the available data did not provide adequate information to determine the overall mechanism by which antimony causes cancer. The relative abundance of the data on each type of characteristic of the substance or biological change could reflect the availability of studies (e.g., genotoxicity has been studied much longer than epigenetic changes), and not the level of its contribution to carcinogenicity. Because antimony trioxide may exert its effects through released trivalent antimony ions, effects observed with other trivalent antimony compounds potentially are relevant to understanding the carcinogenicity of antimony trioxide. Overall, the *in vivo* effects observed were increased DNA damage and micronucleus formation resulting from exposure to antimony trioxide and increased oxidative stress resulting from exposure to an antimony(V) compound. *In vitro* effects included increased oxidative stress (and consequently oxidative damage) induced by antimony trioxide or other compounds containing trivalent antimony (antimony trichloride, antimony potassium tartrate) or pentavalent antimony (meglumine antimoniate); inhibition of DNA repair by antimony trichloride, another trivalent antimony compound; and inhibition of cell differentiation by antimony potassium tartrate, which also contains trivalent antimony.

Antimony trioxide induces reactive oxygen species (ROS) and adversely affects mitochondria and DNA (Mann *et al.* 2006, Lösler *et al.* 2009), and other antimony compounds also cause oxidative damage to proteins (by antimony potassium tartrate, antimony trichloride, meglumine antimoniate) and lipids (by meglumine antimoniate). Antimony trioxide also decreases the levels of antioxidants in cells, which increases their susceptibility to damage by oxidants (such as ROS). Specifically, antimony trioxide lowers the levels of reduced glutathione (GSH), an antioxidant, and inhibits the enzymes involved in GSH functions, which would disrupt the normal cellular balance between oxidation and reduction (the redox system) (Mann *et al.* 2006, Lösler *et al.* 2009). Cells are partially protected from oxidative

damage induced by trivalent antimony through the addition of external antioxidants and ROS scavengers (Hashemzaei *et al.* 2015). A pentavalent antimony compound (meglumine antimoniate) caused oxidative damage in mice, observed as protein carbonylation, lipid peroxidation (Bento *et al.* 2013), and DNA damage (Cantanhêde *et al.* 2015, Moreira *et al.* 2017).

Although antimony trioxide does not cause mutations in bacterial mutagenicity assays except under very specific conditions, it has been shown to damage DNA, chromosomes, and chromatids in experimental animals and cultured cells. In mice exposed to antimony trioxide by inhalation, lung tissue showed increased DNA damage, and red blood cells showed increased formation of micronuclei (small pieces of nucleus produced by incorrect chromosome segregation or other events), indicating genotoxicity and chromosomal instability (NTP 2017). Increased chromosomal aberrations, micronucleus formation, and sister-chromatid exchange were also seen in cultured mammalian cells exposed to antimony trioxide (NTP 2018). The genotoxicity could be the result of oxidative stress and/or decreased DNA repair or other changes.

The effects of antimony trioxide on DNA repair were not studied; however, antimony trichloride, another trivalent antimony compound, decreased the repair of DNA damage induced by ultraviolet and ionizing radiation (Grosskopf *et al.* 2010, Koch *et al.* 2017), and antimony trioxide is likely to have similar effects. Trivalent antimony can directly disrupt XPA, a key protein in nucleotide excision repair (a specific type of DNA repair pathway), by displacing zinc (a metal essential in stabilizing the protein structure) in the protein's DNA-binding region, thus hindering the protein's function (Grosskopf *et al.* 2010). Other repair proteins also are affected by antimony through alteration of protein concentration, structure, or location (NTP 2018).

Lung tumors induced in rats and mice by long-term inhalation of antimony trioxide showed high incidences of specific mutations in the epidermal growth factor receptor gene (*Egfr*) (NTP 2017). These *Egfr* mutations may lead to increased cell survival, which in turn can lead to cancer growth. The fact that *Egfr* mutations were not seen in spontaneous alveolar/bronchiolar carcinomas in control animals or in non-tumor lung tissues in exposed rats or mice suggests a role for antimony trioxide exposure in their occurrence.

A trivalent antimony compound (antimony potassium tartrate) has been shown to prevent cell differentiation in cultured human skin cells (Patterson and Rice 2007), giving cells the potential to continue proliferating and possibly cause cancer. Once skin cells are fully differentiated, they lose the ability to divide, and are not likely to become cancer cells. Prevention of cell differentiation by antimony potassium tartrate results in part from inhibition of the decrease in the number of epidermal growth factor receptors that normally occurs when cells in culture grow to a certain density (e.g., nearly covering the whole bottom of a petri dish). With an excess of epidermal growth factor receptors, cells can continue to divide even at high cell density (e.g., grow into more than one layer of cells on the same growth surface). Consistent with this potential mechanism, skin tumors were seen in mice exposed to antimony trioxide by inhalation, and dermatitis was reported in workers exposed to antimony trioxide.

Cancer Studies in Humans

The data available from studies in humans are inadequate to evaluate the relationship between human cancer and exposure specifically to antimony trioxide or antimony in general.

The data relevant for evaluating the effects of antimony exposure in humans are two cohort studies of antimony-smelter workers, in the United Kingdom (Jones 1994) and the United States (Schnorr *et al.* 1995), a cohort study of tin-smelter workers in the United Kingdom

(Jones *et al.* 2007), and a case-control study of art-glass workers in Sweden (Wingren and Axelson 1993). For lung cancer, elevated mortality was seen in some analyses of all studies of antimony-exposed smelter worker cohorts. However, it is not clear whether the increased risk of lung cancer was due to exposure to antimony; the results may have been biased through confounding by concurrent exposure to other lung carcinogens. An increased risk of stomach cancer was found in the U.S. antimony-smelter cohort study (Schnorr *et al.* 1995) and the Swedish case-control study (Wingren and Axelson 1993), but not in the U.K. antimony-smelter cohort study (Jones 1994).

Properties

Antimony trioxide is the oxide of trivalent (+3) antimony, and it occurs naturally as well as being produced through human activities. Antimony exists in four main oxidation states: -3, 0, +3, and +5. The most common in environmental, biological, and geochemical systems are Sb(III) (the trivalent form) and Sb(V) (the pentavalent form). In nature, antimony trioxide (Sb₂O₃) exists in minerals such as valentinite and senarmonite (Roper *et al.* 2012, ATSDR 2019). Humans purposely oxidize elemental antimony to produce antimony trioxide for various industrial uses. Other forms of antimony can transform into antimony trioxide during the life cycles of products containing antimony. For instance, at high temperature (e.g., during incineration, combustion, or use of the brakes in vehicles), other forms of antimony can be oxidized to antimony trioxide. Antimony trioxide can also be converted to other forms of antimony in the environment.

Antimony trioxide exists as an odorless white crystalline powder (IPCS 2013). It is slightly soluble in water, dilute sulfuric acid, dilute nitric acid, or dilute hydrochloric acid and soluble in solutions of alkali hydroxides or sulfides and in warm solutions of tartaric acid or of bitartrates (OSHA 1989). Physical and chemical properties of antimony trioxide are listed in the following table.

Property	Information
Molecular weight	291.5
Specific gravity	5.9 at 24°C
Melting point	656°C
Boiling point	1,550°C (partially sublimes)
Water solubility	[3.3 × 10 ⁻⁴] g/100 mL at 22.2°C ^a
Vapor pressure	1 mm Hg at 574°C ^b

Source: IPCS 2013.

^aReported as 0.0033 g/L; brackets denote conversion of units. ^bReported as 130 Pa.

Although antimony trioxide is relatively insoluble in water and is not easily taken up by cells (i.e., its bioaccessibility is low), studies show that antimony trioxide has higher bioaccessibility in some artificial fluids that mimic various body fluids. For example, the bioaccessibility of antimony trioxide was highest (81.7%) in artificial lysosomal fluid, followed by 60.8% in artificial sweat, 56.7% in artificial interstitial lung fluid, and 41.5% in artificial blood serum, and was lowest (13.6%) in artificial gastric fluid (ECHA 2017). These findings are consistent with the observation that inhalation of antimony trioxide leads to more adverse health effects than does ingestion.

Use

The major industrial use of antimony trioxide (EPA 2014, NTP 2017) is as a synergist for halogenated flame retardants in plastics, rubber, and textiles, all of which are used in a wide variety of consumer products. The final concentration of antimony trioxide in textiles as a fire-retardant synergist is 4% to 6%, but fire-retardant coatings on the backs of textiles may contain up to 24% (EU 2008).

Antimony trioxide used in industrial processes often changes its form during those processes. In the production of polyethylene tere-

phthalate (PET) plastics, antimony trioxide, which is added in the preparation of the catalyst solution, is readily converted to antimony glycolate (Carneado *et al.* 2015). The final concentration of antimony in PET plastics, where it is bound as antimony glycolate complexes, is 180 to 550 ppm (EU 2008). Antimony trioxide is used in art glass and other types of specialty glass at a concentration of about 0.8% antimony in finished glass (its main use is as a fining agent to remove gaseous inclusions that could leave bubbles in the glass product). It is also used as a white pigment and an opacifier in paints and pigments, which are used in a broad range of industries, in consumer products such as plastics, coatings, enamels, and ceramics, and in building materials (EU 2008). An additional minor use of antimony trioxide is to reduce the amount of hexavalent chromium used in cement (Mapei 2016).

Antimony trioxide is ultimately disposed of as waste during either production processes or through disposal of the final consumer products. Some products are recycled, such as PET beverage bottles for production of PET fibers, but the antimony itself in these recycled products generally is not recovered for reuse.

Production

Antimony trioxide is produced primarily by re-volatilization of crude antimony trioxide or by oxidation of antimony metal (EU 2008). The only current domestic producer of primary antimony metal and oxide identified was a company in Montana that used imported feedstock (USGS 2018); no marketable antimony has been mined in the United States since 2015 (USGS 2018). Under the U.S. Environmental Protection Agency's (EPA's) Chemical Data Reporting rule, production of antimony trioxide in 2015 was reported to be between 1 million and 10 million pounds (EPA 2017).

Antimony trioxide accounts for 80% of total antimony use in the United States (EPA 2014, NTP 2017). U.S. consumption of antimony trioxide is much higher than U.S. production. In 2017, U.S. imports for consumption were approximately 52.8 million pounds of antimony oxide (by weight of antimony content) (USGS 2018). In each year between 2007 and 2011, U.S. imports were roughly 61 million pounds (equivalent to approximately 87% of the yearly consumption of 70 million pounds) (EPA 2014). In 2012, data reported to EPA identified three companies manufacturing and ten facilities importing antimony trioxide (EPA 2012).

Exposure

A significant number of people in the United States are exposed to antimony trioxide, as evidenced by occupational exposure data and supporting data on industrial and consumer uses, consumption, and predicted environmental exposure. In addition to exposure to antimony trioxide in the workplace, people are potentially exposed when using consumer products containing antimony trioxide or when breathing contaminated air. Because the chemical form of antimony changes during manufacturing, in the environment, and *in vivo*, people can be exposed to antimony trioxide produced by oxidation of other forms of antimony and can be exposed to other antimony forms from sources releasing antimony trioxide. The major sources of antimony trioxide exposure are summarized in the table in the next column and discussed below.

Occupational Exposure

The highest occupational exposure to antimony trioxide occurs in workplaces that produce or use antimony trioxide. In the United States, roughly 70 million pounds of antimony trioxide are used annually as a synergist for halogenated flame retardants in plastics, rubber, and textiles, as a catalyst in PET production, and as an additive in optical and art glass, pigments, paints, ceramics, and cement. Workers

Source of antimony trioxide	Exposure route	Expected form of antimony exposure
Sb ₂ O ₃ production: Occupational	Inhalation of Sb ₂ O ₃	Sb ₂ O ₃
	Dermal	Sb ₂ O ₃
Environmental Sb ₂ O ₃ : Sb ₂ O ₃ and some non-Sb ₂ O ₃ -releasing sources	Inhalation of Sb ₂ O ₃	Sb ₂ O ₃
	Ingestion (from consuming contaminated soil)	Sb ions
	Ingestion (from drinking contaminated water)	Sb(V) ion in oxic environments, and Sb(III) ion in anoxic environments
Sb ₂ O ₃ in flame retardant: Occupational and general-population exposure	Inhalation (from breathing in airborne dust which contains antimony trioxide in the indoor workplace or home)	Mainly Sb ₂ O ₃
	Dermal (workplace and from sitting on flame-retardant-treated upholstery)	Sb ions
	Ingestion (from mouthing flame-retardant-treated toys)	Sb ions
Sb ₂ O ₃ used in PET production: Occupational and consumer products	Inhalation: Workers in PET production	Sb ₂ O ₃
	Inhalation: Workers in downstream PET operations	Sb ions
	Ingestion (from drinking liquid in PET bottles)	Sb ions
Sb ₂ O ₃ used in glass, paint and other uses: Occupational	Inhalation and dermal	Sb ₂ O ₃ and Sb ions (depending on process step)

Sources: EU 2008, ATSDR 2019.

at an estimated 273 U.S. facilities (based on information from EPA's Toxics Release Inventory [TRI]) were exposed to antimony trioxide in 2010 (EPA 2014). More than 200,000 workers were exposed to antimony trioxide and other antimony compounds according to the 1981 to 1983 U.S. National Occupational Exposure Survey (CDC 2017b), indicating extensive past exposure to antimony.

The highest occupational exposure to antimony trioxide in the United States, exceeding current regulatory levels by at least tenfold, occurred during smelting and refining operations and production of antimony trioxide in the 1970s and 1980s (when antimony air levels ranged from 50 to over 5,000 µg/m³) (Donaldson 1976). Global data collected since the 1980s suggest that the highest exposure to antimony trioxide occurs during production of antimony trioxide; mean exposure at an antimony trioxide manufacturing facility was 766 µg/m³ (ATSDR 2017), and worst-case exposure was estimated at 790 µg/m³ (EU 2008). The next-highest exposures have been reported for the flame-retardant industry, at up to 200 µg/m³ (ATSDR 1992), with worst-case exposure estimated at 570 µg/m³ (EU 2008). Lower exposures occur during the use of antimony trioxide in the PET industries, with an estimated worst-case exposure of 26 µg/m³ when it is used to generate the catalyst antimony glycolate, and in the glass industries, where 1980s measurements were 40 to 840 µg/m³ (Lüdersdorff *et al.* 1987), with an estimated worst-case exposure of 15 µg/m³ (EU 2008).

Because other forms of antimony can be oxidized to antimony trioxide, workers in industries using other forms of antimony as raw material can also be exposed to antimony trioxide. For example, antimony accounts for up to 2% of an automobile battery's total weight, and when antimonial lead in automobile batteries is recycled, the metals frequently are oxidized to antimony trioxide (Grund *et al.*

2011, Dupont *et al.* 2016). The table above provides information on the forms of antimony in these various industries.

Workers can also be exposed to antimony trioxide in automobile-generated air pollution in high-traffic areas. Antimony trisulfide is used as a lubricant in the abrasive material of brakes and can be oxidized to antimony trioxide by the frictional heat resulting from braking. A study in Valparaíso City, Chile (Quiroz *et al.* 2009) reported very high levels of antimony in the blood of port workers exposed to high vehicular traffic (average concentration = 27 ± 9 ng/kg of body weight); the levels were 5 to 10 times higher than in control groups from either another part of the city or a rural area outside Valparaíso.

Exposure of the General Population

Antimony has been detected in urine, whole blood, and saliva from U.S. residents. Data from the National Health and Nutrition Examination Survey reported low levels of urinary antimony (0.043 µg/L for 2013 to 2014 [CDC 2017a]; 0.047 µg/L for 2015 to 2016 [CDC 2021]), and levels might have been decreasing over time. Higher urinary antimony levels were found in lower-income individuals living in economically deprived neighborhoods than those in more affluent neighborhoods (Belova *et al.* 2013, Tyrrell *et al.* 2013, Gonzales *et al.* 2016), and higher levels were found in younger populations (aged 6 to 11 or 12 to 19) than in adults (aged 20 or older) (CDC 2017a, 2021). These biomonitoring studies measure total antimony; the proportion of urinary antimony that resulted from exposure to antimony trioxide is not known.

Members of the general population are exposed to antimony trioxide primarily by breathing contaminated indoor and outdoor air. In air, antimony is present almost entirely in the particulate matter. In 2010, EPA estimated from TRI data that 11,635 lb of antimony were released into the air from 273 U.S. facilities that likely produced, processed, or used antimony trioxide-containing flame retardants (EPA 2014). Antimony concentrations in outdoor air are highest near facilities that release antimony trioxide into the air, such as mines and smelting operations; levels reported in the 1970s ranged from 0.146 to 300,000 µg/m³. People can also be exposed to antimony trioxide released into the air by oxidation of various forms of antimony: antimony trisulfide in brake pads is oxidized during braking of automobiles, and antimony trioxide is the primary species released to air by burning of coal and petroleum and incineration of waste containing antimony (Health Canada 2010, NTP 2017). In U.S. cities, levels of antimony in the air that are not associated with specific sources are low (approximately 0.001 µg/m³) (ATSDR 2019).

Exposure to antimony trioxide from surface water or soil is unlikely, because antimony trioxide in water or soil is converted to different forms. Antimony trioxide in solution produces the trivalent antimony ion, which hydrolyzes to either the neutral trivalent species antimony (III) hydroxide, Sb(OH)₃, or the charged pentavalent species (the antimonate ion), Sb(OH)₆⁻ (EU 2008). Exposure to antimony in the soil is expected to be minimal because of antimony's low solubility and mobility (EPA 2014, Li *et al.* 2014).

Drinking water and food are not considered sources of exposure to antimony trioxide. The European Union risk assessment for antimony trioxide noted that antimony present in drinking water and foods is not in the form of antimony trioxide (EU 2008).

Members of the general population potentially are exposed to antimony trioxide from consumer products containing the compound as a flame-retardant synergist, and more specifically from the dust generated through wear and tear of these products. The estimated worst-case daily exposure to antimony trioxide from inhalation of house dust is 60 µg/g of dust and 0.0032 µg/m³ of air (EU 2008). Exposure of children, especially infants, is likely increased by their closer

proximity to carpet containing antimony trioxide and their mouthing of toys with antimony-containing fabric, paint, or plastics; the estimated worst-case daily exposure of children from eating house dust (e.g., from unwashed hands) is 0.6 µg/kg of body weight (EU 2008).

Regulations

Department of Transportation (DOT)

Antimony compounds (inorganic, liquid, not otherwise specified), antimony compounds (inorganic, solid, not otherwise specified), and other liquid and solid antimony compounds as specified by the DOT are considered hazardous materials, and special requirements have been set for marking, labeling, and transporting these materials.

Environmental Protection Agency (EPA)

Clean Air Act

National Emission Standards for Hazardous Air Pollutants: Antimony compounds are listed as hazardous air pollutants.

Clean Water Act

Effluent Guidelines: Antimony compounds are listed as toxic pollutants.

Antimony trioxide and other antimony compounds as specified by EPA are designated as hazardous substances.

Comprehensive Environmental Response, Compensation, and Liability Act

Reportable quantity (RQ) = 1,000 lb for antimony trioxide.

Emergency Planning and Community Right-To-Know Act

Toxics Release Inventory: Antimony compounds are listed substances subject to reporting requirements.

Resource Conservation and Recovery Act

Listed Hazardous Waste: Waste codes for which the listing is based wholly or partly on the presence of antimony or its compounds = K176, K177.

Antimony compounds are listed as hazardous constituents of waste.

Occupational Safety and Health Administration (OSHA)

This legally enforceable permissible exposure limit (PEL) was adopted from the 1968 American Conference of Governmental Industrial Hygienists threshold limit value – time-weighted average (TLV-TWA) shortly after OSHA was established; it may not reflect the most recent scientific evidence and may not adequately protect worker health.

Permissible exposure limit (PEL) (8-h TWA) = 0.5 mg/m³ for antimony and compounds (as Sb).

Guidelines

American Conference of Governmental Industrial Hygienists (ACGIH)

Threshold limit value – time-weighted average (TLV-TWA) = 0.5 mg/m³ for antimony and compounds (as Sb).

Exposure to antimony trioxide by all routes should be carefully controlled to levels as low as possible.

A revision of the ACGIH TLV-TWA for antimony trioxide from "L" (i.e., exposure by all routes should be carefully controlled to levels as low as possible) to 0.02 mg/m³ for antimony trioxide inhalable particulate matter has been proposed and placed on the 2020 Notice of Intended Changes (NIC).

Environmental Protection Agency (EPA)

IRIS inhalation reference concentration (RfC) = 2×10^{-4} mg/m³ for antimony trioxide.

Regional Screening Levels (formerly Preliminary Remediation Goals): Residential soil = 28,000 mg/kg for antimony trioxide. Industrial soil = 120,000 mg/kg for antimony trioxide. Residential air = 0.021 µg/m³ for antimony trioxide. Industrial air = 0.088 µg/m³ for antimony trioxide.

National Institute for Occupational Safety and Health (NIOSH)

Recommended exposure limit (REL) = 0.5 mg/m³ (10-h TWA) for antimony and other antimony compounds (as Sb).

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