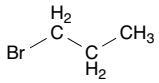
1-Bromopropane

CAS No. 106-94-5

Reasonably anticipated to be a human carcinogen

First listed in the *Thirteenth Report on Carcinogens* (2014)

Also known as *n*-propyl bromide



Carcinogenicity

1-Bromopropane is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in experimental animals. 1-Bromopropane, either directly or via reactive metabolites, causes molecular alterations that typically are associated with carcinogenesis, including genotoxicity, oxidative stress, and glutathione depletion. These alterations, observed mainly *in vitro* and in toxicity studies in rodents, are relevant to possible mechanisms of human carcinogenicity and support the relevance of the cancer studies in experimental animals to human carcinogenicity.

Cancer Studies in Experimental Animals

Inhalation exposure to 1-bromopropane caused tumors in two rodent species and at several different tissue sites, including one tissue site in rats at which tumors are rare (NTP 2011).

In male rats, 1-bromopropane caused significant dose-related increases in the incidences of several types of benign and/or malignant skin tumors (keratoacanthoma; keratoacanthoma and squamous-cell carcinoma combined; and keratoacanthoma, squamous-cell carcinoma, basal-cell adenoma, and basal-cell carcinoma combined). Both female and male rats showed an increased incidence of large-intestine tumors (adenoma of the colon and rectum), which are rare tumors in rats. In females, the incidence was dose-related and statistically significantly higher than in concurrent controls, and it exceeded the historical control range for all routes of exposure used in studies, including inhalation exposure. In males, the incidence of large-intestine adenoma was not significantly increased, but exceeded the historical control range for inhalation-exposure studies, and its occurrence was considered to be biologically significant because of the rarity of these tumors (which occurred in less than 0.2% of the historical controls). Although no carcinoma of the large intestine was observed in male or female rats in this study, adenoma of the large intestine has been shown to progress to carcinoma in other studies, and forms a morphologic continuum with carcinoma (Deschner 1983, Chang 1984, Nigro 1985).

In female mice, 1-bromopropane caused significant dose-related increases in the incidence of benign and malignant lung tumors combined (alveolar/bronchiolar adenoma and carcinoma).

These findings are supported by the observation of additional tumors in rats that may have been related to 1-bromopropane exposure, including malignant mesothelioma of the abdominal cavity and pancreatic islet tumors in males and skin tumors (squamous-cell papilloma, keratoacanthoma, and basal-cell adenoma or carcinoma) in females.

Other Relevant Data

1-Bromopropane is well absorbed following ingestion, inhalation, or dermal exposure. Occupational exposure occurs primarily by inhalation and dermal contact. Unmetabolized 1-bromopropane has been detected in the urine of exposed workers at levels significantly correlated with exposure to 1-bromopropane in air (Kawai *et al.* 2001, Ichihara *et al.* 2004).

1-Bromopropane is metabolized via several pathways; 16 urinary metabolites have been detected in rodents, and several other metabolites have been proposed (Jones and Walsh 1979, Ishidao *et al.* 2002, Garner *et al.* 2006). The primary metabolic pathways in rodents are oxidation reactions catalyzed by cytochrome P450 (primarily CYP2E1) and glutathione conjugation. The available data on human metabolism of 1-bromopropane, although limited, suggest that some of its metabolic pathways in humans are similar to those observed in rodents. Four mercapturic conjugates identified in the urine of rodents were also identified in the urine of workers exposed to 1-bromopropane (Hanley *et al.* 2009). The major metabolite, *N*-acetyl-*S*-(*n*-propyl)-L-cysteine, has been detected in the urine of exposed workers at levels that increased with increasing levels of 1-bromopropane in ambient air (Hanley and Dunn 2006, Valentine *et al.* 2007, Hanley *et al.* 2009, 2010). This metabolite is produced in humans by conjugation of 1-bromopropane with glutathione, and that reaction also releases free bromide ions, another useful biomarker for human exposure to 1-bromopropane (Jones and Walsh 1979, Hanley *et al.* 2006). No studies were identified that tested for the occurrence in humans of the oxidative metabolites that are obligate intermediates to the measured conjugates.

Studies on Mechanisms of Carcinogenesis

The mechanism(s) by which 1-bromopropane causes cancer is not known. However, exposure to 1-bromopropane has been shown to cause molecular alterations related to carcinogenicity, including genotoxicity (mutations and DNA damage), oxidative stress, glutathione depletion, and immunomodulation.

Studies have shown that 1-bromopropane can bind to macromolecules; it formed *S*-propylcysteine–globin adducts in exposed animals and humans (Valentine *et al.* 2007). Although 1-bromopropane did not induce mutations in bacteria under standard assay conditions, it did induce mutations in bacteria both with and without exogenous mammalian metabolic activation in the only reported study whose design was appropriate for testing a highly volatile chemical (Barber *et al.* 1981). It also caused mutations in cultured mammalian cells with or without mammalian metabolic activation (Elf Atochem 1996, as reviewed in NTP 2003) and DNA damage in cultured human cells without metabolic activation (Toraason *et al.* 2006). In addition, there is limited evidence of DNA damage in leukocytes from 1-bromopropane–exposed workers (Toraason *et al.* 2006). In rodents exposed *in vivo*, 1-bromopropane did not increase micronucleus formation in bone marrow (Kim *et al.* 1998, as reviewed in NTP 2003) or peripheral blood erythrocytes (Elf Atochem 1996, cited in NTP 2003, 2011) or cause dominant lethal mutations. However, the dominant lethal mutation assay is generally regarded as relatively insensitive for the detection of mutagenic agents (Saito-Suzuki *et al.* 1982, Yu *et al.* 2008).

There is evidence that metabolic activation plays a role in the genotoxicity and toxicity of 1-bromopropane. Several reactive metabolites (or intermediates) of 1-bromopropane have been identified in rodents, including glycidol and α-bromohydrin, and propylene oxide has been proposed as a metabolite (Garner *et al.* 2006). These compounds cause genotoxic effects *in vitro,* including DNA adduct formation, mutations, and DNA or chromosome damage (Stolzenberg and Hine 1979, IARC 1994, 2000). Glycidol and propylene oxide cause cytogenetic effects *in vivo* and are carcinogenic in experimental animals, and both substances are listed in the Report on Carcinogens as *reasonably anticipated to be human carcinogens*. These reactive and genotoxic metabolites may be responsible for at least some of the carcinogenic effects of 1-bromopropane. As with 1-bromopropane,

oral exposure to glycidol caused rare tumors of the large intestine in rats, as did oral exposure to two halogenated alkane analogues of 1-bromopropane, tribromomethane and bromodichloromethane (NTP 1987, 1989, 1990).

Chronic exposure to 1-bromopropane may produce levels of oxidative metabolites that exceed the glutathione-conjugating capacity or may inhibit enzymes required for glutathione synthesis. Because glutathione is an important cellular defense mechanism, reduced levels can lead to oxidative stress, increased toxicity, and carcinogenicity. Numerous studies have shown that 1-bromopropane induces both oxidative stress and glutathione depletion (Lee *et al.* 2005, 2007, 2010a, Liu *et al.* 2009, 2010, Huang *et al.* 2011). Studies with Cyp2e1$^{-/-}$ knockout mice, cytochrome P450 inhibitors, or a glutathione synthesis inhibitor showed that this metabolic activation pathway is involved in 1-bromopropane–induced toxicity, including neurological and reproductive effects, hepatotoxicity, and immunosuppression (NTP 2003, 2011, Lee *et al.* 2007, 2010a,b). Neurological effects of 1-bromopropane exposure have also been reported in humans (Li *et al.* 2010, Ichihara *et al.* 2012).

It is unclear whether induction of immunotoxicity by 1-bromopropane plays a role in tumor development. Recent studies have shown that 1-bromopropane causes immunosuppression in rodents (Lee *et al.* 2007, Anderson *et al.* 2010). In particular, it reduced the numbers of T cells and T-cell subpopulations. In addition, there is evidence that 1-bromopropane causes an inflammatory response. It induced dose-related increases in gene expression and production of proinflammatory cytokines in mouse macrophages (Han *et al.* 2008) and an inflammatory response in rats (NTP 2011). However, chronic respiratory inflammation and lung tumors were not associated in rodents; respiratory inflammation occurred in rats but not mice, whereas lung tumors occurred in mice but not rats.

Cancer Studies in Humans

No epidemiological studies or case reports were identified that evaluated the relationship between human cancer and exposure specifically to 1-bromopropane.

Properties

1-Bromopropane is a halogenated alkane that exists at room temperature as a colorless to pale-yellow, volatile liquid with a strong, characteristic odor (NTP 2011). It is slightly soluble in water and in most organic solvents, including acetone, ethanol, ether, benzene, chloroform, and carbon tetrachloride. It is less flammable than many other halogenated alkanes at room temperature. Thermal decomposition of 1-bromopropane produces hydrogen bromide. 1-Bromopropane can react with oxidizing agents to form hazardous flammable compounds and with water to produce acids. Physical and chemical properties of 1-bromopropane are listed in the following table.

Property	Information
Molecular weight	123.0[a]
Specific gravity	1.353 at 20°C/20°C[b]
Melting point	–110°C[a]
Boiling point	64.7°C[a]
Log K_{ow}	2.10[b]
Water solubility	2.45 g/L at 20°C[b]
Vapor pressure	110.8 mm Hg at 20°C[a]
Vapor density relative to air	4.25[b]

Sources: [a]NTP 2003, [b]HSDB 2006.

Use

1-Bromopropane is used primarily as a solvent cleaner in vapor and immersion degreasing operations to clean optics, electronics, and metals and as a solvent vehicle in industries using aerosol-applied adhesives, such as foam cushion manufacturing. However, its use as an aerosol solvent or adhesive could be affected by the proposed U.S. Environmental Protection Agency (EPA) rule that finds 1-bromopropane to be unacceptable for these uses (see Regulations). In recent years, 1-bromopropane usage has increased as a result of new industrial and commercial uses as a substitute for ozone-depleting chemicals or suspected carcinogens (e.g., as an alternative to tetrachloroethylene in the drycleaning industry) (Blando *et al.* 2010). 1-Bromopropane also has potential for use as a spot remover in the textile industry; however, an evaluation of 1-bromopropane as a substitute for trichloroethylene concluded that chronic toxicity data were insufficient, and use of 1-bromopropane was not recommended until more data were available (Mirza *et al.* 2000). In the past, 1-bromopropane was used primarily as a solvent for fats, waxes, and resins and as an intermediate in the synthesis of pharmaceuticals, insecticides, quaternary ammonium compounds, flavors, and fragrances in generally well-controlled, closed processes (Hanley *et al.* 2006, NTP 2003).

Production

1-Bromopropane is a high-production-volume chemical. In 2012, 1-bromopropane was manufactured by at least 21 companies worldwide, including at least one company in the United States (SRI 2012). Reported recent and historical volumes of U.S. production, imports, and exports of 1-bromopropane are listed in the following table.

Category		Year	Quantity (lb)
Production + imports[a]		2012	15.3 million
		1998–2006	1 million to < 10 million
		1994	> 500K to 1 million
		1986, 1990	10K to 500K
U.S. imports:[b]			
	recent	2013	9.2 million
	historical	2007	10.9 million
U.S. exports:[b]			
	recent	2013	15.6 million
	historical	2007	8.8 million

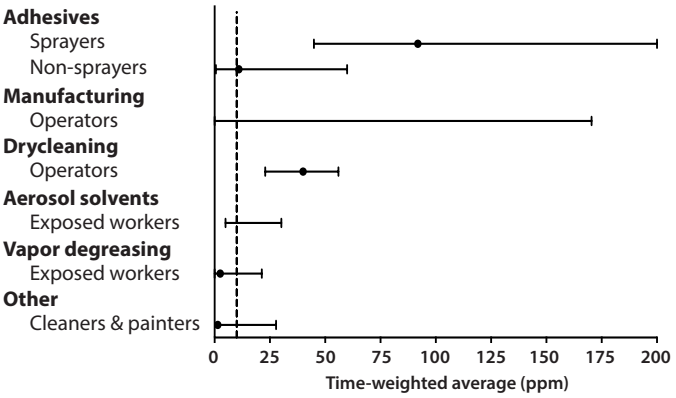
Sources: [a]EPA 2014 (EPA Chemical Data Reporting Rule, formerly the Inventory Update Rule), [b]USITC 2014 (reported as "brominated derivatives of acyclic hydrocarbons").

Exposure

A significant number of people in the United States are exposed to 1-bromopropane as a result of widespread usage, high production volume, and high levels of 1-bromopropane in commercial and industrial settings.

Occupational exposure to 1-bromopropane may occur through inhalation or dermal contact at workplaces where 1-bromopropane is produced or used (HSDB 2006). Concentrations of 1-bromopropane in air (8- to 12-hour time-weighted averages [TWAs]) from all studies identified across several U.S. industrial sectors ranged from not detected to 380 ppm, with the highest concentrations being for adhesive use and the lowest for vapor degreasing. Sprayers in the adhesive industry had the highest exposure, which ranged from 18 to 380 ppm across several studies. Exposure data for 1-bromopropane manufacturing were not available for the United States. Manufacturing exposure reported from China (Ichihara *et al.* 2004) ranged from not detectable to 170.5 ppm for processes that included adding materials to large reaction pots. However, production methods reported in a patent application by a U.S. manufacturer included numerous control processes to contain 1-bromopropane, which would likely reduce potential exposure substantially. The graph below shows TWA 1-bromopropane exposure levels from representative studies of adhesive application (Hanley *et al.* 2006), manufacturing (in China; Ichihara *et al.* 2004), drycleaning (Eisenberg and Ramsey 2010), aero-

sol solvent use (Graul 2012), vapor degreasing (Hanley *et al.* 2010), and cleaning and painting in workshops using 1-bromopropane solvents (Kawai *et al.* 2001).



Occupational exposure to 1-bromopropane, by industry

Time-weighted-average 1-bromopropane exposure levels as geometric means (adhesives, vapor degreasing, and other); arithmetic mean (drycleaning); or not reported (manufacturing and aerosol solvents). The dashed vertical line represents the American Conference of Governmental Industrial Hygienists threshold limit value – time-weighted average (TLV-TWA) of 10 ppm.

Among workers at polyurethane foam furniture cushion manufacturing facilities, geometric mean values for daily urinary bromide excretion and urinary *N*-acetyl-*S*-propylcysteine concentrations were approximately 4 times as high for adhesive sprayers as for non-sprayers (Hanley *et al.* 2006, 2009). Concentrations of 1-bromopropane in exhaled breath also were consistently higher among sprayers than among workers performing other jobs. A National Institute for Occupational Safety and Health (NIOSH) Health Hazard Evaluation (HHE) of a furniture foam cushion manufacturing facility found the average difference between end-of-week and start-of-week serum bromide concentrations to be 23 mg/L for exposed workers, compared with 3 mg/L for unexposed workers (Harney *et al.* 2003). NIOSH HHEs and follow-ups at two facilities showed that 1-bromopropane air concentrations (TWAs) could be reduced by 80% or more through implementation of NIOSH recommendations for engineering controls, such as ventilation improvements and enclosure of spray tables (Reh *et al.* 2002).

The general population may be exposed to 1-bromopropane through inhalation of ambient air in the vicinity of industrial facilities where 1-bromopropane is used as an adhesive. EPA used air dispersion modeling to estimate 1-bromopropane concentrations in ambient air at a distance of 100 m from model facilities. The estimated concentrations were 0.138 mg/m^3 [0.0274 ppm] for facilities with average adhesive use and 1.38 mg/m^3 [0.274 ppm] for facilities with high adhesive use (Morris and Wolf 2003). EPA also estimated daily inhalation uptake of 1-bromopropane for a person living 100 m from a model facility to be 0.0537 mg/kg for average-adhesive-use facilities and 0.537 mg/kg for high-adhesive-use facilities.

Based on its production levels and industrial uses, 1-bromopropane may be released to the environment through various waste streams. 1-Bromopropane has been detected in temperate marine macroalgal tissue and is believed to be transported from these algae to the marine environment (HSDB 2006). No data were found on levels of 1-bromopropane in ambient air, drinking water, surface water, soil, food, or consumer products or on non-occupational exposure to 1-bromopropane.

Regulations

California Occupational Safety and Health Standards Board (OSHSB)

Permissible exposure limit (PEL) = 5 ppm.
Potential for dermal absorption.

Department of Transportation (DOT)

Bromopropanes are considered hazardous materials, and special requirements have been set for marking, labeling, and transporting these materials.

Environmental Protection Agency (EPA)

Emergency Planning and Community Right-To-Know Act

Toxics Release Inventory: Listed substance subject to reporting requirements.

Significant New Alternatives Policy (SNAP) Program

The EPA SNAP program reviews alternatives to ozone-depleting substances and approves the use of alternatives that do not present substantially greater risk to the public health and environment than the substance they replace or other available substitutes. The EPA SNAP program has made the following determinations regarding various end uses of 1-bromopropane:

Solvent in industrial equipment for metals cleaning, electronics cleaning, or precision cleaning as a substitute for CFC-113 and methyl chloroform: acceptable (final rule).

Coatings as a substitute for CFC-113, HCFC-141b, and methyl chloroform: acceptable subject to the condition that use is limited to coatings facilities that have provided EPA data which demonstrate their ability to maintain acceptable workplace exposures (proposed rule).

Aerosol solvents as a substitute for CFC-113, HCFC-141b, and methyl chloroform: unacceptable (proposed rule).

Adhesives as a substitute for CFC-113, HCFC-141b, and methyl chloroform: unacceptable (proposed rule).

Guidelines

American Conference of Governmental Industrial Hygienists (ACGIH)

Threshold limit value – time-weighted average (TLV-TWA) = 0.1 ppm.

Environmental Protection Agency (EPA)

Acceptable exposure limit (8-hour time-weighted average) = 25 ppm.

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