2,3-DIBROMOPROPAN-1-OL

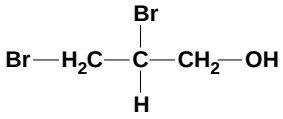
1. Exposure Data

1.1 Chemical and physical data

1.1.1 *Nomenclature*

Chem. Abstr. Serv. Reg. No.: 96-13-9
Deleted CAS Reg. Nos: 116499-75-3; 204570-16-1
Chem. Abstr. Name: 2,3-Dibromo-1-propanol
IUPAC Systematic Name: 2,3-Dibromopropan-1-ol
Synonyms: DBP; DBP (flame retardant); 1,2-dibromopropan-3-ol; 2,3-dibromopropyl alcohol

1.1.2 *Structural and molecular formulae and relative molecular mass*



$C_3H_6Br_2O$ Relative molecular mass: 217.89

1.1.3 *Chemical and physical properties of the pure substance*

(*a*) *Description*: Colourless liquid (Lewis, 1993)
(*b*) *Boiling-point*: 219 °C (Lewis, 1993)
(*c*) *Density*: 2.120 g/cm^3 at 20 °C (Lewis, 1993)
(*d*) *Solubility*: Soluble in water (50–100 g/L), acetone, benzene, diethyl ether and ethanol (National Toxicology Program, 1991; Lewis, 1993)
(*e*) *Spectroscopy data*: infrared (prism [16246], grating [10042]), nuclear magnetic resonance (proton [4214], C-13[2148] and mass spectral data [NIST, 10146] have been reported (Sadtler Research Laboratories, 1980; National Institute of Standards and Technology, 1998)

(*f*) *Stability*: flash point, 112 °C (National Toxicology Program, 1991)
(*g*) *Conversion factor*[1]: $mg/m^3 = 8.91 \times ppm$

1.1.4 *Technical products and impurities*

Trade names for 2,3-dibromopropan-1-ol include: Brominex 257.

1.1.5 *Analysis*

2,3-Dibromopropan-1-ol can be analysed in air, water and food samples by gas chromatography with either electron capture or flame ionization detection (Yurawecz & Puma, 1986; Choudhary, 1987; Matthew & Anastasio, 2000).

1.2 Production

2,3-Dibromopropan-1-ol has been prepared by reaction of allyl alcohol, bromine, and aqueous lithium bromide (Jenkner & Rabe, 1967; Thomas & Levek, 1972).

Information available in 1999 indicated that 2,3-dibromopropan-1-ol was manufactured by three companies in Japan and one company each in Ukraine and the United States (Chemical Information Services, 1999).

1.3 Use

2,3-Dibromopropan-1-ol has been used as an intermediate in the preparation of flame retardants, insecticides and pharmaceuticals (Lewis, 1993). In particular, in the 1970s, the major use of 2,3-dibromopropan-1-ol was in the preparation of the flame retardant tris(2,3-dibromopropyl) phosphate, which was used in textiles; production of this flame retardant other than for research purposes has been discontinued (WHO, 1995; IARC, 1999).

1.4 Occurrence

1.4.1 *Natural occurrence*

2,3-Dibromopropan-1-ol is not known to occur as a natural product.

1.4.2 *Occupational exposure*

No data were available to the Working Group.

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[1] Calculated from: mg/m^3 = (relative molecular mass/24.45) × ppm, assuming a temperature of 25 °C and a pressure of 101 kPa

1.4.3 *Environmental occurrence*

The past use of 2,3-dibromopropan-1-ol as an intermediate in the preparation of the flame retardant tris(2,3-dibromopropyl) phosphate (and as an impurity in tris(2,3-dibromopropyl) phosphate) (Blum & Ames, 1977; Ulsamer *et al*., 1980; WHO, 1995), as a chemical intermediate for insecticidal and pharmaceutical preparations and as a flame retardant itself (Fishbein, 1979) may have resulted in its release into the environment through various waste streams (Ulsamer *et al*., 1980; WHO, 1995).

(*a*) *Industrial effluent discharges*

2,3-Dibromopropan-1-ol has been detected in industrial discharges at levels of 0.5 mg/L (CEC, 1976) and is a hydrolysis product of tris(2,3-dibromopropyl) phosphate (St John *et al.*, 1976). Tris(2,3-dibromopropyl) phosphate has been converted to 2,3-dibromopropan-1-ol by sewage sludge (Alvarez *et al.*, 1982).

(*b*) *Human tissues and secretions*

2,3-Dibromopropan-1-ol, a metabolite of tris(2,3-dibromopropyl) phosphate, has been found in urine samples (at levels of up to 29 ng/mL) from 10 children who were wearing or who had worn tris(2,3-dibromopropyl) phosphate-treated nightwear (Blum *et al*., 1978).

1.5 Regulations and guidelines

No occupational exposure limit or guideline has been established for 2,3-dibromopropan-1-ol.

2. Studies of Cancer in Humans

No data were available to the Working Group.

3. Studies of Cancer in Experimental Animals

3.1 Skin application

3.1.1 *Mouse*

Groups of 50 male and 50 female B6C3F_1 mice, eight weeks of age, were administered doses of 0, 88 or 177 mg/kg bw 2,3-dibromopropan-1-ol (98% purity), applied in 95% ethanol onto the interscapular skin on five days per week for 36–39 weeks for male mice and 39–42 weeks for female mice (the study was terminated

earlier than the planned two years because lymphocytic choriomeningitis virus was detected in serum of sentinel mice). The chemical was administered by pipette at a constant volume of 100 μL. Survival at termination of the study was 100% in the male mice and almost 100% in the females. As shown in Table 1, there were statistically significant increases in the incidence of epithelial tumours of the skin at the site of application of the test substance (squamous papillomas and sebaceous adenomas in high-dose males and females), in squamous papillomas and/or carcinomas of the forestomach in both low- and high-dose females and in adenomas of the liver in high-dose males. In addition, there was a marginal increase in lung adenomas in high-dose males and females, accompanied by a significant increase in focal hyperplasia of the epithelial lining of bronchi, bronchioles or alveoli (National Toxicology Program, 1993; Eustis *et al*., 1995).

3.1.2 *Rat*

Groups of 50 male and 50 male Fischer 344/N rats, eight weeks of age, were administered total doses of 0, 188 or 375 mg/kg bw 2,3-dibromopropan-1-ol (98% purity, confirmed by gas chromatography) applied in 95% ethanol to the interscapular skin on five days per week for 48–51 weeks in male rats and 52–55 weeks in female rats. The study was terminated earlier than the planned two years because of reduced survival of the high-dose groups related to chemical-induced neoplasia, and because sentinel mice housed in the same room as the rats tested positively for lymphocytic choriomeningitis virus. The chemical was administered by pipette at a constant volume of 300 μL. Survival was reduced significantly in treated animals, particularly at the high dose, being 50/50, 41/50 and 17/50 for control, low-dose and high-dose males, respectively, and 48/50, 38/50 and 24/50 for control, low-dose and high-dose females, respectively. As shown in Table 2, there were significantly increased incidences of epithelial tumours of the skin at or near the site of application of the test substance (squamous carcinomas, basal-cell tumours, sebaceous adenomas, keratoacanthomas) in mid- and high-dose males and high-dose females, squamous-cell papillomas or carcinomas of the oral mucosa, oesophagus and/or forestomach in both males and females at both doses, adenocarcinomas of the small intestine of low- and high-dose males, adenomatous polyps of the large intestine in both males and females at both doses, adenomas of the nasal mucosa in both males and females at both doses, Zymbal gland adenomas or adenocarcinomas in either low- and/or high-dose males and females, hepatocellular neoplastic nodules [now generally classified as adenomas] or carcinomas in low- or high-dose females and mammary gland adenocarcinomas and clitoral gland adenocarcinomas in the high-dose females (National Toxicology Program, 1993; Eustis *et al*., 1995).

Table 1. Incidence of primary tumours in B6C3F$_1$ mice exposed to 2,3-dibromopropan-1-ol

Tumour site	Number of animals with tumours					
	Males			Females		
	0 mg/kg bw	88 mg/kg bw	177 mg/kg bw	0 mg/kg bw	88 mg/kg bw	177 mg/kg bw
Skin[a]	0/50	4/50	18/50**	0/50	4/50	9/50**
Forestomach[b]	0/50	14/50**	21/49**	0/50	18/49**	19/50**
Liver[c]	1/50	2/50	11/50*			
Lung[d]	1/50	1/50	6/50*	0/50	3/50	4/50

From National Toxicology Program (1993); Eustis *et al*. (1995)

[a] Squamous-cell papillomas or carcinomas and sebaceous gland adenomas

[b] Squamous-cell papillomas and carcinomas

[c] Hepatocellular adenomas

[d] Alveolar/bronchiolar adenomas

* $p \leq 0.05$ Fisher's exact test

** $p \leq 0.01$ Fisher's exact test

Table 2. Incidence of primary tumours in Fischer 344 rats exposed to 2,3-dibromopropan-1-ol

Tumour site	Number of animals with tumours					
	Males			Females		
	0 mg/kg bw	188 mg/kg bw	375 mg/kg bw	0 mg/kg bw	188 mg/kg bw	375 mg/kg bw
Skin[a]	1/50	22/50**	33/50**	0/50	3/50	18/50**
Mouth[b]	0/50	47/50**	48/50**	0/50	39/50**	49/50**
Oesophagus[c]	0/50	19/50**	33/50**	0/50	9/50**	38/50**
Forestomach[c]	0/50	1/50	17/50**	1/50	3/50	23/50**
Small intestine[d]	0/50	8/50**	11/50**	0/50	3/50	4/50
Large intestine[e]	1/50	13/50**	29/50**	0/50	12/50**	37/50**
Nose[f]	0/50	48/50**	48/50**	0/50	44/50**	49/50**
Liver[g]	0/50	4/50	5/50*	0/50	11/50**	14/50**
Zymbal gland[h]	0/50	9/50**	35/50**	1/50	9/50**	22/50**
Clitoral gland[h]				0/50	1/50	6/50*
Mammary gland[i]				0/50	0/50	5/50*

From National Toxicology Program (1993); Eustis *et al*. (1995)

[a] Squamous-cell papillomas or carcinomas, basal-cell tumours, sebaceous adenomas, keratoacanthomas

[b] Squamous-cell papillomas or carcinomas

[c] Squamous-cell papillomas

[d] Adenocarcinomas

[e] Adenomatous polyps

[f] Adenomas

[g] Neoplastic nodules or carcinomas

[h] Adenomas or adenocarcinomas

[i] Adenocarcinomas

* $p \leq 0.05$, Fisher's exact test

** $p \leq 0.01$, Fisher's exact test

4. Other Data Relevant to an Evaluation of Carcinogenicity and its Mechanisms

4.1 Absorption, distribution, metabolism and excretion

4.1.1 *Humans*

No data were available to the Working Group.

4.1.2 *Experimental systems*

Following oral administration of 50 mg/kg bw 2,3-dibromopropan-1-ol to male Sprague-Dawley rats, two urinary mercapturic acid metabolites were identified as *N*-acetyl-*S*-(2,3-dihydroxypropyl)cysteine and *N*,*N*′-bis-acetyl-*S*,*S*′-(1,3-bis-cysteinyl)-propan-2-ol, respectively. It was inferred that 3-bromo-1,2-propane epoxide is an intermediate in the metabolism of 2,3-dibromopropan-1-ol. In addition, β-bromolactate was produced, presumably as a result of hydrolysis to the α-bromohydrin and subsequent oxidation (Jones & Fakhouri, 1979). Marsden and Casida (1982) identified small amounts of 2-bromoacrylic acid in the urine of rats injected intraperitoneally with 2,3-dibromopropan-1-ol and suggested that this arose by oxidation and dehydrobromination, with 2-bromoacrolein as an unstable intermediate.

4.2 Toxic effects

4.2.1 *Humans*

No data were available to the Working Group.

4.2.2 *Experimental systems*

A single dose of 2,3-dibromopropan-1-ol (61 mg/kg bw) was administered intraperitoneally to male Sprague-Dawley rats (n = 8; 275–325 g) and the rats were killed 48 h later. Serum creatinine, a measure of glomerular filtration rate, was measured. Renal cortical slices were prepared from one kidney and incubated for 90 min in the presence of the organic acid, *para*-aminohippurate, and the organic base, *N*-[^{14}C]methylnicotinamide, to evaluate tubular function. The other kidney was processed by standard histological methods. At this dose, 2,3-dibromopropan-1-ol did not cause any functional or histological change in the kidney (Elliott *et al.*, 1982). In a separate study, male Sprague-Dawley rats (290–310 g) were given an intraperitoneal dose of 39 mg 2,3-dibromopropan-1-ol in 1 mL Emulphor and urine volume was measured for 10 days. There was a two- to threefold increase in urine volume in treated rats, which rapidly returned to normal (Lynn *et al*., 1982).

In a study in which liver microsomes, prepared from male Wistar rats (200–250 g), were incubated with 5×10^{-4} mol/L 2,3-dibromo[^{3}H]propan-1-ol and an NADPH-generating system, covalent binding to protein was determined. Addition of the epoxide hydrolase inhibitor 1,1,1-trichloropropene-2,3-epoxide led to an increase in the protein-binding rate of 2,3-dibromopropan-1-ol (Söderlund *et al*., 1981).

The National Toxicology Program (1993) conducted a series of toxicity studies on 2,3-dibromopropan-1-ol in Fischer 344 rats and B6C3F$_1$ mice. The first study involved 16-day dermal exposure to 2,3-dibromopropan-1-ol. Male and female rats and mice, 52 days and 59 days of age, respectively, were exposed by skin application to 0, 44, 88, 177, 375 or 750 mg/kg bw 2,3-dibromopropan-1-ol in 95% ethanol applied to the shaved subscapular skin. All rodents were treated on five days per week, for a total of 12 exposure days. Two rats and five mice died within two to three days following exposure to 750 mg/kg. However, no treatment-related clinical findings or gross observations were observed in male or female rats or mice exposed to 2,3-dibromopropan-1-ol.

In a 13-week study, male and female Fischer 344 rats and B6C3F$_1$ mice were exposed by skin application to 0, 44, 88, 177, 375 or 750 mg/kg bw 2,3-dibromopropan-1-ol in 95% ethanol applied to the clipped subscapular skin on five days per week for 13 weeks. Animals were approximately seven weeks (rats) or nine weeks (mice) of age when the study began. All rats survived until the end of the study. Eight male mice in the 750-mg/kg bw dose group died during the first four days of the study. Body weight gain decreased significantly in male and female rats exposed to 750 mg/kg bw, by 11% and 13% compared with male and female controls, respectively. Chemical-induced lesions were seen in the kidneys of male rats, the liver of female rats and the liver and lung of both male and female mice. In male rats, nephropathy, characterized by occasional clusters of cortical tubules with thickened basement membranes and basophilic epithelium, was observed in most animals; however, the severity was slightly greater in males rats receiving 375 or 750 mg/kg bw than in the controls. Individual hepatocyte necrosis was observed in all female rats in the 750-mg/kg bw dose group. A dose-related increase in the incidence of pleomorphism of the bronchial and bronchiolar epithelium was observed in male and female mice. This lesion was characterized by loss of nuclear and cellular polarity, cytomegaly with karyomegaly and syncytia formation. The low incidence of this lesion in male mice exposed to 750 mg/kg bw was attributed to the high and early mortality in this group. Centrilobular hepatocellular necrosis was seen in many of the male mice exposed to 750 mg/kg bw that died. There was an increased incidence of hepatocellular necrosis in female mice. Infrequent, scattered individual or small clusters of necrotic hepatocytes, surrounded by a few neutrophils or macrophages, characterized this lesion (National Toxicology Program, 1993; Eustis *et al*., 1995).

In a long-term study, male and female Fischer 344 rats and B6C3F$_1$ mice received skin applications of 0, 188 or 375 mg/kg bw 2,3-dibromopropan-1-ol and 0, 88 or 177 mg/kg bw 2,3-dibromopropan-1-ol, respectively, in 95% ethanol to the subscapular

skin on five days per week. Due to the reduced survival of rats in the high-dose group and because of the detection of antibodies to lymphocytic choriomeningitis virus in sentinel mice, the study was terminated early. Rats received dermal applications for 48–55 weeks and mice received dermal applications for 36–42 weeks. 2,3-Dibromopropan-1-ol caused an increase in the incidence of hyperplasia in the skin, epithelial dysplasia of the forestomach and bronchiolar epithelial pleomorphism and hyperplasia in male and female mice. In rats, 2,3-dibromopropan-1-ol increased the incidences of hyperkeratosis in the skin, forestomach and oesophagus, and epithelial dysplasia in the nose. Cellular pleomorphism and basophilic and clear cell changes were observed in the liver. Nuclear enlargement in the kidney and dose-related increases in the incidences of forestomach ulcers and acanthosis, angiectasis in the liver and renal hyperplasia were also observed in male rats. In female rats, epithelial dysplasia of the forestomach and bile duct hyperplasia in the liver were also observed (National Toxicology Program, 1993).

4.3 Reproductive and developmental effects

No data were available to the Working Group.

4.4 Genetic and related effects

4.4.1 *Humans*

No data were available to the Working Group.

4.4.2 *Experimental systems* (see Table 3 for references)

2,3-Dibromopropan-1-ol was mutagenic in several strains of *Salmonella typhimurium* both in the presence and in the absence of exogenous metabolic systems. It caused preferential cell killing in a polymerase-deficient strain of *Escherichia coli*. It also gave positive results in the mouse lymphoma assay in the absence of S9 activation and caused increases in sister chromatid exchanges and chromosomal aberrations in Chinese hamster ovary cells. It displayed mutagenic activity in male germ cells and caused chromosomal breakage in *Drosophila melanogaster*. However, the compound was inactive in an in-vivo bone-marrow micronucleus assay in male mice.

Table 3. Genetic and related effects of 2,3-dibromopropan-1-ol

Test system	Result[a]		Dose[b] (LED or HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Salmonella typhimurium TA100, reverse mutation	–	+	20 µg/plate	Blum and Ames (1977)
Salmonella typhimurium TA100, TA1535, reverse mutation	+	+	0.1 µL/plate	Carr & Rosenkranz (1978)
Salmonella typhimurium TA100, TA1535, reverse mutation	+	+	218 µg/plate	Nakamura *et al.* (1979)
Salmonella typhimurium TA100, TA1535, reverse mutation	(+)	+	54.5 µg/plate	Lynn *et al.* (1982)
Salmonella typhimurium TA100, TA1535, reverse mutation	+	+	3.3 µg/plate	Haworth *et al.* (1983)
Salmonella typhimurium TA100, reverse mutation	NT	+	10.9	Holme *et al.* (1983)
Salmonella typhimurium TA1535, reverse mutation	+	+	0.1 µL/plate	Prival *et al.* (1977)
Salmonella typhimurium TA1537, TA1538, reverse mutation	–	–	21 800 µg/plate	Nakamura *et al.* (1979)
Salmonella typhimurium TA1537, reverse mutation	–	–	1000 µg/plate	Haworth *et al.* (1983)
Salmonella typhimurium TA1538, reverse mutation	–	–	10 µL/plate	Prival *et al.* (1977)
Salmonella typhimurium TA1538, reverse mutation	–	NT	10 µL/plate	Carr & Rosenkranz (1978)
Salmonella typhimurium TA98, reverse mutation	–	(+)	NR	Nakamura *et al.* (1979)
Salmonella typhimurium TA98, reverse mutation	–	(+)	333 µg/plate	Haworth *et al.* (1983)
Salmonella typhimurium TA102, TA2638, reverse mutation	NT	+	625 µg/plate	Watanabe *et al.* (1998)
Escherichia coli WP2/pKM101, reverse mutation	NT	+	2000 µg/plate	Watanabe *et al.* (1998)
Escherichia coli WP2 *uvr*A/pKM101, reverse mutation	NT	+	313 µg/plate	Watanabe *et al.* (1998)
Drosophila melanogaster, sex-linked recessive lethal mutation	+		500 ppm in feed	Yoon *et al.* (1985); National Toxicology Program (1993)
Drosophila melanogaster, reciprocal translocations	+		440 ppm in feed	Yoon *et al.* (1985)
Drosophila melanogaster, aneuploidy	–[c]		50 mg/mL in feed	Zimmering (1983)

Table 3 (contd)

Test system	Result[a]		Dose[b] (LED or HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Drosophila melanogaster, aneuploidy	(+)		0.5 mg/mL in feed	Zimmering *et al.* (1986)
Drosophila melanogaster, loss of heterozygosity by mitotic recombination	+		21.8 µg/mL in feed	Vogel & Nivard (1993)
Mutation, V79 Chinese hamster lung cells *in vitro*	NT	+	4.36	Holme *et al.* (1983)
Mutation, mouse lymphoma, L5178Y cells *Tk* locus in *vitro*	+	NT	0.0625	National Toxicology Program (1993)
Sister chromatid exchange, Chinese hamster ovary cells *in vitro*	+	+	50.9	National Toxicology Program (1993)
Chromosomal aberrations, Chinese hamster ovary cells *in vitro*	+	+	626.4	National Toxicology Program (1993)
Micronucleus formation, male B6C3F_1 mouse bone-marrow cells *in vivo*	–		100 ip × 3	National Toxicology Program (1993)

[a] +, positive; (+), weak positive; –, negative; NT, not tested; NR, not reported
[b] LED, lowest effective dose; HID, highest ineffective dose; in-vitro tests, µg/mL; in-vivo tests, mg/kg bw/day
[c] Positive for DNA repair deficient strain Mei-9[a]

5. Summary of Data Reported and Evaluation

5.1 Exposure data

2,3-Dibromopropan-1-ol was used as an intermediate to produce the flame retardant tris(2,3-dibromopropyl) phosphate. In the past, it was detected in the urine of children wearing nightwear treated with tris(2,3-dibromopropyl) phosphate. It is still produced for use in the manufacture of other chemicals (possibly flame retardants, insecticides and pharmaceuticals).

5.2 Human carcinogenicity data

No data were available to the Working Group.

5.3 Animal carcinogenicity data

2,3-Dibromopropan-1-ol was tested by skin application in one experiment in mice and in one experiment in rats. In mice, it produced tumours of the skin at the site of application and forestomach in both males and females and tumours of the liver in males. In rats, it produced tumours of the skin at the site of application and of the digestive tract including the mouth, oesophagus, forestomach and intestines, nasal mucosa and Zymbal gland in both males and females, and tumours of the liver, mammary gland and clitoral gland in females.

5.4 Other relevant data

Two mercapturic acid metabolites of 2,3-dibromopropan-1-ol have been identified in the urine of treated rats.

Application of 2,3-dibromopropan-1-ol to the skin of rats and mice for 13 weeks caused kidney lesions in male rats, liver lesions in female rats and liver and lung lesions in both male and female mice.

2,3-Dibromopropan-1-ol was mutagenic in bacterial assays both in the presence and absence of exogenous metabolic systems. It gave positive results in a mammalian cell mutagenesis assay and induced sister chromatid exchanges and chromosomal aberrations in Chinese hamster ovary cells. It was mutagenic in *Drosophila melanogaster*. It was inactive in an in-vivo bone-marrow micronucleus assay in male mice.

5.5 Evaluation

No epidemiological data relevant to the carcinogenicity of 2,3-dibromopropan-1-ol were available.

There is *sufficient evidence* in experimental animals for the carcinogenicity of 2,3-dibromopropan-1-ol.

Overall evaluation

2,3-Dibromopropan-1-ol is *possibly carcinogenic to humans (Group 2B).*

6. References

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