

Bromoform; CASRN 75-25-2

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the [IRIS assessment development process](#). Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the [guidance documents located on the IRIS website](#).

STATUS OF DATA FOR Bromoform

File First On-Line 09/30/1987

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	yes	09/30/1987
Inhalation RfC (I.B.)	qualitative discussion	12/01/1993
Carcinogenicity Assessment (II.)	yes	09/01/1990

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Bromoform

CASRN — 75-25-2

Primary Synonym — Tribromomethane

Last Revised — 09/30/1987

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of

substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
Hepatic lesions	NOEL: 25 mg/kg/day (converted to	1000	1	2E-2 mg/kg/day
Rat, Subchronic Oral Gavage Bioassay	17.9 mg/kg/day)			
NTP, 1989	LOAEL: 50 mg/kg/day (converted to 35.7 mg/kg/day)			

*Conversion Factors -- Doses have been adjusted for treatment schedule (5 days/week)

I.A.2. Principal and Supporting Studies (Oral RfD)

NTP (National Toxicology Program). 1989. Toxicology and Carcinogenicity Studies of Tribromomethane and Bromoform in F344/N rats and B6C3F1 mice (gavage study). NTP-350. Research Triangle Park, NC.

Ten F344/N rats/sex were gavaged with 0, 12, 25, 50, 100, or 200 mg/kg bromoform and 10 B6C3F1 mice/sex were gavaged with 0, 25, 50, 100, 200, or 400 mg/kg bromoform 5 days/week for 13 weeks. Complete histology was conducted on high-dose and vehicle control groups of both species. Liver histology was conducted on all rats and on male mice receiving doses greater than 100 mg/kg. Females of both species did not show any chemically-related effects. A decrease in body weight of both sexes of mice was reported, but was not dose-related. The male mice showed fatty metamorphosis of the liver at doses of 200 and 400 mg/kg. The only effect reported for male rats was a dose-related increase in clear cell foci of the liver. A Fisher Exact Test showed that the incidence of the clear cell foci at doses of 50 mg/kg (the LOAEL) or above was statistically elevated relative to the vehicle control ($p=0.035$), therefore, 25 mg/kg is the NOEL for F344/N rats (NTP, 1989).

Subchronic rat studies by Chu et al. (1982a,b) are not considered suitable for derivation of the

RfD because of difficulties in interpretation of study design and statistical methodology. Tobe et al. (1982) described changes in histology in rats that were not supported by changes in function.

I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — Factors of 10 each were employed for use of a subchronic assay, for extrapolation from animal data, and for protection of sensitive human subpopulation.

MF — None

I.A.4. Additional Comments (Oral RfD)

There are no adequate published data on teratogenicity or reproductive effects of trihalomethanes.

I.A.5. Confidence in the Oral RfD

Study — Medium

Database — Medium

RfD — Medium

The NTP (1989) study utilized both sexes of two species of animals. Both species showed liver lesions, but the study did not investigate clinical chemistries or perform urinalysis; thus, confidence in the study is rated medium. Several studies support the choice of hepatic lesions as the critical effect for the basis of the RfD, but the chosen study is of subchronic duration and reproductive effects have not been monitored; thus, the database is rated medium to low. Medium to low confidence in the RfD follows.

I.A.6. EPA Documentation and Review of the Oral RfD

Source Document — U.S. EPA, 1985

The 1985 Drinking Water Criteria Document for Trihalomethanes is currently undergoing Agency Review.

Other EPA Documentation — None

Agency Work Group Review — 12/02/1985, 02/05/1986, 05/14/1986, 08/13/1987

Verification Date — 08/13/1987

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfD for Bromoform conducted in September 2002 identified one or more significant new studies. IRIS users may request the references for those studies from the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Bromoform

CASRN — 75-25-2

Primary Synonym — Tribromomethane

The health effects data for bromoform (tribromomethane) were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for the derivation of an inhalation RfC. The verification status for this chemical is currently NOT VERIFIABLE. For additional information on the health effects of this chemical, interested parties are referred to the U.S. EPA documentation listed below.

NOT VERIFIABLE status indicates that the U.S. EPA RfD/RfC Work Group deemed the database at the time of review to be insufficient to derive an inhalation RfC according to the Interim Methods for Development of Inhalation Reference Concentrations (U.S. EPA, 1990). This status does not preclude the use of information in cited references for assessment by others.

Tribromomethane is a colorless, heavy liquid with a sweetish odor. Currently, tribromomethane is generated in small amounts for use in laboratories and in electronics testing (ATSDR, 1990). The health effects of exposure to tribromomethane have been reviewed (U.S. EPA, 1991).

Low-level inhalation exposure of humans to tribromomethane results in irritation, lacrimation, and reddening of the face (Sax and Lewis, 1989), suggesting the potential for portal-of-entry effects. The information available on inhalation exposure in laboratory animals comes primarily from older studies that employed high concentrations for acute durations. In these studies, the CNS, liver, and kidney appear to be the major target organs following acute inhalation

exposures. Exposure to tribromomethane vapors may also cause irritation to the respiratory tract and lacrimation (von Oettingen, 1955).

The only studies of subchronic-duration inhalation exposure are reported in abstracts that do not provide sufficient detail for critical evaluation. Tribromomethane had a narcotic effect on rabbits administered single inhalation exposures of 1064-1741 ppm tribromomethane (Dykan, 1964). Rats administered 240 ppm tribromomethane vapor for 10 days developed CNS effects and dystrophic and vascular alterations of the liver and kidney (Dykan, 1964). Vapor concentrations of 24 ppm for 2 months also induced hepatic disorders, characterized by decreased blood clotting and impaired glycogenesis, and altered renal filtration capacity (Dykan, 1962). A concentration of 4.8 ppm tribromomethane in rats did not elicit any adverse effects in rats after 2 months of exposure (Dykan, 1964). These reports provide no details regarding exposure generation or characterization, specific effect measures, or results and are inadequate for derivation of an RfC.

Severe CNS depression was observed in dogs and guinea pigs following acute exposure to extremely high levels (concentrations not specified) (Graham, 1915). Clinical signs included deep sedation, narcosis, and sleep. The CNS depression was rapid in onset and transient, disappearing within a day following cessation of exposure. This study is of limited value because of poor descriptive details on the protocol and unquantified exposure concentrations.

In the early 1900s, tribromomethane was administered as a sedative to children suffering from whooping cough, and several deaths resulted from accidental overdoses (von Oettingen, 1955). The most obvious clinical sign in these fatal cases was profound depression of the CNS that was manifested as unconsciousness, stupor, and loss of reflexes. Death was usually the result of respiratory failure (von Oettingen, 1955). These case reports are of limited value because doses were not quantified; however, the doses were likely in the range of 20-40 drops (150-300 mg/kg/day).

The principal cause of death in laboratory animals following acute oral exposure to tribromomethane is CNS depression (Bowman et al., 1978). Moody and Smuckler (1986) exposed Sprague-Dawley rats (n = 3) to single gavage doses of 1000 mg/kg tribromomethane and observed significant reductions in the liver microsomal cytochrome P-450 content and aminolevulinic acid-dehydratase activity and increases in porphyrin and glutathione content. These effects suggest disturbances in hepatic heme metabolism, because porphyrins are major intermediates in heme synthesis. Chu et al. (1980) reported that female rats were more sensitive than male rats to lethal doses of tribromomethane based on LD50 values of 1388 and 1147 for males and females, respectively.

Studies of 14-90 days duration in rats and mice exposed by gavage have reported liver, kidney, and thyroid effects, as well as transient lethargy at high concentrations (Chu et al., 1982; Condie

et al., 1983; NTP, 1989a; Munson et al., 1982). In the NTP (1989a) study, the incidences of hepatocyte vacuolization in male rats were 3/10, 6/10, 5/10, 8/10, 8/10, and 10/10 for the control, 12-, 25-, 50-, 100-, and 200-mg/kg groups, respectively. In mice, 5/10 male mice that received 200 mg/kg and 8/10 male mice that received 400 mg/kg tribromomethane developed cytoplasmic vacuolization. This dose-related, minimal-to-moderate change involved only a few cells or was diffuse. Behavioral effects (decreased response rate in an operant-conditioning test) were also reported in mice treated by gavage with 100 or 400 mg/kg/day for 60 days (Balster and Borzelleca, 1982).

A 2-year chronic gavage bioassay was conducted in F344/N rats and B6C3F1 mice (50/sex/group), in which doses of 0, 100, or 200 mg/kg (rats and female mice) or 0, 50, or 100 mg/kg (male mice) tribromomethane were administered 5 days/week for 103 weeks (NTP, 1989a). In the high-dose rats (both sexes), mean body weights were significantly (10-28%) lower than controls throughout the second year of the study. Survival of the male rats administered 200 mg/kg was significantly reduced ($p < 0.001$) after week 91. Dose-related lethargy was observed in male and female rats. Nonneoplastic changes, including fatty change and scattered minimal necrosis (males) and mixed cell foci (females), occurred in the liver of treated rats. The incidence of focal or diffuse fatty change in both sexes was increased (males: 23/50, 49/50, and 50/50; females: 19/50, 39/50, and 46/50). The lowest dose tested in this bioassay (100 mg/kg/day) induced effects on the liver, salivary gland, prostate gland, lungs, and forestomach, and thus is considered a LOAEL for rats.

High-dose female mice developed an increased incidence of follicular cell hyperplasia of the thyroid gland (5/49, 4/49, and 19/47). Female mice in both groups exhibited increased incidences of minimal-to-mild fatty change of the liver consisting of scattered hepatocyte foci with vacuolated cytoplasm (1/49, 9/50, and 24/50). Thus, 100 mg/kg also is considered a LOAEL for liver changes in female mice.

No studies were available regarding the respiratory tract absorption of tribromomethane in humans or laboratory animals. After oral administration, tribromomethane is rapidly absorbed. Gastrointestinal absorption following oral exposure has been estimated to be 60-90% complete following a single gavage dose; this percentage of the administered dose was recovered in the expired air, urine, or in the tissues (Mink et al., 1986).

The metabolism of tribromomethane is similar to the metabolism of other trihalomethanes (Anders et al., 1978; Stevens and Anders, 1979, 1981).

Developmental effects were monitored following gavage administration of 50, 100, or 200 mg/kg/day tribromomethane to pregnant Sprague-Dawley rats (15/group) from days 6-15 of gestation (Ruddick et al., 1983). Slight increases in several skeletal anomalies were observed in

treated animals and, to a lesser extent, in controls. No other significant maternal toxicity, fetotoxicity, or teratogenicity was observed.

In a reproductive study, effects of tribromomethane were assessed in Swiss CD-1 mice (n = 17-20/group) exposed by gavage to 0, 50, 100, or 200 mg/kg/day (NTP, 1989b). Postnatal survival was significantly decreased in the 200- mg/kg/day group. No other reproductive effects were seen in the F1 or F2 generations.

The database for tribromomethane is inadequate for the derivation of an RfC. No chronic or subchronic inhalation studies on tribromomethane, and no reproductive or developmental studies that employed an inhalation exposure regimen were found. The toxicokinetic data for the inhalation route are insufficient for route-to-route extrapolation from oral data, and the potential for portal-of-entry (respiratory tract) toxicity has not been adequately characterized.

Anders, M.W., J.L. Stevens, R.W. Sprague, Z. Shaath, and A.E. Ahmed. 1978. Metabolism of haloforms to carbon monoxide. II. In vivo studies. *Drug. Metab. Dispos.* 6(5): 556-560.

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Chu, I., V. Secours, I. Marino, and D.C. Villeneuve. 1980. The acute toxicity of four trihalomethanes in male and female rats. *Toxicol. Appl. Pharmacol.* 52(2): 351-353.

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Graham, E.A. 1915. Late poisoning with chloroform and other alkyl halides in relationship to the halogen acids formed by their chemical dissociation. *J. Exp. Med.* 221: 48-75.

Mink, F.L., T.J. Brown, and J. Rickabaugh. 1986. Absorption, distribution, and excretion of 14C-trihalomethanes in mice and rats. *Bull. Environ. Contam. Toxicol.* 37(5): 752-758.

Moody, D.E. and E.A. Smuckler. 1986. Disturbances in hepatic heme metabolism in rats administered alkyl halides. *Toxicol. Lett.* 32(3): 209-214.

Munson, A.E., L.E. Sain, V.M. Sanders, et al. 1982. Toxicology of organic drinking-water contaminants--trichloromethane, bromodichloromethane, dibromochloromethane, and tribromomethane. *Environ. Health Perspectives*. 46: 117-126.

NTP (National Toxicology Program). 1989a. Toxicology and carcinogenesis studies of tribromomethane (bromoform) (CAS No. 75-25-2) in F344/N rats and B6C3F1 mice (gavage studies). Technical Report Series No. 350. U.S. DHHS, Public Health Service, National Institute of Health, Research Triangle Park, NC. NIH Pub. No. 88-2805. NTIS/PB90-110149.

NTP (National Toxicology Program). 1989b. Bromoform reproduction and fertility assessment in Swiss CD-1 mice when administered by gavage. NTP-89- 068. NTIS/PB89-169254.

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Agency Work Group Review — 02/11/1993

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfC for Bromoform conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

EPA Contacts:

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II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Bromoform

CASRN — 75-25-2

Primary Synonym — Tribromomethane

Last Revised — 09/01/1990

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day.

The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification — B2; probable human carcinogen

Basis — Based on inadequate human data and sufficient evidence of carcinogenicity in animals, namely an increased incidence of tumors after oral administration of bromoform in rats and intraperitoneal administration in mice. Bromoform is genotoxic in several assay systems. Also, bromoform is structurally related to other trihalomethanes (e.g., chloroform, bromodichloromethane, dibromochloromethane) which have been verified as either probable or possible carcinogens.

II.A.2. Human Carcinogenicity Data

Inadequate. Cantor et al. (1978) suggests a positive correlation between levels of trihalomethane in drinking water and the incidence of several human cancers. Additional geographic studies of bromoform indicate that there may be an association between the levels of trihalomethanes in drinking water and the incidence of bladder, colon, rectal, or pancreatic cancer in humans (Kraybill, 1980; Cotruvo, 1981; Carlo and Mettlin, 1980; Isacson et al., 1983; Crump, 1983). However, the information from these studies is considered incomplete and preliminary because their designs do not permit consideration of several possible variables which may be involved (e.g., personal habits, information on residential histories, and past exposures) (NTP, 1988).

II.A.3. Animal Carcinogenicity Data

Sufficient. Bromoform has been tested for carcinogenicity in two species, rat and mouse, by oral or intraperitoneal administration.

In a gavage study (NTP, 1988), F344/N rats (50/sex/group) and B6C3F1 mice (50/sex/group) were administered bromoform in corn oil by gavage 5 days/week for 2 years at 0, 100, or 200 mg/kg (rats and female mice) or 0, 50, or 100 mg/kg (male mice). Decreased body weight and survival in rats and female mice suggest that the MTD was reached. In male rats, mean body weight was decreased in the high- and low-dose groups by 12-28% and 5-14%, respectively. Survival was significantly lower in the high-dose males after week 91. In female rats, body weight was decreased in the high-dose group by 10-25%. In male mice, body weight and survival were comparable to controls. In female mice, however, body weight was decreased in the high- and low-dose groups by 5-16% and 6-11%, respectively; survival was significantly lower in both dose groups after week 77.

Neoplastic lesions (adenomatous polyps or adenocarcinomas) were observed in the large intestine (colon or rectum) of male rats (0/50, 0/50, 3/50) and female rats (0/50, 1/50, 8/50) rats. Adenocarcinomas alone were not significantly increased compared with controls. The reduced survival of male rats in the high-dose group may account for the lower incidence of lesions in this group. No treatment-related tumors were observed in mice at either dose level. Under the conditions of this study, the NTP judged there was clear evidence of carcinogenicity for female rats, some evidence of carcinogenicity for male rats, and no evidence of carcinogenicity for male and female mice.

Theiss et al. (1977) administered bromoform by i.p. injection to male A/St mice (20/group). Doses of 100, 48, and 4 mg/kg were given 3 times/week for a total of 24, 23 or 18 injections, respectively. Mice in the control group received 24 i.p. injections of the vehicle, tricapyrin. Animals were sacrificed 24 weeks after the first injection and the lungs were examined for surface adenomas. Some surface nodules were examined histologically to confirm the morphological appearance of adenomas. The number of lung tumors/mouse for the control, low-, mid-, and high-dose groups were 0.27, 0.53, 1.13, and 0.67 respectively. Only the ratio of the mid-dose group was statistically significantly elevated over that of controls.

In a feeding study with microencapsulated bromoform, Kurokawa (1987) observed no evidence of carcinogenicity in male or female Wistar rats exposed for 24 months at concentrations of 400, 1600, or 6500 ppm.

II.A.4. Supporting Data for Carcinogenicity

Pereira et al. (1982a,b) determined that bromoform did not induce GGTase- positive foci in the rat liver at 1 mM (253 mg/kg) or 0.8 mM (202 mg/kg) following a 2/3 hepatectomy and promotion with phenobarbital. However, Pereira (1983) found that bromoform is a potent inducer of ornithine decarboxylase, which is an indication of tumor promotion activity in the skin and liver.

Bromoform has been shown to produce mutations in *Salmonella typhimurium* strains TA97, TA98, TA100, and TA1535 with and without rat hepatic homogenates (NTP, 1988; Simmon and Tardiff, 1978; Simmon, 1977, 1981; Simmon et al., 1977; Tardiff et al., 1978). Bromoform also produces mutations at the TK locus in mouse cells (NTP, 1988); SCE induction in Chinese hamster ovary cells, human lymphocytes (in vitro) and mouse bone marrow cells (in vivo) (Galloway et al., 1985; Morimoto and Koizumi, 1983; NTP, 1988); chromosomal aberrations in Chinese hamster ovary cells (Galloway et al., 1985); cell cycle delay in human lymphocytes (Morimoto and Koizumi, 1983); and an increased incidence of micronuclei in bone marrow erythrocytes from mice given bromoform i.p. (NTP, 1988).

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

II.B.1. Summary of Risk Estimates

Oral Slope Factor — 7.9E-3 per (mg/kg)/day

Drinking Water Unit Risk — 2.3E-7 per (ug/L)

Extrapolation Method — Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E+2 ug/L
E-5 (1 in 100,000)	4E+1 ug/L
E-6 (1 in 1,000,000)	4E+0 ug/L

II.B.2. Dose-Response Data (Carcinogenicity, Oral Exposure)

Tumor Type: Neoplastic lesions in the large intestine

Test animals: F344/N rat, female

Route: gavage in corn oil

Reference: NTP, 1988

Administered Dose (ppm)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0	0	0/50
100	10.6	1/50
200	20.5	8/50

II.B.3. Additional Comments (Carcinogenicity, Oral Exposure)

The unit risk should not be used if the water concentration exceeds 4E+4 ug/L, since above this concentration the unit risk may not be appropriate. Pharmacokinetic data indicate that gastrointestinal absorption of bromoform in rats is greater than or equal to 80%. However, in the absence of more definitive data, gastrointestinal absorption of 100% was assumed.

II.B.4. Discussion of Confidence (Carcinogenicity, Oral Exposure)

An adequate number of animals was treated for an adequate duration of exposure by a relevant route at two non-zero dose levels. Comprehensive histopathological and statistical analyses were performed.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

II.C.1. Summary of Risk Estimates

Inhalation Unit Risk — 1.1E-6 per ug/cu.m

Extrapolation Method — Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	9E+1 ug/cu.m
E-5 (1 in 100,000)	9 ug/cu.m
E-6 (1 in 1,000,000)	9E-1 ug/cu.m

II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

This unit risk was calculated from the oral data presented in II.B.2.

II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

The inhalation quantitative risk estimate is based on tumor incidence in rats treated by gavage. Several factors suggest that the tumorigenic response is a systemic rather than portal-of-entry effect. Pharmacokinetic data suggest that gastrointestinal absorption is rapid and biotransformation is an activating mechanism. A default value of 50% absorption was used because no data are available to quantify the extent of respiratory absorption.

II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)

Kinetic data for the metabolism of bromoform are insufficient to establish route-specific metabolized doses. Because these data are insufficient, the inhalation cancer unit risk is based on internal dosage estimated from an oral study.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1988

The 1988 Health and Environmental Effects Document is an external draft for review purposes only and has not received Agency Review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 08/02/1989

Verification Date — 08/02/1989

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the cancer assessment for Bromoform conducted in September 2002 identified one or more significant new studies. IRIS users may request the references for those studies from the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

III. [reserved]

IV. [reserved]

V. [reserved]

VI. Bibliography

Substance Name — Bromoform

CASRN — 75-25-2

Primary Synonym — Tribromomethane

VI.A. Oral RfD References

Chu, I., D.C. Villeneuve, V.E. Secours and G.C. Becking. 1982a. Toxicity of trihalomethanes: I. The acute and subacute toxicity of chloroform, bromodichloromethane, chlorodibromomethane and bromoform in rats. *J. Environ. Sci. Health.* B17(3): 205-224.

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VII. Revision History

Substance Name — Bromoform

CASRN — 75-25-2

Primary Synonym — Tribromomethane

Date	Section	Description
09/01/1990	II.	Carcinogen assessment on-line
12/01/1993	I.B.	Inhalation RfC message on-line
12/03/2002	I.A.6., I.B., II.D.2.	Screening-Level Literature Review Findings message has been added.

VIII. Synonyms

Substance Name — Bromoform

CASRN — 75-25-2

Primary Synonym — Tribromomethane

Last Revised — 12/01/1993

- 75-25-2
- Bromoform
- Methane, tribromo-
- Methenyl tribromide
- Tribromomethane