Acute Exposure Guideline Levels for Selected Airborne Chemicals

VOLUME 17

Committee on Acute Exposure Guideline Levels

Committee on Toxicology

Board on Environmental Studies and Toxicology

Division on Earth and Life Studies

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Preface

Extremely hazardous substances (EHSs)[2] can be released accidentally as a result of chemical spills, industrial explosions, fires, or accidents involving railroad cars and trucks transporting EHSs. Workers and residents in communities surrounding industrial facilities where EHSs are manufactured, used, or stored and in communities along the nation's railways and highways are potentially at risk of being exposed to airborne EHSs during accidental releases or intentional releases by terrorists. Pursuant to the Superfund Amendments and Reauthorization Act of 1986, the U.S. Environmental Protection Agency (EPA) has identified approximately 400 EHSs on the basis of acute lethality data in rodents.

As part of its efforts to develop acute exposure guideline levels for EHSs, EPA and the Agency for Toxic Substances and Disease Registry (ATSDR) in 1991 requested that the National Research Council (NRC) develop guidelines for establishing such levels. In response to that request, the NRC published *Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances* in 1993. Subsequently, *Standard Operating Procedures for Developing Acute Exposure Guideline Levels for Hazardous Substances* was published in 2001, providing updated procedures, methodologies, and other guidelines used by the National Advisory Committee (NAC) on Acute Exposure Guideline Levels for Hazardous Substances and the Committee on Acute Exposure Guideline Levels (AEGLs) in developing the AEGL values.

Using the 1993 and 2001 NRC guidelines reports, the NAC—consisting of members from EPA, the Department of Defense (DOD), the Department of Energy (DOE), the Department of Transportation (DOT), other federal and state governments, the chemical industry, academia, and other organizations from the private sector—has developed AEGLs for more than 270 EHSs.

In 1998, EPA and DOD requested that the NRC independently review the AEGLs developed by NAC. In response to that request, the NRC organized within its Committee on Toxicology (COT) the Committee on Acute Exposure Guideline Levels, which prepared this report. This report is the seventeenth vol*xiv Preface*

ume in that series. AEGL documents for acrylonitrile, carbon tetrachloride, cyanogen, epichlorohydrin, ethylene chlorohydrin, toluene, trimethylacetyl chloride, hydrogen bromide, and boron tribromide are each published as an appendix in this report. The committee concludes that the AEGLs developed in these appendixes are scientifically valid conclusions based on the data reviewed by NAC and are consistent with the NRC guideline reports. AEGL reports for additional chemicals will be presented in subsequent volumes.

[2] As defined pursuant to the Superfund Amendments and Reauthorization Act of 1986.

The committee's review of the AEGL documents involved both oral and written presentations to the committee by the authors of the documents. The committee examined the draft documents and provided comments and recommendations for how they could be improved in a series of interim reports. The authors revised the draft AEGL documents based on the advice in the interim reports and presented them for reexamination by the committee as many times as necessary until the committee was satisfied that the AEGLs were scientifically justified and consistent with the 1993 and 2001 NRC guideline reports. After these determinations have been made for an AEGL document, it is published as an appendix in a volume such as this one.

The interim reports of the committee that led to this report were reviewed in draft form by individuals selected for their diverse perspectives and technical expertise, in accordance with procedures approved by the NRC's Report Review Committee. The purpose of this independent review is to provide candid and critical comments that will assist the institution in making its published report as sound as possible and to ensure that the report meets institutional standards for objectivity, evidence, and responsiveness to the study charge. The review comments and draft manuscript remain confidential to protect the integrity of the deliberative process. We wish to thank the following individuals for their review of the committee interim reports, which summarize the committee's conclusions and recommendations for improving NAC's AEGL documents for acrylonitrile (interim reports 19b, 21a, and 22), carbon tetrachloride (interim reports 13, 14, 18, and 22), cyanogen (interim report 19a), epichlorohydrin (interim reports 15, 19a, 20a, and 21a), ethylene chlorohydrin (interim reports 20a and 21a), toluene (interim reports 12, 18, and 22), trimethylacetyl chloride (interim reports 20a and 21a), hydrogen bromide (interim reports 16, 18, and 22), and boron tribromide (interim reports 19a and 22): Deepak Bhalla (Wayne State University), Harvey Clewell (The Hamner Institutes for Health Sciences), Jeffrey Fisher (U.S. Food and Drug Administration), David Gaylor (Gaylor and Associates, LLC), Sam Kacew (University of Ottawa), A. Wallace Hayes (Harvard School of Public Health), Rogene Henderson (Lovelace Respiratory Research Institute [retired]), James McDougal (Wright State University [retired]), Charles Reinhardt (DuPont Haskell Laboratory [retired]), Andrew Salmon (California Environmental Protection Agency), Joyce Tsuji (Exponent, Inc.), Bernard Wagner (New York University Medical Center [retired]), and Judith Zelikoff (New York University).

Although the reviewers listed above have provided many constructive comments and suggestions, they were not asked to endorse the conclusions or recommendations, nor did they see the final draft of this volume before its re

lease. The review of interim reports was overseen by David Gaylor (Gaylor and Associates, LLC), Sidney Green, Jr., (Howard University), and Robert Goyer (University of Western Ontario [retired]). Appointed by the NRC, they were responsible for making certain that an independent examination of the interim reports was carried out in accordance with institutional procedures and that all review comments were carefully considered. Responsibility for the final content of this report rests entirely with the authoring committee and the institution.

The committee gratefully acknowledges the valuable assistance provided by Ernest Falke and Iris A. Camacho from EPA. The committee also acknowledges Susan Martel, the project director for her work this project. Other staff members who contributed to this effort are James J. Reisa (director of the Board on Environmental Studies and Toxicology), Radiah Rose (manager of editorial projects), Mirsada Karalic-Loncarevic (manager of the Technical Information Center), and Tamara Dawson (program associate). Finally, I would like to thank all members of the committee for their expertise and dedicated effort throughout the development of this report.

Edward C. Bishop, *Chair*
Committee on Acute Exposure
Guideline Levels

Contents

Acute Exposure Guideline Levels for Selected Airborne Chemicals

VOLUME 17

National Research Council Committee Review of Acute Exposure Guideline Levels for Selected Airborne Chemicals

This report is the seventeenth volume in the series *Acute Exposure Guideline Levels for Selected Airborne Chemicals*.

In the Bhopal disaster of 1984, approximately 2,000 residents living near a chemical plant were killed and 20,000 more suffered irreversible damage to their eyes and lungs following accidental release of methyl isocyanate. The toll was particularly high because the community had little idea what chemicals were being used at the plant, how dangerous they might be, or what steps to take in an emergency. This tragedy served to focus international attention on the need for governments to identify hazardous substances and to assist local communities in planning how to deal with emergency exposures.

In the United States, the Superfund Amendments and Reauthorization Act (SARA) of 1986 required that the U.S. Environmental Protection Agency (EPA) identify extremely hazardous substances (EHSs) and, in cooperation with the Federal Emergency Management Agency and the U.S. Department of Transportation, assist local emergency planning committees (LEPCs) by providing guidance for conducting health hazard assessments for the development of emergency response plans for sites where EHSs are produced, stored, transported, or used. SARA also required that the Agency for Toxic Substances and Disease Registry (ATSDR) determine whether chemical substances identified at hazardous waste sites or in the environment present a public health concern.

As a first step in assisting the LEPCs, EPA identified approximately 400 EHSs largely on the basis of their immediately dangerous to life and health values, developed by the National Institute for Occupational Safety and Health. Although several public and private groups, such as the Occupational Safety and Health Administration and the American Conference of Governmental Industrial Hygienists, have established exposure limits for some substances and some exposures (e.g., workplace or ambient air quality), these limits are not easily or directly translated into emergency exposure limits for exposures at high levels

but of short duration, usually less than 1 hour (h), and only once in a lifetime for the general population, which includes infants (from birth to 3 years of age), children, the elderly, and persons with diseases, such as asthma or heart disease.

The National Research Council (NRC) Committee on Toxicology (COT) has published many reports on emergency exposure guidance levels and spacecraft maximum allowable concentrations for chemicals used by the U.S. Department of Defense (DOD) and the National Aeronautics and Space Administration (NASA) (NRC 1968, 1972, 1984a,b,c,d, 1985a,b, 1986a, 1987, 1988, 1994, 1996a,b, 2000a, 2002a, 2007a, 2008a). COT has also published guidelines for developing emergency exposure guidance levels for military personnel and for astronauts (NRC 1986b, 1992, 2000b). Because of COT's experience in recommending emergency exposure levels for short-term exposures, in 1991 EPA and ATSDR requested that COT develop criteria and methods for developing emergency exposure levels for EHSs for the general population. In response to that request, the NRC assigned this project to the COT Subcommittee on Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances. The report of that subcommittee, *Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances* (NRC 1993), provides step-by-step guidance for setting emergency exposure levels for EHSs. Guidance is given on what data are needed, what data are available, how to evaluate the data, and how to present the results.

In November 1995, the National Advisory Committee (NAC)[3] for Acute Exposure Guideline Levels for Hazardous Substances was established to identify, review, and interpret relevant toxicologic and other scientific data and to develop acute exposure guideline levels (AEGLs) for high-priority, acutely toxic chemicals. The NRC's previous name for acute exposure levels—community emergency exposure levels (CEELs)—was replaced by the term AEGLs to reflect the broad application of these values to planning, response, and prevention in the community, the workplace, transportation, the military, and the remediation of Superfund sites.

AEGLs represent threshold exposure limits (exposure levels below which adverse health effects are not likely to occur) for the general public and are applicable to emergency exposures ranging from 10 minutes (min) to 8 h. Three levels—AEGL-1, AEGL-2, and AEGL-3—are developed for each of five exposure periods (10 min, 30 min, 1 h, 4 h, and 8 h) and are distinguished by

[3] NAC completed its chemical reviews in October 2011. The committee was composed of members from EPA, DOD, many other federal and state agencies, industry, academia, and other organizations. From 1996 to 2011, the NAC discussed over 300 chemicals and developed AEGLs values for at least 272 of the 329 chemicals on the AEGLs priority chemicals lists. Although the work of the NAC has ended, the NAC-reviewed technical support documents are being submitted to the NRC for independent review and finalization.

varying degrees of severity of toxic effects. The three AEGLs are defined as follows:

AEGL-1 is the airborne concentration (expressed as ppm [parts per million] or mg/m^3 [milligrams per cubic meter]) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic nonsensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.

AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.

AEGL-3 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience life-threatening adverse health effects or death.

Airborne concentrations below AEGL-1 represent exposure levels that can produce mild and progressively increasing but transient and nondisabling odor, taste, and sensory irritation or certain asymptomatic nonsensory adverse effects. With increasing airborne concentrations above each AEGL, there is a progressive increase in the likelihood of occurrence and the severity of effects described for each corresponding AEGL. Although the AEGL values represent threshold levels for the general public, including susceptible subpopulations, such as infants, children, the elderly, persons with asthma, and those with other illnesses, it is recognized that individuals, subject to idiosyncratic responses, could experience the effects described at concentrations below the corresponding AEGL.

SUMMARY OF REPORT ON GUIDELINES FOR DEVELOPING AEGLS

As described in *Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances* (NRC 1993) and the NRC guidelines report *Standing Operating Procedures for Developing Acute Exposure Guideline Levels for Hazardous Chemicals* (NRC 2001a), the first step in establishing AEGLs for a chemical is to collect and review all relevant published and unpublished information. Various types of evidence are assessed in establishing AEGL values for a chemical. These include information from (1) chemicalphysical characterizations, (2) structure-activity relationships, (3) in vitro toxicity studies, (4) animal toxicity studies, (5) controlled human studies, (6) observations of humans involved in chemical accidents, and (7) epidemiologic studies. Toxicity data from human studies are most applicable and are used when

available in preference to data from animal studies and in vitro studies. Toxicity data from inhalation exposures are most useful for setting AEGLs for airborne chemicals because inhalation is the most likely route of exposure and because extrapolation of data from other routes would lead to additional uncertainty in the AEGL estimate.

For most chemicals, actual human toxicity data are not available or critical information on exposure is lacking, so toxicity data from studies conducted in laboratory animals are extrapolated to estimate the potential toxicity in humans. Such extrapolation requires experienced scientific judgment. The toxicity data for animal species most representative of humans in terms of pharmacodynamic and pharmacokinetic properties are used for determining AEGLs. If data are not available on the species that best represents humans, data from the most sensitive animal species are used. Uncertainty factors are commonly used when animal data are used to estimate risk levels for humans. The magnitude of uncertainty factors depends on the quality of the animal data used to determine the noobserved-adverse-effect level (NOAEL) and the mode of action of the substance in question. When available, pharmacokinetic data on tissue doses are considered for interspecies extrapolation.

For substances that affect several organ systems or have multiple effects, all end points (including reproductive [in both genders], developmental, neurotoxic, respiratory, and other organ-related effects) are evaluated, the most important or most sensitive effect receiving the greatest attention. For carcinogenic chemicals, excess carcinogenic risk is estimated, and the AEGLs corresponding to carcinogenic risks of 1 in 10,000 (1×10^{-4}), 1 in 100,000 (1×10^{-5}), and 1 in 1,000,000 (1×10^{-6}) exposed persons are estimated.

REVIEW OF AEGL REPORTS

As NAC began developing chemical-specific AEGL reports, EPA and DOD asked the NRC to review independently the NAC reports for their scientific validity, completeness, and consistency with the NRC guideline reports (NRC 1993, 2001a). The NRC assigned this project to the COT Committee on Acute Exposure Guideline Levels. The committee has expertise in toxicology, epidemiology, occupational health, pharmacology, medicine, pharmacokinetics, industrial hygiene, and risk assessment.

The AEGL draft reports were initially prepared by ad hoc AEGL development teams consisting of a chemical manager, chemical reviewers, and a staff scientist of the NAC contractors—Oak Ridge National Laboratory and subsequently SRC, Inc. The draft documents were then reviewed by NAC and elevated from "draft" to "proposed" status. After the AEGL documents were approved by NAC, they were published in the *Federal Register* for public comment. The reports were then revised by NAC in response to the public

comments, elevated from "proposed" to "interim" status, and sent to the NRC Committee on Acute Exposure Guideline Levels for final evaluation.

The NRC committee's review of the AEGL reports prepared by NAC and its contractors involves oral and written presentations to the committee by the authors of the reports. The NRC committee provides advice and recommendations for revisions to ensure scientific validity and consistency with the NRC guideline reports (NRC 1993, 2001a). The revised reports are presented at subsequent meetings until the committee is satisfied with the reviews.

Because of the enormous amount of data presented in AEGL reports, the NRC committee cannot verify all of the data used by NAC. The NRC committee relies on NAC and the contractors for the accuracy and completeness of the toxicity data cited in the AEGL reports. Thus far, the committee has prepared sixteen reports in the series *Acute Exposure Guideline Levels for Selected Airborne Chemicals* (NRC 2001b, 2002b, 2003, 2004, 2007b, 2008b, 2009, 2010a,b, 2011, 2012a,b,c, 2013a,b, 2014). This report is the seventeenth volume in that series. AEGL documents for acrylonitrile, carbon tetrachloride, cyanogen, epichlorohydrin, ethylene chlorohydrin, toluene, trimethylacetyl chloride, hydrogen bromide, and boron tribromide are each published as an appendix in this report. The committee concludes that the AEGLs developed in these appendixes are scientifically valid conclusions based on the data reviewed by NAC and are consistent with the NRC guideline reports. AEGL reports for additional chemicals will be presented in subsequent volumes.

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Appendixes

9

Boron Tribromide[4]

Acute Exposure Guideline Levels

PREFACE

Under the authority of the Federal Advisory Committee Act (FACA) P.L. 92-463 of 1972, the National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL Committee) has been established to identify, review, and interpret relevant toxicologic and other scientific data and develop AEGLs for high-priority, acutely toxic chemicals.

AEGLs represent threshold exposure limits for the general public and are applicable to emergency exposure periods ranging from 10 minutes (min) to 8 hours (h). Three levels—AEGL-1, AEGL-2, and AEGL-3—are developed for each of five exposure periods (10 and 30 min and 1, 4, and 8 h) and are distinguished by varying degrees of severity of toxic effects. The three AEGLs are defined as follows:

AEGL-1 is the airborne concentration (expressed as parts per million or milligrams per cubic meter [ppm or mg/m^3]) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, nonsensory

[4] This document was prepared by the AEGL Development Team composed of Sylvia Talmage (Oak Ridge National Laboratory), Lisa Ingerman (SRC, Inc.), Chemical Manager Robert Benson (National Advisory Committee [NAC] on Acute Exposure Guideline Levels for Hazardous Substances), and Ernest V. Falke (U.S. Environmental Protection Agency). The NAC reviewed and revised the document and AEGLs as deemed necessary. Both the document and the AEGL values were then reviewed by the National Research Council (NRC) Committee on Acute Exposure Guideline Levels. The NRC committee has concluded that the AEGLs developed in this document are scientifically valid conclusions based on the data reviewed by the NRC and are consistent with the NRC guidelines reports (NRC 1993, 2001).

effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.

AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.

AEGL-3 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience life-threatening health effects or death.

Airborne concentrations below the AEGL-1 represent exposure concentrations that could produce mild and progressively increasing but transient and nondisabling odor, taste, and sensory irritation or certain asymptomatic, nonsensory effects. With increasing airborne concentrations above each AEGL, there is a progressive increase in the likelihood of occurrence and the severity of effects described for each corresponding AEGL. Although the AEGL values represent threshold concentrations for the general public, including susceptible subpopulations, such as infants, children, the elderly, persons with asthma, and those with other illnesses, it is recognized that individuals, subject to idiosyncratic responses, could experience the effects described at concentrations below the corresponding AEGL.

SUMMARY

Boron tribromide is a colorless, fuming liquid with a sharp or acrid, irritating odor. It hydrolyzes or decomposes violently in the presence of water or moist air, producing heat, hydrogen bromide, and boric acid. In the presence of water, conversion to hydrogen bromide is complete. Boron tribromide is used as a catalyst in the manufacture of diborane, ultrahigh purity boron, and semiconductors. It is an excellent demethylating or dealkylating agent for ethers, particularly in the production of pharmaceuticals. As a Lewis acid catalyst it finds applications in olefin polymerization and in Friedel-Crafts chemistry. Theoretically, one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide.

No human or animal data were available to derive AEGL values for boron tribromide, as the reactive nature of boron tribromide precludes toxicity testing. Hydrogen bromide is considered the irritant hydrolysis product as boric acid has been used in topical antiseptic powders and ointments, and dilute solutions are used in eye and mouthwash solutions. On the basis that boron tribromide hydrolyzes into hydrogen bromide, the AEGL values for boron tribromide were based on the AEGL values for hydrogen bromide. The boron tribromide values were derived by dividing the hydrogen bromide AEGL values by 3. See Chapter

8 for the technical support document on hydrogen bromide. The AEGL values for boron tribromide are presented in Table 9-1.

TABLE 9-1 AEGL Values for Boron Tribromide

Classification	10 min	30 min	1 h	4 h	8 h	End Point[a]
AEGL-1 (nondisabling)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	Analogy with hydrogen bromide
AEGL-2 (disabling)	83 ppm (850 mg/m^3)	28 ppm (290 mg/m^3)	13 ppm (130 mg/m^3)	3.3 ppm (34 mg/m^3)	1.7 ppm (17 mg/m^3)	Analogy with hydrogen bromide
AEGL-3 (lethal)	250 ppm (2,600 mg/m^3)	83 ppm (850 mg/m^3)	40 ppm (410 mg/m^3)	10 ppm (100 mg/m^3)	5 ppm (51 mg/m^3)	Analogy with hydrogen bromide

[a] On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide, the AEGL values for hydrogen bromide were divided by three.

1. INTRODUCTION

Boron tribromide is a colorless, fuming liquid with a sharp or acrid, irritating odor. It hydrolyzes or decomposes in the presence of water or moist air, producing heat, hydrogen bromide, and boric acid (ACGIH 2001; O'Neil et al. 2006; Krzystowczyk 2007; Ball et al. 2012). Boron tribromide is nonflammable (BOC 1996). However, as a result of the strong Lewis acid properties of bromide, the reaction with water is violent and results in risk of explosion. This reactivity, resulting in caustic action at the site of exposure, makes it impossible to determine systemic toxicity. Breakdown to hydrogen bromide in water is complete (Krzystowczyk 2007). Theoretically, three moles of hydrogen bromide are produced from one mole of boron tribromide. Additional chemical and physical properties are listed in Table 9-2.

The boron trihalides are important industrial chemicals that are used as Lewis acid catalysts and in chemical vapor deposition processes. As a Lewis acid catalyst, boron tribromide finds applications in olefin polymerization and in Friedel-Crafts chemistry. Boron tribromide is used as a catalyst in the manufacture of diborane and ultrahigh purity boron. Boron tribromide is an excellent demethylating or dealkylating agent for ethers in the production of pharmaceuticals. The electronics industry uses boron tribromide as a source of boron in predeposition processes for doping in the manufacture of semi-conductors (Albemarle Corporation 2004; HSDB 2013). Boron tribromide is produced on a large scale by the reaction of bromine and granulated boron carbide (Alam et al. 2003). It is commercially available neat or in solution with

dichloromethane or hexanes (Doyaguez 2005). Boron tribromide is shipped in 70-kg stainless-steel drums (Albemarle Corporation 2004).

2. HUMAN TOXICITY DATA

By analogy with hydrogen bromide, the acrid odor of boron tribromide should be detectable at 2 ppm (Ball et al. 2012). Data were insufficient to set a level of odor awareness. Boron tribromide is considered irritating to the skin and mucus membranes and corrosive to the eyes (HSDB 2013). No inhalation data on lethal concentrations, developmental or reproductive toxicity, genotoxicity, or carcinogenicity of boron tribromide in humans were found. Data on the breakdown products, hydrogen bromide and boric acid, were available.

The Connecticut State Department of Health (unpublished data, 1955) evaluated responses of human subjects to hydrogen bromide vapors. Six volunteers inhaled hydrogen bromide at 2-6 ppm for durations of several minutes (see Table 9-3). The odor was detectable by all subjects at all concentrations. None of the subjects experienced ocular irritation. Only one subject experienced nasal and throat irritation at 3 ppm. One subject experienced throat irritation at the higher concentrations, and all subjects experienced nasal irritation at 5 and 6 ppm. Although exposure at 5 ppm caused nasal and throat irritation in a majority of the volunteers, the report stated that "it was considered unlikely that noticeable disturbances will occur if peak concentrations do not exceed this value for brief periods."

TABLE 9-2 Chemical and Physical Properties of Boron Tribromide

Parameter	Value	References
Synonyms	Boron bromide; tribromoborane	HSDB 2013
CAS registry no.	10294-33-4	HSDB 2013
Chemical formula	BBr_3	HSDB 2013
Molecular weight	250.57	HSDB 2013
Physical state	Liquid	HSDB 2013
Boiling point	91.3ºC	HSDB 2013
Melting point	–46ºC	HSDB 2013
Density (water =1)	2.60 g/mL	HSDB 2013
Solubility in water	Hydrolyzes violently	HSDB 2013
Vapor pressure	69 mm Hg at 25ºC	Barber et al. 1964; ACGIH 2001
Flammability limits	Non-flammable	BOC Gases 1996

Conversion factors	1 ppm = 10.25 mg/m^3 1 mg/m^3 = 0.097 ppm	ACGIH 2001

TABLE 9-3 Human Responses to Hydrogen Bromide Vapor

	Number of Subjects with Response (n = 6)				
Response	2 ppm	3 ppm	4 ppm	5 ppm	6 ppm
Detectable odor	6	6	6	6	6
Nasal irritation	0	1	3	6	6
Throat irritation	0	1	1	1	1
Ocular irritation	0	0	0	0	0

Source: Connecticut State Department of Health, unpublished data, 1955.

Although the inhalation toxicity of boron oxide and borates is well established (ATSDR 2010), no information on the inhalation toxicity of boric acid in humans was found. Boric acid is used as an astringent and antiseptic. Borates in general are considered either nonirritating or mild dermal and ocular irritants (Hubbard 1998). Oral exposure to boric acid has low acute toxicity in adults (Hubbard 1998), but there are some reports of fatalities (Jordan and Crissey 1957). Death has occurred from intake of less than 5 g in infants and from 5-20 g in adults (O'Neil et al. 2006). Wong et al. (1964) reported that five of 14 infants were killed within 2-3 day after ingesting boric acid; the infants that died consumed 4.6-14 g of the chemical, whereas those that survived consumed 2-4.5 g. Mortality was 70% among infants who were accidentally poisoned with boric acid (Goldbloom and Goldbloom 1953).

Boric acid has been held responsible for systemic intoxication after ingestion, injection, application to damaged skin, or enema (McIntyre and Burke 1937; Brooke and Boggs 1951; Ducey and Williams 1953; Johnstone et al. 1955; Rosen and Haggerty 1956; Jordan and Crissey 1957). There is no evidence that boric acid or borates are absorbed through intact skin (Sciarra 1958). Whether the apparent increased susceptibility of infants and children is due to immaturity of the kidneys (which accounts for the primary route of elimination) (Locksley and Sweet 1954) or is related to the relatively high dose on a body weight basis (Young et al. 1949) is not clear. Autopsy is generally unremarkable with deaths occurring several days after exposure, but pancreatic lesions and those in kidneys and brain have been described (McNally and Rust 1928; Valdes-Dapena and Arey 1962). Although seizures can precede death, the hyperchloremic metabolic acidosis is a characteristic feature (Wong et al. 1964).

3. ANIMAL TOXICITY DATA

No data on the lethality, developmental or reproductive effects, genotoxicity, or chronic toxicity or carcinogenicity of boron tribromide were available. Data on the breakdown products, boric acid and hydrogen bromide were available. Toxicity data on other hydrogen halides, such as hydrogen chloride and hydrogen fluoride, are also relevant.

Inhalation exposure of male Swiss-Webster mice to boric acid aerosol at 300 mg/m^3 (approximately 120 ppm), the highest achievable concentration, resulted in a decrease in respiratory rate by less than 20%. The effect was attributed to sensory irritation, as there was no indication of pulmonary effects (Krystofiak and Schaper 1996). The oral LD_{50} (lethal dose, 50% lethality) for boric acid in rats is 5 g/kg (O'Neil et al. 2006).

Groups of five to eight Fisher 344 rats were exposed by inhalation to hydrogen chloride or hydrogen bromide at approximately 1,300 ppm for 30 min (Stavert et al. 1991). Animals were placed in body plethysmographs for noseonly exposure. Mortality rates were 6% in the hydrogen-chloride group and 8% in the hydrogen-bromide group. Lesions were confined to the nasal passages.
Moderate to severe fibrinonecrotic rhinitis was observed only in the anterior most region of the nasal passages. The same authors (Kusewitt et al. 1989) exposed rats to hydrogen chloride or hydrogen bromide at concentrations of 1001,000 ppm for 30 min. No deaths occurred at 1,000 ppm before the animals were killed after 24 h. Lesions were confined to the nasal passages with no damage to the lungs. No further details were reported in the abstract.

MacEwen and Vernot (1972) exposed groups of 10 male Sprague-Dawley rats to hydrogen bromide at 2,205-3,822 ppm for 1 h. Groups of 10 ICR-derived mice were exposed at 507-1,163 ppm for 1 h. Mortalities from these exposures are summarized in Table 9-4. The 1-h LC_{50} for hydrogen bromide in rats was 2,858 ppm (95% confidence limits: 2,481-3,164 ppm), and the 1-h LC_{50} in mice was 814 ppm (95% confidence limits: 701-947 ppm). Responses in the animals were dose-related, and followed a sequence of nasal and ocular irritation, labored breathing, gasping, and convulsions. The fur turned orange-brown during the exposures, and burns were observed on the exposed skin of both species.

Barrow et al. (1977) exposed groups of four male Swiss-Webster mice to hydrogen chloride at concentrations of 40, 99, 245, 440, or 943 ppm for 10 min. An RD_{50} (concentration that reduces the respiratory rate by 50%) of 309 ppm was calculated. At 99 ppm, approximately one-third of the RD_{50}, the decrease in respiratory rate was 25-30%.

4. SPECIAL CONSIDERATIONS

4.1. Metabolism and Disposition

Boron tribromide undergoes rapid hydrolysis in the presence of water or moist air, producing heat, hydrogen bromide, and boric acid (ACGIH 2001). No information on the hydrolysis half-life was found, but reaction with water or moisture in the air is rapid and complete (Krzystowczyk 2007).

4.2. Mechanism of Toxicity

The mechanism of toxicity of boron tribromide appears to be related to the formation of hydrobromic acid. Hydrogen bromide is a severe irritant to the eyes, skin, and nasal passages; high concentration may penetrate to the lungs resulting in edema and hemorrhage (Kusewitt et al. 1989; Stavert et al. 1991; see Chapter 8).

Boric acid is used as an astringent and antiseptic. Orally, it is of low acute toxicity to adult humans. Effects include nausea, vomiting, abdominal pain, diarrhea, depression of the central nervous system, and convulsions. Death has occurred from intakes of less than 5 g in infants and from 5-20 g in adults (ACGIH 2005). In the occupational setting, exposure to airborne boric acid and borax dusts is associated with respiratory and ocular irritation without measurable changes in pulmonary function (ATSDR 2010). No studies were available that describe the mechanism of toxicity of systemic effects.

TABLE 9-4 One-Hour Inhalation Studies of Hydrogen Bromide

Species	Concentration (ppm)	Mortality Ratio
Rat	2,205	1/10
	2,328	4/10
	2,759	4/10
	3,253	6/10
	3,711	7/10
	3,822	10/10
Mouse	507	0/10
	875	7/10
	1,036	9/10
	1,163	10/10

Source: Adapted from McEwen and Vernot 1972.

4.3. Structure-Activity Relationships

Because one mole of boron tribromide breaks down into three moles of hydrogen bromide, the toxicity of hydrogen bromide and related hydrogen halides

are relevant. On the basis of lethality, hydrogen fluoride is the most toxic, followed by hydrogen bromide and then hydrogen chloride, although the values for hydrogen bromide and hydrogen chloride were similar (MacEwen and Vernot 1972). At sublethal concentrations, the severity and extent of lesions in the upper respiratory tract of rats exposed to hydrogen halides by inhalation were greatest for hydrogen fluoride, followed by hydrogen chloride and then hydrogen bromide. However, the severity and extent of lesions were similar among the three chemicals (Kusewitt et al. 1989; Stavert et al. 1991).

The halides chlorine, bromine, and iodine, are exceptionally good leaving groups, readily hydrolyzing to their acid forms in the aqueous environment. The exception is boron trifluoride. The lack of outer orbitals on the fluoride atom results in a shorter and, thus, stronger bond than what is present with the other halides (Krzystowczyk 2007). Toxicity comparisons of the boron trihalides with their breakdown products are summarized in Table 9-5. The 4-h LC_{50} for boron trifluoride in rats is 1.21 mg/L (approximately 436 ppm) (Rusch et al. 1986). The 1-h LC_{50} for hydrogen fluoride ranges from 966 ppm to 1,395 ppm (Vernot et al. 1977; NRC 2004). The 1-h LC_{50} for boron trichloride in rats is 2,541 ppm (Vernot et al. 1977). The 1-h LC_{50} for hydrogen chloride in rats is 3,124 ppm (Vernot et al. 1977). The similarity in toxicity values for boron trifluoride and boron trichloride with the hydrolysis products tends to support limited hydrolysis.

4.4. Other Relevant Information

No information on species variability, susceptible populations, or concentration-exposure duration relationships for boron tribromide was available. For hydrogen halides, such as hydrogen fluoride and hydrogen chloride, the mouse is more susceptible than the rat to the lethal effects (NRC 1991).

5. DATA ANALYSIS FOR AEGL-1

5.1. Human Data Relevant to AEGL-1

No human data on boron tribromide relevant to AEGL-1 end points were available.

5.2. Animal Data Relevant to AEGL-1

No animal data on boron tribromide relevant to AEGL-1 end points were available.

5.3. Derivation of AEGL-1 Values

No human or animal data on boron tribromide were available to derive AEGL-1 values. On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide in moist air, the AEGL-1 values for boron tribromide were derived by dividing the hydrogen bromide AEGL-1 values by 3. See Chapter 8 of this report for how AEGL-1 values were derived for hydrogen bromide. The AEGL-1 values for boron tribromide are presented in Table 9-6, and the calculations are in Appendix A.

TABLE 9-5 Comparison of LC_{50} Values for Boron Trihalides and Acid Halides in Rats

Chemical	LC_{50} Value	Reference
Boron trifluoride	436 ppm (4 h)	Rusch et al. 1986
Hydrogen fluoride	500 ppm (4 h)[a]	Vernot et al. 1977
Boron trichloride	2,541 ppm (1 h)	Vernot et al. 1977
Hydrogen chloride	3,124 ppm (1 h)	Vernot et al. 1977
Boron tribromide	No data	—
Hydrogen bromide	2,858 ppm (1 h)	MacEwen and Vernot 1972

[a] Value was time scaled from 1 h to 4 h using the equation $C^2 \times t = k$ (NRC 2004).

TABLE 9-6 AEGL-1 Values for Boron Tribromide

10 min	30 min	1 h	4 h	8 h
0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)

6. DATA ANALYSIS FOR AEGL-2

6.1. Human Data Relevant to AEGL-2

No human data on boron tribromide relevant to AEGL-2 end points were available.

6.2. Animal Data Relevant to AEGL-2

No animal data on boron tribromide relevant to AEGL-2 end points were available.

6.3. Derivation of AEGL-2 Values

No human or animal data on boron tribromide were available to derive AEGL-2 values. On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide in moist air, the AEGL-2 values for boron tribromide were derived by dividing the hydrogen bromide AEGL-2 values by 3. See Chapter 8 of this report for how AEGL-2 values were derived for hydrogen bromide. The AEGL-2 values for boron tribromide are presented in Table 9-7.

7. DATA ANALYSIS FOR AEGL-3

7.1. Human Data Relevant to AEGL-3

No human data on boron tribromide relevant to AEGL-3 end points were available.

7.2. Animal Data Relevant to AEGL-3

No animal data on boron tribromide relevant to AEGL-3 end points were available.

7.3. Derivation of AEGL-3 Values

No human or animal data on boron tribromide were available to derive AEGL-3 values. On the basis that one mole of boron tribromide hydrolyzes to form three moles of hydrogen bromide in moist air, the AEGL-3 values for boron tribromide were derived by dividing the hydrogen bromide AEGL-3 values by three. See Chapter 8 of this report for how AEGL-3 values were derived for hydrogen bromide. AEGL-3 values for boron tribromide are presented in Table 9-8.

TABLE 9-7 AEGL-2 Values for Boron Tribromide

10 min	30 min	1 h	4 h	8 h
83 ppm (850 mg/m^3)	28 ppm (290 mg/m^3)	13 ppm (130 mg/m^3)	3.3 ppm (34 mg/m^3)	1.7 ppm (17 mg/m^3)

TABLE 9-8 AEGL-3 Values for Boron Tribromide

10 min	30 min	1 h	4 h	8 h
250 ppm (2600 mg/m^3)	83 ppm (850 mg/m^3)	40 ppm (410 mg/m^3)	10 ppm (100 mg/m^3)	5 ppm (51 mg/m^3)

The toxicity of boric acid liberated during hydrolysis of boron tribromide was considered. The intake of boric acid at the AEGL-3 values by infants, the most susceptible population, can be calculated. The 8-h AEGL-3 is 51 mg/m^3. The breathing rate of a child is 12 m^3/day. Boron tribromide is 4.32% boron. Assuming complete uptake of boron from the respiratory tract, the resulting uptake for a child is:

51 mg/m^3 × 12 m^3/24 h × 8 h × 0.0432 = 8.8 mg of boron potentially absorbed.

This value is low when compared with the 2-5 g of boron needed for lethality in a child.

8. SUMMARY OF AEGL VALUES

8.1. AEGL Values and Toxicity End Points

AEGL values for boron tribromide are presented in Table 9-9.

8.2. Comparison with Other Standards and Guidelines

Workplace guidelines exist for boron tribromide (see Table 9-10). The American Conference of Governmental Industrial Hygienists has established a TLV-ceiling value of 1 ppm for boron tribromide, which is based on analogy with hydrogen bromide (ACGIH 2012, 2001). ACGIH recommends ceiling values for primary irritants with no known chronic effects. The ceiling value is a concentration that should not be exceeded during any part of the working day. The National Institute for Occupational Safety and Health (NIOSH 2011) recommended exposure limit-ceiling and the Netherlands MAC value are also 1 ppm (MSZW 2004). These guidelines are higher than the AEGL-1 value of 0.33 ppm. The ACGIH TLV-ceiling for hydrogen bromide is 2 ppm (ACGIH 2012), and the ACGIH TLV-TWA for boric acid is 2 mg/m^3 as inhalable particulate mass (ACGIH 2012).

TABLE 9-9 AEGL Values for Boron Tribromide

	Exposure Duration				
Classification	10 min	30 min	1 h	4 h	8 h
AEGL-1 (nondisabling)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)	0.33 ppm (3.4 mg/m^3)

AEGL-2 (disabling)	83 ppm (850 mg/m^3)	28 ppm (290 mg/m^3)	13 ppm (130 mg/m^3)	3.3 ppm (34 mg/m^3)	1.7 ppm (17 mg/m^3)
AEGL-3 (lethal)	250 ppm (2600 mg/m^3)	83 ppm (850 mg/m^3)	40 ppm (410 mg/m^3)	10 ppm (100 mg/m^3)	5 ppm (51 mg/m^3)

TABLE 9-10 Standards and Guidelines for Boron Tribromide

	Exposure Duration				
Guideline	10 min	30 min	1 h	4 h	8 h
AEGL-1	0.33 ppm	0.33 ppm	0.33 ppm	0.33 ppm	0.33 ppm
AEGL-2	83 ppm	28 ppm	13 ppm	3.3 ppm	1.7 ppm
AEGL-3	250 ppm	83 ppm	40 ppm	10 ppm	5 ppm
TLV-C (ACGIH)[a]	1 ppm	1 ppm	1 ppm	1 ppm	1 ppm
REL-C (NIOSH)[b]	1 ppm	1 ppm	1 ppm	1 ppm	1 ppm
MAC (The Netherlands)[c]	–	–	–	–	10 mg/m^3 1 ppm

[a] TLV-C (threshold limit value – ceiling, American Conference of Governmental Industrial Hygienists) (ACGIH 2012) is a concentration that should not be exceeded during the working day.

[b] REL-C (recommended exposure limit – ceiling, National Institute for Occupational Safety and Health) (NIOSH 2011) is defined analogous to the ACGIH TLV-ceiling.

[c] MAC (maximaal aanvaarde concentratie [maximal accepted concentration], Dutch Expert Committee for Occupational Standards, The Netherlands) (MSZW 2004) is defined analogous to the ACGIH TLV-TWA.

8.3. Data Adequacy and Research Needs

The reactive nature of boron tribromide precludes toxicity testing. In the absence of empirical data on boron tribromide, and on the basis that one mole of boron tribromide theoretically hydrolyzes into three moles of hydrogen bromide, the AEGL values for boron tribromide were based on those for hydrogen bromide. The database for hydrogen bromide was combined with the more robust data base for the related chemical, hydrogen chloride.

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APPENDIX A

DERIVATION OF AEGL VALUES FOR BORON TRIBROMIDE

Derivation of AEGL-1 Values

Inadequate data were available on boron tribromide, so AEGL-1 values were based on the AEGL-1 values for hydrogen bromide.

Calculation: On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide, the hydrogen bromide AEGL-1 value was divided by 3. For all AEGL-1 durations: 1 ppm ÷ 3 = 0.33 ppm

Derivation of AEGL-2 Values

Inadequate data were available on boron tribromide, so AEGL-2 values were based on the AEGL-2 values for hydrogen bromide.

Calculation: On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide, the hydrogen bromide AEGL-2 values were divided by 3.

10-min AEGL-2: 250 ppm ÷ 3 = 83 ppm

30-min AEGL-2: 83 ppm ÷ 3 = 28 ppm

1-h AEGL-2: 40 ppm ÷ 3 = 13 ppm

4-h AEGL-2 10 ppm ÷ 3 = 3.3 ppm

8-h AEGL-2: 5 ppm ÷ 3 = 1.7 ppm

Derivation of AEGL-3 Values

Inadequate data were available on boron tribromide, so AEGL-3 values were based on the AEGL-3 values for hydrogen bromide.

Calculation: On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide, the hydrogen bromide AEGL-3 values were divided by 3.

10-min AEGL-3: 740 ppm ÷ 3 = 250 ppm

30-min AEGL-3: 250 ppm ÷ 3 = 83 ppm 1-h AEGL-3 120 ppm ÷ 3 = 40 ppm

4-h AEGL-3: 31 ppm ÷ 3 = 10 ppm

8-h AEGL-3: 15 ppm ÷ 3 = 5 ppm

APPENDIX B

ACUTE EXPOSURE GUIDELINE LEVELS FOR BORON TRIBROMIDE

Derivation Summary

AEGL-1 VALUES

10min	30 min	1 h	4 h	8 h
0.33 ppm	0.33 ppm	0.33 ppm	0.33 ppm	0.33 ppm

Data adequacy: Inadequate data were available on boron tribromide, so values were based on the AEGL-1 values for hydrogen bromide. On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide, the hydrogen bromide AEGL-1 values were divided by 3.

AEGL-2 VALUES

10 min	30 min	1 h	4 h	8 h
83 ppm	28 ppm	13 ppm	3.3 ppm	1.7 ppm

Data adequacy: Inadequate data were available on boron tribromide, so values were based on the AEGL-2 values for hydrogen bromide. On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide, the hydrogen bromide AEGL-2 values were divided by 3.

AEGL-3 VALUES

10 min	30 min	1 h	4 h	8 h
250 ppm	83 ppm	40 ppm	10 ppm	5 ppm

Data adequacy: Inadequate data were available on boron tribromide, so values were based on the AEGL-3 values for hydrogen bromide. On the basis that one mole of boron tribromide hydrolyzes into three moles of hydrogen bromide, the hydrogen bromide AEGL-3 values were divided by 3.