

BROMINE

ICSC: 0107

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CAS # 7726-95-6 Br₂
RTECS # EF9100000 Molecular
mass:
159.8

UN # 1744
EC Annex 1 035-001-
Index # 00-5



EC/EINECS 231-778-1

TYPES OF HAZARD / EXPOSURE	ACUTE HAZARDS / SYMPTOMS	PREVENTION	FIRST AID / FIRE FIGHTING
FIRE	Not combustible but enhances combustion of other substances. Many reactions may cause fire or explosion. Heating will cause rise in pressure with risk of bursting. Gives off irritating or toxic fumes (or gases) in a fire.	NO contact with incompatible materials; see Chemical Dangers.	In case of fire in the surroundings: use appropriate extinguishing media.
EXPLOSION	Risk of fire and explosion (See Chemical Dangers).	NO contact with incompatible materials; see Chemical Dangers.	In case of fire: keep cylinder cool by spraying with water.

EXPOSURE		AVOID ALL CONTACT!	IN ALL CASES CONSULT A DOCTOR!
Inhalation	Cough. Sore throat. Shortness of breath. Dizziness. Headache. Wheezing. Laboured breathing. Symptoms may be delayed (see Notes).	Breathing protection. Closed system and ventilation.	Fresh air, rest. Half-upright position. Artificial respiration may be needed. Refer immediately for medical attention.
Skin	Redness. Burning sensation. Pain. Serious skin burns.	Protective gloves. Protective clothing.	First rinse with plenty of water for at least 15 minutes, then remove contaminated clothes and rinse again. Put clothes in sealable container. Refer immediately for medical

			attention.
Eyes	Causes watering of the eyes. Redness. Blurred vision. Pain. Burns.	Safety goggles, face shield and eye protection in combination with breathing protection.	Rinse with plenty of water (remove contact lenses if easily possible). Refer immediately for medical attention.
Ingestion	Burns in mouth and throat. Burning sensation in the throat and chest. Abdominal pain. Shock or collapse.	Do not eat, drink, or smoke during work.	Rinse mouth. Do NOT induce vomiting. Refer immediately for medical attention.

SPILLAGE DISPOSAL	PACKAGING & LABELLING
Evacuate danger area! Consult an expert! Personal protection: gas-tight chemical protection suit including self-contained breathing apparatus. Do NOT let this chemical enter the environment. Ventilation. Remove vapour with fine water spray. Collect leaking liquid in sealable containers with fluorinated coating. Do NOT absorb in saw-dust or other combustible absorbents. Absorb remaining liquid in dry sand or inert absorbent and remove to safe place.	Special material. Unbreakable packaging; put breakable packaging into closed unbreakable container. Do not transport with food and feedstuffs. EU Classification Symbol: T+, C, N R: 26-35-50 S: (1/2-)7/9-26-45-61 UN Classification UN Hazard Class: 8 UN Subsidiary Risks: 6.1 UN Pack Group: I GHS Classification Danger May be corrosive to metals Fatal if inhaled May be harmful if swallowed Causes severe skin burns and eye damage May cause respiratory irritation Very toxic to aquatic life
EMERGENCY RESPONSE	STORAGE
Transport Emergency Card: TEC (R)-80S1744 NFPA Code: H 3; F 0; R 0; OX	Provision to contain effluent from fire extinguishing. Separated from food and feedstuffs, see Chemical Dangers. Cool. Dry. Well closed. Keep in a well-ventilated room. Store only in original container. Store in an area without drain or sewer access.

IPCS

International Programme on Chemical Safety



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BROMINE

ICSC: 0107

IMPORTANT DATA

PHYSICAL STATE; APPEARANCE:
FUMING RED TO BROWN LIQUID, WITH PUNGENT ODOUR.

ROUTES OF EXPOSURE:
Serious local effects by all routes of exposure.

<p>PHYSICAL DANGERS: The vapour is heavier than air.</p> <p>CHEMICAL DANGERS: Upon heating, toxic fumes are formed. The substance is a strong oxidant and reacts violently with combustible and reducing materials. The substance reacts with most organic and inorganic compounds, causing fire and explosion hazard. Attacks metal, some forms of plastic, rubber and coatings.</p> <p>OCCUPATIONAL EXPOSURE LIMITS: TLV: 0.1 ppm as TWA; 0.2 ppm as STEL; (ACGIH 2008). EU OEL: 0.1 ppm, 0.7 mg/m³ as TWA; (EU 2006).</p>	<p>INHALATION RISK: A harmful contamination of the air will be reached very quickly on evaporation of this substance at 20°C.</p> <p>EFFECTS OF SHORT-TERM EXPOSURE: Lachrymation. The substance is corrosive to the eyes, the skin and the respiratory tract. Corrosive on ingestion. Inhalation may cause asthma-like reactions. Inhalation may cause pneumonitis. Inhalation may cause lung oedema, but only after initial corrosive effects on eyes and/or airways have become manifest. The effects may be delayed. Medical observation is indicated. Exposure may result in death.</p> <p>EFFECTS OF LONG-TERM OR REPEATED EXPOSURE: The substance may have effects on the respiratory tract and lungs, resulting in chronic inflammations and impaired functions.</p>
<p>PHYSICAL PROPERTIES</p>	
<p>Boiling point: 58.8°C Melting point: -7.2°C Relative density (water = 1): 3.1 Solubility in water, g/100 ml at 20°C: 4.0 Vapour pressure, kPa at 20°C: 23.3 Relative vapour density (air = 1): 5.5</p>	<p>Relative density of the vapour/air-mixture at 20°C (air = 1): 2.0 Viscosity, mm²/s at 40 °C: 0.264</p>
<p>ENVIRONMENTAL DATA</p>	
<p>The substance is very toxic to aquatic organisms. It is strongly advised that this substance does not enter the environment.</p>	
<p>NOTES</p>	
<p>The symptoms of lung oedema often do not become manifest until a few hours have passed and they are aggravated by physical effort. Rest and medical observation are therefore essential. Immediate administration of an appropriate inhalation therapy by a doctor or a person authorized by him/her, should be considered.</p>	
<p>ADDITIONAL INFORMATION</p>	
<p>LEGAL NOTICE</p>	<p>Neither the CEC nor the IPCS nor any person acting on behalf of the CEC or the IPCS is responsible for the use which might be made of this information</p>
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See Also:

[Toxicological Abbreviations](#)
[Bromine \(PIM 080\)](#)

BROMINE

International Programme on Chemical Safety
Poisons Information Monograph 080
Chemical

1. NAME

1.1 Substance

Bromine

1.2 Group

Halogen

1.3 Synonyms

Brom (German);
Brome (French);
Bromo (Italian);
Broom (Dutch).

1.4 Identification numbers

1.4.1 CAS number

7726-95-6

1.4.2 Other numbers

DOT: 1744 59 (designated corrosive material)
IMIS: 0390
NIOSH RTECS: EF 9100000
UN: 1744

1.5 Main brand names, main trade names

1.6 Main manufacturers, main importers

United States Manufacturers (Weiss, 1980):

Arkansas Chemicals Inc., El Dorado, Arkansas 71730
Bromet Company, Magnolia, Arkansas 71753
Dow Chemical Company, Midland, Michigan 48640

2. SUMMARY

2.1 Main risks and target organs

Inhalation of the irritant bromine vapours and/or direct contact (liquid or vapour) with skin and mucous membranes will produce direct tissue injury. Injury may occur at various levels of the respiratory tract depending on the

concentration of bromine and duration of exposure. Target organs include the upper and lower respiratory tract, skin, and eyes.

In theory, although never demonstrated in the literature, the potential exists for bromine to accumulate in body tissues as bromide after an inhalation or direct skin contact. If accumulated to sufficiently high concentrations, bromide would subsequently produce neurotoxic and acne-like effects as seen with the ingestion of bromide compounds or the chronic inhalation of methyl bromide. However, due the

extreme irritant nature of bromine, the duration of exposure is generally severely limited, reducing any likely body burden of bromide.

2.2 Summary of clinical effects

Acute

Dermal: Pure bromine (liquid or vapour) is extremely irritating to the skin. Unlike most other chemical agents, there is no immediate visible skin reaction after contact. The delay before initial signs of injury become apparent often results in more extensive damage. The most common local effects are blister formation, brownish discoloration of the skin and slow-healing ulcers (Sagi et al., 1985).

Mucous Membranes: Exposure to low concentrations produces lacrimation, rhinorrhoea, eye irritation with mucous secretions from the oropharyngeal and upper airways, coughing, dyspnoea, choking, wheezing, epistaxis, and headache. A brownish discoloration of the tongue and buccal mucosa may occur and be accompanied by a characteristic breath odour. Inflammatory lesions of the upper airway, photophobia and blepharospasm are seen with higher concentrations (Parmeggiani, 1983). **Upper and lower respiratory tract:** Initial irritant symptoms of bromine vapour inhalation include: dyspnoea, coughing, choking, and wheezing. In addition, immediate or delayed bronchoconstriction and the development of laryngeal spasm, glottal oedema, asthma and cheobronchitis. With increased parenchymal penetration, there may be associated peribronchiolar abscesses, pulmonary infiltrates consistent with chemical pneumonitis, bronchiolitis obliterans and pulmonary oedema (Edelman, 1991; Rom & Barkman, 1983). Acute obstructive ventilatory impairment may lead to severe hypoxaemia, metabolic acidosis, measles-like rash and subsequent death. It should be noted that more severe respiratory symptoms may be delayed for several hours after the exposure (Lossos et al., 1990; Kraut & Lilis, 1988).

Central nervous system: The bromide ion is a central nervous system depressant producing ataxia, slurred speech, tremor, nausea, vomiting, lethargy, dizziness, visual disturbances, unsteadiness, headaches, impaired memory and concentration, disorientation and hallucinations. This has only been documented in the literature with reference to overdoses of bromide-containing medications and the inhalation of bromide-containing fumigants. Although theoretically possible, this has not been reported in the medical literature from acute (or chronic) exposure to bromine.

Respiratory: There are few reports about the chronic complications of an acute exposure to bromine. However, the literature has described the chronic manifestations of chlorine inhalation. Bromine is potentially capable of extensive damage to the lower respiratory tract. Limited studies have reported diffuse interstitial pulmonary fibrosis, emphysema and/or airway hyperreactivity secondary to acute exposure to bromine (Lossos et al., 1990; Kraut & Lilis, 1988).

Dermal: There is a rare cutaneous manifestation of bromide accumulation known as bromoderma tuberosum, which progresses from red papules to pustules that enlarge and develop into indurated lesions with a central ulcer (Sticht & Kaferstein, 1988). This effect is related to the ingestion of bromides formerly used in medications and, in theory, to chronic inhalation of low-level concentrations of bromine (Sax, 1984).

Other: Most available data come from studies involving chronic exposure to bromides in the form of oral medications. This type of exposure has led to depression, ataxia and psychoses (bromism). Chronic exposure to methyl bromide has caused peripheral neuropathy. Soviet literature has described loss of corneal reflexes, joint pain, vegetative disorders, thyroid dysfunction and depression of bone marrow (Sticht & Kaferstein, 1988). However, there is no published evidence that these chronic occur effects with the inhalation and/or dermal absorption of bromine liquid or vapour.

2.3 Diagnosis

Signs characteristic of acute bromine poisoning include:

- * after exposure of skin, delayed onset of burns
- * brownish discolouration of tongue and buccal mucosa
- * characteristic odour on the breath

Bromine gas blood concentrations are not clinically useful. Bromine is generally unmeasurable since it changes directly to hydrobromic and bromic acid. No specific laboratory

studies are needed unless otherwise indicated by the severity of symptoms (see Section 2.4.)

2.4 First-aid measures and management principles

Acute contact with bromine liquid or vapour requires removal from the source of the bromine contamination.

Eye: The eye(s) should be irrigated with copious amounts of water for at least 15 minutes. If irritation, pain, swelling, lacrimation or photophobia persist, further medical evaluation is recommended.

Dermal: Remove contaminated clothing and wash affected area thoroughly with copious volumes of water for 20 minutes. Since effects may be delayed, close observation for blistering and discoloration of the skin is required for the next 24 hours (Sagi et al., 1985).

Inhalation: Respiratory support in accordance with symptomatology, including: maintenance of an adequate airway, oxygen, antibronchospasm therapy (beta adrenergic agonist, aerosols, aminophylline and/or short course of corticosteroids) and antibiotics if there is evidence of infection. Initial testing should include: chest x-ray to view inflammatory changes in the parenchyma, spirometry (flow-volume loop with and without bronchodilator) to determine air flow capacity and reversibility, diffusing

capacity (DLco) to assess changes in the alveolar-capillary permeability, arterial blood gases to evaluate blood oxygenation (Po₂) and ventilation (Pco₂), and complete white blood count. Repeat chest radiograph and spirometry are recommended to determine the progression or resolution of residual effects. Ventilation-perfusion scanning does not usually provide significant additional information, except to rule out other processes, such as pulmonary emboli. Careful examination is important to detect pathology (fine wheezes in subtle or early asthma and fine crackles in early pulmonary oedema) not revealed by the studies noted above.

Ingestion: Not relevant

3. PHYSICO-CHEMICAL PROPERTIES

3.1 Origin of the substance

Bromine occurs naturally in the earth's crust as a non-metallic element. Like other halogens, it is very reactive and principally found in the form of inorganic bromides (Na, K, NH₄, Ca and Mg) and as a component secondary to chlorine in minerals and biological systems (humans and animals). A pure bromine-containing mineral, bromite (AgBr)

is found in Mexico (Sticht & Kaferstein, 1988). Bromine is derived from sea water and naturally occurring brines.

Most sources outside of the United States (United Kingdom, France, Japan Israel and Soviet Union) recover bromine from salt lakes and sea water (Stokinger, 1981). The bromine concentration in sea water averages about 0.0065%, while the Dead Sea contains an average concentration of 1.5% (Sticht & Kaferstein, 1988).

Before the bromine can be steamed out of the sea water, the bromine is concentrated by vaporization and the addition of sulphur dioxide. Water is then added to the resultant hydrogen bromide to produce a concentrated bromide solution. The remainder of the process is similar to bromine recovery from brines. The by-products of hydrochloric and sulfuric acid are recycled to the incoming water to reduce the pH sufficiently to promote efficient chlorination.

Most production in the United States is limited to naturally occurring brines in Michigan and Arkansas and accounts for about two-thirds of the world's annual production (Sticht & Kaferstein, 1988). The oil-field brines in Arkansas are more concentrated (5000 ppm) than in Michigan and represent the principal area of bromine production in the United States (Leddy, 1983). The brine (bromine-containing carnallite) is oxidized with elemental chlorine to liberate the bromine which is then condensed, distilled and dried. The bromine is stored and shipped in glass bottles with lead caps and lead-lined metal barrels, drums and tanks. Steam and chlorine are emitted from steaming-out towers and the spent brine is neutralized and pumped back into the subterranean strata (Leddy, 1983). In some cases, the debrominated brine is processed further to produce calcium and magnesium brine chemicals (Meyer, 1977).

3.2 Chemical structure

Bromine (Br): Atomic Weight 79.904; Atomic Number 35; Valences 1 to 7; Elemental State Br₂, molecular weight 159.82; two stable isotopes ⁷⁹Br and ⁸¹Br (Budivari, 1996).

3.3 Physical properties

3.3.1 Colour

Dark reddish brown.

3.3.2 State/form

3.3.3 Description

Boiling Point:	-59.47°C
Melting Point:	-7.25°C
Flash Point:	-7.3°C
Autoignition Temperature:	Not found
Specific Gravity:	Liquid = 3.12 at 20°C Vapour = 5.5 at 20°C
Vapour Pressure:	175 mmHg at 20°C, 3.385 psia (0.2303 atm)
Solubility (g/100 mL at 20°C):	Water 3.55g; Freely soluble in alcohol, ether, CHCl ₃ , CCl ₄ and CS ₂ .
Explosive Limits:	Not combustible, but may cause fire on contact with combustibles
pH:	Not found
Viscosity:	Not found

(Sax, 1984; Sticht & Kaferstein, 1988; OSHA, 1989; Budivari, 1996; Meyer, 1977; Weiss, 1980)

3.4 Hazardous characteristics

Bromine is a dark reddish-brown, volatile, diatomic liquid with a suffocating odour at room temperature. It is the only liquid non-metallic element. Because the vapour pressure is so high, the dark red vapours are immediately detectable when a container is opened.

The corrosive property of bromine is considered a major hazard by the United States Department of Transportation. Bromine is capable of dissolving metals and non-metals and spontaneously combines with aluminum, titanium copper, phosphorus, arsenic, gold and antimony. It will not corrode platinum, lead or nickel. The corrosive reaction results in a non-hazardous bromide. (Sittig, 1985; Budivari, 1996; Meyer, 1977)

As an oxidizer, bromine will react with inorganic matter, such as wood or sawdust; tremendous heat is produced increasing the risk of combustion following bromine spills. Bromine spills should be neutralized with a 5 to 10% solution of sodium thiosulphate. Sawdust should never be used to

absorb bromine. Explosions are also possible if ammonium hydroxide is used in an attempt to neutralize a spill (Meyer, 1977).

Bromine is slightly soluble in water, producing hydrogen bromide. Hydrogen bromide is a corrosive colourless gas with a pungent odor that is extremely soluble in water. In the presence of sunlight and humid air or hot water, it forms

hydrobromic acid with concentrations up to about 60% (Sticht & Kaferstein, 1988). Although less toxic than bromine, it has all the irritant qualities of bromine.

Bromine should be stored in its original container, separated from combustible, organic or other readily oxidizable materials and protected against physical damage and sunlight. Bromine should be kept above 20°F (- 6.6°C) to prevent freezing but heating above atmospheric temperatures should be avoided as raised vapour pressure could rupture the container (OSHA, 1989).

When handling bromine in significant quantities, full body protection (constructed of resistant material) should be worn.

Bromine will readily dissolve in alcohol, ether and other organic solvents.

When elemental chlorine is used in the treatment of waste water, it releases free bromine and bromine chloride from the bromide solution. Acute toxicity of bromine and chlorine have been studied in fish; however, no US drinking water limits have been set for bromine (Calabrese & Kenyon, 1991). Although bromine has been used as a water disinfectant for swimming pools, it is not recommended for this purpose in drinking water due to its cumulative neurotoxicity and the lack of sufficient research (NAS, 1977).

4. USES

4.1 Uses

4.1.1 Uses

4.1.2 Description

Pure bromine is used in the synthesis of a variety of bromine-containing substances. In the early 1970s, about 75% of the production of bromine went into the making of ethylene dibromide (EDB) as an antiknock agent in leaded gasoline. With the United States' mandate to reduce vehicle exhaust emissions, EDB use significantly declined in the 1980s. Export of EDB has maintained production in the US at about 40% (Leddy, 1983).

Fumigant production in the form of methyl bromide and ethylene dibromide account for about 10% of the total bromine marketed, while high density bromine fluids account for about 25%. The high density bromine fluids (calcium bromide and zinc bromide) are used

around the world as completion fluids in oil wells

(Leddy, 1983). The remaining market for bromine is in the manufacture of various organic compounds including tetrabromobisphenol A, decarbromodiphenyl oxide, hexabromocyclododecane and pentabromochlorocyclohexane which are blended with various polymeric materials to modify the finished products.

Other uses for bromine include flame retardants, cleaning agents, dyestuffs, photography, water sanitation, pharmaceuticals, bleaching fibers and silk, and chemical warfare gas (Sticht & Kaferstein, 1988; Sittig, 1985).

4.2 High risk circumstance of poisoning

Because of bromine's high reactivity with other elements, the forms of inorganic bromides found in the environment pose no danger of poisoning. Primary risk of poisoning is through industrial sources during bromine recovery and/or when bromine is applied to the synthesis of bromine-containing substances noted above. It is estimated that 20,000 workers in the US have the potential for occupational exposure to bromine (Broderick & Schwartz, 1992)

4.3 Occupationally exposed populations

Professions that would be potentially at risk of exposure include:

Drug Makers
Dye Makers
Gold Extractors
Gasoline Additive Makers
Organic Chemical Synthesizers
Petroleum Refinery Workers
Photographic Chemical Makers
Silk and Fibre Bleachers
(NIOSH, 1977)

5. ROUTES OF ENTRY

5.1 Oral

Unknown

5.2 Inhalation

During an accidental spill or leak during transportation or manufacturing.

5.3 Dermal

During an accidental spill or leak during transportation or manufacturing and/or improper handling or use of protective equipment.

5.4 Eye

During an accidental spill or leak during transportation or manufacturing and/or improper handling or use of protective equipment.

5.5 Parenteral

Unknown.

5.6 Others

No data available.

6. KINETICS

6.1 Absorption by route of exposure

The reactivity of bromine in biological systems makes it difficult to study the pharmacokinetics and to separate the effects of the bromine from those of the bromine compounds and metabolites.

Inhalation

Absorption of bromine vapours by other routes is usually minimal compared with the dose delivered by inhalation. The physical characteristics of the bromine are extremely important in determining the site and depth of lung penetration and systemic absorption, as well as the local effects of the exposure. The appearance or odour of bromine are usually adequate to alert the person to the presence of the material and afford time for escape.

The toxic effects of the bromine vapour on the respiratory tract are primarily due to its water solubility. Bromine is slightly more water soluble than chlorine and will produce immediate irritation of the upper airways (Broderick & Schwartz, 1992). However, when the person is caught in a confined space or in an overwhelming vapour cloud, inhalation may cause irritation to the lower airways.

6.2 Distribution by route of exposure

No data available.

6.3 Biological half-life by route of exposure

The biological half-life for bromide through ingestion is 12 to 30 days; however, there are no data available on the inhalation of bromine (Sticht & Kaferstein, 1988).

6.4 Metabolism

Due to its reactivity, bromine does not persist as an element in living tissue but quickly forms bromide. In this form, it may be deposited in the tissues, displacing other halogens (Sticht & Kaferstein, 1988). However, there are no data available regarding the metabolism of inhaled bromine.

6.5 Elimination by route of exposure

No data available.

7. TOXICOLOGY

7.1 Mode of Action

The injurious effects of bromine are generally felt to be similar to those of chlorine (Rom & Barkman, 1983; Broderick & Schwartz, 1992; Schwartz, 1987). Due to its potent oxidising action, bromine liberates nascent oxygen or oxygen free radicals from the water present in mucous membranes. Nascent oxygen is a potent oxidizer, capable of producing tissue damage. The extent of the damage is dependent on the dose of bromine and the availability of water to react with it. In addition, the formation of hydrobromic and bromic acids will result in secondary irritation during the reaction. Contact with the respiratory epithelium produces initial alveolar capillary congestion followed by focal and confluent patches of high-fibrinogen oedematous fluid. The fluid is interstitial at first but can fill the alveoli. Once this occurs, copious frothy, blood-tinged sputum is seen. A granulocyte response may occur several hours after inhalation. Hyaline membrane formation can occur later resulting in clinical deterioration at a time when signs of improvement have occurred. Poor oxygen diffusion, hypoxia and hypercapnia result from development of atelectasis, emphysema and membrane formation. Acute obstructive ventilatory impairment leads to severe hypoxaemia, metabolic acidosis and death usually due to cardiac arrest secondary to the hypoxaemia.

7.2 Toxicity

7.2.1 Human data

7.2.1.1 Adults

Exposure Effects Levels (ppm)

0.05 to 3.5	Odour Threshold
0.1	TLV-TWA Limit
>1.0	Irritation Level
40 to 60	Toxic pneumonitis & pulmonary oedema
1000	Fatal within a few minutes

(Calabrese & Kenyon, 1991; Fazzalari, 1978)

7.2.1.2 Children

No data available.

7.2.2 Relevant animal data

Post mortem on guinea pigs and rabbits exposed to bromine at 300 ppm for three hours revealed the presence of pulmonary oedema; pseudomembranous deposit on the trachea; and bronchi and haemorrhage of the gastric mucosa (Stokinger, 1981).

The mortality of mice exposed to 240 or 750 ppm bromine was dependent on the duration of exposure (Bitron & Aharonson, 1978).

7.2.3 Relevant in vitro data

Not relevant.

7.2.4 Workplace standards

TLV-TWA: 0.1 ppm (approximately 0.66 mg/m³) adopted in 1986 (ACGIH, 1991).

TLV-STEL: 0.3 ppm (approximately 2.0 mg/m³) adopted in 1986 (ACGIH, 1991).

OSHA PEL: TWA 0.1ppm (approximately 0.7 mg/m³); STEL 0.3 ppm (approximately 2.0 mg/m³) (ACGIH, 1991)

NIOSH REL: TWA 0.1 ppm (approximately 0.7 mg/m³); STEL 0.3 ppm (approximately 2.0mg/m³) (ACGIH, 1991)

Country/ Organization	TWA		STEL	
	ppm	mg/m ³	ppm	mg/m ³
Australia	0.1	0.7	0.3	2
Belgium	0.1	0.66	0.3	2
Denmark	0.1	0.7	-	-
Finland	-	-	0.1	0.7
France	-	-	0.1	0.7
Germany	0.1	0.7	"I"	"I"
Hungary	-	-	-	0.7
Japan	0.1	0.65	-	-
Poland	-	0.7	-	-
Sweden	0.1	0.7	0.3	2
Switzerland	0.1	0.7	0.2	1.4
United Kingdom	0.1	0.7	0.3	2
US: ACGIH	0.1	0.66	0.3	2
US: NIOSH/OSHA	-	0.7	-	2
USSR	0.1	-	-	0.5

(ILO, 1991)

"I" = Local Irritant

7.2.5 Acceptable daily intake (ADI) and other guideline levels

Acceptable Daily Intake (ADI)

Oral: 1 mg bromide/kg body weight (Sticht & Kaferstein, 1988).

7.3 Carcinogenicity

No data have been found implicating bromine as a carcinogen (Calabrese & Kenyon, 1991; Alderson, 1986).

7.4 Teratogenicity

No data have been found implicating bromine as a teratogen (Calabrese & Kenyon, 1991).

7.5 Mutagenicity

No data have been found implicating bromine as a mutagen (Calabrese & Kenyon, 1991).

7.6 Interactions

No data available.

8. TOXICOLOGICAL ANALYSES AND BIOMEDICAL INVESTIGATIONS

8.1 Material sampling plan

8.1.1 Sampling and specimen collection

8.1.1.1 Toxicological analyses

8.1.1.2 Biomedical analyses

8.1.1.3 Arterial blood gas analysis

8.1.1.4 Haematological analyses

8.1.1.5 Other (unspecified) analyses

8.1.2 Storage of laboratory samples and specimens

8.1.2.1 Toxicological analyses

8.1.2.2 Biomedical analyses

8.1.2.3 Arterial blood gas analysis

8.1.2.4 Haematological analyses

8.1.2.5 Other (unspecified) analyses

8.1.3 Transport of laboratory samples and specimens

8.1.3.1 Toxicological analyses

8.1.3.2 Biomedical analyses

8.1.3.3 Arterial blood gas analysis

8.1.3.4 Haematological analyses

8.1.3.5 Other (unspecified) analyses

8.2 Toxicological analyses and their interpretation

8.2.1 Tests on toxic ingredient(s) of material

8.2.1.1 Simple qualitative test(s)

- 8.2.1.2 Advanced qualitative confirmation test(s)
- 8.2.1.3 Simple quantitative method(s)
- 8.2.1.4 Advanced quantitative method(s)
- 8.2.2 Tests for biological specimens
 - 8.2.2.1 Simple qualitative test(s)
 - 8.2.2.2 Advanced qualitative confirmation test(s)
 - 8.2.2.3 Simple quantitative method(s)
 - 8.2.2.4 Advanced quantitative method(s)
 - 8.2.2.5 Other dedicated method(s)
- 8.2.3 Interpretation of toxicological analyses
- 8.3 Biomedical investigations and their interpretation
 - 8.3.1 Biochemical analysis
 - 8.3.1.1 Blood, plasma or serum
 - 8.3.1.2 Urine
 - 8.3.1.3 Other fluids
 - 8.3.2 Arterial blood gas analyses
 - 8.3.3 Haematological analyses
 - 8.3.4 Interpretation of biomedical investigations
- 8.4 Other biomedical (diagnostic) investigations and their interpretation
- 8.5 Overall interpretation of all toxicological analyses and toxicological investigations

Biomedical Analysis

Initial testing should include: chest x-ray to view inflammatory changes in the parenchyma, spirometry (flow-volume loop with and without bronchodilator) to determine air flow capacity and reversibility, diffusing capacity (DLco) to assess changes in the alveolar-capillary permeability, arterial blood gases to evaluate blood oxygenation (Po₂) and ventilation (Pco₂) and complete white blood cell count. Repeat chest radiographs and spirometry are recommended to determine the progression and resolution of residual effects. Careful examination is important to detect pathology (fine wheezes in subtle or early asthma and fine crackles in early pulmonary oedema) not evident from the studies noted above.

9. CLINICAL EFFECTS

9.1 Acute poisoning

9.1.1 Ingestion

Not relevant.

9.1.2 Inhalation

Upper and Lower Respiratory Tract: Initial symptoms due to irritation by bromine vapour inhalation include: dyspnoea, coughing, choking, and wheezing; immediate or delayed bronchoconstriction; and the development of laryngeal spasm, glottal oedema, asthma and tracheobronchitis. With increased parenchymal penetration, there may be associated peribronchiolar abscesses, pulmonary infiltrates consistent with chemical pneumonitis, bronchiolitis obliterans and pulmonary oedema (Edelman, 1991; Rom & Barkman, 1983). Acute obstructive ventilatory impairment may lead to severe hypoxaemia, metabolic acidosis, measles-like rash and subsequent death. It should be noted that more severe respiratory symptoms may be delayed for several hours after the exposure (Lossos et al., 1990; Kraut & Lillis, 1988).

9.1.3 Skin exposure

Pure bromine (liquid or vapour) is extremely irritating to the skin. Unlike most other chemical agents, there is no immediate visible skin reaction with contact. Lack of action due to the delay in initial signs of injury often results in more extensive damage. The most common local effects are blister formation, brownish discoloration of the skin and slowly healing ulcers (Sagi et al., 1985).

9.1.4 Eye contact

Exposure to low concentrations produce irritation of the eye with lacrimation. Photophobia and blepharospasm are seen with higher concentrations (Parmeggiani, 1983).

9.1.5 Parenteral exposure

Not relevant.

9.1.6 Other

Not relevant.

9.2 Chronic poisoning

9.2.1 Ingestion

Not relevant.

9.2.2 Inhalation

There are few reports on the chronic complications of acute exposure to bromine. However, the literature has described the chronic

manifestations of chlorine inhalation. Bromine is potentially capable of extensive damage to the lower respiratory tract. Limited studies have reported diffuse interstitial pulmonary fibrosis, emphysema and/or airway hyperreactivity secondary to acute exposure to bromine (Lossos et al., 1990; Kraut & Lilis, 1988).

9.2.3 Skin exposure

There is a rare cutaneous manifestation of bromide accumulation known as bromoderma tuberosum, which progresses from red papules to pustules that enlarge and develop into indurated lesions with a central ulcer (Sticht & Kaferstein, 1988). This effect is related to the ingestion of bromides formerly used in medications and may, in theory, occur after chronic inhalation of low concentrations of bromine (Sax, 1984).

9.2.4 Eye contact

No data available.

9.2.5 Parenteral exposure

No data available.

9.2.6 Other

No data available.

9.3 Course, prognosis, cause of death

The course and prognosis of a bromine exposure is depends on the concentration and duration of the exposure. Effects may vary from mild irritation of mucous membranes to severe damage of the skin and lung parenchyma. Death is secondary to severe hypoxaemia/metabolic acidosis due to acute obstructive ventilatory impairment.

9.4 Systematic description of clinical effects

9.4.1 Cardiovascular

In an acute bromine exposure, significant hypoxaemia and hypercapnia secondary to marked respiratory obstruction and/or restriction are responsible for the cardiovascular changes. Initial sinus tachycardia and cardiac arrhythmias may progress to cardiac arrest with progressive pulmonary obstruction.

9.4.2 Respiratory

Initial irritant symptoms of bromine vapour inhalation include: dyspnoea, coughing, choking, and wheezing, in addition, immediate or delayed bronchoconstriction and the development of laryngeal spasm, glottal oedema, asthma and tracheobronchitis. With increased parenchyma penetration, there may be

associated peribronchiolar abscesses, pulmonary infiltrates consistent with chemical pneumonitis, bronchiolitis obliterans and pulmonary oedema (Edelman, 1991; Rom & Barkman, 1983). Acute obstructive ventilatory impairment may lead to severe hypoxaemia, metabolic acidosis, measles-like rash and subsequent death. It should be noted that the onset of severe respiratory symptoms may be delayed for several hours after the exposure (Lossos et al., 1990; Kraut & Lilis, 1988).

There are few reports on the chronic complication of acute exposure to bromine. However, the literature has extensively described the chronic manifestations of chlorine inhalation. Bromine is potentially capable of extensive damage to the lower respiratory tract. Limited studies have reported diffuse interstitial pulmonary fibrosis, emphysema and/or airway hyperreactivity secondary to acute exposure to bromine (Lossos et al., 1990; Kraut & Lilis, 1988).

9.4.3 Neurological

9.4.3.1 Central Nervous System (CNS)

The bromide ion is a central nervous system depressant producing ataxia, slurred speech, tremor, nausea, vomiting, lethargy, dizziness, visual disturbances, unsteadiness, headaches, impaired memory and concentration, disorientation and hallucinations. This has only been documented in the literature with

reference to overdoses of bromide-containing medications and the inhalation of bromide-containing fumigants.

9.4.3.2 Peripheral nervous system

Chronic exposure to methyl bromide has caused peripheral neuropathy; however, there are no data on pure bromine exposure.

9.4.3.3 Autonomic nervous system

No data available.

9.4.3.4 Skeletal and smooth muscle

No data available.

9.4.4 Gastrointestinal

Not relevant.

9.4.5 Hepatic

No data available.

9.4.6 Urinary

9.4.6.1 Renal

No data available.

9.4.6.2 Others

No data available

9.4.7 Endocrine and reproductive systems

No data available.

9.4.8 Dermatological

Pure bromine (liquid or vapour) is extremely irritating to the skin. Unlike most other chemical agents, there are no immediate visible skin reaction with contact. Lack of action due to the delay in initial signs of injury often results in more extensive damage. The most common local effects are blister formation, brownish discoloration of the skin and slowly healing ulcers (Sagi et al., 1985).

There is a rare cutaneous manifestation of bromide accumulation known as bromoderma tuberosum, which progresses from red papules to pustules that enlarge and develop into indurated lesions with a central ulcer (Sticht & Kaferstein, 1988). This effect is related to the ingestion of bromides formerly used in medications and, in theory, to chronic inhalation of low level concentrations of bromine (Sax, 1984).

9.4.9 Eye, ears, nose, throat: local effects

Exposure to low concentrations produce irritation of the eye with lacrimation, rhinorrhoea, choking and burning sensation of the throat. With higher concentrations, there may be photophobia and blepharospasm, epistaxis, hoarseness, stridor and laryngeal oedema.

9.4.10 Haematological

No data available.

9.4.11 Immunological

No data available.

9.4.12 Metabolic

9.4.12.1 Acid-base disturbances

With severe exposure to bromine, metabolic acidosis may follow acute respiratory acidosis secondary to acute pulmonary obstruction and pulmonary oedema. With poor pulmonary function, inadequate

ventilation leads to excess carbonic acid.

9.4.12.2 Fluid and electrolyte disturbances

No data available.

9.4.12.3 Others

No data available

9.4.13 Allergic reactions

No data available.

9.4.14 Other clinical effects

No data available.

9.4.15 Special risks

There is one report of neonatal bromism (neurological depression and hypotonia) secondary to maternal exposure to bromides at a photographic laboratory. However, no data are available on pure bromine exposure.

9.5 Others

No data available.

9.6 Summary

10. MANAGEMENT

10.1 General principles

Acute contact with bromine liquid or vapour requires removal from the source of the bromine contamination.

Eye: The eye(s) should be irrigated with copious amounts of tepid water for at least 15 minutes. If irritation, pain, swelling, lacrimation or photophobia persist, further medical evaluation is recommended.

Dermal: Remove of contaminated clothing and thoroughly wash the affected area with copious volumes of water for 20 minutes. Since the effects may be delayed, close observation for blistering and discoloration of the skin is required for the next 24 hours (Sagi et al., 1985).

Inhalation: Respiratory support in accordance with symptomatology, including: maintenance of an adequate airway, oxygen, antibronchospasm therapy (inhaled beta adrenergic agonist, aminophylline and/or short course of corticosteroids) and antibiotics if there is evidence of infection. Assisted or supported ventilation with tracheal intubation and positive pressure ventilation may be needed.

10.2 Life supportive procedures and symptomatic treatment

Acute contact with bromine liquid or vapour requires removal from the source of the bromine contamination.

Eye: The eye(s) should be irrigated with copious amounts of water for at least 15 minutes. If irritation, pain,

swelling, lacrimation or photophobia persist, further medical evaluation is recommended.

Dermal: Remove contaminated clothing and wash affected area thoroughly with copious volumes of water for 20 minutes. Since effects may be delayed, close observation for blistering and discoloration of the skin is required for the next 24 hours (Sagi et al., 1985).

Inhalation: Respiratory support in accordance with symptomatology, including: maintenance of an adequate airway, oxygen, antibrachospasm therapy (beta adrenergic agonist, aerosols, aminophylline and/or short course of corticosteroids) and antibiotics if there is evidence of infection. Initial testing should include: chest x-ray to view inflammatory changes in the parenchyma, spirometry (flow-volume loop with and without bronchodilator) to determine air flow capacity and reversibility, diffusing capacity (DLco) to assess changes in the alveolar-capillary permeability, arterial blood gases to evaluate blood oxygenation (Po₂) and ventilation (Pco₂), and complete white blood count. Repeat chest radiograph and spirometry are recommended to determine the progression or resolution of residual effects. Ventilation-perfusion scanning does not usually provide significant additional information, except to rule out other processes, such as pulmonary emboli. Careful examination is important to detect pathology (fine wheezes in subtle or early asthma and fine crackles in early pulmonary oedema) not revealed by the studies noted above.

Ingestion: Not relevant

10.3 Decontamination

Acute contact with bromine liquid or vapour requires removal from the source of the bromine contamination.

Eye: The eye(s) should be irrigated with copious amounts of tepid water for at least 15 minutes.

Dermal: Removal of contaminated clothing and thorough washing of affected area with copious volumes of water for 20 minutes.

Inhalation: Maintain adequate fresh air source.

10.4 Enhanced elimination

Contaminated clothing should be discarded. When bromide-containing medications are ingested; the compounds are degraded to bromide and excreted in the urine over a period of weeks (biological half-life of 12-20 days). Bromide also occurs naturally in the urine (Schaller, 1985).

However, there are no data on excretion of bromide after a bromine exposure (inhalation or dermal).

10.5 Antidote treatment

10.5.1 Adults

No data available.

10.5.2 Children

No data available.

10.6 Management discussion

No data available.

11. ILLUSTRATIVE CASES

11.1 Case reports from literature

Burns caused by bromine and some of its compounds:

Three cases of exposure to bromine-containing compounds were reviewed which emphasise the significance of the delay in the appearance of clinical signs and symptoms. Prompt first aid, including copious irrigation with water, reduced the extent and depth of the injury (Sagi et al., 1985).

Accidental Bromine Exposure in an Urban Population: An Acute Epidemiological Assessment:

In 1984, a large number of people in Geneva, Switzerland, was exposed to bromine gas for several hours at concentrations above the short-term exposure limit (0.3 ppm); 91 exposed individuals were evaluated. The main symptoms were: acute conjunctivitis (90%); upper respiratory tract irritation (68%); cough (47%); and headache (46%). Follow-up one month later revealed that, in all cases, the moderate eye and upper airway irritation was self-limiting (Morabia et al., 1988).

Pneumomediastinum: Complication of exposure to bromine

A maintenance technician in a chemical company developed a cough with severe bronchospasm and spontaneous pneumomediastinum following an accidental exposure to bromine. The chest radiograph on admission was normal and only a surveillance chest x-ray taken a few hours later demonstrated the presence of pneumomediastinum. This indicates the need for vigilance and serial chest radiographs after an exposure (Lossos et al., 1990).

Chemical Pneumonitis Due to Exposure to Bromine Compounds

A laboratory technician developed chemical pneumonitis following an accidental exposure to bromide compounds (hydrogen bromide and phosphorus tribromide). The patient's course was protracted with recurrent pulmonary infiltrates despite having no subsequent exposures. The case illustrated

the importance of close medical follow-up after an irritant respiratory exposure (Kraut & Lilis, 1988).

12. ADDITIONAL INFORMATION

12.1 Specific preventive measures

Although bromine is not combustible, it may react with other chemicals causing fire and explosion. Good ventilation, local exhaust or breathing protection are recommended; also protective gloves and clothing, face-shield or eye protection in combination with breathing protection (Chemical Safety Sheets, 1991).

12.2 Other

An International Chemical Safety Card (ICSC), produced by the IPCS, exists - No. 0107.

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