

**SULFUR MUSTARD****ICSC: 0418**

**Date of Peer  
Review:  
April 2005**

HD  
Mustard gas  
Bis(2-chloroethyl)sulfide  
1,1'-Thiobis(2-chloroethane)

CAS # 505-60-2 C<sub>4</sub>H<sub>8</sub>Cl<sub>2</sub>S  
RTECS # WQ0900000 Molecular mass: 159.1  
UN # 2810  
EC #

<b>TYPES OF HAZARD / EXPOSURE</b>	<b>ACUTE HAZARDS / SYMPTOMS</b>	<b>PREVENTION</b>	<b>FIRST AID / FIRE FIGHTING</b>
<b>FIRE</b>	Combustible under specific conditions.	NO open flames.	In case of fire in the surroundings: use appropriate extinguishing media.
<b>EXPLOSION</b>			
<b>EXPOSURE</b>		<b>AVOID ALL CONTACT!</b>	<b>IN ALL CASES CONSULT A DOCTOR!</b>
<b>Inhalation</b>	Cough. Burning sensation. Sore throat. Shortness of breath. Laboured breathing. Symptoms may be delayed (see Notes).	Breathing protection.	Fresh air, rest. Half-upright position. Artificial respiration may be needed. No mouth-to-mouth artificial respiration. Administer oxygen by trained personnel. Refer for medical attention.
<b>Skin</b>	MAY BE ABSORBED! Pain. Redness. Blisters. Serious skin burns.	Protective gloves. Protective clothing.	Remove contaminated clothes. Rinse and then wash skin with water and soap. Wear protective gloves when administering first aid. Refer for medical attention.
<b>Eyes</b>	Causes watering of the eyes. Redness. Pain. Severe deep burns. Permanent loss of vision.	Safety goggles and face shield or eye protection in combination with breathing protection.	First rinse with plenty of water for several minutes (remove contact lenses if easily possible), then take to a doctor.
<b>Ingestion</b>	Abdominal pain. Nausea. Vomiting.	Wash hands before eating. Do not eat,	Do NOT induce vomiting. No mouth-to-

	Burning sensation. Shock or collapse. (See Inhalation).	drink, or smoke during work.	mouth artificial respiration. Administer oxygen by trained personnel. Refer for medical attention.
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<b>SPILLAGE DISPOSAL</b>	<b>PACKAGING &amp; LABELLING</b>
Evacuate danger area! Consult an expert! Personal protection: complete protective clothing including self-contained breathing apparatus. Collect leaking and spilled liquid in sealable containers as far as possible. Absorb remaining liquid in sand or inert absorbent and remove to safe place. Do NOT let this chemical enter the environment.	Do not transport with food and feedstuffs. <b>EU Classification</b> <b>UN Classification</b> UN Hazard Class: 6.1 UN Pack Group: I
<b>EMERGENCY RESPONSE</b>	<b>STORAGE</b>
Transport Emergency Card: TEC (R)-61GT1-I NFPA Code: H4; F1; R1; 0	Separated from water, food and feedstuffs. Well closed. Ventilation along the floor. Store in an area without drain or sewer access.

**IPCS**  
International  
Programme  
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Chemical  
Safety



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## SULFUR MUSTARD

**ICSC: 0418**

### IMPORTANT DATA

**PHYSICAL STATE; APPEARANCE:**  
COLOURLESS TO YELLOW OILY LIQUID OR CRYSTALS, WITH CHARACTERISTIC ODOUR.

**CHEMICAL DANGERS:**  
The substance decomposes on heating producing toxic fumes. Reacts with water. Attacks metal.

**OCCUPATIONAL EXPOSURE LIMITS:**  
TLV not established.  
MAK: skin absorption (H); Carcinogen category: 1; (DFG 2004).

**ROUTES OF EXPOSURE:**  
The substance can be absorbed into the body by inhalation, through the skin and by ingestion.

**INHALATION RISK:**  
A harmful contamination of the air can be reached very quickly on evaporation of this substance at 20°C.

**EFFECTS OF SHORT-TERM EXPOSURE:**  
Blistering agent. Lachrymation. The substance is severely irritating to the eyes, the skin and the respiratory tract. Inhalation of the substance may cause lung oedema (see Notes). The effects may be delayed. Medical observation is indicated.

**EFFECTS OF LONG-TERM OR REPEATED EXPOSURE:**  
Repeated or prolonged contact with skin may cause dermatitis. Lungs may be affected by repeated or prolonged exposure. The substance may have effects on the eyes, resulting in impaired functions. This substance is

	carcinogenic to humans.
<b>PHYSICAL PROPERTIES</b>	
Boiling point: 216°C Melting point: 13.5°C Relative density (water = 1): 1.27 Solubility in water, g/100 ml at 25°C: 0.0068 (very poor) Vapour pressure, Pa at 20°C: 9.33 Relative vapour density (air = 1): 5.5	Flash point: 105°C Octanol/water partition coefficient as log Pow: 1.37-2.41
<b>ENVIRONMENTAL DATA</b>	
Persistence of this chemical may occur in soil and snow.	
<b>NOTES</b>	
Do NOT take working clothes home. Depending on the degree of exposure, periodic medical examination is suggested. Common name: Yperite, Lost.	
<b>ADDITIONAL INFORMATION</b>	
<b>LEGAL NOTICE</b> 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	
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# International Agency for Research on Cancer (IARC) - Summaries & Evaluations

## MUSTARD GAS (SULPHUR MUSTARD) (Group 1)

For definition of Groups, see [Preamble Evaluation](#).

**Supplement 7:** (1987) (p. 259)

**CAS No.:** 505-60-2

**Chem. Abstr. Name:** 1,1'-Thiobis(2-chloroethane)

### **A. Evidence for carcinogenicity to humans** (*sufficient*)

The mortality of British and American veterans who were exposed to mustard gas during the First World War has been compared with that of other veterans who experienced respiratory infections; the effect of smoking could not be directly controlled for in either group. Cumulative lung cancer risk was not affected in UK veterans and was only modestly elevated (relative risk, 1.5, compared with the effect of cigarette smoking, roughly 10) in US veterans [ref: 1].

In contrast, mustard gas production workers in Japan during the Second World War have been found to have experienced an increase in the proportion of deaths attributed to lung cancer (three fold) compared to the local population [ref: 1,2], and especially in respiratory cancer (40 fold) in comparison with the general population [ref: 1]. Although sophisticated analytical methods were not used, the prevalence of smoking appeared to be comparable in the exposed and unexposed groups, and there was increased risk with increased duration of exposure [ref: 3]. British workers engaged in mustard gas production during the Second World War have also been followed up. Among 511 individuals, 11 cases of cancer (nine of the larynx and two of the pharynx) were identified, whereas one would have been expected [ref: 4].

### **B. Evidence for carcinogenicity to animals** (*limited*)

Mustard gas was tested for carcinogenicity in mice, producing lung tumours after its inhalation or intravenous injection and local sarcomas after its subcutaneous injection [ref: 1].

### **C. Other relevant data**

Mustard gas is a bifunctional alkylating agent [ref: 5]. No data were available on its genetic and related effects in humans.

Evidence of covalent binding to cellular DNA, RNA and protein *in vivo* was obtained in mice injected intraperitoneally with <sup>35</sup>S-labelled mustard gas. It induced chromosomal aberrations and DNA damage in rodent cells *in vitro* and mutation in mouse lymphoma cells *in vitro* and in a host-mediated assay. It induced aneuploidy, heritable translocations, dominant lethal mutations and sex-linked recessive lethal mutations in *Drosophila*. It was mutagenic to fungi and induced DNA damage in bacteria [ref: 5].

### **Overall evaluation**

Mustard gas (sulphur mustard) is *carcinogenic to humans (Group 1)*.

For definition of the italicized terms, see [Preamble Evaluation](#).

**Also see previous evaluation:** [Vol. 9 \(1975\)](#)

## References

1. IARC Monographs, 9, 181-192, 1975
2. Shigenobu, T. (1980) Occupational cancer of the lungs - cancer of the respiratory tract among workers manufacturing poisonous gases (Jpn.). Jpn. J. thorac. Dis., 18, 880-885
3. Nishimoto, Y., Yamakido, M., Shigenobu, T., Onari, K. & Yukutake, M. (1983) Long term observation of poison gas workers with special reference to respiratory cancers. J. Univ. occup. environ. Health, 5 (Suppl.), 89-94
4. Manning, K.P., Skegg, D.C.G., Stell, P.M. & Doll, R. (1981) Cancer of the larynx and other occupational hazards of mustard gas workers. Clin. Otolaryngol., 6, 165-170
5. IARC Monographs, Suppl. 6, 403-405, 1987

## Synonyms

- Bis(2-chloroethyl)sulphide
- Bis(  $\beta$ -chloroethyl)sulphide
- 1-Chloro-2-(  $\beta$ -chloroethylthio)ethane
- 2,2'-Dichlorodiethyl sulphide
- Di-2-chloroethyl sulphide
- $\beta,\beta'$ -Dichloroethyl sulphide
- Schwefel-lost
- S-Lost
- S-Mustard
- Sulphur mustard
- Sulphur mustard gas
- Yellow cross liquid
- Yperite

## 1. Name

### 1.1 Substance

Mustard gas (sulphur mustard)

### 1.2 Group

Alkylating agents

### 1.3 Synonyms

1, 1' thiobis [2 chloroethane]  
bis-(2-chloroethyl) sulphide  
beta, beta' dichloroethyl sulphide  
2, 2' dichloroethyl sulphide  
bis (beta-chloroethyl) sulphide  
1-chloro-2 (beta-chlorodiethylthio) ethane  
sulphur mustard  
yellow cross liquid  
Kampfstoff "Lost"  
Yperite  
H  
HT  
HD

### 1.4 Identification numbers

#### 1.4.1 CAS

505-60-2

#### 1.4.2 Other numbers

No other numbers found.

### 1.5 Brand names/Trade names

To be filled in by centre using the monograph.

### 1.6 Manufacturers, importers

To be filled in by centre using the monograph.

## 2. SUMMARY

### 2.1 Main risks and target organs

Acute poisoning - Main risks

Mustard gas is a powerful irritant and vesicant, used as a chemical warfare agent. The main risk of acute poisoning exists with the use of sulphur mustard vapour in war and occasionally during laboratory work. However, due to its persistence poisoning may occur at a later stage.

Target organs: Skin, eyes, respiratory tract, bone marrow.

Chronic toxicity - Main risks

Workers employed in the manufacture of mustard gas are at an increased risk to develop cancer. Risk of mortality from influenza, pneumonia and chronic respiratory disease was also reported to be higher in persons occupationally exposed to mustard gas.

Delayed toxic effects of sulphur mustard may occur months and years after exposure, mainly with respiratory disorders.

Target organs: Lung, larynx, pharynx, oral cavity, bone marrow and sexual organs.

## 2.2 Summary of clinical effects

### Acute poisoning

Effects of exposure to mustard gas vapour or liquid are typically delayed for several hours. The delay is shorter in case of liquid contamination. In the first hour after exposure to mustard gas vapour or liquid no signs or symptoms are usually produced, but nausea, retching, vomiting and eye smarting have been occasionally reported.

Exposure to superlethal concentrations may induce convulsions, coma and death within one hour after exposure.

Nausea, fatigue, headache, eye inflammation with intense eye pain, lachrymation, blepharospasm, photophobia and rhinorrhoea, followed by reddening of face and neck, soreness of throat and increased pulse and respiratory rate develop at two to six hours post exposure.

Six to twenty four hours post exposure the above symptoms are generally increased in severity and are accompanied by skin inflammation followed by blister formation in the warmest areas such as genito-perineal area, buttocks, axillae and on the inner aspects of thighs.

In the next twenty four hours the condition generally worsens, blistering becomes more marked, coughing appears. Mucus, pus and necrotic slough may be expectorated. Intense itching of skin and increased skin pigmentation occur.

The blood count may reveal anaemia and neutropenia four days post exposure. In general, initial leukocytosis on the first 2 to 3 days after exposure is followed by leukopenia in severe intoxicated patients.

A few hours after the ingestion of mustard contaminated food or water, the following signs and symptoms develop: nausea, vomiting, abdominal pain, bloody vomiting and diarrhoea with signs of shock and prostration in severe poisoning. The patients who are severely intoxicated may die during the second week after exposure due to respiratory complications and septic shock.

### Chronic toxicity

Increased risk of cancer of oral cavity and respiratory tract has been observed in workers chronically exposed to mustard

gas. Development of cancer is likely after a single exposure. Cases of leukaemia, lung and stomach cancers were observed in Iranian combatants who were exposed once to sulphur mustard.

### 2.3 Relevant laboratory analyses/sample collection

Blood, urine and blister fluids should be collected for haematological, biochemical and toxicological analyses.

#### Acute overdose

Full blood count, serum electrolytes, urea, protein levels. Arterial blood gasses determination is indicated in cases of pulmonary oedema and Adult respiratory distress syndrome. Culture of sputum and eye exudate. Blood cultures where indicated.

Skin blisters may be aspirated and the fluid obtained analysed for thioidiglycol. The same estimation may be performed in blood and urine in order to differentiate blistering produced by mustard gas from that produced by other agents such as Lewisite. Contents of blisters are not toxic to attendants (Sulzberger, 1943). However, secondary exposure of the nursing and technical staff occurred in Iran after caring for the patients and handling the blister fluid.

#### Chronic toxicity

Mutagenicity can be evaluated by counting sister chromatid exchanges in lymphocytes (Wulf et al., 1985).

### 2.4 First-aid measures and management principles

#### Life support

Support respiratory and cardiovascular function. Treat pulmonary oedema and respiratory distress syndrome. The patients with severe leukopenia ( $<1000$  WBC/mm) should be isolated to avoid secondary infection and septic shock.

#### Eye decontamination

Irrigate the eyes immediately with copious amounts of normal saline or water for at least 15 minutes. Since sulphur mustard is lipid soluble, it is advisable to use diluted infant shampoo as well.

#### Skin decontamination

Remove any contaminated clothing. Wash exposed area thoroughly with water and neutral soap. Areas of liquid contamination should be decontaminated using Fullers' earth. Washing with organic solvents such as paraffin followed by the use of soap and water has also been recommended.

#### Gut decontamination

Emesis should not be induced. Gastric lavage is indicated after ingestion of food or water contaminated with mustard

gas. Airways should be protected by cuffed endotracheal intubation. Prior to gastric lavage stomach contents should be diluted by 100 to 200 mL of milk or water.

Activated charcoal is of unproven benefit, but may be used.

#### Symptomatic treatment

Provide adequate analgesia. Routine use of morphine is not indicated due to its depression of respiration. Correct fluid and electrolyte imbalance carefully, avoiding a net positive fluid balance. Systemic and inhaled corticosteroids are effective in antagonizing pulmonary toxicity.

Treat eyes with antibiotics, preferably sulphacetamide 20% solution, and mydriatics. In case of keratitis the use of corticosteroid eye drops is contraindicated.

Dark glasses are helpful, but contact lenses are contraindicated. Reassure the patient that visual recovery is usual. Seek ophthalmological opinion.

Treat skin lesions with standard therapy for severe chemical burns, preferably with silver sulphadiazine cream.

Treat infection with appropriate antibiotic.

Observe patients who ingested contaminated food or water with mustard gas for the development of complications caused by gastrointestinal tract burns, such as haemorrhage and perforation.

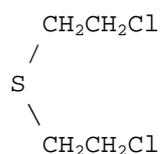
Blood transfusion may be required in patients with bone marrow depression.

### 3. PHYSICO-CHEMICAL PROPERTIES

#### 3.1 Origin of the substance

Mustard gas is a synthetic substance prepared by treating ethylene with sulfur chloride (Levinstein process) or by treating 2, 2'- dihydroxyethyl sulfide with HCl gas (German process) (Merck Index, 1989). The synthesis was first reported by Victor Meyer in 1886. Mustard gas was first used as a chemical warfare agent in 1917.

#### 3.2 Chemical structure (formula, molecular weight)



MW 159.08

### 3.3 Physical properties

#### 3.3.1 Colour

Colourless or yellow liquid (1 atm. and 15°C).

#### 3.3.2 State/form

Oily liquid at 1 atm. and 15°C.

#### 3.3.3 Description

Melting point: 13 to 14°C

Boiling point: 215 to 217°C

Vapour pressure at: 0°C 0.025 mm  
30°C 0.090 mm

Solubility: very sparingly in water; soluble in fats and organic solvents.

Heavier than water.

Weak mustard or garlic like odour.

### 3.4 Other characteristics

Hydrolysed by water to thiodiglycol and hydrochloric acid. Half life for hydrolysis is 5 minutes at 37°C. Hydrolysis is catalysed by increased temperature and presence of alkalies. Oxidized by bleaching powder and chloramines into sulfoxides (harmless) and sulfones (possess vesicant activity) and sulfides.

Vapour has marked penetrating power; penetrates cloth, leather, wood and paint on metallic surfaces. Metal, glass and glazed tiles are impermeable. Persistent in the environment. Hydrolyses with water only occurs after thorough mixing. Since it is heavier than water it sinks and it is believed that it can provide a continuing local source of poison for some time. Dangerous oily film of sulphur mustard remains on the water surface.

Contact with sea water turns mustard gas from its normal liquid state to viscous or even solid one. It is believed that large quantities lie loose in lumps on the sea bed of Baltic sea where gas bombs were dumped at the end of the second world war (Perera & Thomas, 1987).

It decomposes at high temperatures and produces toxic compounds containing sulphur and chlorine oxides with strong lachrymatory actions.

## 4. USES/HIGH RISK CIRCUMSTANCES OF POISONING

### 4.1 Uses

#### 4.1.1 Uses

#### 4.1.2 Description

In chemical warfare with intention to:

(a) to prevent or delay certain activities within restricted areas;

(b) to disturb or exhaust personnel by compelling them to wear respirators or to remain on gas-proof premises for lengthy periods (under special weather conditions only) (Lundquist, 1983).

It may be used as liquid or vapour and may be delivered by artillery shell, rocket, bomb or aircraft spray.

The use is likely to be influenced by the following meteorological factors:

(a) Temperature. High temperatures increase the toxicity of mustard gas. Low temperature may freeze it and so increase its persistence. The danger of carrying such an agent into a warm building on boots and equipment and so giving off toxic vapour, should be born in mind.

(b) Rain. Heavy rain reduces its toxicity.

(c) Atmospheric stability. Persistence of vapour is prolonged by inversion (the air temperature higher than that of the ground).

#### 4.2 High risk circumstances of poisoning

Mustard gas is suitable for tactical attacks in limited operations or restricted areas putting army personnel at the highest risk of exposure. Civilian inhabitants in the neighbourhood of military activities may be exposed to some risk of being affected by both chemical contamination and drifting chemical clouds (Lundquist, 1983). Due to its persistency mustard gas can remain in the ground and in water for a long time especially so in cold conditions. Exposure may therefore occur some time after the attack.

Furthermore 'accidental' release following an attack on stocks of mustard gas can cause several casualties as well as severe environmental damage (Marshall, 1987).

Accidental exposure may occur during dumping of unused mustard gas or during coincidental encounters with improperly disposed of containers.

Food and water are easily contaminated when exposed to liquid or vapour forms of mustard gas. Intoxication is likely following ingestion of contaminated food and water. Mustard gas is freely soluble in oils and fats and large quantities can be absorbed by food with a high fat content. Only food sealed in impermeable containers such as tins, glass or glazed earthenware jars and foil wrappings is completely protected (See Section 12.2 for Decontamination of food and water).

Laboratory workers involved in organic synthesis of sulphur mustard and also health professionals, who are involved in the caring of the patients and handling of blister fluids,

are at risk.

#### 4.3 Occupationally exposed populations

Military staff in the battle field are at risk of acute exposure in case of chemical warfare. Chemical accidents with sulphur mustard may occur during manufacturing, transportation, storage and use.

Workers employed in the manufacture of mustard gas are believed to be at risk of developing cancers of the oral cavity and the respiratory tract (Wada et al. 1968, Easton et al. 1988).

### 5. ROUTES OF ENTRY

#### 5.1 Oral

Oral exposure is likely if food or water are contaminated with mustard gas. Air that is polluted with sulphur mustard may cause oesophagogastric damage if swallowed.

#### 5.2 Inhalation

Usual route of entry.

#### 5.3 Dermal

Mustard gas has a marked penetrating power making skin a common route of entry.

#### 5.4 Eye

Usual route of entry.

#### 5.5 Parenteral

Unknown.

#### 5.6 Others

Unknown.

### 6. KINETICS

#### 6.1 Absorption by route of exposure

Mustard gas is absorbed in the respiratory tract when inhaled.

It has been demonstrated that 80% of sulphur mustard applied to the skin evaporates, 10% remains in the skin and 10% gets absorbed systemically (Renshaw, 1946). It can penetrate the skin by contact with either the liquid or vapour. The rate of penetration is proportional to dose, temperature and humidity.

#### 6.2 Distribution by route of exposure

Equilibrium between blood and tissues was achieved within 5 minutes after perfusion of the lung in dog (IARC, 1975).

Mustard gas is highly fat soluble and expected to accumulate in those tissues with a high fat content.

Levels of mustard in the tissues of an Iranian patient who died 7 days after exposure to mustard gas were qualitatively analysed (Drasch et al., 1987). The concentrations of sulphur mustard determined by GC/MS and atomic absorption spectrophotometer in the tissues were as follows:

fat (from thigh)	15.1 mg/kg
brain	10.7 mg/kg
abdominal skin	8.4 mg/kg
kidney	5.6 mg/kg
muscle	3.9 mg/kg
liver	2.4 mg/kg
cerebrospinal fluid	1.9 mg/L
spleen	1.5 mg/kg
blood	1.1 mg/L.

### 6.3 Biological half-life by route of exposure

The precise half life is not known.

The parent compound could be detected in human urine up to a week after acute exposure but not several days later (Vycudilik, 1985).

### 6.4 Metabolism

Metabolic studies with radioactively labelled mustard gas were performed in rodents (Somani & Babu, 1989). The following metabolic pathways were proposed: hydrolyses to thiodiglycol and S-oxidation to sulfoxide and sulfone, followed by conjugation. Urinary metabolites in rats consisted of thiodiglycol and conjugates (15%), glutathione-bis-beta-chloroethylsulfide conjugates (45%), glutathione-bis-beta chloroethylsulfone (7%), bis-beta-chloroethylsulfone and conjugates (8%), and small amounts of cysteine conjugates.

Urinary metabolites formed from intraperitoneal injection in rats were bis-cysteinylethylsulfone and thiodiglycol.

### 6.5 Elimination and excretion by route of exposure

The urinary excretion of unmetabolized sulphur mustard is low (Drasch et al., 1987). This is probably caused by its strong fixation to the lipid compartments of the body (See 6.2).

The major portion of mustard gas excreted in the urine represents compounds formed by alkylation, followed by metabolites formed by enzymatic action.

The majority of an intravenously-injected dose of 1.5 mg/kg 35S-mustard gas was excreted in the urine within 72 hours in mice and rats; approximately 6% was eliminated in the faeces

(IARC, 1975).

## 7. TOXICOLOGY

### 7.1 Mode of action

Sulphur mustard is an alkylating agent. Alkylating agents bind covalently to various nucleophilic molecules such as DNA, RNA, proteins and components of cell membranes (Somani & Babu, 1989).

DNA: Mustard gas causes cross linking of DNA strands. Alkylation of DNA can result in the disruption of its function, i.e. coding errors, breakage of the strand, low fidelity repair, inhibition of replication and cell death.

It has been proposed that DNA damage can be followed by release of plasminogen activator which may play a role in skin blistering by disrupting the dermal-epidermal junction.

RNA: Alkylation of RNA molecules can result in altered translation and altered protein synthesis resulting in cell death.

Proteins: Binding to proteins mainly with the thiol group of cysteine produces structural changes which may alter the normal physiology of the cell i.e. altered enzyme activity.

Membranes: Mustard gas can either alkylate structural proteins located in the cell membrane or induce lipid peroxidation which may result in irreversible changes and cell death.

NAD<sup>+</sup> depletion: The cell has a capacity to repair damaged DNA but the repair process can further disrupt functioning of the cell. Enzymes involved in the DNA repair mechanism utilise NAD<sup>+</sup> and cause NAD<sup>+</sup> depletion. Consequently glycolysis becomes inhibited which could lead to cell death (Papirmeister, 1983).

### 7.2 Range of toxicity

#### 7.2.1 Human data

##### 7.2.1.1 Adults

Death has been recorded by dermal exposure after 1 hour at 64 mg/kg, and by inhalation at 1500 mg min/m<sup>3</sup> (Marshall, 1987).

The toxicity of mustard gas vapour is expressed in terms of the profile of exposure (Ct) in mg min/m<sup>3</sup>, and that of liquid in µg/cm<sup>2</sup>.

Effects on eyes:

50 mg min/m<sup>3</sup> maximum safe dosage  
70 mg min/m<sup>3</sup> mild reddening of the eyes  
100 mg min/m<sup>3</sup> partial incapacitation due to  
eye effects  
200 mg min/m<sup>3</sup> total incapacitation due to  
temporary blindness

Effects on skin:

100 to 400 mg min/m<sup>3</sup> erythema  
200 to 1000 mg min/m<sup>3</sup> skin burns  
750 to 10000 mg min/m<sup>3</sup> severe incapacitating  
skin burn  
50 µg/cm<sup>2</sup> erythema in 5 min.  
250-500 µg/cm<sup>2</sup> blistering in 5 min.

Increased temperature enhances the effects.

7.2.1.2 Children

Not known.

7.2.2 Relevant animal data

LD50 (percutaneous): rat 9 mg/kg  
dog 20 mg/kg  
rabbit 100 mg/kg

LC50 (inhalation): rat 100 mg/m<sup>3</sup> for 10 minutes  
rabbit 280 mg/m<sup>3</sup> for 10 minutes  
monkey 80 mg/m<sup>3</sup> for 10 minutes

7.2.3 Relevant in vitro data

Mutagenicity and genotoxicity of mustard gas were observed in several in vitro tests such as bacterial screening tests, human Hela cells and mouse lymphocytes (Dabney, 1989).

Increased sister chromatid exchanges were seen in lymphocytes of exposed fishermen (Wulf, et al. 1985).

7.2.4 Workplace standards

McNamara et al., (1975) proposed the following workplace standards with personal protective devices:

Ceiling	0.4	mg/m <sup>3</sup>
CL - 6 minutes	0.3	mg/m <sup>3</sup>
CL - 3 hours	0.01	mg/m <sup>3</sup>
CL - five 8 hour days	0.003	mg/m <sup>3</sup> .

A ceiling is the concentration that must not be exceeded for any period of time and places a limit on the maximum upper excursion of concentration during the averaging hours.

A control limit (CL) is the maximum average airborne concentration of a substance to which it is believed that essentially all members of a specified population can be exposed for a specified period without adverse effects.

#### 7.2.5 Acceptable daily intake (ADI) and other guideline levels

ADI is not determined.

Mc Namara et al. (1975) have also proposed the levels of exposure for the general population:

Ceiling	0.01 mg/m <sup>3</sup>
CL - 3 hours	0.00033 mg/m <sup>3</sup>
CL - 8 hours	0.00017 mg/m <sup>3</sup>
CL - 72 hours	0.00001 mg/m <sup>3</sup>

The definitions for a ceiling and a control limit (CL) are given in Section 7.2.4.

### 7.3 Carcinogenicity

Mustard gas is a well documented animal carcinogen and is listed as an accepted human carcinogen (IARC, 1975). Increased mortality from oral cavity and respiratory tract cancer has been shown in several studies in humans exposed to mustard gas, with risk of mortality being greater from chronic occupational exposure than from sporadic exposure (Case & Lea, 1955; Wada et al. 1968; Easton et al., 1988)

### 7.4 Teratogenicity

Teratogenic potential of orally administered sulphur mustard was evaluated in rats and rabbits. Maternal toxicity was observed at all concentrations of mustard gas but significant fetal effects such as decreased weights, reduced ossification and skeletal anomalies were observed only at the highest dose (2 mg/kg). It was concluded that sulphur mustard is not teratogenic in rats and rabbits (Somani & Babu, 1989).

However, nitrogen mustards, compounds related to sulphur mustard and used in cancer chemotherapy were shown to be teratogenic in all laboratory species tested (Schardein, 1985). Multiple defects including cleft palate, central nervous system, jaw, limb and digit abnormalities were observed. In humans, digital defects and kidney malformations were described.

### 7.5 Mutagenicity

Simple mutations, structural chromosomal aberrations, sex chromosome loss and nondisjunction and heritable translocations have all been observed in numerous in vitro tests (Dabney, 1989).

Sister chromatid exchanges were measured in fishermen exposed

to leaking mustard gas shells (Wulf et al., 1985) and were found to be significantly higher than in a control group matched for sex, age and tobacco consumption.

#### 7.6 Interactions

No information found.

#### 8. Toxicological analyses

To be completed.

#### 9. CLINICAL EFFECTS

##### 9.1 Acute poisoning

A characteristic feature of exposure to sulphur mustard is an asymptomatic period which may last for up to two hours. The duration of this latent period depends on the mode of exposure, environmental temperature and individual sensitivity.

###### 9.1.1 Ingestion

A few hours after ingestion nausea, vomiting, abdominal pain, bloody vomiting and diarrhoea, and in cases of severe poisoning shock and prostration, may be expected. Systemic toxic effects in the respiratory tract, skin, eye, and bone marrow may occur thereafter.

###### 9.1.2 Inhalation

20 to 60 minutes post exposure: Usually none, but nausea, retching, vomiting and eye smarting can occur. Respiratory irritation with coughing and dyspnoea may also occur.

2 to 6 hours post exposure: Nausea, fatigue, headache, rhinorrhoea, sore throat. Voice becomes hoarse and may be completely lost. Pulse and respiratory rate are increased. Eye symptoms and signs are described in Section 9.1.4.

6 to 24 hours post exposure: Increase in severity of above effects.

48 hours post exposure: Severe coughing, mucus, pus and slough may be expectorated.

4 days or more post exposure: Initial leukocytosis followed by leucopenia.

###### 9.1.3 Skin exposure

20 to 60 minutes post exposure: Usually none, but a mild erythema with pruritus may occur.

2 to 6 hours post exposure: Nausea, fatigue, headache, reddening of the face and neck, increased pulse and

respiratory rate.

6 to 24 hours: Increase in severity of above effects. Inflammation of inner thighs, genitalia, perineum, buttocks, axillae followed by blister formation. Blisters vary in size are pendulous and filled with clear yellow fluid.

48 hours post exposure: condition worsened. Marked blistering, intense itching of the skin and increased skin pigmentation occur.

4 days or more post exposure: leucopenia.

#### 9.1.4 Eye contact

20 to 60 minutes post exposure: usually none, but eye smarting is possible.

2 to 6 hours post exposure: inflammation, intense pain, lachrymation, blepharospasm, photophobia.

1 to 10 days post exposure: corneal epithelial loss and stromal opacification. Secondary infection and uveitis may occur, but are uncommon. Late: abnormalities of limbal and vascular bed, ischemia and ulceration.

#### 9.1.5 Parenteral exposure

Not known.

#### 9.1.6 Other

Not known.

## 9.2 Chronic poisoning

### 9.2.1 Ingestion

Not known.

### 9.2.2 Inhalation

Increased incidence of cancer of oral cavity and respiratory tract was reported among workers employed in the manufacture of mustard gas (Wada et al. 1968, Easton et al. 1988).

Nishimoto et al. (1970) found high prevalence of chronic obstructive lung disease in workers exposed repeatedly to mustard gas or lewisite.

Delayed toxic effects, mainly on the respiratory tract (obstructive and restrictive lung disease), and malignancy may occur years after a single exposure.

### 9.2.3 Skin exposure

Exposure may result in diffuse changes of skin

pigmentation with areas of hyperpigmentation and hypopigmentation and scarring of the skin.

#### 9.2.4 Eye contact

Direct exposure of sulphur mustard droplets to the eyes have occurred amongst Iranian combatants in the field. As a result erosive keratoconjunctivitis and blepharospasms occurred.

#### 9.2.5 Parenteral exposure

Not known.

#### 9.2.6 Other

Not known.

### 9.3 Course, prognosis, cause of death

Most of the victims with minimal exposure to mustard gas recover without any consequences.

The majority of eye lesions are resolved in 28 days post exposure.

Superficial skin lesions heal in 14 to 21 days, while deep skin lesions may be expected to heal in up to 60 days. However, residual scarring with itching may last up to 10 years and longer.

The time course for the resolution of respiratory tract lesions is difficult to predict but lung function tests may provide a useful guide. Initial obstructive lung disease followed by restrictive lung disease.

Initial leukocytosis followed by leucopenia is a usual finding in mustard gas poisoning and recovers within 14 days. Marked leucopenia is a sign of sinister prognosis, leading to overwhelming infection and multiple organ failure and death.

Death may also result due to pulmonary oedema, Adult respiratory distress syndrome, airway obstruction, arrhythmias and cardiac arrest.

### 9.4 Systematic description of clinical effects

#### 9.4.1 Cardiovascular

Acute: Arrhythmias - AV block and possibly cardiac arrest (NATO, 1973).

Chronic: None described.

Acute-on-chronic: None described.

#### 9.4.2 Respiratory

Acute: Coughing and tachypnoea. Inflammation

of bronchial mucosa with bleeding, purulent secretions, and sloughing of the necrotic mucosa. Haemorrhagic oedema in peribronchial alveoli. In severe cases a syndrome similar to adult respiratory distress syndrome can develop.

Chronic: Chronic bronchitis. Bronchial stenosis. Significantly increased incidence ( $p < 0.001$ ) of lung cancer was reported in workers employed in the manufacture of mustard gas (Easton et al., 1988). Wada et al. (1968) observed an increased incidence of cancer, while Nishimoto et al. (1970) reported an increased incidence of chronic obstructive lung disease in Japanese factory workers involved in production of mustard gas.

Acute respiratory tract infections were shown to be a more common cause of death in the elderly exposed to mustard gas compared to the un-exposed (Easton et al., 1988).

#### 9.4.3 Neurological

##### 9.4.3.1 CNS

Acute: Apathy, mental disturbance and anxiety states were reported among soldiers exposed to mustard gas during the First World War. Neuropsychiatric disorders including insomnia, anxiety, agitation, depression and acute psychosis were observed in Iranian combatants.

Chronic: The above features may persist for some time.

Occasionally chronic psychosis with a poor response to treatment has occurred.

##### 9.4.3.2 Peripheral nervous system

Cases of peripheral polyneuropathy have been observed.

##### 9.4.3.3 Autonomic nervous system

None described.

##### 9.4.3.4 Skeletal and smooth muscle

None described.

#### 9.4.4 Gastrointestinal

Acute: Nausea, vomiting, abdominal pain, bloody diarrhoea.

Chronic: Perforation, bleeding and late stricture formation can result from burns in the gastrointestinal tract.

#### 9.4.5 Hepatic

None described.

Transient elevation of transaminases were observed in Iranian patients with sulphur mustard poisoning.

#### 9.4.6 Urinary

##### 9.4.6.1 Renal

None described.

Transient elevation of proteinuria and haematuria were observed in Iranian patients.

##### 9.4.6.2 Other

None described.

#### 9.4.7 Endocrine and reproductive systems

None described.

Cases of abnormal sperm shape, oligospermia and occasionally azospermia were observed in Iranian patients.

#### 9.4.8 Dermatological

Acute: (a) Striking erythema of the skin, accompanied by intense itching particularly in axillary and genito perineal areas. As erythema fades, areas of increased pigmentation appear; (b) Blistering of the skin. Blisters vary in size, are delicate and can be easily rubbed off. Rubbing can lead to the development of new crops of blisters (Nikolsky's sign), which can appear as late as two weeks post exposure. Blisters are uncomfortable and may feel tense, but are not painful. However, when they appear over joints, they are reputedly painful and may hinder movement of these joints. Blisters are filled with fluid which may cause blistering if applied to skin. Healing is characterised by hyper and hypo-pigmentation changes; (c) Deep burning, which can lead to full thickness skin loss, accompanied with severe pain. Skin burns characteristically take longer to heal than typical thermal burns.

Chronic: Diffuse changes of skin pigmentation with areas of hyperpigmentation. Scarring of the skin.

#### 9.4.9 Eyes, ears, nose, throat: local effects

Acute:

Eyes: marked conjunctivitis, local oedema,

blepharospasm, lachrymation, miosis, photophobia, severe eye pain.

Nose: profuse rhinorrhea and rarely epistaxis.

Oral cavity, pharynx, larynx: inflammation and ulceration of the palate, nasopharynx, oropharynx and larynx, with hoarseness of voice and temporary aphonia.

Chronic:

Eyes: corneal ulceration, adhesions of the iris to the lens capsule, visual impairment and permanent blindness.

Oral cavity, pharynx, larynx: Increased incidence of cancer was reported in British workers employed in the production of mustard gas (Easton et al., 1988).

#### 9.4.10 Haematological

Acute: Early leucocytosis, followed by mild leucopenia. Severe leukopenia, trombocytopenia and erythropenia indicate bone marrow depression.

Chronic: Bone marrow depression leading to leukaemia.

#### 9.4.11 Immunological

Immunosuppression, by sulphur mustard, observed either as cellular (mainly T-cells) or humoral (IgA suppression) was observed in Iranian patients.

Acute: See 9.4.13.

Chronic: See 9.4.13.

#### 9.4.12 Metabolic

##### 9.4.12.1 Acid-base disturbances

Acute: Tachypnoea may cause respiratory alkalosis which may be followed by acidosis due to pulmonary oedema and chemical burns.

Chronic: None described.

##### 9.4.12.2 Fluid and electrolyte disturbances

Acute: Dehydration.

Chronic: None described.

##### 9.4.12.3 Other

None described.

#### 9.4.13 Allergic reactions

Acute: Cutaneous sensitization may occur from repeated exposure of 1 to 3 weeks.

Chronic: Sensitivity to mustard gas. A morbilliform rash and eczematoid dermatitis around old skin lesions are characteristic of sensitization reactions (NATO, 1973).

Acute-on-chronic: Sensitized individuals may have a shorter latent period than nonsensitized persons for development of dermal symptoms (NATO, 1973).

#### 9.4.14 Other clinical effects

None described.

#### 9.4.15 Special risks

Pregnancy: The risk for the development of fetal malformations following the exposure to nitrogen mustards was estimated to 1:3 (Schardein, 1985), but no information was found on sulphur mustard. Fetal abnormalities such as cleft lip was observed in a few cases of the Iranian victims.

Breast feeding: No information found, but due to high solubility of mustard gas in fats it could be expected that it would appear in the milk of the exposed lactating mothers.

Enzyme deficiency: No data available.

### 9.5 Other

None described.

## 10. MANAGEMENT

### 10.1 General principles

Management of mustard gas poisoning consists of decontamination and symptomatic treatment. The importance of rapid and efficient decontamination can not be overemphasized.

### 10.2 Relevant laboratory analyses

#### 10.2.1 Sample collection

To be completed.

#### 10.2.2 Biomedical analysis

Full blood count, serum electrolytes, urea, protein levels. Arterial blood gasses determination is indicated in case of pulmonary toxicity.

Culture of sputum and eye exudate.

Blood cultures where indicated.

Skin blisters may be aspirated and the fluid obtained analysed for thioglycol. The same estimation may be performed in blood and urine in order to differentiate blistering produced by mustard gas from that produced by other agents such as Lewisite. Contents of blisters are not toxic to attendants. (Sulzberger, 1943).

#### 10.2.3 Toxicological analysis

To be completed.

### 10.3 Life support procedures and symptomatic/specific treatment

Life support:

Maintain respiratory and circulatory function.

Replace extracellular fluid loss, electrolytes and proteins.

Blood transfusion is indicated in case of bone marrow depression.

Symptomatic treatment:

GENERAL:

Analgesics. The choice of analgesic depends on the severity of pain in each individual. Mild analgesics can be given together with diazepam to dissociate pain from panic. Routine use of morphine is contraindicated due to its depression of respiration. Carbamazepine 200 mg thrice daily has been reported to control intense burning pain during skin healing (Newman-Taylor & Morris, 1991). Give appropriate antibiotics as indicated. Antiemetics, i.e. phenothiazines if vomiting persists.

SKIN:

Bland lotions (Prapoderm) for erythema and mild blistering. Silver sulphadiazine. Corticosteroid preparations (i.e. Hydrocortisone lotion, Beclomethasone dipropionate) reduce irritation and itching. Antihistamines (i.e. promethazine, dimethidine) can be of value in reducing itching. Skin grafting may occasionally be necessary in full thickness burns.

EYES:

Start immediate irrigation with normal saline or water. Use Vaseline on follicular margins to prevent sticking. Avoid cocaine because it may produce sloughing of the corneal epithelium. Topical corticosteroids (i.e. 1% prednisolone four times a day) should be continued until all signs of inflammation have gone in order to prevent late corneal dissolution. In case of corneal erosion and keratitis topical corticosteroid must not be used. Use Chloramphenicol eye

drops, as appropriate. Mydriatics i.e. hyoscine (0.5%) is administered to prevent sticking of the iris to the lens. Dark glasses may alleviate photophobia. Give reassurance that ocular and visual recovery is usual. Contact lenses are contraindicated. Topical ascorbate and citrate drops are indicated when severe burns with limbal ischaemia and epithelial defects persist for more than 5 days and are accompanied by structural changes. Apply 10% potassium ascorbate and 10% sodium citrate alternatively each once an hour (half hourly drops) for 14 hours. These drops can be safely discontinued when a stable epithelial covering develops. Seek ophthalmological opinion.

#### 10.4 Decontamination

Remove victims from the source of decontamination.

Eye decontamination: Irrigate the eyes immediately with copious amounts of normal saline or water for at least 15 minutes.

A solution of diluted infant shampoo may be useful for eye decontamination.

Skin decontamination: Remove any contaminated clothing. Wash exposed area thoroughly with water and neutral soap. Areas of liquid contamination should be decontaminated using Fullers' earth. Washing with organic solvents such as paraffin oil followed by the use of soap and water has also been recommended.

Gut decontamination: Emesis should not be induced. Gastric lavage is indicated after ingestion of mustard gas. Airways should be protected by cuffed endotracheal intubation. Prior to gastric lavage stomach contents should be diluted by 100 to 200 ml of milk or water. Activated charcoal is of unproven benefit but may be used.

#### 10.5 Elimination

No reliable method established.

#### 10.6 Antidote treatment

##### 10.6.1 Adults

No specific antidote is available. See 10.7.

##### 10.6.2 Children

No specific antidote is available. See 10.7.

#### 10.7 Management discussion

Bone marrow depression in severe intoxicated patients may be seen as an irreversible consequence of mustard gas poisoning. Granulocyte, platelet and red cell transfusions as well as bone marrow transplantation have been recommended for treatment of aplastic anaemia. The value of Granulocyte Colony Stimulating Factor has not been assessed but may be of use.

Several independent scientists (Callaway & Pearce, 1958; Fasth & Sorbo, 1973; Vojvodic et al., 1985) have shown that cysteine, thiosulfate and other thiols reduce the toxicity of sulphur and nitrogen mustards (Nitrogen mustards are used in chemotherapy of malignant diseases). The use of thiols has been proposed in the treatment of mustard gas poisoning, but has not been established.

Numerous other supportive measures were used in treating casualties from the Iran-Iraq war: H<sub>2</sub> antagonists to prevent stress ulceration. Heparin has been used to prevent deep venous thrombosis. A single large dose of methyl prednisolone (2 g) may prevent general tissue damage. Administration of Vitamins C, B12 and folate may be of use.

Haemodialysis and haemoperfusion have been suggested, although there is no firm theoretical basis for such therapy.

## 11. ILLUSTRATIVE CASES

### 11.1 Case reports from the literature

Mustard gas was used for the first time by Germans in 1917 at Ypres. More than 14,000 British casualties were produced in the first three months and by the end of the first world war more than 120,000 British mustard casualties had occurred. The most commonly injured areas of the body were: eyes (86.1%), respiratory tract (75.3%), scrotum (42.1%), face (26.6%), anus (23.9%), back (12.9%), armpits (12.5%), neck (12%).

Adolph Hitler was exposed to mustard gas during the first world war. He described his personal experience in "Mein Kampf" (Vol. 1, 1924): "During the night of October 13 to 14th (1918) the British opened an attack with gas on the front south of Ypres. They used the yellow gas whose effect was unknown to us, at least from personal experience. I was destined to experience it that very night. On a hill south of Werwick, in the evening of 13 October, we were subjected to several hours of heavy bombardment with gas bombs, which continued through the night with more or less intensity. About midnight a number of us were put out of action, some for ever. Towards morning I also began to feel pain. It increased with every quarter of an hour, and about seven o'clock my eyes were scorching as I staggered back and delivered the last dispatch I was destined to carry in this war. A few hours later my eyes were like glowing coals, and all was darkness around me."

During the second world war mustard gas was not used but Lundquist (1983) reports of a large number of Allied soldiers and sailors who were exposed to mustard gas towards the ends of second world war as a result of German bombing of the harbour at Bari in Italy. Of the two dozen ships destroyed, one was carrying a cargo of about 100 thousand kilograms of mustard-gas bombs. Much of the mustard gas was released into the water and some of it dissolved in the floating oil. More than 1000 people were killed and of these deaths more than

100 were determined to have been specifically caused by mustard-gas poisoning and many more to have been due to various indirectly associated reasons, such as disablement followed by drowning.

Eleven fishermen have become exposed to mustard gas from leaking shells that were dumped into the Baltic sea after the Second World War (Wulf et al., 1985). They presented with very inflamed skin, especially in the axilla and in the genitofemoral regions, yellow blisters on the hands and legs, painful irritation of the eyes and transient blindness. In two pulmonary oedema developed. Haemoglobin values, leucocyte, differential and platelet counts, lactate dehydrogenase and aspartate aminotransferase activities and serum creatinine were normal. Sister chromatid exchange count was significantly higher than in a control group. All the fishermen recovered, but might have an increased cancer risk.

It is believed that mustard gas was used by Iraq in the recent war against Iran. Dunn (1986) reports of an attack in 1984 using aerial bombs which, upon exploding at ground level, released a grey cloud with a garlic like smell. Victims of the attack suffered severe eye, bronchial and lung damage accompanied by a skin rash. Several deaths followed acute pulmonary dysfunction. It has been suggested that during this attack mustard gas had been released from a bomb in a form of micronized aerosol particles which were sufficiently small to create only a skin rash, rather than the typical skin lesions.

Bockmeyer (1985) reports of three Iranian patients who were treated in Germany following exposure to mustard gas during the Iran-Iraq war. They all suffered from first or second degree skin burns, corrosive changes in the mouth and pharynx and damage to the respiratory tract. In the second week changes in the blood picture appeared with the impairment of clotting. All three patients had to be ventilated and two of them required tracheotomy. Daily lavage with Prednisolon-21 hemisuccinate sodium and Dexapanthenol solution was also used. N-acetylcystein was applied intravenously and as an aerosol. All three patients recovered in three months.

Leipner et al. (1987) describe a late sequel of poisoning with mustard gas, not described in the literature to date, in a 22 years old patient. The patient was admitted to the hospital two years post exposure for dermatomyositis. Chest X-ray revealed lung fibrosis, mediastinal emphysema and pulmonary hypertension. CAT scan of the thorax confirmed the X-ray findings. Fibrosis was located in the dorsobasal periphery of the right lung. Perivascular collections of air were observed in the left lung communicating via the left hilus with the mediastinal emphysema. This finding suggested the presence of bronchial or lung parenchyma fistulas. Bronchoscopy did not reveal any large defects in the trachea and large bronchi. Lung function tests showed a restrictive ventilatory disorder without a considerable obstructive component.

Thirty nine Iranian soldiers and doctors exposed to sulphur mustard in the Iran-Iraq war were treated in the UK during

1985 and 1986 (Rees et al., 1991). They all suffered from skin burns and most had mucosal damage and eye inflammation. Severe cough which responded poorly to symptomatic treatment was the most common respiratory effect. Airflow obstruction was a prominent feature. Low arterial PO<sub>2</sub> was a common finding. Chest x rays showed various abnormalities such as lobar consolidation and widespread ill-defined opacities. Three patients required artificial ventilation and two of these died with respiratory, renal and bone marrow failure 7 and 14 days after exposure. In these patients remarkable sloughing was shown in the tracheal and bronchial mucosa. Bone marrow depression was common in patients with evidence of moderate exposure. One patient died due to adrenal haemorrhage associated with thrombocytopenia.

Newman-Taylor and Morris (1991) reported about another five Iranian soldiers treated in the UK in 1988, 11 days after exposure to mustard gas. They had injuries of the skin, eyes, mouth, upper respiratory tract and lungs. Skin burns involved the exposed areas, axillae, buttocks and genitalia. The treatment of skin burns included twice daily saline baths, silver sulphadiazine cream dressings and paraffin gauze. Pain was a prominent feature and was treated with opiates and antihistamines. Carbamazepine 600 mg daily was successfully used to control the pain unresponsive to opiates and antihistamines. The eyes of the patients were severely inflamed with a non-ulcerated keratitis and haemorrhagic conjunctivitis which were treated with dexamethasone and ascorbate drops 3-hourly. Three patients suffered severe inflammation and ulceration of oral and laryngeal mucosae and were treated with simple mouth washes. All patients had productive cough, four of them had inspiratory crackles, airway obstruction and hypoxemia. One patient had a lung abscess caused by Methicillin resistant *Staphylococcus aureus*. This patient also showed suppressed leucocyte response to infection. Respiratory infections were treated with antibiotics. The skin and the eyes recovered in 2 to 6 weeks without damage. The respiratory function improved slowly but steadily during the six weeks of hospitalisation. Nebulized bronchodilators were used but did not provide much symptomatic or objective benefit.

A follow up study of Japanese workers who were engaged in the manufacture of mustard gas between 1929 and 1945 showed that they had experienced 33 deaths from cancer of the respiratory tract, compared with 0.9 expected. The tumours occurred centrally and were of squamous or undifferentiated cell type (Wada et al., 1968).

Easton et al. (1988) also provided evidence that chronic exposure to mustard gas can cause cancers of respiratory tract. Significant excesses of malignant tumours of oral cavity, pharynx, larynx and lungs were observed among British workers employed in manufacture of mustard gas during the second world war. Incidence of deaths from nonmalignant acute and chronic respiratory diseases was also increased.

The reports on the possible long term respiratory effects of acute exposure to mustard gas are controversial. One follow up study on soldiers who were exposed to mustard gas during the first world war suggests that mustard gas had no effect

on the development of lung cancer later in life (Case & Lea, 1955), while the other provided evidence that the incidence was slightly increased among those war veterans exposed to mustard gas (Beebe, 1960). It is probably unlikely that a single exposure to mustard gas can cause cancer.

#### 11.2 Internally-extracted data on cases

None available.

### 12. ADDITIONAL INFORMATION

#### 12.1 Availability of antidotes

None established. See also 10.7.

#### 12.2 Specific preventative measures

When food is suspected to be contaminated it should be destroyed. However, where food is scarce the following measures have been proposed (Ministry of Defence, 1987):

All food that was exposed to liquid forms of mustard gas should be destroyed.

All high fat content food such as butter, fat, milk, cheese, meat, bacon should be destroyed.

Low fat content food that has been contaminated with the vapour form of mustard gas should be washed with 2% sodium bicarbonate solution, peeled where applicable and cooked by boiling. Low fat content dry foods should be exposed to the air for 48 hours.

Sugar, salt and foods of high water content such as fruit and vegetables, sugar, salt may be made unpalatable by the formation of acid products of hydrolyses.

Open water sources may become contaminated, but there is no practicable means of decontaminating water in the field. Water from deep sources such as springs and wells is likely to be contaminated.

#### 12.3 Other

Special equipment is available to test for the presence of sulphur mustard in the environment.

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#### 14. AUTHOR(S), REVIEWER(S), ADDRESS(ES), DATE(S)

Authors: Dr L. Perharic Walton  
National Poisons Unit  
Guy's Hospital, London  
Avonely Rd  
London SE14 6TE

Dr R.L. Maynard  
Department of Health  
Hannibal House  
Elephant & Castle  
London SE 1 5ER

Dr V.S.G. Murray

National Poisons Unit  
Guy's Hospital, London  
Avonely Rd  
London SE14 6TE

Date: May 1991  
Reviewer: Dr J. Szajewski  
Warsaw Poison Centre  
  
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