

# AEROSOL SPECIALTIES CLEANER

Chemwatch Independent Material Safety Data Sheet

Issue Date: 1-Nov-2010

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## Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

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### PRODUCT NAME

AEROSOL SPECIALTIES CLEANER

### SYNONYMS

GP3, "General Purpose Cleaner"

### PROPER SHIPPING NAME

AEROSOLS

### PRODUCT USE

■ Application is by spray atomisation from a hand held aerosol pack.  
All-purpose cleaner.

### SUPPLIER

Company: Appco Group Prosales Pty Ltd

Address:

Level 6, 80 Cooper Street

Surry Hills

NSW, 2007

Australia

Telephone: +61 2 8219 7900

Fax: +61 2 9280 4878

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## Section 2 - HAZARDS IDENTIFICATION

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### STATEMENT OF HAZARDOUS NATURE

**DANGEROUS GOODS. NON-HAZARDOUS SUBSTANCE.** According to ADG Code and NOHSC Criteria.

### RISK

Risk Codes

R44

Risk Phrases

■ Risk of explosion if heated under confinement.

### SAFETY

Safety Codes

S23

S53

S60

Safety Phrases

■ Do not breathe gas/ fumes/ vapour/ spray.

■ Avoid exposure - obtain special instructions before use.

■ This material and its container must be disposed of as hazardous waste.

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## Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

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NAME	CAS RN	%
ethylene glycol monobutyl ether	111-76-2	<10
ingredients nonhazardous proprietary		>60
hydrocarbon propellant	68476-85-7.	4

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## Section 4 - FIRST AID MEASURES

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### SWALLOWED

- Not considered a normal route of entry.
- If swallowed do NOT induce vomiting.
- If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- Observe the patient carefully.
- Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- Seek medical advice.

### EYE

- If aerosols come in contact with the eyes:
  - Immediately hold the eyelids apart and flush the eye with fresh running water.
  - Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
  - Seek medical attention without delay; if pain persists or recurs seek medical attention.
  - Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

### SKIN

- If solids or aerosol mists are deposited upon the skin:
  - Flush skin and hair with running water (and soap if available).
  - Remove any adhering solids with industrial skin cleansing cream.
  - DO NOT use solvents.
  - Seek medical attention in the event of irritation.

### INHALED

- If aerosols, fumes or combustion products are inhaled:
  - Remove to fresh air.
  - Lay patient down. Keep warm and rested.
  - Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
  - If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
  - Transport to hospital, or doctor.

### NOTES TO PHYSICIAN

- Treat symptomatically.

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## Section 5 - FIRE FIGHTING MEASURES

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### EXTINGUISHING MEDIA

- SMALL FIRE:
  - Water spray, dry chemical or CO2
- LARGE FIRE:
  - Water spray or fog.

### FIRE FIGHTING

- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- If safe, switch off electrical equipment until vapour fire hazard removed.
- Use water delivered as a fine spray to control fire and cool adjacent area.

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Section 5 - FIRE FIGHTING MEASURES

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- DO NOT approach containers suspected to be hot.
  - Cool fire exposed containers with water spray from a protected location.
  - If safe to do so, remove containers from path of fire.
  - Equipment should be thoroughly decontaminated after use.
- When any large container (including road and rail tankers) is involved in a fire, consider evacuation by 100 metres in all directions.

## FIRE/EXPLOSION HAZARD

- Non combustible.
  - Not considered to be a significant fire risk.
  - Heating may cause expansion or decomposition leading to violent rupture of containers.
  - Aerosol cans may explode on exposure to naked flames.
  - Rupturing containers may rocket and scatter burning materials.
  - Hazards may not be restricted to pressure effects.
  - May emit acrid, poisonous or corrosive fumes.
  - Decomposes on heating and may emit toxic fumes of carbon monoxide (CO).
- Decomposition may produce toxic fumes of: carbon dioxide (CO<sub>2</sub>), other pyrolysis products typical of burning organic material.

## FIRE INCOMPATIBILITY

- None known.

## HAZCHEM

2YE

## Personal Protective Equipment

Breathing apparatus.

Gas tight chemical resistant suit.

Limit exposure duration to 1 BA set 30 mins.

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## Section 6 - ACCIDENTAL RELEASE MEASURES

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### MINOR SPILLS

- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Wear protective clothing, impervious gloves and safety glasses.
- Shut off all possible sources of ignition and increase ventilation.
- Wipe up.
- If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated.
- Undamaged cans should be gathered and stowed safely.

### MAJOR SPILLS

- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water courses
- No smoking, naked lights or ignition sources.
- Increase ventilation.
- Stop leak if safe to do so.
- Water spray or fog may be used to disperse / absorb vapour.
- Absorb or cover spill with sand, earth, inert materials or vermiculite.
- If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated.
- Undamaged cans should be gathered and stowed safely.
- Collect residues and seal in labelled drums for disposal.

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Section 6 - ACCIDENTAL RELEASE MEASURES

Personal Protective Equipment advice is contained in Section 8 of the MSDS.

## Section 7 - HANDLING AND STORAGE

### PROCEDURE FOR HANDLING

- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- DO NOT enter confined spaces until atmosphere has been checked.
- Avoid smoking, naked lights or ignition sources.
- Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- DO NOT incinerate or puncture aerosol cans.
- DO NOT spray directly on humans, exposed food or food utensils.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately.
- Use good occupational work practice.
- Observe manufacturer's storing and handling recommendations.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

### SUITABLE CONTAINER

- Aerosol dispenser.
- Check that containers are clearly labelled.

### STORAGE INCOMPATIBILITY

- None known.

### STORAGE REQUIREMENTS

- Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can.
- Store in original containers.
- Store in an upright position.
- DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
- No smoking, naked lights, heat or ignition sources.
- Keep containers securely sealed.
- Contents under pressure.
- Store in a cool, dry, well ventilated area; away from incompatible materials.
- Avoid storage at temperatures higher than 40 deg C.
- Protect containers against physical damage.
- Check regularly for leaks.
- Observe manufacturer's storing and handling recommendations.

## Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

### EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m <sup>3</sup>	STEL ppm	STEL mg/m <sup>3</sup>	Notes
Australia Exposure Standards	ethylene glycol monobutyl ether (2-Butoxyethanol)	20	96.9	50	242	Sk

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## Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

Source	Material	TWA ppm	TWA mg/m <sup>3</sup>	STEL ppm	STEL mg/m <sup>3</sup>	Notes
Australia Exposure Standards	hydrocarbon propellant (LPG (liquified petroleum gas))	1000	1800			

### EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m <sup>3</sup> )	Revised IDLH Value (ppm)
ethylene glycol monobutyl ether		700 [Unch]
hydrocarbon propellant		2,000 [LEL]

### NOTES

Values marked LEL indicate that the IDLH was based on 10% of the lower explosive limit for safety considerations even though the relevant toxicological data indicated that irreversible health effects or impairment of escape existed only at higher concentrations.

### MATERIAL DATA

AEROSOL SPECIALTIES CLEANER:

Not available

ETHYLENE GLYCOL MONOBUTYL ETHER:

- For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL).

Exposed individuals are reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class A or B.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV- TWA for example) is being reached, even when distracted by working activities
B	26- 550	As " A" for 50- 90% of persons being distracted
C	1- 26	As " A" for less than 50% of persons being distracted
D	0.18- 1	10- 50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached

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## Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

E <0.18 As " D" for less than 10% of persons aware of being tested

### PERSONAL PROTECTION

#### EYE

■ No special equipment for minor exposure i.e. when handling small quantities.

OTHERWISE: For potentially moderate or heavy exposures:

- Safety glasses with side shields.
- NOTE: Contact lenses pose a special hazard; soft lenses may absorb irritants and ALL lenses concentrate them.

#### HANDS/FEET

• No special equipment needed when handling small quantities.

• OTHERWISE:

- For potentially moderate exposures:
- Wear general protective gloves, eg. light weight rubber gloves.
- For potentially heavy exposures:
- Wear chemical protective gloves, eg. PVC. and safety footwear.

#### OTHER

■ No special equipment needed when handling small quantities.

OTHERWISE:

- Overalls.
- Skin cleansing cream.
- Eyewash unit.
- Do not spray on hot surfaces.

#### RESPIRATOR

■ Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Breathing Zone Level ppm (volume)	Maximum Protection Factor	Half- face Respirator	Full- Face Respirator
1000	10	AX- AUS	-
1000	50	-	AX- AUS
5000	50	Airline *	-
5000	100	-	AX- 2
10000	100	-	AX- 3
	100+		Airline**

\* - Continuous Flow

\*\* - Continuous-flow or positive pressure demand.

The local concentration of material, quantity and conditions of use determine the type of personal protective equipment required. For further information consult site specific CHEMWATCH data (if available), or your Occupational Health and Safety Advisor.

#### ENGINEERING CONTROLS

■ General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.

Provide adequate ventilation in warehouse or closed storage areas.

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## Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

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### APPEARANCE

■ Supplied as an aerosol pack. Contents under PRESSURE. Contains highly flammable hydrocarbon propellant. Clear liquid with sweet odour; mixes with water.

### PHYSICAL PROPERTIES

Liquid.

Gas.

Mixes with water.

State	Liquid	Molecular Weight	Not Applicable
Melting Range (°C)	Not Available	Viscosity	Not Available
Boiling Range (°C)	<- 18 to 172	Solubility in water (g/L)	Misc ible
Flash Point (°C)	- 87 propellant	pH (1% solution)	Not Availab le
Decomposition Temp (°C)	Not Available	pH (as supplied)	11.4
Autoignition Temp (°C)	Not Available	Vapour Pressure (kPa)	Not Available
Upper Explosive Limit (%)	10.6 propellant	Specific Gravity (water=1)	0.96
Lower Explosive Limit (%)	1.1 propellant	Relative Vapour Density (air=1)	>1
Volatile Component (%vol)	99	Evaporation Rate	>1 Ether = 1
ethylene glycol monobutyl ether			
■ log Kow (Prager 1995):		0.83	
■ log Kow (Sangster 1997):		0.8	

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## Section 10 - CHEMICAL STABILITY AND REACTIVITY INFORMATION

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### CONDITIONS CONTRIBUTING TO INSTABILITY

- Elevated temperatures.
- Presence of open flame.
- Product is considered stable.
- Hazardous polymerisation will not occur.

*For incompatible materials - refer to Section 7 - Handling and Storage.*

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## Section 11 - TOXICOLOGICAL INFORMATION

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### POTENTIAL HEALTH EFFECTS

#### ACUTE HEALTH EFFECTS

##### SWALLOWED

■ Not normally a hazard due to physical form of product.

Ingestion may result in nausea, abdominal irritation, pain and vomiting.

##### EYE

■ The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

##### SKIN

■ The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

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Section 11 - TOXICOLOGICAL INFORMATION

## INHALED

- Mist may be an irritant.

Acute effects from inhalation of high vapour concentrations may be chest and nasal irritation with coughing, sneezing, headache and even nausea.

WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.

## CHRONIC HEALTH EFFECTS

- Long-term exposure to the product is not thought to produce chronic effects adverse to the health (as classified by EC Directives using animal models); nevertheless exposure by all routes should be minimised as a matter of course.

## TOXICITY AND IRRITATION

HYDROCARBON PROPELLANT:

AEROSOL SPECIALTIES CLEANER:

- Not available. Refer to individual constituents.

ETHYLENE GLYCOL MONOBUTYL ETHER:

- unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

### TOXICITY

Oral (rat) LD50: 470 mg/kg

Dermal (rabbit) LD50: 220 mg/kg

Inhalation (human) TClO: 100 ppm

Inhalation (human) TClO: 195 ppm/8h \* [Union Carbide]

Inhalation (Rat) LC50: 450 ppm \*

- The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow.

Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO<sub>2</sub>, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO<sub>2</sub>, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

**Respiratory Effects.** Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

**Cardiovascular Effects.** Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12-24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with

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ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

**Gastrointestinal Effects.** Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

**Musculoskeletal Effects.** Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

**Hepatic Effects.** Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

**Renal Effects.** Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

**Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

**Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

**Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

**Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

**Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

**Genotoxic Effects:** Studies in humans have not addressed the genotoxic effects of ethylene glycol. However,

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available in vivo and in vitro laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m<sup>3</sup>) for EGHE, LC50 > 400ppm (2620 mg/m<sup>3</sup>) for EGBEA to LC50 > 2132 ppm (9061 mg/m<sup>3</sup>) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitizers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE in vitro than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA in vitro and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA in vitro.

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. In vitro cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m<sup>3</sup> and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m<sup>3</sup>), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m<sup>3</sup>), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m<sup>3</sup>) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m<sup>3</sup> (rabbit-EGPE), 100 ppm or 425 mg/m<sup>3</sup> (rat-EGPE), 50 ppm or 241 mg/m<sup>3</sup> (rat EGBE) and 100 ppm or 483 mg/m<sup>3</sup> (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m<sup>3</sup> (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

At least one researcher has stated that the reproductive effects were less than that of other monoalkyl

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Section 11 - TOXICOLOGICAL INFORMATION

ethers of ethylene glycol.

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility. Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes.

## Section 12 - ECOLOGICAL INFORMATION

Refer to data for ingredients, which follows:

ETHYLENE GLYCOL MONOBUTYL ETHER:

HYDROCARBON PROPELLANT:

AEROSOL SPECIALTIES CLEANER:

■ DO NOT discharge into sewer or waterways.

ETHYLENE GLYCOL MONOBUTYL ETHER:

■ Fish LC50 (96hr.) (mg/l):	1490
■ BCF<100:	0.4
■ log Kow (Prager 1995):	0.83
■ log Kow (Sangster 1997):	0.8
■ Half- life Soil - High (hours):	672
■ Half- life Soil - Low (hours):	168
■ Half- life Air - High (hours):	32.8
■ Half- life Air - Low (hours):	3.28
■ Half- life Surface water - High (hours):	672
■ Half- life Surface water - Low (hours):	168
■ Half- life Ground water - High (hours):	1344
■ Half- life Ground water - Low (hours):	336
■ Aqueous biodegradation - Aerobic - High (hours):	672
■ Aqueous biodegradation - Aerobic - Low (hours):	168
■ Aqueous biodegradation - Anaerobic - High (hours):	2688
■ Aqueous biodegradation - Anaerobic - Low (hours):	672
■ Photooxidation half- life air - High (hours):	32.8
■ Photooxidation half- life air - Low (hours):	3.28
■ Fish LC50 (96hr.) (mg/l):	1250- 1650
■ Daphnia magna EC50 (48hr.) (mg/l):	600- 1000

■ For ethylene glycol monoalkyl ethers and their acetates:

Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE)

Environmental fate:

The ethers, like other simple glycol ethers possess no functional groups that are readily subject to

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hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions.

Level III fugacity modeling indicates that category members, when released to air and water, will partition predominately to water and, to a lesser extent, to air and soil. Estimates of soil and sediment partition coefficients (Kocs ranging from 1- 10) suggest that category members would exhibit high soil mobility. Estimated bioconcentration factors (log BCF) range from 0.463 to 0.732. Biodegradation studies indicate that all category members are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment.

Ecotoxicity:

Glycol ether acetates do not hydrolyse rapidly into their corresponding glycol ethers in water under environmental conditions. The LC50 or EC50 values for EGHE are lower than those for EGPE and EGBE (which have shorter chain lengths and lower log Kow values). Overall, the LC50 values for the glycol ethers in aquatic species range from 94 to > 5000 mg/L. For EGHE, the 96-hour LC50 for *Brachydanio rerio* (zebra fish) is between 94 and mg/L, the 48-hour EC50 for *Daphnia magna* was 145 mg/L and the 72-hour EC50 values for biomass and growth rate of algae (*Scenedesmus subspicatus*) were 98 and 198 mg/L, respectively. LC50/EC50 values for EGPE and EGBE in aquatic species are 835 mg/l or greater.

Aquatic toxicity data for EGBEA show a 96-hour LC50 of 28.3 mg/L for rainbow trout (*Oncorhynchus mykiss*), a 48-hour LC50 of 37-143 mg/L for *Daphnia magna*, a 72-hour EC50 of greater than 500 mg/L for biomass or growth rate of algae (*Scenedesmus subspicatus* and *Pseudokirchneriella subcapitata*, respectively), and a 7-day EC10 of 30.4 mg/L and a NOEC of 16.4 mg/L for reproduction in *Ceriodaphnia dubia*.

■ For glycol ethers:

Environmental fate:

Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity:

Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.

Glycols exert a high oxygen demand for decomposition and once released to the environments cause the death of aquatic organisms if dissolved oxygen is depleted.

log Kow: 0.76-0.83

Koc: 67

Half-life (hr) air: 17

Henry's atm m<sup>3</sup>/mol: 2.08E-08

BOD 5 if unstated: 0.71

COD: 2.2

Log BCF: 0.4

Fish LC50 (24 h): 983-1650 mg/L

Fish LC50 (96 h): fathead minnow 1700 mg/L \*\*

Invertebrate toxicity:

cell mult. inhib.91-900mg/L

(*Daphnia*) 48h LC50: >1000 mg/L \*\*

Bioaccumulation: not sig

Effects on algae and plankton: cell mult. inhib.35-900mg/L

Degradation Biological: rapid

processes Abiotic: no hydrol&photonol,RxnOH\*

\*\* [Union Carbide]

HYDROCARBON PROPELLANT:

■ For hydrocarbons:

Environmental fate:

The lower molecular weight hydrocarbons are expected to form a "slick" on the surface of waters after release in calm sea conditions. This is expected to evaporate and enter the atmosphere where it will be degraded through reaction with hydroxy radicals.

Some hydrocarbon will become associated with benthic sediments, and it is likely to be spread over a fairly

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wide area of sea floor. Marine sediments may be either aerobic or anaerobic. The material, in probability, is biodegradable, under aerobic conditions (isomerised olefins and alkenes show variable results). Evidence also suggests that the hydrocarbons may be degradable under anaerobic conditions although such degradation in benthic sediments may be a relatively slow process.

Under aerobic conditions hydrocarbons degrade to water and carbon dioxide, while under anaerobic processes they produce water, methane and carbon dioxide.

Alkenes have low log octanol/water partition coefficients ( $K_{ow}$ ) of about 1 and estimated bioconcentration factors (BCF) of about 10; aromatics have intermediate values (log  $K_{ow}$  values of 2-3 and BCF values of 20-200), while C5 and greater alkanes have fairly high values (log  $K_{ow}$  values of about 3-4.5 and BCF values of 100-1,500).

The estimated volatilisation half-lives for alkanes and benzene, toluene, ethylbenzene, xylene (BTEX) components were predicted as 7 days in ponds, 1.5 days in rivers, and 6 days in lakes. The volatilisation rate of naphthalene and its substituted derivatives were estimated to be slower.

Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds. Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media.

The final products of microbial degradation are carbon dioxide, water, and microbial biomass. The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics, and the aromatics in the C10-C22 range are the most readily biodegradable; n-alkanes, n-alkyl aromatics, and aromatics in the C5-C9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilisation and thus are unavailable in most environments; n-alkanes in the C1-C4 ranges are biodegradable only by a narrow range of specialised hydrocarbon degraders; and n-alkanes, n-alkyl aromatics, and aromatics above C22 are generally not available to degrading microorganisms. Hydrocarbons with condensed ring structures, such as PAHs with four or more rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is, greater than 7.

All biological transformations are affected by temperature. Generally, as the temperature increases, biological activity tends to increase up to a temperature where enzyme denaturation occurs.

Atmospheric fate: Alkanes, isoalkanes, and cycloalkanes have half-lives on the order of 1-10 days, whereas alkenes, cycloalkenes, and substituted benzenes have half-lives of 1 day or less. Photochemical oxidation products include aldehydes, hydroxy compounds, nitro compounds, and peroxyacyl nitrates. Alkenes, certain substituted aromatics, and naphthalene are potentially susceptible to direct photolysis.

Ecotoxicity:

Hydrocarbons are hydrophobic (high log  $K_{ow}$  and low water solubility). Such substances produce toxicity in aquatic organisms by a mechanism referred to as "non-polar narcosis" or "baseline" toxicity. The hydrophobicity increases and water solubility decreases with increasing carbon number for a particular class of hydrocarbon. Substances with the same carbon number show increased hydrophobicity and decreased solubility with increasing saturation. Quantitative structure activity relationships (QSAR), relating both solubility and toxicity to  $K_{ow}$  predict that the water solubility of single chemical substances decreases more rapidly with increasing  $K_{ow}$  than does the acute toxicity.

Based on test results, as well as theoretical considerations, the potential for bioaccumulation may be high. Toxic effects are often observed in species such as blue mussel, daphnia, freshwater green algae, marine copepods and amphipods.

The values of log  $K_{ow}$  for individual hydrocarbons increase with increasing carbon number within homologous series of generic types. Quantitative structure activity relationships (QSAR), relating log  $K_{ow}$  values of single hydrocarbons to toxicity, show that water solubility decreases more rapidly with increasing  $K_{ow}$  than does the concentration causing effects. This relationship varies somewhat with species of hydrocarbon, but it follows that there is a log  $K_{ow}$  limit for hydrocarbons, above which, they will not exhibit acute toxicity; this limit is at a log  $K_{ow}$  value of about 4 to 5. It has been confirmed experimentally that for fish and invertebrates, paraffinic hydrocarbons with a carbon number of 10 or higher (log  $K_{ow} > 5$ ) show no acute toxicity and that alkylbenzenes with a carbon number of 14 or greater (log  $K_{ow} > 5$ ) similarly show no acute toxicity.

QSAR equations for chronic toxicity also suggest that there should be a point where hydrocarbons with high log  $K_{ow}$  values become so insoluble in water that they will not cause chronic toxicity, that is, that there is also a solubility cut-off for chronic toxicity. Thus, paraffinic hydrocarbons with carbon numbers of greater

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than 14 (log Kow >7.3) should show no measurable chronic toxicity. Experimental support for this cut-off is demonstrated by chronic toxicity studies on lubricant base oils and one "heavy" solvent grade (substances composed of paraffins of C20 and greater) which show no effects after exposures to concentrations well above solubility.

The initial criteria for classification of substances as dangerous to the aquatic environment are based upon acute toxicity data in fish, daphnids and algae. However, for substances that have low solubility and show no acute toxicity, the possibility of a long-term or chronic hazard to the environment is recognised in the R53 phrase or so-called "safety net". The R53 assignment for possible long-term harm is a surrogate for chronic toxicity test results and is triggered by substances that are both bioaccumulative and persistent. The indicators of bioaccumulation and persistence are taken as a BCF > 100 (or log Kow > 3 if no BCF data) and lack of ready biodegradability. For low solubility substances which have direct chronic toxicity data demonstrating no chronic toxicity at 1 mg/L or higher, these data take precedence such that no classification for long term toxicity is required.

■ Drinking Water Standards: hydrocarbon total: 10 ug/l (UK max.).

## Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
ethylene glycol monobutyl ether	LOW	LOW	LOW	HIGH

## Section 13 - DISPOSAL CONSIDERATIONS

- Consult State Land Waste Management Authority for disposal.
- Discharge contents of damaged aerosol cans at an approved site.
- Allow small quantities to evaporate.
- DO NOT incinerate or puncture aerosol cans.
- Bury residues and emptied aerosol cans at an approved site.

## Section 14 - TRANSPORTATION INFORMATION



Labels Required: NON-FLAMMABLE COMPRESSED GAS

### HAZCHEM:

2YE (ADG7)

### ADG7:

Class or division	2.2	Subsidiary risk:	None
UN No.:	1950	UN packing group:	None
Special provisions:	63; 190; 277; 327; 344	Packing Instructions:	None
Limited quantities:	See SP 277	Portable tanks and bulk containers - Instructions:	None
Portable tanks and bulk containers - Special provisions:	None	Packagings and IBCs - Packing instruction:	P003; LP02

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Section 14 - TRANSPORTATION INFORMATION

Packagings and IBCs - PP17, PP87, L2

Special packing

provisions:

Name and description: AEROSOLS

## Land Transport UNDG:

Class or division 2.2 Subsidiary risk: None

UN No.: 1950 UN packing group: None

Shipping Name: AEROSOLS

## Air Transport IATA:

ICAO/IATA Class: 2.2 ICAO/IATA Subrisk: None

UN/ID Number: 1950 Packing Group: -

Special provisions: A98

Shipping Name: AEROSOLS, NON-FLAMMABLE

## Maritime Transport IMDG:

IMDG Class: 2 IMDG Subrisk: SP63

UN Number: 1950 Packing Group: None

EMS Number: F- D , S- U Special provisions: 63 190 277 327 959

Limited Quantities: See SP277

Shipping Name: AEROSOLS

## Section 15 - REGULATORY INFORMATION

POISONS SCHEDULE None

## REGULATIONS

### Regulations for ingredients

#### ethylene glycol monobutyl ether (CAS: 111-76-2) is found on the following regulatory lists;

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "International Fragrance Association (IFRA) Survey: Transparency List", "OECD Representative List of High Production Volume (HPV) Chemicals"

#### hydrocarbon propellant (CAS: 68476-85-7, 68476-86-8) is found on the following regulatory lists;

"Australia Exposure Standards", "Australia Hazardous Substances", "Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "OECD Representative List of High Production Volume (HPV) Chemicals"

No data for Aerosol Specialties Cleaner (CW: 4686-76)

## Section 16 - OTHER INFORMATION

### INGREDIENTS WITH MULTIPLE CAS NUMBERS

Ingredient Name	CAS
hydrocarbon propellant	68476- 85- 7, 68476- 86- 8

### REPRODUCTIVE HEALTH GUIDELINES

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
ethylene glycol monobutyl ether	3.6 mg/m3	100	D	NA	-

■ These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified

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Section 16 - OTHER INFORMATION

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otherwise.

CR = Cancer Risk/10000; UF = Uncertainty factor:

TLV believed to be adequate to protect reproductive health:

LOD: Limit of detection

Toxic endpoints have also been identified as:

D = Developmental; R = Reproductive; TC = Transplacental carcinogen

Jankovic J., Drake F.: A Screening Method for Occupational Reproductive

American Industrial Hygiene Association Journal 57: 641-649 (1996).

■ Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

A list of reference resources used to assist the committee may be found at:

[www.chemwatch.net/references](http://www.chemwatch.net/references).

■ The (M)SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

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*This is the end of the MSDS.*