

**JASOL SOLVAKLEEN****Hazard Alert Code:
MODERATE**

Chemwatch Material Safety Data Sheet

Revision No: 4

Chemwatch 51367

Issue Date: 25-Feb-2008

CD 2009/3

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION**PRODUCT NAME**

Jasol Solvakeen

SYNONYMS

"Detsol Spray & Wipe"

PRODUCT USE

Used for the removal of soils and greases from most inert surfaces. General cleaning.

SUPPLIER

Company: Jasol Australia

Address:

41-45 Tarnard Drive

Braeside VIC 3195

Telephone: +613 9580 5722

Emergency Tel: 1 800 629 953

Fax: +613 9580 9902

Company: Jasol New Zealand

Address:

151B Marua Road

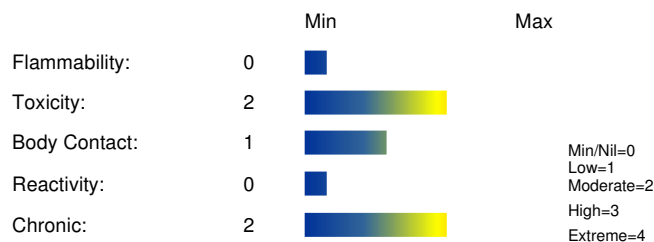
Auckland

Telephone: +64 9580 2105

Emergency Tel: 0800 243 622 (Chemcall)

Emergency Tel: 0800 764 766 (National Poisons Dunedin)

Fax: +64 9571 4388

HAZARD RATINGS**Section 2 - HAZARDS IDENTIFICATION****STATEMENT OF HAZARDOUS NATURE**

NON-HAZARDOUS SUBSTANCE. NON-DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.
--

POISONS SCHEDULE

S5

RISK

- Ingestion may produce health damage*.

* (limited evidence).

SAFETY

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

- Avoid contact with skin.

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
alkaline salts	Not avail.	0-10
ethylene glycol monobutyl ether	111-76-2	0-10
non-hazardous surfactants		0-10
other non-hazardous ingredients		0-10
water	7732-18-5	>60

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Section 4 - FIRST AID MEASURES**SWALLOWED**

-
- 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 this product comes in contact with the eyes:
- Wash out immediately 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.
- 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 skin or hair contact occurs:
- Flush skin and hair with running water (and soap if available).
- Seek medical attention in event of irritation.

INHALED

-
- If fumes or combustion products are inhaled remove from contaminated area.
- Other measures are usually unnecessary.

NOTES TO PHYSICIAN

- Treat symptomatically.
- Followed acute or short term repeated exposures to ethylene glycol monoalkyl ethers and their acetates:
- Hepatic metabolism produces ethylene glycol as a metabolite.
 - Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.
 - Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure. [Ellenhorn and Barceloux: Medical Toxicology]. For acute or short term repeated exposures to ethylene glycol:
 - Early treatment of ingestion is important. Ensure emesis is satisfactory.
 - Test and correct for metabolic acidosis and hypocalcaemia.
 - Apply sustained diuresis when possible with hypertonic mannitol.
 - Evaluate renal status and begin haemodialysis if indicated. [I.L.O]
 - Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
 - Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
 - Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
 - Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
 - Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis. [Ellenhorn and Barceloux: Medical Toxicology] It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures. Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600.

Section 5 - FIRE FIGHTING MEASURES**EXTINGUISHING MEDIA**

- The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas. Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances. In such an event consider:

- foam.
- dry chemical powder.
- carbon dioxide.

FIRE FIGHTING

-
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear breathing apparatus plus protective gloves for fire only.
- Prevent, by any means available, spillage from entering drains or water courses.
- Use fire fighting procedures suitable for surrounding area.
- DO NOT approach containers suspected to be hot.

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- 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.

FIRE/EXPLOSION HAZARD

-
- The material is not readily combustible under normal conditions.
- However, it will break down under fire conditions and the organic component may burn.
- Not considered to be a significant fire risk.
- Heat may cause expansion or decomposition with violent rupture of containers.
- Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).
- May emit acrid smoke.

Decomposes on heating and produces toxic fumes of: carbon dioxide (CO₂), nitrogen oxides (NO_x), other pyrolysis products typical of burning organic material.

May emit poisonous fumes.

FIRE INCOMPATIBILITY

- None known.

HAZCHEM

None

Section 6 - ACCIDENTAL RELEASE MEASURES**EMERGENCY PROCEDURES****MINOR SPILLS**

-
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact by using protective equipment.
- Contain and absorb spill with sand, earth, inert material or vermiculite.
- Wipe up.
- Place in a suitable, labelled container for waste disposal.

MAJOR SPILLS

- Moderate hazard.
- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- Stop leak if safe to do so.
- Contain spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Neutralise/decontaminate residue.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- If contamination of drains or waterways occurs, advise emergency services.

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.
- DO NOT allow material to contact humans, exposed food or food utensils.
- Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- Keep containers securely sealed when not in use.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately. Launder contaminated clothing before re-use.
- 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.

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SUITABLE CONTAINER

-
- Polyethylene or polypropylene container.
- Packing as recommended by manufacturer.
- Check all containers are clearly labelled and free from leaks.

STORAGE INCOMPATIBILITY

- Reacts with aluminium / zinc producing flammable, explosive hydrogen gas.
- Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

STORAGE REQUIREMENTS

-
- Store in original containers.
- Keep containers securely sealed.
- Store in a cool, dry, well-ventilated area.
- Store away from incompatible materials and foodstuff containers.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storing and handling recommendations.

SAFE STORAGE WITH OTHER CLASSIFIED CHEMICALS

X: Must not be stored together

O: May be stored together with specific preventions

+: May be stored together

Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION**EXPOSURE CONTROLS**

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

The following materials had no OELs on our records

- water: CAS:7732-18-5

EMERGENCY EXPOSURE LIMITS

Material	Revised IDLH Value (mg/m ³)	Revised IDLH Value (ppm)
ethylene glycol monobutyl ether		700 [Unch]

ODOUR SAFETY FACTOR (OSF)

OSF=2E2 (2-BUTOXY ETHANOL)

MATERIAL DATA

JASOL SOLVAKLEEN:

- None assigned. Refer to individual constituents.

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

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ETHYLENE GLYCOL MONOBUTYL ETHER:

- Not available. Refer to individual constituents.

WATER:

- 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:

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

PERSONAL PROTECTION**EYE**

- Safety glasses with side shields
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59]

HANDS/FEET

- Suitability and durability of glove type is dependent on usage. Factors such as:
 - frequency and duration of contact,
 - chemical resistance of glove material,
 - glove thickness and
 - dexterity,
 are important in the selection of gloves.
- Wear chemical protective gloves, eg. PVC.
- Wear safety footwear or safety gumboots, eg. Rubber

OTHER

- Overalls.
- P.V.C. apron.
- Barrier cream.
- Skin cleansing cream.
- Eye wash unit.

RESPIRATOR

- Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature

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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	A-AUS P	-
1000	50	-	A-AUS P
5000	50	Airline *	-
5000	100	-	A-2 P
10000	100	-	A-3 P
	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 operating 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. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood - local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Section 9 - PHYSICAL AND CHEMICAL PROPERTIES**APPEARANCE**

A clear to amber mobile alkaline liquid with a characteristic odour; mixes with water.

PHYSICAL PROPERTIES

Liquid.

Mixes with water.

Molecular Weight: Not Applicable	Boiling Range (°C): 100	Melting Range (°C): Not Available
Specific Gravity (water=1): 1.0	Solubility in water (g/L): Miscible	pH (as supplied): 12.2-12.5
pH (1% solution): Not Available	Vapour Pressure (kPa): Not Available	Volatile Component (%vol): >60
Evaporation Rate: Not Available	Relative Vapour Density (air=1): Not Available	Flash Point (°C): Not Applicable
Lower Explosive Limit (%): Not Applicable	Upper Explosive Limit (%): Not Applicable	Autoignition Temp (°C): Not Applicable
Decomposition Temp (°C): Not Available	State: Liquid	Viscosity: Not Available

Material	Value
log Kow	0.76-0.83

Section 10 - CHEMICAL STABILITY

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CONDITIONS CONTRIBUTING TO INSTABILITY

-
- Presence of incompatible materials.
- Product is considered stable.
- Hazardous polymerisation will not occur.

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

Section 11 - TOXICOLOGICAL INFORMATION**POTENTIAL HEALTH EFFECTS****ACUTE HEALTH EFFECTS****SWALLOWED**

- Accidental ingestion of the material may be damaging to the health of the individual. Ingestion may result in nausea, abdominal irritation, pain and vomiting.

EYE

- Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn). 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 is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. 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.

INHALED

- The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Not normally a hazard due to non-volatile nature of product.

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. Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS].

TOXICITY AND IRRITATION

- Not available. Refer to individual constituents.

ALKALINE SALTS:

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

TOXICITY**IRRITATION**

for sodium carbonate:

Oral (rat) LD50: 4090 mg/kg

Skin (rabbit): 500 mg/24h Mild

for potassium carbonate:

Oral (rat) LD50: 1870 mg/kg

for sodium metasilicate:

Oral (rat) LD50: 1153 mg/kg

Skin (human): 250 mg/24h - SEVERE

for trisodium phosphate dodecahydrate

Oral (rat) LD50: 6500 mg/kg Skin (rabbit): FSHA 3.3 / 8.0

Dermal (rat) LD50: 7940 mg/kg Eye (rabbit): FSHA CORROSIVE

ETHYLENE GLYCOL MONOBUTYL ETHER:

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

TOXICITY**IRRITATION**

Oral (rat) LD50: 470 mg/kg

Skin (rabbit): 500 mg, open; Mild

Dermal (rabbit) LD50: 220 mg/kg

Eye (rabbit): 100 mg/24h-Moderate

Inhalation (human) TCLo: 100 ppm

Eye (rabbit): 100 mg SEVERE

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.

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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 CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, 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 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

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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, 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/m³) for EGHE, LC50 > 400ppm (2620 mg/m³) for EGBEA to LC50 > 2132 ppm (9061 mg/m³) 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/m³ and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m³), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m³), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m³) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m³ (rabbit-EGPE), 100 ppm or 425 mg/m³ (rat-EGPE), 50 ppm or 241 mg/m³ (rat EGBE) and 100 ppm or 483 mg/m³ (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m³ (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

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not observed in other species.

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

WATER:

- No significant acute toxicological data identified in literature search.

CARCINOGEN

2-Butoxyethanol International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs Group 3

SKIN

ethylene glycol monobutyl ether

Australia Exposure Standards - Skin

Notes

Sk

Section 12 - ECOLOGICAL INFORMATION

- DO NOT discharge into sewer or waterways.

Refer to data for ingredients, which follows:

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ALKALINE SALTS:

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

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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 th 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 m³ /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&photol,RxnOH* ** [Union Carbide]

WATER:

Ecotoxicity

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
Jasol		No		
Solvakleen		data		
alkaline		No		
salts		data		
ethylene				
glycol		No		
monobutyl		data		
ether				
water		No		
		data		

Section 13 - DISPOSAL CONSIDERATIONS

■ Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:

- Reduction,
- Reuse
- Recycling
- Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- DO NOT allow wash water from cleaning or process equipment to enter drains.

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- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Recycle wherever possible.
- Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- Dispose of by: Burial in a licenced land-fill or incineration in a licenced apparatus (after admixture with suitable combustible material).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

Section 14 - TRANSPORTATION INFORMATION

HAZCHEM: None (ADG6)

NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS: UN, IATA, IMDG

Section 15 - REGULATORY INFORMATION**POISONS SCHEDULE**

S5

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","OECD Representative List of High Production Volume (HPV) Chemicals"

water (CAS: 7732-18-5) is found on the following regulatory lists;

"Australia Inventory of Chemical Substances (AICS)","GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships","IMO IBC Code Chapter 18: List of products to which the Code does not apply","OECD Representative List of High Production Volume (HPV) Chemicals"

No data for Jasol Solvakeen (CW: 51367)

No data for alkaline salts (CAS: , Not avail)

Section 16 - OTHER INFORMATION**REPRODUCTIVE HEALTH GUIDELINES**

Ingredient	ORG	UF	Endpoint	CR	Adeq TLV
ethylene glycol monobutyl ether	3.6 mg/m ³	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 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).

EXPOSURE STANDARD FOR MIXTURES

■ "Worst Case" computer-aided prediction of spray/ mist or fume/ dust components and concentration: ■ Composite Exposure Standard for Mixture (TWA) :100 mg/m³.

■ 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.

■ 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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