Varn UV Deglazer

Hazard Alert Code: MODERATE

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT INFORMATION

Product Name
Varn UV Deglazer

Molecular Weight
Not Applicable

COMPANY IDENTIFICATION
Company: Day International Sdn Bhd
Address:
Lot 30 Jalan Anggerik Mokara 31/59, Seksyen 31,
Kota Kemuning
Selangor Darul Ehsan, 40460
Malaysia
Telephone: +603 5122 4418
Telephone: +603 5121 2950
Fax: +603 5121 8418

CONTACT POINT
POISON CENTRE MALAYSIA
1800 888 099 (office hours)

Section 2 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
ethylene glycol monobutyl ether	111-76-2	>60
isopropanol	67-63-0	10-30

Section 3 - PHYSICAL AND CHEMICAL PROPERTIES

State: Liquid
Solubility in Water (g/L): Miscible
Boiling Point (°C): Not Available
Melting Range (°C): Not Available
Pressure (kPa): 0.6 @ 20 degC
Relative Density (air=1): >1
Specific Gravity (water=1): 0.89
Flash Point (°C): 43.3 (TCC)
Auto Ignition Temperature (°C): Not Available

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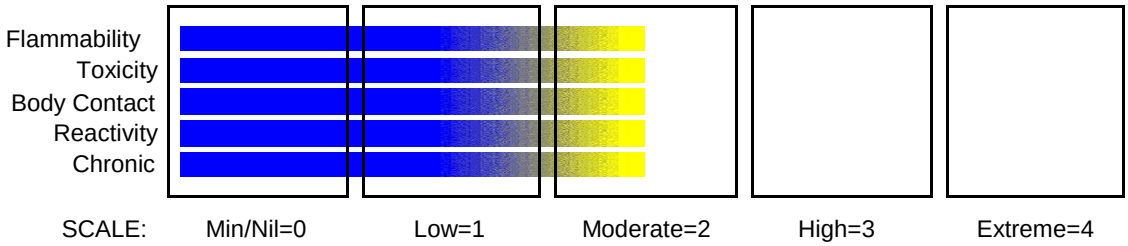
Lower Explosive Limit (%): 1.0
Upper Explosive Limit (%): 13.5
pH(1% solution): Not Available
pH(as supplied): Not Available
Volatile component (%vol): Not Available
Evaporation rate: <1 BuAC = 1 Viscosity:
Not Available

APPEARANCE
Clear liquid with a mild odour; mixes with water.

Section 4 - HAZARD IDENTIFICATION

CHEMWATCH HAZARD RATINGS



RISK

■ Flammable.
■ May form explosive peroxides.
■ Harmful by inhalation, in contact with skin and if swallowed.
■ Irritating to eyes and skin.
■ HARMFUL- May cause lung damage if swallowed.
■ Vapours may cause drowsiness and dizziness.
■ Cumulative effects may result following exposure*.
■ May produce discomfort of the respiratory system*.
■ Limited evidence of a carcinogenic effect*.
■ May be harmful to the foetus/ embryo*.
■ May possibly affect fertility*.

■ Repeated exposure potentially causes skin dryness and cracking*.
* (limited evidence).

SAFETY

• Do not breathe gas/fumes/vapour/spray.
• Avoid contact with skin.
• Avoid contact with eyes.

• Wear suitable protective clothing.
• Wear suitable gloves.

• Wear eye/face protection.

• Handle and open container with care.

• Use only in well ventilated areas.

• Keep container in a well ventilated place.

• Avoid exposure - obtain special instructions before use.
• To clean the floor and all objects contaminated by this material, use water.
• Keep container tightly closed.

• Keep away from food, drink and animal feeding stuffs.
• In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.
• If swallowed, IMMEDIATELY contact Doctor or Poisons Information Centre. (show this container or label).
• This material and its container must be disposed of as hazardous waste.

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POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

■ Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
■ Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.
Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).
■ Severe acute exposure to ethylene glycol monobutyl ether, by ingestion, may cause kidney damage, haemoglobinuria, (blood in urine) and is potentially fatal.
■ Following ingestion, a single exposure to isopropyl alcohol produced lethargy and non-specific effects such as weight loss and irritation. Ingestion of near-lethal doses of isopropanol produces histopathological changes of the stomach, lungs and kidneys, incoordination, lethargy, gastrointestinal tract irritation, and inactivity or anaesthesia.
Swallowing 10 ml. of isopropanol may cause serious injury; 100 ml. may be fatal if not promptly treated. The adult single lethal doses is approximately 250 ml. The toxicity of isopropanol is twice that of ethanol and the symptoms of intoxication appear to be similar except for the absence of an initial euphoric effect; gastritis and vomiting are more prominent. Ingestion may cause nausea, vomiting, and diarrhoea.
There is evidence that a slight tolerance to isopropanol may be acquired.
■ Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols.
Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.
Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality
Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 - Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.
Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in he case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.

EYE

■ Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant

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inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
■ When instilled in rabbit eyes ethylene glycol monobutyl ether produced pain, conjunctival irritation, and transient corneal injury.
■ Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage. Eye contact may cause tearing or blurring of vision.

SKIN

■ Skin contact with the material may be harmful; systemic effects may result following absorption.
■ The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either
- produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or
- produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period.

Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.
■ Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.
■ Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.
■ Open cuts, abraded or irritated skin should not be exposed to this material.
■ Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds 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.
■ Ethylene glycol monobutyl ether (2-butoxyethanol) penetrates the skin easily and toxic effects via this route may be more likely than by inhalation. Percutaneous uptake rate in the guinea pig was estimated to be 0.25 umole/min/cm2.

INHALED

■ Limited evidence exists, or practical experience predicts, that the material produces irritation of the respiratory system in a significant number of individuals following inhalation.
■ Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.
■ Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.
■ Inhalation hazard is increased at higher temperatures.
■ Ethylene glycol monobutyl ether (2-butoxyethanol) and its metabolite butoxyacetic acid are haemolytic agents, causing red blood cell destruction.
On the basis of industrial experience and volunteer short-term exposure humans are shown to be less

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susceptible than experimental animals to exposure. In 8-hour exposures at concentrations of 200 or 100 ppm no objective effects were seen other than raised urinary excretion of the metabolite butoxyacetic acid. No increased osmotic fragility of the red blood cell is observed. Subjectively these concentrations were uncomfortable with mild eye, nose and throat irritation occurring. No clinical signs of adverse effects nor subjective complaints were produced when male volunteers were exposed for 2 hours to 20 ppm during light physical exercise. Other studies have established that the most sensitive indicators of toxic effect observed from many of the glycol ethers is an increase in erythrocyte osmotic fragility in rats. This appears to be related to the development of haemoglobinuria at higher exposure levels.
■ The odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal.
■ Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination.
■ Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.

CHRONIC HEALTH EFFECTS
■ On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.
Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure.
Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain.

Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals.

There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol.

Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects.
NOTE: Commercial isopropanol does not contain "isopropyl oil". An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct "isopropyl oil". Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.
Ethylene glycol esters and their ethers cause wasting of the testicles, reproductive changes, infertility and changes to kidney function. Shorter chain compounds are more dangerous. They are also associated with the formation of stones in the urine.

Section 5 - 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.

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- 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.
- Avoid giving milk or oils.
- Avoid giving alcohol.
- If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

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.
- 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 skin contact occurs:
- Immediately remove all contaminated clothing, including footwear.
- 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.
- 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.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- Transport to hospital, or doctor.

ADVICE TO DOCTOR

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.
for poisons (where specific treatment regime is absent):

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 L/min.
- Monitor and treat, where necessary, for pulmonary oedema .
- Monitor and treat, where necessary, for shock.
- Anticipate seizures .
- DO NOT use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

ADVANCED TREATMENT

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- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

BRONSTEIN, A.C. and CURRANCE, P.L.
EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994.

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.

For acute or short term repeated exposures to isopropanol:

- Rapid onset respiratory depression and hypotension indicates serious ingestions that require careful cardiac and respiratory monitoring together with immediate intravenous access.
- Rapid absorption precludes the usefulness of emesis or lavage 2 hours post-ingestion. Activated charcoal and cathartics are not clinically useful. Ipecac is most useful when given 30 mins. post-ingestion.
- There are no antidotes.
- Management is supportive. Treat hypotension with fluids followed by vasopressors.
- Watch closely, within the first few hours for respiratory depression; follow arterial blood gases and tidal volumes.
- Ice water lavage and serial haemoglobin levels are indicated for those patients with evidence of gastrointestinal bleeding.

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Section 6 - FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA

- Alcohol stable foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

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.
- Avoid spraying water onto liquid pools.
- 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.

When any large container (including road and rail tankers) is involved in a fire,
consider evacuation by 500 metres in all directions.

FIRE/EXPLOSION HAZARD

- Liquid and vapour are flammable.
- Moderate fire hazard when exposed to heat or flame.
- Vapour forms an explosive mixture with air.
- Moderate explosion hazard when exposed to heat or flame.
- Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO).

Combustion products include: carbon dioxide (CO_2), other pyrolysis products typical of burning organic material.
WARNING: Long standing in contact with air and light may result in the formation
of potentially explosive peroxides.

FIRE INCOMPATIBILITY

- Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result.

Section 7 - ACCIDENTAL RELEASE MEASURES

Spills & Disposal:
Eliminate ignition sources.
Prevent from entering drains.
Contain spillage by any means.
To clean the floor and all objects contaminated by this material, use water.

MINOR SPILLS

- Remove all ignition sources.
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.

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- Control personal contact with the substance, by using protective equipment.
- Contain and absorb small quantities with vermiculite or other absorbent material.
- Wipe up.
- Collect residues in a flammable waste container.

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 course.
- Consider evacuation (or protect in place).
- 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.
- Contain spill with sand, earth or vermiculite.
- Use only spark-free shovels and explosion proof equipment.
- Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite.
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

Section 8 - HANDLING AND STORAGE

Keep container tightly closed.
Keep container in a well ventilated place.
Keep away from food, drink and animal feeding stuffs.
Handle and open container with care.
Store in cool, dry, protected area.
Restrictions on Storage apply.
Refer to Full Report.

SUITABLE CONTAINER

- DO NOT use aluminium or galvanised containers.
- Packing as supplied by manufacturer.
- Plastic containers may only be used if approved for flammable liquid.
- Check that containers are clearly labelled and free from leaks.
- For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure.
- For materials with a viscosity of at least 2680 cSt. (23 deg. C)
- For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)
- Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C)

(i) : Removable head packaging;
(ii) : Cans with friction closures and
(iii) : low pressure tubes and cartridges may be used.

- Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages
- In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.

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STORAGE INCOMPATIBILITY

Ethylene glycol monobutyl ether (2-butoxyethanol) and its acetate:

- May form unstable peroxides in storage
- is incompatible with oxidisers, permanganates, peroxides, ammonium persulfate, bromine dioxide, nitrates, strong acids, sulfuric acid, nitric acid, perchloric acid.

Alcohols

- are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents.
- reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen
- react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium
- should not be heated above 49 deg. C. when in contact with aluminium equipment.
- Glycol ethers may form peroxides under certain conditions; the potential for peroxide formation is enhanced when these substances are used in processes such as distillation where they are concentrated or even evaporated to near-dryness or dryness; storage under a nitrogen atmosphere is recommended to minimise the possible formation of highly reactive peroxides
- Nitrogen blanketing is recommended if transported in containers at temperatures within 15 deg C of the flash-point and at or above the flash-point - large containers may first need to be purged and inerted with nitrogen prior to loading
- In the presence of strong bases or the salts of strong bases, at elevated temperatures, the potential exists for runaway reactions.
- Contact with aluminium should be avoided; release of hydrogen gas may result- glycol ethers will corrode scratched aluminium surfaces.
- May discolour in mild steel/ copper; lined containers, glass or stainless steel is preferred
- Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water . Investigation of the hazards associated with use of 2-butoxyethanol for alloy electropolishing showed that mixtures with 50-95% of acid at 20 deg C, or 40-90% at 75 C, were explosive and initiable by sparks. Sparking caused mixtures with 40-50% of acid to become explosive, but 30% solutions appeared safe under static conditions of temperature and concentration.

Secondary alcohols and some branched primary alcohols may produce potentially explosive peroxides after exposure to light and/ or heat.

STORAGE REQUIREMENTS

- Store in original containers in approved flammable liquid storage area.
- Store away from incompatible materials in a cool, dry, well-ventilated area.
- DO NOT store in pits, depressions, basements or areas where vapours may be trapped.
- No smoking, naked lights, heat or ignition sources.
- Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access.
- Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.
- Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
- Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors.
- Keep adsorbents for leaks and spills readily available.
- Protect containers against physical damage and check regularly for leaks.
- Observe manufacturer's storage and handling recommendations contained within this MSDS.

In addition, for tank storages (where appropriate):

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- Store in grounded, properly designed and approved vessels and away from incompatible materials.
- For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.
- Storage tanks should be above ground and diked to hold entire contents.

TRANSPORTATION

Class 3 - Flammable liquids shall not be loaded in the same vehicle or packed in the same vehicle or packed in the same freight container with:
Class 1 - Explosives;
Class 2.1 - Flammable gases (where both flammable liquids and flammable gases are in bulk);
Class 2.3 - Poisonous gases;
Class 4.2 - Spontaneously combustible substances;
Class 5.1 - Oxidising agents;
Class 5.2 - Organic peroxides;
Class 7 - Radioactive substances.

SAFE STORAGE WITH OTHER CLASSIFIED CHEMICALS

+	X	+	X	X	+

+: May be stored together
O: May be stored together with specific preventions
X: Must not be stored together

Section 9 - EXPOSURE CONTROLS AND PERSONAL PROTECTION

EXPOSURE CONTROLS

Source	Material	TWA ppm	TWA mg/m³	STEL ppm	STEL mg/m³	Peak ppm	Peak mg/m³	TWA F/CC	Notes
Singapore Permissible Exposure Limits of Toxic Substances	(2-Butoxyethanol (EGBE))	25	121						
Malaysia Permissible Exposure Limits	(2-Butoxyethanol (EGBE))	20	96.7						skin
Singapore Permissible Exposure Limits of Toxic Substances	(Isopropyl alcohol)	400	983	1230	500				
Malaysia Permissible Exposure Limits	(Isopropyl alcohol)	400	983						

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Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.

Engineering Controls:
Local Exhaust Ventilation recommended.

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], [AS/NZS 1336 or national equivalent].

HANDS/FEET
- Wear chemical protective gloves, eg. PVC.
- Wear safety footwear or safety gumboots, eg. Rubber.

The selection of the suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and
has to be observed when making a final choice.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Contaminated gloves should be replaced.

continued...

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

OTHER

- Overalls.
- PVC Apron.
- PVC protective suit may be required if exposure severe.
- Eyewash unit.
- Ensure there is ready access to a safety shower.
- Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.
- For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets), non sparking safety footwear.

RESPIRATOR

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.

Section 10 - STABILITY

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

For incompatible materials - refer to Section 8 - Handling and Storage.

Section 11 - TOXICOLOGICAL INFORMATION

TOXICITY AND IRRITATION

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

■ The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

:

■ Not available. Refer to individual constituents.

ETHYLENE GLYCOL MONOBUTYL ETHER:

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 *
Oral (Rat) LD50: 300 mg/kg **
Dermal (Guinea pig) LD50: 210 mg/kg **
Inhalation (Rat) LC50: 2210 mg/m³ **

IRRITATION
Skin (rabbit): 500 mg, open; Mild
Eye (rabbit): 100 mg/24h- Moderate
Eye (rabbit): 100 mg SEVERE

■ The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or

continued...

prolonged exposure to irritants may produce conjunctivitis.
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 sensitisers 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 cytogenicity 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

continued...

ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity 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 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.
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_2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO_2, 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

continued...

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

ISOPROPANOL:

TOXICITY
Oral (human) LDLo: 3570 mg/kg
Oral (human) TDLo: 223 mg/kg
Oral (man) TDLo: 14432 mg/kg
Oral (rat) LD50: 5045 mg/kg
Dermal (rabbit) LD50: 12800 mg/kg
Oral (Human) TDLo: 14432 mg/kg
Oral (Human) LD: 5272 mg/kg
Oral (Human) LD: 3570 mg/kg
Intraperitoneal (Rat) LD50: 2735 mg/kg
Intravenous (Rat) LD50: 1088 mg/kg
Oral (Mouse) LD50: 3600 mg/kg
Intraperitoneal (Mouse) LD50: 4477 mg/kg
Intravenous (Mouse) LD50: 1509 mg/kg
Oral (Dog) LD: 1537 mg/kg
Intravenous (Dog) LD: 1024 mg/kg
Intravenous (Cat) LD: 1963 mg/kg
Oral (Rabbit) LD50: 6410 mg/kg
Intraperitoneal (Rabbit) LD50: 667 mg/kg
Intravenous (Rabbit) LD50: 1184 mg/kg
Intraperitoneal (Guinea pig) LD50: 2560 mg/kg
Inhalation (Mouse) LC50: 53000 mg/m^3/4h
Oral (Rat) LD50: 5000 mg/kg
Intraperitoneal (Rat) TDLo: 800 mg/kg
Inhalation (Rat) LC50: 72600 mg/m^3/4h
Oral (Human) TDLo: 286 mg/kg
Inhalation (Human) TCLo: 35 ppm/4h
Inhalation (Human) TCLo: 150 ppm/2h

IRRITATION
Skin (rabbit): 500 mg - Mild
Eye (rabbit): 10 mg - Moderate
Eye (rabbit): 100mg/24hr- Moderate
Eye (rabbit): 100 mg - SEVERE

■ For isopropanol (IPA):
Acute toxicity: Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat.
Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred.
Repeat dose studies: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified
from these studies were to the kidney.
Reproductive toxicity: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of

continued...

the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.
Developmental toxicity: The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity
Genotoxicity: All genotoxicity assays reported for isopropanol have been negative
Carcinogenicity: rodent inhalation studies were conduct to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment.
The substance is classified by IARC as Group 3:
NOT classifiable as to its carcinogenicity to humans.
Evidence of carcinogenicity may be inadequate or limited in animal testing.

CARCINOGEN

International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group	3
International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs	Group	1

SENSITISER

IUCLID Photodegradation Data	Sensitiser	NO3
IUCLID Photodegradation Data	Sensitiser	OH
IUCLID Photodegradation Data	Sensitiser	

SKIN

Malaysia Permissible Exposure Limits - Skin	Notes	skin
GESAMP/EHS Composite List - GESAMP Hazard Profiles	D1: skin irritation/corrosion	1
GESAMP/EHS Composite List - GESAMP Hazard Profiles	D1: skin irritation/corrosion	0

continued...

Section 12 - ECOLOGICAL INFORMATION

ISOPROPANOL:
ETHYLENE GLYCOL MONOBUTYL ETHER:
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 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:

continued...

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^3 /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]

ISOPROPANOL:

log Kow (Sangster 1997):	0.05
log Pow (Verschueren 1983):	- 0.5714285
BOD5:	60%
BOD20:	78%
COD:	2.23
ThOD:	2.4
Half- life Soil - High (hours):	168
Half- life Soil - Low (hours):	24
Half- life Air - High (hours):	72
Half- life Air - Low (hours):	6.2
Half- life Surface water - High (hours):	168
Half- life Surface water - Low (hours):	24
Half- life Ground water - High (hours):	336
Half- life Ground water - Low (hours):	48
Aqueous biodegradation - Aerobic - High (hours):	168
Aqueous biodegradation - Aerobic - Low (hours):	24
Aqueous biodegradation - Anaerobic - High (hours):	672
Aqueous biodegradation - Anaerobic - Low (hours):	96
Photooxidation half- life water - High (hours):	1.90E+05
Photooxidation half- life water - Low (hours):	4728
Photooxidation half- life air - High (hours):	72
Photooxidation half- life air - Low (hours):	6.2

For isopropanol (IPA):

continued...

log Kow : -0.16- 0.28
Half-life (hr) air : 33-84
Half-life (hr) H2O surface water : 130
Henry's atm m3 /mol: 8.07E-06
BOD 5: 1.19,60%
COD : 1.61-2.30,97%
ThOD : 2.4
BOD 20: >70% * [Akzo Nobel]
Environmental Fate
Based on calculated results from a lever 1 fugacity model,IPA is expected to partition primarily to the aquatic compartment (77.7%) with the remainder to the air (22.3%). IPA has been shown to biodegrade rapidly in aerobic, aqueous biodegradation tests and therefore, would not be expected to persist in aquatic habitats. IPA is also not expected to persist in surface soils due to rapid evaporation to the air. In the air, physical degradation will occur rapidly due to hydroxy
radical (OH) attack. Overall, IPA presents a low potential hazard to aquatic or terrestrial biota.
IPA is expected to volatilise slowly from water based on a calculated Henry's Law constant of 7.52 x 10 -6 atm.m 3 /mole. The calculated half-life for the volatilisation from surface water (1 meter depth) is predicted to range from 4 days (from a river) to 31 days (from a lake). Hydrolysis is not considered a significant degradation process for IPA. However, aerobic biodegradation of IPA has been shown to occur rapidly under non-acclimated conditions, based on a result of 49% biodegradation from a 5 day BOD test. Additional biodegradation data developed using standardized test methods show that IPA is readily biodegradable in both freshwater and saltwater media (72 to 78% biodegradation in 20 days).
IPA will evaporate quickly from soil due to its high vapor pressure (43 hPa at 20°C), and is not expected to partition to the soil based on a calculated soil adsorption coefficient (log Koc) of 0.03.
IPA has the potential to leach through the soil due to its low soil adsorption
In the air, isopropanol is subject to oxidation predominantly by hydroxy radical attack. The room temperature rate constants determined by several investigators are in good agreement for the reaction of IPA with hydroxy radicals. The atmospheric half-life is expected to be 10 to 25 hours, based on measured degradation rates ranging from 5.1 to 7.1 x 10 -12 cm3 /molecule-sec, and an OH concentration of 1.5 x 106 molecule/cm3 , which is a commonly used default value for calculating atmospheric half-lives. Using OH concentrations representative of polluted (3 x 106) and pristine (3 x 105) air, the atmospheric half-life of IPA would range from 9 to 126 hours, respectively. Direct photolysis is not expected to be an important transformation process for the degradation of IPA.
Ecotoxicity:
IPA has been shown to have a low order of acute aquatic toxicity. Results from 24- to 96-hour LC50 studies range from 1,400 to more than 10,000 mg/L for freshwater and saltwater fish and invertebrates. In addition, 16-hour to 8-day toxicity threshold levels (equivalent to 3% inhibition in cell growth) ranging from 104 to 4, 930 mg/L have been demonstrated for various microorganisms.
Chronic aquatic toxicity has also been shown to be of low concern, based on 16- to 21-day NOEC values of 141 to 30 mg/L, respectively, for a freshwater invertebrate. Bioconcentration of IPA in aquatic organisms is not expected to occur based on a measured log octanol/water partition coefficient (log Kow) of 0.05, a calculated bioconcentration factor of 1 for a freshwater fish, and the unlikelihood of constant, long-term exposures.
Toxicity to Plants
Toxicity of IPA to plants is expected to be low, based on a 7-day toxicity threshold value of 1,800 mg/L for a freshwater algae, and an EC50 value of 2,100 mg/L from a lettuce seed germination test.

Ecotoxicity

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

continued...

Section 13 - DISPOSAL INFORMATION

Eliminate ignition sources.
Prevent from entering drains.
Contain spillage by any means.
To clean the floor and all objects contaminated by this material, use water.
• Containers may still present a chemical hazard/ danger when empty.
• Return to supplier for reuse/ recycling if possible.
Otherwise:
• If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
• Where possible retain label warnings and MSDS and observe all notices pertaining to the product.
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.
• 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 land-fill specifically licenced to accept chemical and / or pharmaceutical wastes 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

Shipping Class	FLAMMABLE LIQUID N.O.S. (contains isopropanol)
Hazard Name	3, None
UN/NA Number	1993
Packing Class	III
Labels Required	flammable liquid

continued...

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International Transport Regulations	
IMO	1993
IMDG Page Number	3

Air Transport IATA:

ICAO/IATA Class:	3	ICAO/IATA Subrisk:	None
UN/ID Number:	1993	Packing Group:	III
Special provisions:	A3		
Cargo Only			
Packing Instructions:	366	Maximum Qty/Pack:	220 L
Passenger and Cargo		Passenger and Cargo	
Packing Instructions:	355	Maximum Qty/Pack:	60 L
Passenger and Cargo Limited Quantity		Passenger and Cargo Limited Quantity	
Packing Instructions:	Y344	Maximum Qty/Pack:	10 L

Shipping name:FLAMMABLE LIQUID, N.O.S.(contains isopropanol)

Maritime Transport IMDG:

IMDG Class:	3	IMDG Subrisk:	None
UN Number:	1993	Packing Group:	III
EMS Number:	F-E,S-E	Special provisions:	223 274 955
Limited Quantities:	5 L		

Shipping name:FLAMMABLE LIQUID, N.O.S.(contains isopropanol)

Section 15 - REGULATORY INFORMATION

RISK

Risk Codes	Risk Phrases
R10	• Flammable.
R19	• May form explosive peroxides.
R20/21/22	• Harmful by inhalation, in contact with skin and if swallowed.
R36/38	• Irritating to eyes and skin.
R65	• HARMFUL- May cause lung damage if swallowed.
R67	• Vapours may cause drowsiness and dizziness.
R33?	• Cumulative effects may result following exposure*.
R37?	• May produce discomfort of the respiratory system*.
R40(3)?	• Limited evidence of a carcinogenic effect*.
R61?	• May be harmful to the foetus/ embryo*.
R62?	• May possibly affect fertility*.
R66?	• Repeated exposure potentially causes skin dryness and cracking*.

continued...

SAFETY

Safety Codes	Safety Phrases
S23	Do not breathe gas/fumes/vapour/spray.
S24	Avoid contact with skin.
S25	Avoid contact with eyes.
S36	Wear suitable protective clothing.
S37	Wear suitable gloves.
S39	Wear eye/face protection.
S18	Handle and open container with care.
S51	Use only in well ventilated areas.
S09	Keep container in a well ventilated place.
S53	Avoid exposure - obtain special instructions before use.
S40	To clean the floor and all objects contaminated by this material, use water.
S07	Keep container tightly closed.
S13	Keep away from food, drink and animal feeding stuffs.
S26	In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.
S46	If swallowed, IMMEDIATELY contact Doctor or Poisons Information Centre (show this container or label).
S60	This material and its container must be disposed of as hazardous waste.

REGULATIONS

Regulations for ingredients

ethylene glycol monobutyl ether (CAS: 111-76-2) is found on the following regulatory lists;
"ASEAN Cosmetic Directive Annex III – Part 1 List of substances which cosmetic products must not contain except subject to restrictions and conditions laid down", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "IMO IBC Code Chapter 17: Summary of minimum requirements", "IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances", "IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "International Fragrance Association (IFRA) Survey: Transparency List", "Malaysia Occupational Safety and Health Act - Chemicals for which medical surveillance is appropriate", "Malaysia Permissible Exposure Limits", "OECD List of High Production Volume (HPV) Chemicals", "OSPAR National List of Candidates for Substitution – Norway", "Singapore Odour Thresholds and Irritation Concentration of Chemicals", "Singapore Permissible Exposure Limits of Toxic Substances"

isopropanol (CAS: 67-63-0) is found on the following regulatory lists;
"GESAMP/EHS Composite List - GESAMP Hazard Profiles", "IMO IBC Code Chapter 17: Summary of minimum requirements", "IMO IBC Code Chapter 18: List of products to which the Code does not apply", "IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances", "IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only mixtures containing at least 99% by weight of components already assessed by IMO", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "International Fragrance Association (IFRA) Survey: Transparency List", "Malaysia Food Regulations - Permitted Food Conditioners", "Malaysia Permissible Exposure Limits", "OECD List of High Production Volume (HPV) Chemicals", "OSPAR National List of Candidates for Substitution – Norway", "Singapore Fire Safety (Petroleum and Flammable Materials) - Licensable Products - Flammable Materials", "Singapore Odour Thresholds and Irritation Concentration of Chemicals", "Singapore Permissible Exposure Limits of Toxic Substances"

continued...

No data for Varn UV Deglazer (CW: 6634-83)

Section 16 - OTHER INFORMATION

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 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.

■ 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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