



UltraColor Red Rubber Stamp Ink

UltraColor Products

Chemwatch: 47240

Version No: 4.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: 21/02/2013

Print Date: 08/10/2015

Initial Date: Not Available

L.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	UltraColor Red RS Ink
Synonyms	Manufacturer's Code 3020
Proper shipping name	FLAMMABLE LIQUID, N.O.S. (contains ethanol)
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Rubber stamp ink.
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Details of the supplier of the safety data sheet

Registered company name	Zeus Chemical Products
Address	3 Anderson Place South Windsor 2756 NSW Australia
Telephone	+61 2 4577 4866
Fax	+61 2 4577 6919
Website	www.ultracolor.com.au
Email	sales@ultracolor.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	+61 2 4577 4866 (Mon-Fri, 8am-5pm)
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the Model WHS Regulations and the ADG Code.

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	2	
Toxicity	2	
Body Contact	2	
Reactivity	1	
Chronic	2	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
GHS Classification [1]	Flammable Liquid Category 3, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Carcinogen Category 2, STOT - SE (Resp. Irr.) Category 3, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2

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Legend:

1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	   
SIGNAL WORD	WARNING

Hazard statement(s)

H226	Flammable liquid and vapour
H302	Harmful if swallowed
H315	Causes skin irritation
H319	Causes serious eye irritation
H351	Suspected of causing cancer
H335	May cause respiratory irritation
H401	Toxic to aquatic life
H411	Toxic to aquatic life with long lasting effects

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P271	Use only outdoors or in a well-ventilated area.
P281	Use personal protective equipment as required.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P362	Take off contaminated clothing.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

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Precautionary statement(s) Disposal

P501 Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64-17-5	30-60	ethanol
107-21-1	10-30	ethylene glycol
56-81-5	10-30	glycerol
112-34-5	1-10	diethylene glycol monobutyl ether
989-38-8	1-10	C.I. Basic Red 1
2465-27-2	<1	C.I. Basic Yellow 2
7732-18-5	1-10	water
Not Available	NotSpec.	NOTE: Manufacturer has supplied full ingredient
Not Available	NotSpec.	information to allow CHEMWATCH assessment.

NOTE: Manufacturer has supplied full ingredient information to allow CHEMWATCH assessment.

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> 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 Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> 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.
Inhalation	<ul style="list-style-type: none"> 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, without delay.
Ingestion	<ul style="list-style-type: none"> 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. Avoid giving milk or oils. Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

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.

As in all cases of suspected poisoning, follow the ABCDEs of emergency medicine (airway, breathing, circulation, disability, exposure), then the ABCDEs of toxicology (antidotes, basics, change absorption, change distribution, change elimination).

For poisons (where specific treatment regime is absent):

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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

- 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

Treat symptomatically.

For acute or short term repeated exposures to ethanol:

- Acute ingestion in non-tolerant patients usually responds to supportive care with special attention to prevention of aspiration, replacement of fluid and correction of nutritional deficiencies (magnesium, thiamine pyridoxine, Vitamins C and K).
- Give 50% dextrose (50-100 ml) IV to obtunded patients following blood draw for glucose determination.
- Comatose patients should be treated with initial attention to airway, breathing, circulation and drugs of immediate importance (glucose, thiamine).
- Decontamination is probably unnecessary more than 1 hour after a single observed ingestion. Cathartics and charcoal may be given but are probably not effective in single ingestions.
- Fructose administration is contra-indicated due to side effects.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- Alcohol stable foam.
 - Dry chemical powder.
 - BCF (where regulations permit).
 - Carbon dioxide.
 - Water spray or fog - Large fires only.
- Do not use** a water jet to fight fire.

Special hazards arising from the substrate or mixture

Fire Incompatibility

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

Advice for firefighters

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.

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 (CO₂), carbon monoxide (CO), acrolein, hydrogen chloride, phosgene, nitrogen oxides (NO_x), other pyrolysis products typical of burning organic material

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	<ul style="list-style-type: none">Remove all ignition sources.Clean up all spills immediately.Avoid breathing vapours and contact with skin and eyes.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. <p>Slippery when spilt.</p>																													
Major Spills	Chemical Class: alcohols and glycols For release onto land: recommended sorbents listed in order of priority.																													
	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS																									
	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS																									
	LAND SPILL - SMALL																													
	cross-linked polymer - particulate	1	shovel	shovel	R, W, SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R,I, P	wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT	treated wood fiber - pillow	3	throw	pitchfork	DGC, RT	foamed glass - pillow	4	throw	pichfork	R, P, DGC, RT
	cross-linked polymer - particulate	1	shovel	shovel	R, W, SS																									
	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT																									
	sorbent clay - particulate	2	shovel	shovel	R,I, P																									
	wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT																									
	treated wood fiber - pillow	3	throw	pitchfork	DGC, RT																									
foamed glass - pillow	4	throw	pichfork	R, P, DGC, RT																										
LAND SPILL - MEDIUM																														
cross-linked polymer - particulate	1	blower	skiploader	R,W, SS	polypropylene - particulate	2	blower	skiploader	W, SS, DGC	sorbent clay - particulate	2	blower	skiploader	R, I, W, P, DGC	polypropylene - mat	3	throw	skiploader	DGC, RT	expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC	polyurethane - mat	4	throw	skiploader	DGC, RT	
cross-linked polymer - particulate	1	blower	skiploader	R,W, SS																										
polypropylene - particulate	2	blower	skiploader	W, SS, DGC																										
sorbent clay - particulate	2	blower	skiploader	R, I, W, P, DGC																										
polypropylene - mat	3	throw	skiploader	DGC, RT																										
expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC																										
polyurethane - mat	4	throw	skiploader	DGC, RT																										
Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988 Slippery when spilt.																														
<ul style="list-style-type: none">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.																														
Personal Protective Equipment advice is contained in Section 8 of the SDS.																														

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SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> Containers, even those that have been emptied, may contain explosive vapours. Do NOT cut, drill, grind, weld or perform similar operations on or near containers. DO NOT allow clothing wet with material to stay in contact with skin Avoid all personal contact, including inhalation. Wear protective clothing when risk of overexposure occurs. Use in a well-ventilated area. Prevent concentration in hollows and sumps. DO NOT enter confined spaces until atmosphere has been checked. Avoid smoking, naked lights or ignition sources. Avoid generation of static electricity. DO NOT use plastic buckets. Earth all lines and equipment. Use spark-free tools when handling. 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. Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<ul style="list-style-type: none"> 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 SDS. <p>In addition, for tank storages (where appropriate):</p> <ul style="list-style-type: none"> 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.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> 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.
Storage incompatibility	<p>Alcohols</p> <ul style="list-style-type: none"> 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

Continued...

▸ Avoid strong acids, bases.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION**Control parameters****OCCUPATIONAL EXPOSURE LIMITS (OEL)****INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	ethanol	Ethyl alcohol	1880 mg/m3 / 1000 ppm	Not Available	Not Available	Not Available
Australia Exposure Standards	ethylene glycol	Ethylene glycol (particulate) / Ethylene glycol (vapour)	10 mg/m3 / 52 mg/m3 / 20 ppm	104 mg/m3 / 40 ppm	Not Available	Sk
Australia Exposure Standards	glycerol	Glycerin mist (a)	10 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
ethanol	Ethyl alcohol; (Ethanol)	Not Available	Not Available	Not Available
ethylene glycol	Ethylene glycol	10 ppm	40 ppm	60 ppm
glycerol	Glycerine (mist); (Glycerol; Glycerin)	30 mg/m3	310 mg/m3	2500 mg/m3
diethylene glycol monobutyl ether	Butoxyethoxyethanol, 2-(2-; (Diethylene glycol monobutyl ether)	10 ppm	10 ppm	170 ppm
C.I. Basic Red 1	Basic Red 1; (Rhodamine 6G extra base)	0.0081 mg/m3	0.089 mg/m3	1.3 mg/m3
C.I. Basic Yellow 2	Auramine; (4,4'-(Imidocarbonyl)bis(N,N-dimethylamine) monohydrochloride)	0.49 mg/m3	5.4 mg/m3	53 mg/m3

Ingredient	Original IDLH	Revised IDLH
ethanol	15,000 ppm	3,300 [LEL] ppm
ethylene glycol	Not Available	Not Available
glycerol	Not Available	Not Available
diethylene glycol monobutyl ether	Not Available	Not Available
C.I. Basic Red 1	Not Available	Not Available
C.I. Basic Yellow 2	Not Available	Not Available
water	Not Available	Not Available
NOTE: Manufacturer has supplied full ingredient	Not Available	Not Available
information to allow CHEMWATCH assessment.	Not Available	Not Available

MATERIAL DATA

for ethylene glycol:

Odour Threshold: 25 ppm

NOTE: Detector tubes for ethylene glycol, measuring in excess of 10 mg/m3, are commercially available.

It appears impractical to establish separate TLVs for ethylene glycol vapour and mists. Atmospheric concentration that do not cause discomfort are unlikely to cause adverse effects. The TLV-C is thought to be protective against throat and respiratory irritation and headache reported in exposed humans. NIOSH has not established a limit for this substance due to the potential teratogenicity associated with exposure and because respiratory irritation reported at the TLV justified a lower value

For diethylene glycol monobutyl ether:

CEL TWA: 15.5 ppm, 100 mg/m3

(CEL = Chemwatch Exposure Limit)

In studies involving the inhalation toxicity of diethylene glycol monobutyl ether, exposure for 6 hours daily at 100 mg/m3 had no effect. This concentration is in the range of the saturated vapour concentration.

Local damage was produced following inhalation of concentrations higher than the saturated vapour concentrations, that is, during inhalation of the aerosol (350 mg/m3). Since the only potential effects of inhalation are restricted to local discomfort (in the aerosol concentration range) the substance is classified in category 1 for the limitation of exposure peaks.

Teratogenicity studies have not revealed prenatal toxic effects at high oral doses and this ether is classified in pregnancy risk group C.

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For ethanol:

Odour Threshold Value: 49-716 ppm (detection), 101 ppm (recognition)

Eye and respiratory tract irritation do not appear to occur at exposure levels of less than 5000 ppm and the TLV-TWA is thought to provide an adequate margin of safety against such effects. Experiments in man show that inhalation of 1000 ppm caused slight symptoms of poisoning and 5000 ppm caused strong stupor and morbid sleepiness. Subjects exposed to 5000 ppm to 10000 ppm experienced smarting of the eyes and nose and coughing. Symptoms disappeared within minutes. Inhalation also causes local irritating effects to the eyes and upper respiratory tract, headaches, sensation of heat intraocular tension, stupor, fatigue and a need to sleep. At 15000 ppm there was continuous lachrymation and coughing.

Exposure controls

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.

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.)

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.

Personal protection



Eye and face protection

- 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 lenses 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]

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Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> Wear chemical protective gloves, e.g. PVC. Wear safety footwear or safety gumboots, e.g. Rubber <p>The selection of 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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> frequency and duration of contact, chemical resistance of glove material, glove thickness and dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> 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. Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. Contaminated gloves should be replaced. <p>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.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> Overalls. PVC Apron. PVC protective suit may be required if exposure severe. Eyewash unit. Ensure there is ready access to a safety shower. <p>Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.</p> <p>For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).</p> <p>Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot and shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.</p>
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

Zeus 3020 Z112 Red RS Ink / MTW Red

Material	CPI
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
TEFLON	C
VITON	C
##ethylene	glycol

* CPI - Chemwatch Performance Index

Respiratory protection

Type A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	A-2 P2	A-PAPR-2 P2
up to 50 x ES	-	A-3 P2	-
50+ x ES	-	Air-line**	-

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO₂), G = Agricultural chemicals, K = Ammonia(NH₃), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

Continued...

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Red flammable liquid with a mild odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.038
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	<61	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	>90
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	>1	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none">▸ Unstable in the presence of incompatible materials.▸ Product is considered stable.▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to
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protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.

Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.

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.

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

One case report describes kidney and liver damage in two people working in a closed room with paint containing diethylene glycol monobutyl ether and at the same time consuming large quantities of alcoholic beverages.

It has as yet not been established whether the glycol ether and alcohol have synergistic effects but it is possible that oxidation and elimination of both substances probably involves alcohol dehydrogenases; competitive inhibition would be the result.

The most common signs of inhalation overexposure to ethanol, in animals, include ataxia, incoordination and drowsiness for those surviving narcosis. The narcotic dose for rats, after 2 hours of exposure, is 19260 ppm.

Ingestion

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).

The toxic effects of glycols (dihydric alcohols), following ingestion are similar to those of alcohol, with depression of the central nervous system (CNS), nausea, vomiting and degenerative changes in liver and kidney.

Cyanosis, rapid breathing and heart beat, low blood pressure, muscle tenderness and unconsciousness may follow ingestion of diethylene glycol monobutyl ether. Swallowing large or repeated doses may affect kidney function.

Ingestion of ethanol may produce nausea, vomiting, gastrointestinal bleeding, abdominal pain and diarrhoea. Systemic effects:

Blood concentration:	Effects:
<1.5 g/l	Mild: Impaired visual acuity, coordination and reaction time, emotional lability
1.5-3.0 g/l	Moderate: Slurred speech, confusion, ataxia, emotional lability, perceptual and sensation disturbances possible blackout spells, and incoordination with impaired objective performance in standardised tests. Possible diplopia, flushing, tachycardia, sweating and incontinence. Bradypnoea may occur early and tachypnoea may develop in cases of metabolic acidosis, hypoglycaemia and hypokalaemia. CNS depression may progress to coma.
3-5 g/l	Severe: Cold clammy skin, hypothermia and hypotension. Atrial fibrillation and atrioventricular block have been reported. Respiratory depression may occur, respiratory failure may follow serious intoxication, aspiration of vomitus may result in pneumonitis and pulmonary oedema. Convulsions due to severe hypoglycaemia may also occur. Acute hepatitis may develop.

Skin Contact

The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either
 ▶ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or
 ▶ produces significant, but moderate, 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

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	<p>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.</p> <p>There are indications that diethylene glycol monobutyl ether is absorbed through intact skin. Toxic effects only occur at very high doses.</p>
Eye	<p>Direct contact of the eye with ethanol may cause immediate stinging and burning with reflex closure of the lid and tearing, transient injury of the corneal epithelium and hyperaemia of the conjunctiva. Foreign-body type discomfort may persist for up to 2 days but healing is usually spontaneous and complete.</p> <p>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 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.</p>
Chronic	<p>On the basis, primarily, of animal experiments, concern has been expressed 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.</p> <p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>There is some evidence to provide a presumption that human exposure to the material may result in impaired fertility on the basis of: some evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.</p> <p>There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</p> <p>Human volunteers exposed to ethylene glycol, 20 to 22 hours/day at mean daily concentrations ranging from 1.4 to 27 ppm for about 4 weeks complained of throat irritation, mild headache and low backache. Complaints became marked when the concentration in the exposure chamber was raised above 56 mg/m³ for part of the day. The most common complaint was irritation of the upper respiratory tract. Concentrations above 80 ppm were intolerable with a burning sensation along the trachea and a burning cough. Excessively exposed workers have reported drowsiness.</p> <p>Long-term exposure to ethanol may result in progressive liver damage with fibrosis or may exacerbate liver injury caused by other agents.</p> <p>Repeated ingestion of ethanol by pregnant women may adversely affect the central nervous system of the developing foetus, producing effects collectively described as foetal alcohol syndrome. These include mental and physical retardation, learning disturbances, motor and language deficiency, behavioural disorders and reduced head size.</p> <p>Consumption of ethanol (in alcoholic beverages) may be linked to the development of Type I hypersensitivities in a small number of individuals. Symptoms, which may appear immediately after consumption, include conjunctivitis, angioedema, dyspnoea, and urticarial rashes. The causative agent may be acetic acid, a metabolite (1).</p> <p>(1) Boehncke W.H., & H.Gall, Clinical & Experimental Allergy, 26, 1089-1091, 1996</p> <p>Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).</p>

Zeus 3020 Z112 Red RS Ink / MTW Red	TOXICITY	IRRITATION
	Not Available	Not Available
ethanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 17100 mg/kg [1]	Eye (rabbit): 500 mg SEVERE
	Inhalation (rat) LC50: 64000 ppm/4h [2]	Eye (rabbit):100mg/24hr-moderate
	Oral (rat) LD50: >11872769 mg/kg [1]	Skin (rabbit):20 mg/24hr-moderate
ethylene glycol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 9530 mg/kgD [2]	Eye (rabbit): 100 mg/1h - mild
	Inhalation (rat) LC50: 50.1 mg/L/8 hr[2]	Eye (rabbit): 12 mg/m ³ /3D
	Oral (rat) LD50: 4700 mg/kgd [2]	Eye (rabbit): 1440mg/6h-moderate
		Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 555 mg(open)-mild
glycerol	TOXICITY	IRRITATION
	dermal (guinea pig) LD50: 54000 mg/kg [1]	Not Available
	Oral (rat) LD50: >20<39800 mg/kg [1]	

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diethylene glycol monobutyl ether	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2700 mg/kg [2]	Eye (rabbit): 20 mg/24h moderate
	Oral (rat) LD50: 3306 mg/kg [1]	Eye (rabbit): 5 mg - SEVERE
C.I. Basic Red 1	TOXICITY	IRRITATION
	dermal (rat) LD50: >2500 mg/kg** [2]	Eye (rabbit): irritating *
	Oral (rat) LD50: 250 mg/kg** [2]	Skin (rabbit): non-irritating *
C.I. Basic Yellow 2	TOXICITY	IRRITATION
	dermal (mouse) LD50: 300 mg/kg [2]	Not Available
	Oral (mouse) LD50: 480 mg/kg [2]	
water	TOXICITY	IRRITATION
	Oral (rat) LD50: >90000 mg/kg [2]	Not Available
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

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RS Ink / MTW Red

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

No significant acute toxicological data identified in literature search.

For glycerol:

Acute toxicity: Glycerol is of a low order of acute oral and dermal toxicity with LD50 values in excess of 4000 mg/kg bw. At very high dose levels, the signs of toxicity include tremor and hyperaemia of the gastro-intestinal tract. Skin and eye irritation studies indicate that glycerol has low potential to irritate the skin and the eye. The available human and animal data, together with the very widespread potential for exposure and the absence of case reports of sensitisation, indicate that glycerol is not a skin sensitizer.

Repeat dose toxicity: Repeated oral exposure to glycerol does not induce adverse effects other than local irritation of the gastro-intestinal tract. The overall NOEL after prolonged treatment with glycerol is 10,000 mg/kg bw/day (20% in diet). At this dose level no systemic or local effects were observed. For inhalation exposure to aerosols, the NOAEC for local irritant effects to the upper respiratory tract is 165 mg/m³ and 662 mg/m³ for systemic effects.

Genotoxicity: Glycerol is free from structural alerts, which raise concern for mutagenicity. Glycerol does not induce gene mutations in bacterial strains, chromosomal effects in mammalian cells or primary DNA damage *in vitro*. Results of a limited gene mutation test in mammalian cells were of uncertain biological relevance. *In vivo*, glycerol produced no statistically significant effect in a chromosome aberrations and dominant lethal study. However, the limited details provided and the absence of a positive control, prevent any reliable conclusions to be drawn from the *in vivo* data. Overall, glycerol is not considered to possess genotoxic potential.

Carcinogenicity: The experimental data from a limited 2 year dietary study in the rat does not provide any basis for concerns in relation to carcinogenicity. Data from non-guideline studies designed to investigate tumour promotion activity in male mice suggest that oral administration of glycerol up to 20 weeks had a weak promotion effect on the incidence of tumour formation.

Reproductive and developmental toxicity: No effects on fertility and reproductive performance were observed in a two generation study with glycerol administered by gavage (NOAEL 2000 mg/kg bw/day). No maternal toxicity or teratogenic effects were seen in the rat, mouse or rabbit at the highest dose levels tested in a guideline comparable teratogenicity study (NOEL 1180 mg/kg bw/day).

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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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

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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 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.

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	<p>Genotoxic Effects : Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available <i>in vivo</i> and <i>in vitro</i> laboratory studies provide consistently negative genotoxicity results for ethylene glycol.</p>
<p>ETHANOL</p>	<p>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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
<p>ETHYLENE GLYCOL</p>	<p>For ethylene glycol:</p> <p>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.</p> <p>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).</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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).</p> <p>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</p>

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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. [Estimated Lethal Dose (human) 100 ml; RTECS quoted by Orica] Substance is reproductive effector in rats (birth defects). Mutagenic to rat cells.

GLYCEROL

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

For glycerol:

Acute toxicity: Glycerol is of a low order of acute oral and dermal toxicity with LD50 values in excess of 4000 mg/kg bw. At very high dose levels, the signs of toxicity include tremor and hyperaemia of the gastro-intestinal tract. Skin and eye irritation studies indicate that glycerol has low potential to irritate the skin and the eye. The available human and animal data, together with the very widespread potential for exposure and the absence of case reports of sensitisation, indicate that glycerol is not a skin sensitiser.

Repeat dose toxicity: Repeated oral exposure to glycerol does not induce adverse effects other than local irritation of the gastro-intestinal tract. The overall NOEL after prolonged treatment with glycerol is 10,000 mg/kg bw/day (20% in diet). At this dose level no systemic or local effects were observed. For inhalation exposure to aerosols, the NOAEC for local irritant effects to the upper respiratory tract is 165 mg/m3 and 662 mg/m3 for systemic effects.

Genotoxicity: Glycerol is free from structural alerts, which raise concern for mutagenicity. Glycerol does not induce gene mutations in bacterial strains, chromosomal effects in mammalian cells or primary DNA damage *in vitro*. Results of a limited gene mutation test in mammalian cells were of uncertain biological relevance. *In vivo*, glycerol produced no statistically significant effect in a chromosome aberrations and dominant lethal study. However, the limited details provided and the absence of a positive control, prevent any reliable conclusions to be drawn from the *in vivo* data. Overall, glycerol is not considered to possess genotoxic potential.

Carcinogenicity: The experimental data from a limited 2 year dietary study in the rat does not provide any basis for concerns in relation to carcinogenicity. Data from non-guideline studies designed to investigate tumour promotion activity in male mice suggest that oral administration of glycerol up to 20 weeks had a weak promotion effect on the incidence of tumour formation.

Reproductive and developmental toxicity: No effects on fertility and reproductive performance were observed in a two generation study with glycerol administered by gavage (NOAEL 2000 mg/kg bw/day). No maternal toxicity or teratogenic effects were seen in the rat, mouse or rabbit at the highest dose levels tested in a guideline comparable teratogenicity study (NOEL 1180 mg/kg bw/day).

DIETHYLENE GLYCOL
MONOBUTYL ETHER

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

For diethylene glycol monoalkyl ethers and their acetates:

This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates.

Acute toxicity: There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals

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	<p>and/or humans were negative.</p> <p>Repeat dose toxicity : Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than continuous exposure regimens.</p> <p>Mutagenicity: DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in <i>S. typhimurium</i> strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in <i>E. coli</i> WP2uvrA, with and without metabolic activation. <i>In vitro</i> cytogenicity and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and <i>in vivo</i> micronucleus or cytogenicity tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic.</p> <p>Reproductive and developmental toxicity: Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGPE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA.</p> <p>Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m³) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during gestation; however a transient decrease in body weight was observed, which reversed by Day 21. In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus</p>
C.I. BASIC RED 1	<p>The substance is classified by IARC as Group 3:</p> <p>NOT classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p> <p>* BASF Canada</p>
C.I. BASIC YELLOW 2	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>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.</p> <p>Long term animal feeding studies with auramine show a dose-dependent reduction in food consumption and delayed body-weight gain and an increase in relative liver weights. Histologically detectable chronic toxic organ damage such as hyperplasia, cirrhotic changes, bile duct proliferation and cholangiofibrosis are only found after administration of relatively high concentrations in the diet for 2-years or for the whole life-span of animals and are generally associated with the development of hepatomas, cholangiomas and hepatocellular carcinomas. Tests for the initiating and promoting activity of auramine yield positive results. The currently available data indicate that exposure to auramine and the working conditions prevailing to the production process involve a cancer risk to man.</p> <p>NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p> <p>Liver and kidney tumours, tumours at sites of application recorded. Equivocal tumorigenic agent by RTECS criteria.</p>
WATER	No significant acute toxicological data identified in literature search.

Acute Toxicity	✓	Carcinogenicity	✓
Skin Irritation/Corrosion	✓	Reproductivity	⊖
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	⊖	STOT - Repeated Exposure	⊖
Mutagenicity	⊖	Aspiration Hazard	⊖

Legend: ✓ – Data required to make classification available
✗ – Data available but does not fill the criteria for classification
⊖ – Data Not Available to make classification

Continued...

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
ethanol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
ethylene glycol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
glycerol	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
diethylene glycol monobutyl ether	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Basic Red 1	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Basic Yellow 2	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
water	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
NOTE: Manufacturer has supplied full ingredient	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
information to allow CHEMWATCH assessment.	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For glycerol

log Kow : -2.66- -2.47

BOD 5: 0.617-0.87,31-51%

COD : 1.16,82-95%

ThOD : 1.217-1.56

Completely biodegradable.

Environmental fate:

Based on the relevant physical-chemical properties and the fact that glycerol is readily biodegradable, glycerol will partition primarily to water.

Biodegradability: Glycerol is considered to be readily biodegradable in the aquatic environment. Pre-adapted microorganisms can degrade glycerol rapidly under both aerobic and anaerobic conditions.

Bioaccumulation: Based on Log Kow -1.76, glycerol will have a low bioaccumulation potential and is not expected to bioaccumulate.

Photodegradation: The calculated half-life for the photo-oxidation (reaction with hydroxyl radicals) of glycerol in air is 6.8 hours (EPIWIN vs 3.04).

Stability in Water: Glycerol does not contain functional groups that are expected to react with water.

Transport between Environmental Compartments: From the EQC model (Mackay level III), it can be deduced that 100% of glycerol will end up in the water phase. Negligible amounts will be distributed towards soil, air and sediment

Ecotoxicity:

Fish LC50: >5000 mg/l

Algae IC50: >2900 mg/l

Bacteria EC50: .10000 mg/l (*Pseudomonas putida*)

The weight of evidence indicates that glycerol is of low toxicity to aquatic organisms and this conclusion is supported by QSAR predictions. The lowest LC50 for fish is a 24-h LC50 of >5000 mg/l for *Carassius auratus* (Goldfish) and for aquatic invertebrates, a 24 h EC50 of >10000 mg/l for *Daphnia magna* is the lowest EC50. Several tests on algae are available, which suggest very low toxicity to a range of species, however their validity is uncertain. A QSAR prediction for the 96h EC50 to algae was 78000 mg/l. No toxicity towards the microorganism *Pseudomonas putida* was observed at 10000 mg/l after exposure for 16 hours. No long-term aquatic toxicity data is available. Screening studies are available on frog and carp embryos which indicate some effects on growth and hatching rates respectively at very high concentrations of glycerol, >7000 mg/l. However, their ecological relevance is not clear.

For organic cationics

Cationic substances, and their polymers and those polymers that are reasonably anticipated to become cationic in the natural aquatic environment (pH range 4-9) may be environmental hazards.

Exempt from this concern are those polymers to be used only in solid phase, such as ion-exchange resins, and where the FGEW (Functional Group Equivalent Weight) of cationic groups is not 5000 and above.

The numerous studies of aquatic toxicity, many of which were conducted in natural waters with and without added effluents, indicate that the source and composition of the test water dramatically affects the toxicity of the test substance. These results are consistent with the known behavior of these materials in the environment. Cationic substances in the environment instantaneously form complexes with naturally occurring negatively charged constituents in sewage, soils, sediments, and with dissolved humic substances in surface waters. This complexation behavior results in reduced bioavailability in actual environmental conditions that is not adequately represented by standard laboratory assays and/or predictions by various QSAR models.

Ecotoxicity:

These chemicals, by the nature of their surfactant properties, are toxic to aquatic organisms at low concentrations. Cationic groups such as alkylsulfoniums, alkylphosphoniums and quaternary ammonium polymers are highly toxic to fish and other aquatic organisms. Similarly potentially cationic groups such as amines and isocyanates are of concern. Some cationics, however, may fall into the category of PLCs (polymers of low concern)

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provided they possess low charge density, and/or are not water-soluble or are not self-dispersing polycarboxylates or poly- (aromatic or aliphatic) sulfonate polymers.

The toxicity of quaternary ammonium compounds is known to be greatly reduced in the environment because of preferential binding to dissolved organics in surface water

for ethylene glycol:

log Kow : -1.93- -1.36

Half-life (hr) air : 24

Henry's atm m³/mol: 6.00E-08

BOD 5 : 0.15-0.81,12%

COD : 1.21-1.29

ThOD : 1.26

BCF : 10-190

In the atmosphere ethylene glycol exists mainly in the vapour phase. It is degraded in the atmosphere by reaction with photochemically produced hydroxy radicals (estimated half-life 24-50 hours).

Ethylene glycol does not concentrate in the food chain.

Environmental fate:

Ethylene glycol has a low vapour pressure (7.9 Pa at 20 C); it is expected to exist almost entirely in the vapour phase if released to the atmosphere. The Henry's law constant for ethylene glycol is 1.41×10^{-3} or 6.08×10^{-3} Pa.m³/mol, depending on method of calculation, indicating a low capacity for volatilisation from water bodies or soil surfaces.

Ethylene glycol adsorbed onto silica gel and irradiated with light (wavelength >290 nm) degraded by 12.1% over 17 h. Photodegradation is not expected, as the molecule should not absorb at these wavelengths; the mechanism of this breakdown is, therefore, unknown. Estimated half-life in the atmosphere for reaction with hydroxyl radicals from various reports is 2.1 days, 8-84 h or 1 day.

Ethylene glycol released to the atmosphere will be degraded by reaction with hydroxyl radicals; the half-life for the compound in this reaction has been estimated at between 0.3 and 3.5 days. No hydrolysis of ethylene glycol is expected in surface waters.

The compound has little or no capacity to bind to particulates and will be mobile in soil. Soil partition coefficients (log Koc) of 0-0.62 were determined.

Migration rates in five soil types were measured at between 4 and 27 cm per 12 h

The low octanol/water partition coefficient (log Kow -1.93 to -1.36) and measured bioconcentration factors in a few organisms indicate low capacity for bioaccumulation. Bioconcentration factors of 190 for the green algae (*Chlorella fusca*), up to 0.27 in specific tissues of the crayfish (*Procambarus* sp.), and 10 for the golden orfe (*Leuciscus idus melanotus*) confirm low bioaccumulation.

Ethylene glycol is readily biodegradable in standard tests using sewage sludge. Many studies show biodegradation under both aerobic and anaerobic conditions. Some studies suggest a lag phase before degradation, but many do not. Degradation occurs in both adapted and unadapted sludges. Rapid degradation has been reported in surface waters (less in salt water than in fresh water), groundwater, and soil inocula. Several strains of microorganisms capable of utilising ethylene glycol as a carbon source have been identified.

Ethylene glycol has been identified as a metabolite of the growth regulator ethylene in a number of higher plants and as naturally occurring in the edible fungus *Tricholoma matsutake*

Ecotoxicity:

Fish LC50 (96 h): 118-550 mg/L

Ethylene glycol has generally low toxicity to aquatic organisms. Toxic thresholds for microorganisms are above 1000 mg/litre. EC50s for growth in microalgae are 6500 mg/litre or higher. Acute toxicity tests with aquatic invertebrates where a value could be determined show LC50s above 20 000 mg/litre, and those with fish show LC50s above 17 800 mg/litre. An amphibian test showed an LC50 for tadpoles at 17 000 mg/litre. A no-observed-effect concentration (NOEC) for chronic tests on daphnids of 8590 mg/litre (for reproductive end-points) has been reported. A NOEC following short-term exposure of fish has been reported at 15 380 mg/litre for growth. Tests using deicer containing ethylene glycol showed greater toxicity to aquatic organisms than observed with the pure compound, indicating other toxic components of the formulations. Laboratory tests exposing aquatic organisms to stream water receiving runoff from airports have demonstrated toxic effects and death. Field studies in the vicinity of an airport have reported toxic signs consistent with ethylene glycol poisoning, fish kills, and reduced biodiversity. These effects cannot definitively be ascribed to ethylene glycol. Terrestrial organisms are much less likely to be exposed to ethylene glycol and generally show low sensitivity to the compound. Concentrations above 100 000 mg/litre were needed to produce toxic effects on yeasts and fungi from soil. Very high concentrations and soaking of seeds produced inhibition of germination in some experiments; these are not considered of environmental significance. A no-observed-effect level (NOEL) for orally dosed ducks at 1221 mg/kg body weight and reported lethal doses for poultry at around 8000 mg/kg body weight indicate low toxicity to birds.

When ethanol is released into the soil it readily and quickly biodegrades but may leach into ground water; most is lost by evaporation. When released into water the material readily evaporates and is biodegradable.

Ethanol does not bioaccumulate to an appreciable extent.

The material is readily degraded by reaction with photochemically produced hydroxy radicals; release into air will result in photodegradation and wet deposition.

Environmental Fate:

TERRESTRIAL FATE: An estimated Koc value of 1 indicates that ethanol is expected to have very high mobility in soil. Volatilisation of ethanol from moist soil surfaces is expected to be an important fate process given a Henry's Law constant of 5×10^{-6} atm-m³/mole. The potential for volatilisation of ethanol from dry soil surfaces may exist based upon an extrapolated vapor pressure of 59.3 mmHg. Biodegradation is expected to be an important fate process for ethanol based on half-lives on the order of a few days for ethanol in sandy soil/groundwater microcosms.

AQUATIC FATE: An estimated Koc value of 1 indicates that ethanol is not expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon a Henry's Law constant of 5×10^{-6} atm-m³/mole. Using this Henry's Law constant and an estimation method, volatilisation half-lives for a model river and model lake are 3 and 39 days, respectively. An estimated BCF= 3, from a log Kow of -0.31 suggests bioconcentration in aquatic organisms is low. Hydrolysis and photolysis in sunlit surface waters is not expected to be an important environmental fate process for ethanol since this compound lacks functional groups that hydrolyse or absorb light under environmentally relevant conditions. Ethanol was degraded with half-lives on the order of a few days in aquatic studies conducted using microcosms constructed with a low organic sandy soil and groundwater, indicating it is unlikely to be persistent in aquatic environments(8).

ATMOSPHERIC FATE: Ethanol, which has an extrapolated vapor pressure of 59.3 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapour-phase ethanol is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 5 days, calculated from its rate constant of 3.3×10^{-12} m³/molecule-sec at 25 deg C.

Ecotoxicity:

log Kow: -0.31- -0.32

Half-life (hr) air: 144

Half-life (hr) H₂O surface water: 144

Continued...

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Henry's atm m3 /mol: 6.29E-06
BOD 5 if unstated: 0.93-1.67,63%
COD: 1.99-2.11,97%
ThOD: 2.1

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethanol	LOW (Half-life = 2.17 days)	LOW (Half-life = 5.08 days)
ethylene glycol	LOW (Half-life = 24 days)	LOW (Half-life = 3.46 days)
glycerol	LOW	LOW
diethylene glycol monobutyl ether	LOW	LOW
C.I. Basic Yellow 2	HIGH	HIGH
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
ethanol	LOW (LogKOW = -0.31)
ethylene glycol	LOW (BCF = 200)
glycerol	LOW (LogKOW = -1.76)
diethylene glycol monobutyl ether	LOW (BCF = 46)
C.I. Basic Yellow 2	LOW (BCF = 16)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
ethanol	HIGH (KOC = 1)
ethylene glycol	HIGH (KOC = 1)
glycerol	HIGH (KOC = 1)
diethylene glycol monobutyl ether	LOW (KOC = 10)
C.I. Basic Yellow 2	LOW (KOC = 24750)
water	LOW (KOC = 14.3)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible.
	<p>Otherwise:</p> <ul style="list-style-type: none"> 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 SDS and observe all notices pertaining to the product. <p>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.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> Reduction Reuse Recycling Disposal (if all else fails) <p>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.</p> <ul style="list-style-type: none"> 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.



Continued...

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- 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 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Y

Land transport (ADG)

UN number	1993
Packing group	III
UN proper shipping name	FLAMMABLE LIQUID, N.O.S. (contains ethanol)
Environmental hazard	No relevant data
Transport hazard class(es)	Class 3 Subrisk Not Applicable
Special precautions for user	Special provisions 223 274 Limited quantity 5 L

Air transport (ICAO-IATA / DGR)

UN number	1993
Packing group	III
UN proper shipping name	Flammable liquid, n.o.s. * (contains ethanol)
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class 3 ICAO / IATA Subrisk Not Applicable ERG Code 3L
Special precautions for user	Special provisions A3 Cargo Only Packing Instructions 366 Cargo Only Maximum Qty / Pack 220 L Passenger and Cargo Packing Instructions 355 Passenger and Cargo Maximum Qty / Pack 60 L Passenger and Cargo Limited Quantity Packing Instructions Y344 Passenger and Cargo Limited Maximum Qty / Pack 10 L

Sea transport (IMDG-Code / GGVSee)

UN number	1993
Packing group	III
UN proper shipping name	FLAMMABLE LIQUID, N.O.S. (contains ethanol)
Environmental hazard	Not Applicable

Continued...

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Transport hazard class(es)	IMDG Class	3
	IMDG Subrisk	Not Applicable
Special precautions for user	EMS Number	F-E , S-E
	Special provisions	223 274 955
	Limited Quantities	5 L

Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	ethylene glycol	Y

SECTION 15 REGULATORY INFORMATION

Safety, health and environmental regulations / legislation specific for the substance or mixture

ETHANOL(64-17-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

ETHYLENE GLYCOL(107-21-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

GLYCEROL(56-81-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
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DIETHYLENE GLYCOL MONOBUTYL ETHER(112-34-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists	Australia Inventory of Chemical Substances (AICS)
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C.I. BASIC RED 1(989-38-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
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C.I. BASIC YELLOW 2(2465-27-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists	Australia Inventory of Chemical Substances (AICS)
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WATER(7732-18-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (C.I. Basic Red 1; diethylene glycol monobutyl ether; glycerol; ethanol; water; ethylene glycol; C.I. Basic Yellow 2)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (water)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
glycerol	29796-42-7, 30049-52-6, 37228-54-9, 56-81-5, 75398-78-6, 78630-16-7, 8013-25-0

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

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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