SDS 101

Version No: 6.1.1.1
Issue Date: 12/01/2022
Initial Date: Not Available

Safety Data Sheet according to NOHSC and ADG requirements

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

Product Identifier

Product Name:	Liquid Intelligence 101 Red Coolant
Chemical Name:	Not Applicable
Synonyms	Coolant Anti-Freeze. Part Number 101
Proper shipping name	Ethylene Glycol Coolant
Chemical formula	Not Applicable
Other means of identification	Not Available
CAS number	Not Applicable

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

Relevant identified uses	Automotive radiator coolant.

Details of the manufacturer/importer

Registered company name	Liquid Intelligence Pty Ltd
Address	unit 8 / 12 Eddie Road Minchinbury 2770 NEW Australia
Telephone	1800 441 163
Fax	Not Available
Website	www.liquidintelligence.com.au
Email	peter@liquidintelligence.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	1800 441 163

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

Flammability	1 = Low
Toxicity	2 = Moderate
Body Contact	1 = Low
Reactivity	1 = Low
Chronic	0 = Minimum

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

Relevant risk statements are found in section 2

Poisons Schedule	S5
Classification	Acute Toxicity (Oral) Category 4
Risk Phrases	R22 Harmful if swallowed.
Indication(s) of danger	Xn
Legend:	1. Classification drawn from HSIS ; 2. Classification drawn from EC Directive 1272/2008 - Annex VI

Hazard statement(s)
H302 Harmful if swallowed

Precautionary statement(s) Prevention
P264 Wash all exposed external body areas thoroughly after handling
P270 Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response
P301+P312 IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P330 Rinse mouth.

Precautionary statement(s) Storage
Not Applicable

Precautionary statement(s) Disposal
P501 Dispose of contents/container in accordance with local regulations.

SAFETY ADVICE
S13 Keep away from food, drink and animal feeding stuffs.
S23 Do not breath gas/fumes/vapour/spray.
S36 Wear suitable protective clothing.
S37 Wear suitable gloves.
S40 To clean the floor and all objects contaminated by this material, use water and detergent.
S46 If swallowed, seek medical advice immediately and show this container or label.
S56 Dispose of this material and its container at hazardous or special waste collection point.

Other hazards
May produce discomfort of the eyes and skin*.
Possible respiratory and skin sensitizer*.
Inhalation may produce health damage*.
Cumulative effects may result following exposure*.
May affect fertility*.
May be harmful to the foetus/ embryo*.
Repeated exposure potentially causes skin dryness and cracking*.
Vapours potentially cause drowsiness and dizziness*.

HAZARD PICTOGRAM
Signal Word Warning

SECTION 3 COMPOSITIONS / INFORMATION ON INGREDIENTS

Substances
See section below for composition of Mixtures

Chemical Name	CAS Number	Proportion
Ethylene Glycol	107-21-1	>30%
DDDA C12, C11, and C10 diacids	693-23-2	>4%
Undecanedioic Acid	1852-04-6	>1%
Sebacic Acid,	111-20-6	<1%
Cyclododecanol/cyclododecanone	7262-23-3	>4%
Alkanolamin	102-71-6	>5%
Denatonium Benzoate	3734-33-6	<1%

SECTION 4 FIRST AID MEASURES
Description of first aid measures

Eye Contact
If in eyes, hold eyelids apart and flush the eye continuously with running water.
Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.
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
If skin contact occurs:
Immediately remove all contaminated clothing, including footwear.
Flush skin and hair with running water (and soap if available).
Seek medical attention in event of irritation.

Inhalation
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.

Ingestion
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.
For advice, contact a Poisons Information Centre or a doctor.

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.

For acute or short term, repeated exposures to ethylene glycol:
Early treatment of ingestion is important. Ensure emesis is satisfactory.
Test and correct for metabolic acidosis and hypocalcaemia.
Apply sustained diuresis when possible with hypertonic mannitol.
Evaluate renal status and begin haemodialysis if indicated.
Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
Ethanol therapy prolongs the half-life of glycol and reduces the formation of toxic metabolites.
Pyridoxine and thiamine are cofactors for glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.
[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a work shift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to glycol ethers. This arises from the finding that an increase injurinary stones may be associated with such exposures.
Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600

To treat poisoning by the higher aliphatic alcohols (up to C7):
Gastric lavage with copious amounts of water.
It may be beneficial to instil 60 ml of mineral oil into the stomach.
Oxygen and artificial respiration as needed.
Electrolyte balance: it may be useful to start 500ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5)

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 shock.
Monitor and treat, where necessary, for pulmonary oedema.
Anticipate and treat, where necessary, for 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.
Give activated charcoal.

ADVANCED TREATMENT
Consider orotracheal or nontracheal 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.
If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
Drug therapy should be considered for pulmonary oedema.
Treat seizures with diazepam.
Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
Acidosis may respond to hyperventilation and bicarbonate therapy.
Haemodialysis might be considered in patients with severe intoxication.
Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2ndEd. 1994
For C8 alcohols and above.
Symptomatic and supportive therapy is advised in managing patients.

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.

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.
Wear full body protective clothing with breathing apparatus.
Prevent, by any means available, spillage from entering drains or water course.
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

Combustible.
Slight fire hazard when exposed to heat or flame.
Heating may cause expansion or decomposition leading to violent rupture of containers.
On combustion, may emit toxic fumes of carbon monoxide (CO).
May emit acrid smoke.
Mists containing combustible materials may be explosive.
Combustion products include carbon dioxide (CO_2), other pyrolysis products typical of burning organic material may emit poisonous fumes.

SECTION 6 ACCIDENTAL RELEASE MEASURES
Personal precautions, protective equipment and emergency procedures

Spills
Slippery when spilt.
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 spill with sand, earth, inert material or vermiculite.
Wipe up.
Place in a suitable, labelled container for waste disposal.

SECTION 7 HANDLING AND STORAGE
Precautions for safe handling

Safe handling
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 exposure occurs.
Use in a well-ventilated area.
Prevent concentration in hollows and sumps.
DO NOT enter confined spaces until atmosphere has been checked.
Avoid smoking, naked lights or ignition sources.
Avoid contact with incompatible materials.
When handlings, 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 MSDS.
Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.

Other information
Material is hygroscopic, i.e. absorbs moisture from the air. Keep containers well sealed in storage.
Store in original containers.
Keep containers securely sealed.
No smoking, naked lights or ignition sources.
Store in a cool, dry, well-ventilated area.
Store away from incompatible materials and foodstuff containers.
Protect containers against physical damage and check regularly for leaks.
Observe manufacturer's storage and handling recommendations contained within this MSDS.

Conditions for safe storage, including any incompatibilities

Suitable container
DO NOT use aluminium or galvanised containers
Metal can or drum
Packaging as recommended by manufacturer.
Check all containers are clearly labelled and free from leaks.

Storage incompatibility
Avoid reaction with oxidising agents
Avoid strong acids, bases.

Package material incompatibilities
Not Available

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	Glycol	Dihydroxy Diethyl Ether (CH2CH2OH)2O	10 mg/m3 / 52 mg/m3/20 ppm	104 mg/m3 / 40 ppm	Not Available	Sk

MATERIAL DATA
Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However, this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.
OSHA (USA) concluded that exposure to sensory irritants can:
Cause inflammation
Cause increased susceptibility to other irritants and infectious agents
Lead to permanent injury or dysfunction
Permit greater absorption of hazardous substances and
Acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

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]

Skin protection
See Hand protection below

Hands/feet protection
See Hand protection below
Hands/feet protection
Wear chemical protective gloves, e.g. PVC.
Wear safety footwear or safety gumboots, e.g. Rubber
NOTE:
The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
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 cannot be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
frequency and duration of contact, chemical resistance of glove material, glove thickness and dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
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.
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.

Body protection
See Other protection below

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

Thermal hazards
Not Available

Recommended material(s)
GLOVE SELECTION INDEX
Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".

Material	CPI
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A
NEOPRENE/NATURAL	A
NITRILE	A
NITRILE+PVC	A
PE/EVAL/PE	A
PVC	A
TEFLON	A
PVA	B

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	Viscous yellow liquid with a mild odour; miscible with water and alcohol.

Physical state	Liquid	**Relative density (Water = 1)**	1.1167
Odour	low	**Partition coefficient n-octanol / water**	Not required
Odour threshold	Not required	**Auto-ignition temperature (°C)**	Not Available
pH	8.1	**Viscosity (cSt)**	Not Applicable
Freezing point (°C)	-56	**Decomposition temperature**	Not Available
Initial boiling point and boiling range (°C)	245	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	163°C	**Taste**	Bitter
Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Applicable
Upper Explosive Limit (%)	12.8	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	3.2	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Negligible	**Gas group**	Not Available
Solubility in water	Total	**pH as a solution (1%)**	7.4
Vapour density (Air =1)	2.2	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	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

Inhaled	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 neuro-behavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, and 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 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.

Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 grams 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). Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general, the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased. Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 - Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However, the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat, even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema. Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent. 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.
Skin Contact	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 The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either: produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non-allergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering 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.
Eye	Limited evidence or practical experience suggests that the material may cause moderate 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 of experimental animals. Repeated or prolonged exposure may cause moderate inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. 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. 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. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

	TOXICITY	IRRITATION
Liquid Intelligence 100 Coolant Concentrate	Not Available	Not Available
Ethylene Glycol	Dermal (rabbit) LD50: 9530 mg/kg Inhalation (human) TCLo:10000 mg/m3 Inhalation (rat) LC50: 50100 mg/m3/8 hr Oral (child) TDLo: 5500 mg/kg Oral (human) LDLo: 398 mg/kg Oral (rat) LD50: 4700 mg/kg	Eye (rabbit): 100 mg/1h - mild Eye (rabbit): 12 mg/m3/3D Eye (rabbit): 1440mg/6h-moderate Eye (rabbit): 500 mg/24h - mild Skin (rabbit): 555 mg(open)-mild

Ethylene Glycol:
Ethylene Glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, Ethylene Glycol is initially metabolised by alcohol dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of
Ethylene Glycol and is considered to be part of a second stage in Ethylene Glycol poisoning the symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of Dihydroxy Diethyl Ether 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 Dihydroxy Diethyl Ether. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of Dihydroxy Diethyl Ether.
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 diarrhoea 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 Dihydroxy Diethyl Ether intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

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 Dihydroxy Diethyl Ether. Other evidence of embyrotoxicity 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 (birthdefects). Mutagenic to rat cells.

SECTION 12 ECOLOGICAL INFORMATION

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
Ethylene Glycol	low (Half-life = 24 #days)	low (Half-life = 3.46 #days)

Bioaccumulative potential

Ingredient	Bioaccumulation
Ethylene Glycol	low (BCF = 3.162)

Mobility in soil

Ingredient	Mobility
Ethylene Glycol	high (KOC = 1)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal

Containers may still present a chemical hazard/ danger when empty.

Otherwise:
If container cannot be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
Where possible retain label warnings and MSDS and observe all notices pertaining to the product.

Recycle wherever possible or consult manufacturer for recycling options.
Consult State Land Waste Authority for disposal.
Bury or incinerate residue at an approved site.
Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION
Labels Required

Marine Pollutant	**NO**
HAZCHEM	**Not Applicable**

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

SECTION 15 REGULATORY INFORMATION
Safety, health and environmental regulations / legislation specific for the substance or mixture

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"

SECTION 16 OTHER INFORMATION
Other information

The 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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