Hi-Tec Oil Traders Pty Ltd ABN 28 053 837 362
5 Tarlington Place Smithfield NSW 2164
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SAFETY DATA SHEET

Issue Date: 24 June 2020
Bug Rid
Version: 4

Product name: **Bug Rid**

1. COMPANY DETAILS AND PRODUCT IDENTIFICATION

COMPANY:	Hi-Tec Oil Traders Pty Ltd. (ABN 28 053 837 362)
ADDRESS:	PO Box 322 Castle Hill NSW 1765 5 Tarlington Place, Smithfield NSW 2164
TELEPHONE NUMBER:	1300 796 009
FAX NUMBER:	(02) 9604 1611
EMERGENCY TELEPHONE NUMBER:	1300 796 009
PRODUCT NAME:	Bugrid
OTHER NAMES:	None
MANUFACTURER'S PRODUCT CODE:	HI8-3060
USE:	Diesel fuel biocide
ADDITIONAL INFORMATION:	Refer to Product Information Sheet for additional information.
OTHER INFORMATION:	Visit our website: www.hi-tecoils.com.au Email: hitecoils@hi-tecoils.com.au

2. HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE:	HAZARDOUS SUBSTANCE DANGEROUS GOODS Hazard classification according to criteria of NOHSC and GHS. Dangerous Goods classification according to the Australian Dangerous Goods (ADG) Code, IATA and IMDG criteria.
POISON SCHEDULE:	S6

GHS LABEL ELEMENTS

SIGNAL WORD(S): **DANGER**

2. HAZARDS IDENTIFICATION (CONT)

GHS HAZARD CLASSIFICATIONS

HAZARD CLASSIFICATION:	Category 4: Flammable Liquid Category 4: Acute Toxicity (Oral) Category 4: Acute Toxicity (Dermal) Category 4: Acute Toxicity (Inhalation) Category 2: Skin Corrosion / Irritation Category 1: Serious Eye Damage Category 1: Skin Sensitizer Category 2: Cacinogenicity Category 1: Aspriation Hazard Category 2: Acute Aquatic Hazard Category 2: Chronic Aquatic Hazard
HAZARD STATEMENTS:	H227: Combustible liquid H302: Harmful if swallowed. H312: Harmful in contact with skin. H332: Harmful if inhaled. H315: Causes skin irritation. H318: Causes serious eye damage. H317: May cause an allergic skin reaction. H351: Suspected of causing cancer. H304: May be fatal if swallowed and enters airways. H411: Toxic to aquatic life with long lasting effects. AUH066: Repeated exposure may cause skin dryness and cracking
PREVENTION STATEMENTS:	P201: Obtain special instructions before use. P210: Keep away from heat/sparks/open flames/hot surfaces. - No smoking. P271: Use only outdoors or in a well-ventilated area. P280: Wear protective gloves/protective clothing/eye protection/face protection. P281: Use personal protective equipment as required. P261: Avoid breathing mist/vapours/spray. P270: Do not eat, drink or smoke when using this product. P273: Avoid release to the environment. P272: Contaminated work clothing should not be allowed out of the workplace
RESPONSE STATEMENTS:	P301+P310: IF SWALLOWED: Immediately call the POISON INFORMATION CENTER on 13 11 26 or doctor/physician. P305+P351+P338: IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. P308+P313: IF exposed or concerned: Get medical advice/attention. P331: Do NOT induce vomiting. P362: Take off contaminated clothing and wash before reuse. P370+P378: In case of fire: Use alcohol resistant foam or normal protein foam for extinction.

2. HAZARDS IDENTIFICATION (CONT)

RESPONSE STATEMENTS:

P302+P352: IF ON SKIN: Wash with plenty of soap and water.
P333+P313: If skin irritation or rash occurs: Get medical advice/attention.
P391: Collect spillage.
P301+P312: IF SWALLOWED: Call the POISON INFORMATION CENTER on 13 11 26 or doctor/physician if you feel unwell.
P304+P340: IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330: Rinse mouth.

STORAGE STATEMENTS:

P403+P235: Store in a well-ventilated place. Keep cool.
P405: Store locked up.

DISPOSAL STATEMENT:

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

3. IDENTIFICATION / COMPOSITION OF INGREDIENTS

Ingredients	CAS No	Conc, %
Middle distillate	68476-34-6	30 – 60
Kerosene, (petroleum), hydrosulfurised	64742-81-0	10 – 30
Ethylene glycol monobutyl ether	111-76-2	10 – 30
Aromatic 150	64742-95-6	1 – 10
Nonylphenol, ethoxylated	9016-45-9	< 10
Benzyl-C12-16-alkyldimethylammonium chloride	68424-85-1	< 5
Isothiazolinones, mixed	55965-84-9	< 1

4. FIRST AID MEASURES

GENERAL INFORMATION:

You should call the POISONS INFORMATION CENTRE if you feel that you may have been poisoned, burned or irritated by this product. The number is 13 11 26 from anywhere in Australia (0800 764 766 in New Zealand) and is available at all times. Have this SDS with you when you call.

EYE CONTACT:

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:

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.

4. FIRST AID MEASURES (CONT)

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.

Indication of any immediate medical attention and special treatment needed-
For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 50 mm Hg) should be intubated.
Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

5. FIRE FIGHTING MEASURES

EXTINGUISHING MEDIA:	Foam. Dry chemical powder. BCF (where regulations permit). Carbon dioxide. Water spray or fog - Large fires only.
INCOMPATIBILITIES:	Avoid contamination with strong oxidising agents as ignition may result.
FIRE FIGHTING:	Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water courses. Use water delivered as a fine spray to control fire and cool adjacent area. **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.

5. FIRE FIGHTING MEASURES (CONT)

FIRE AND EXPLOSION HAZARDS:	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. Other combustion products include:, carbon dioxide (CO2), nitrogen oxides (NOx), chlorides, other pyrolysis products typical of burning organic material.
HAZCHEM:	•3Z

6. ACCIDENTAL RELEASE MEASURES

MINOR 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.
MAJOR SPLILLS:	Slippery when spilt. Moderate hazard. Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. No smoking, naked lights or ignition sources. Increase ventilation. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. 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.

7. HANDLING AND STORAGE

SAFE HANDLING:	Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. Prevent concentration in hollows and sumps. **DO NOT** enter confined spaces until atmosphere has been checked. Avoid smoking, naked lights or ignition sources. Avoid contact with incompatible materials. When handling, **DO NOT** eat, drink or smoke. 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.

7. HANDLING AND STORAGE (CONT)

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 SDS.

SUITABLE CONTAINER: Metal can or drum. Packaging as recommended by manufacturer.
Check all containers are clearly labelled and free from leaks.

STORAGE INCOMPATIBILITY: Avoid storage with oxidisers. Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

8. EXPOSURE CONTROLS / PERSONAL PROTECTION

CONTROL PARAMETERS
OCCUPATIONAL EXPOSURE LIMITS: Australia Exposure Standards

Ingredient	Material name	TWA	STEL	Peak	Notes
Ethylene glycol monobutyl ether	2-Butoxyethanol	96mg/m^3 / 20 ppm	242 mg/m^3 / 50 ppm	N/A	Sk

EMERGENCY LIMITS:

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
Middle distillates	Diesel fuels; (includes diesel fuel No.4 (68476-31-3), fuel oil No. 2 (68476-30-2), fuel oil residual (68476-33-5)	300 mg/m^3	3,300 mg/m^3	20,000 mg/m^3
Ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
Nonylphenol, ethoxylated	Glycols, polyethylene, mono(p-nonylphenyl) ether	4.5 mg/m^3	49 mg/m^3	300 mg/m^3
Nonylphenol, ethoxylated	Ethoxylated nonylphenol; (Nonyl phenyl polyethylene glycol ether)	1 mg/m^3	11 mg/m^3	260 mg/m^3
Benzyl-C12-16- alkyldimethylammonium chloride	Quaternary ammonium compounds, benzyl-C12-C16-alkyldimethyl, chlorides	1.3 mg/m^3	14 mg/m^3	84 mg/m^3

Ingredient	Original IDLH	Revised IDLH
Middle distillate	Not available	Not available
Kerosene, (petroleum), hydrosulfurised	Not available	Not available
Ethylene glycol monobutyl ether	700 ppm	700 (Unch) ppm
Aromatic 150	Not available	Not available
Nonylphenol, ethoxylated	Not available	Not available
Benzyl-C12-16- alkyldimethylammonium chloride	Not available	Not available
Isothiazolinones, mixed	Not available	Not available

8. EXPOSURE CONTROLS / PERSONAL PROTECTION (CONT)

MATERIAL DATA:

None assigned. Refer to individual constituents.
NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP.
NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP.
NOTE N: The classification as a carcinogen need not apply if the full refining history is known and it can be shown that the substance from which it is produced is not a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP.

ENGINEERING 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.
General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

8. EXPOSURE CONTROLS / PERSONAL PROTECTION (CONT)

ENGINEERING CONTROLS:

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

Within each range the appropriate value depends on:

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

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

EYE/FACE PROTECTION:	Safety glasses with side shields; or as required. 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].
HANDS / FEET PROTECTION:	Butyl rubber gloves. Neoprene gloves. PVC gloves. Safety footwear. PVC boots
BODY PROTECTION:	Safety gloves: Butyl rubber, neoprene and PVC. Safety footwear. PVC aprons or overalls. Respirator with type A filter. Barrier cream.
THERMAL HAZARDS:	Not available.

8. EXPOSURE CONTROLS / PERSONAL PROTECTION (CONT)

RECOMMENDED MATERIAL (S): 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: Hi-Tec Bug Rid.

Material	*CPI	Material	*CPI
BUTYL	C	PE/EVAL/PE	C
NATURAL+NEOPRENE+NITRILE	C	PVA	C
NATURAL RUBBER	C	PVC	C
NEOPRENE	C	SARANEX-23	C
NITRILE	C		

* CPI - Chemwatch Performance Index

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.

RESPIRATORY PROTECTION: Type AK-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	AK-AUS / Class 1 P3	-	AK-PAPR-AUS / Class 1 P3
Up to 25 x ES	Air-line*	AK-2 P23	A-PAPR-2 P3
Up to 50 x ES	-	AK-3 P23	-
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).

9. PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE:	Brown liquid, does not mix with water
PHYSICAL STATE:	Liquid
ODOUR:	Petroleum odour
ODOUR THRESHOLD:	Not available
pH (as supplied):	Not applicable
FREEZING/MELTING POINT (°C):	Not available
INITIAL BOILING POINT/RANGE (°C):	Not available
FLASH POINT (°C):	> 61
EVAPORATION RATE:	Not available
FLAMMABILITY:	Combustible
EXPLOSION LIMITS (%):	Not available
VAPOUR PRESSURE (kPa):	Not available
SOLUBILITY IN WATER (g/L):	Immiscible
VAPOUR DENSITY (AIR=1):	> 1
RELATIVE DENSITY (WATER = 1):	~ 0.82
PARTITION COEEFICIENT: (N-OCTANOL / WATER)	Not available
AUTO-IGNITION TEMPERATURE (°C):	Not available
DECOMPOSITION TEMPERATURE:	Not available
VISCOSITY (cSt):	Not available
MOLECULAR WEIGHT (g/mol):	Not applicable
TASTE:	Not available
EXPLOSION PROPERTIES:	Not available

9. PHYSICAL AND CHEMICAL PROPERTIES (CONT)

OXIDISING PROPERTIES:	Not available
SURFACE TENSION (dyn/cm or mN/m):	Not available
VOLATILE COMPONENT (%vol):	Not available
GAS GROUP:	Not available
pH (as a solution 1%):	Not applicable
VOC (g/L):	Not available

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.
POSSIBLE HAZARDOUS REACTIONS:	See section 7.
CONDITIONS TO AVOID:	See section 7.
INCOMPATIBLE MATERIALS:	See section 7.
HAZARDOUS DECOMPOSITION: PRODUCTS	See section 5.

11. TOXICOLOGICAL INFORMATION

INHALED:	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 hazard is increased at higher temperatures. Acute effects from inhalation of high concentrations of gas/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.
INGESTION:	The liquid is highly discomforting. Ingestion may result in nausea, pain, vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis.

11. TOXICOLOGICAL INFORMATION (CONT)

SKIN CONTACT:

Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant 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. The material may accentuate any pre-existing skin condition.

EYE:

When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.

CHRONIC:

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. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

	TOXICITY	IRRITATION
Hi-Tec Bug Rid	Not available	Not available
Middle distillate	Dermal (rabbit) LD50: > 4200 mg/kg[1] Oral (rat) LD50: 7560 mg/kg[1]	Not available
Kerosene, (petroleum), hydrosulfurised)	Dermal (rabbit) LD50: >2000 mg/kg[1] Oral (rat) LD50: > 5000 mg/kg[1]	Not available

11. TOXICOLOGICAL INFORMATION (CONT)

	TOXICITY	IRRITATION
Ethylene glycol monobutyl ether	Dermal (rat) LD50: > 2000 mg/kg[1] Inhalation (rat) LC50: 450 ppm/4hr[2] Oral (rat) LD50: 250 mg/kg[2]	Eye (rabbit): 100mg SEVERE Eye (rabbit): 100mg/24h-moderate Skin (rabbit): 500 mg, open; mild
Aromatic 150	Dermal (rabbit) LD50: > 1900 mg/kg[1] Dermal (rat) LD50: > 2000 mg/kg[1] Inhalation (rat) LC50: > 0.59 mg/L/4hr[2] Inhalation (rat) LC50: > 3670 ppm/8h*[2] Oral (rat) LD50: >2000 mg/kg[1] Oral (rat) LD50: >4500 mg/kg[1]	Not available
Nonylphenol, ethoxylated	Dermal (rabbit) LD50: 2080 mg/kg[2] Oral (rat) LD50: 1310 mg/kg[2]	Eye (rabbit): 5 mg SEVERE Skin (human): 15 mg/3D mild Skin (rabbit): 500 mg mild
Benzyl-C12-16-alkyldimethylammonium chloride	Oral (rat) LD50: 426 mg/kg[2]	Not available
Isothiazolinones, mixed	Oral (rat) LD50: 53 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*

KEROSENE, (PETROLEUM), HYDRODESULFURISED:

Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than that of iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.

11. TOXICOLOGICAL INFORMATION (CONT)

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 "kerosenes"
Acute toxicity: Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg The dermal LD50s of the same three kerosenes were all >2.0 g//kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No. 8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same material produced an LC50 of >6.4 mg/l. When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced “moderate” to “severe” irritation. Six additional skin irritation studies on a range of kerosenes produced “mild” to “severe” irritation. An eye irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed and washed eyes) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurized kerosene and jet fuel. These materials produced more irritation in the unwashed eyes at 1 hour than had the straight run kerosene. The eye irritation persisted longer than that seen with straight run kerosene, but by day 7 had resolved. Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurized kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in guinea pigs.
Repeat-Dose toxicity: Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects. Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24 respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment-related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from “slight” to “moderate” in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss. Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related:
• increased relative heart weights for the mid- and high- dose males and females,
• increased absolute and relative spleen weights in treated females, and
• differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stress-related and therefore, indirectly related to treatment).
Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group.

11. TOXICOLOGICAL INFORMATION (CONT)

These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes. In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Opthalomological examination of all animals also found no treatment-related effects. There were no treatment-related effects on growth rates, haematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin. A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study . In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m3 kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues.

Carcinogenicity: In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. .Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation . If the equivalent dose of kerosene was applied to the skin in manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred . Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration . The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor. A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice . Animal survivals were not effected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity.

***In-Vitro* (Genotoxicity):** The potential *in vitro* genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation . Modified Ames assays on four kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation . The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results . Hydrodesulfurized kerosene tested in a sister chromatid exchange assay produced negative results (with/without activation).

11. TOXICOLOGICAL INFORMATION (CONT)

***In-Vivo* Genotoxicity:** Multiple *in vivo* genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in *in vivo* bone marrow cytogenetic tests in Sprague-Dawley rats . One of the kerosene samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay . Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation.

Reproductive/Developmental Toxicity Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days premating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compound-related effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/ developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day. Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported . There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds.

ETHYLENE GLYCOL MONOBUTYL ETHER:

For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):
Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids

11. TOXICOLOGICAL INFORMATION (CONT)

containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

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

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

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

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE). Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species. At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol. Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility. Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1).

11. TOXICOLOGICAL INFORMATION (CONT)

The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia wer associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.
1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:
Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol, dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate 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 change 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.

11. TOXICOLOGICAL INFORMATION (CONT)

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.

11. TOXICOLOGICAL INFORMATION (CONT)

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

AROMATIC 150:

for petroleum:
This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.
This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents.
Carcinogenicity: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.
Mutagenicity: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.
Reproductive Toxicity: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus.

11. TOXICOLOGICAL INFORMATION (CONT)

However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.
Human Effects: Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.

NONYLPHENOL ETHOXYLATED:

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture. On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69. Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products . Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity . Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

11. TOXICOLOGICAL INFORMATION (CONT)

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture. On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficultto diagnose ACD to these compounds by patch testing.
Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:
EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)
EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41
EO > 15-20 gives Harmful (Xn) with R22-41
>20 EO is not classified (CESIO 2000)
Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin) .
AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC.
In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO2).Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO2)). The metabolism of C12 AE yields PEG, carboxylic acids, and CO2 as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.
The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed.

11. TOXICOLOGICAL INFORMATION (CONT)

The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations. AEs are not contact sensitisers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust. In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.
For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):
Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm2/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/ cm2/hr . Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series , the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl; ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.
Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo.* The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid . Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

11. TOXICOLOGICAL INFORMATION (CONT)

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur.
Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.
Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.
Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity
In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation . Due to a high incidence of similar spontaneous Changes in normal New Zealand White rabbits , the testicular effects were considered not to be related to treatment . Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.
A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day . In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats. In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity.
Mutagenicity: Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

11. TOXICOLOGICAL INFORMATION (CONT)

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater that the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).
Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with . 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

BENZYL-C12-16-ALKYLDIMETHYLAMMONIUM CHLORIDE:

The material may produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
For alkyldimethylbenzylammonium chlorides (ADMBAC):
Alkyldimethylbenzylammonium chlorides (ADMBAC) are included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC with the following classification: C8-18 ADMBAC are classified as Harmful (Xn) with the risk phrases R21/22 (Harmful in contact with skin and if swallowed) and Corrosive (C) with R34 (Causes burns) and (N) with R50 (Very toxic to aquatic organisms).
Acute toxicity: Absorption of these alkyldimethylbenzylammonium (ADMBAC) cationic surfactants through the skin is anticipated to be low. Different homologues of ADMBAC showed a moderate acute toxicity in experiments with rats and mice. The relationship between alkyl chain length and the acute toxicity of various ADMBAC homologues (C8 to C19) has been studied in mice. The studies indicated that chain lengths above C16 had a markedly lower acute toxicity and that even-numbered alkyl chain homologues appeared to be less toxic than odd-numbered carbon chains. It was suggested that the decrease in toxicity above C16 was due to a decreased water-solubility.
Irritation studies: ADMBAC is a skin irritant in animals at concentrations above 0.1%). A nonspecified ADMBAC caused skin irritation and minor to moderate eye irritation at 0.625 and 1.25% concentrations. Inflammation of the eye and deterioration of vision occurred 3 days after change of soaking solution for a soft contact lens to a solution containing C8-18 ADMBAC.
Sensitisation: The sensitisation potential of ADMBAC has been examined in an experiment including 2,295 patients with suspected allergic contact dermatitis. Some of the patients (5.5%) showed positive reactions after exposure to 0.1% ADMBAC.

11. TOXICOLOGICAL INFORMATION (CONT)

These results were surprising as ADMBAC was not suspected to be a sensitiser. The high irritating potential of ADMBAC, even at low concentrations, could be an explanation of the observed results as the patch test reactions may have been false positives. However, another group of 2,806 patients with eczema was patch tested with 0.1% ADMBAC, and 2.13% of these patients appeared to be sensitised. Skin sensitisation was noted in patients patch tested with ADMBAC in aqueous solutions at 0.07 to 0.1% surfactant. However, there was no incidence of skin sensitisation in a population of normal individuals tested with 0.1% ADMBAC. This indicates that individuals with diseased skin may be at risk for sensitisation to ADMBAC.

Genetic toxicity: C16 ADMBAC did not induce transformation of the cells in an in vitro bioassay for carcinogenesis by using cultures of Syrian golden hamster embryo cells. The mutagenic potential of this surfactant was also examined by using *Salmonella typhimurium* strains - no mutagenic effects were seen). In other short-term genotoxicity assays (Salmonella/microsome assay) and rec-assay (bacterial DNA repair test) C16 ADMBAC was tested for ability to cause DNA damage in bacteria. None of the data indicated any mutagenic effects.

Carcinogenicity: Lifetime studies of ADMBAC were conducted in mice and rabbits that were treated with 8.5 to 17% surfactant dissolved in acetone or methanol. ADMBAC was applied repeatedly to the skin and ADMBAC caused ulceration, inflammations and scars in many animals, but no tumours.

Developmental toxicity: No embryotoxic activity was detected when C18 ADMBAC was applied topically to pregnant rats during the period of major organogenesis (day 6-15) at doses up to 6.6%, which was sufficient to cause adverse maternal reactions. Intravaginal instillation of ADMBAC (single doses up to 200 mg/kg) to pregnant rats on day one of the gestation caused abnormal foetal development and embryotoxicity.

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben
Madsen et al: Miljoministeriet (Danish Environmental Protection Agency)

For quaternary ammonium compounds (QACs):

Quaternary ammonium compounds (QACs) are cationic surfactants. They are synthetic organically tetra-substituted ammonium compounds, where the R substituents are alkyl or heterocyclic radicals. A common characteristic of these synthetic compounds is that one of the R's is a long-chain hydrophobic aliphatic residue. The cationic surface active compounds are in general more toxic than the anionic and non-ionic surfactants. The positively-charged cationic portion is the functional part of the molecule and the local irritation effects of QACs appear to result from the quaternary ammonium cation. Due to their relative ability to solubilise phospholipids and cholesterol in lipid membranes, QACs affect cell permeability which may lead to cell death. Further QACs denature proteins as cationic materials precipitate protein and are accompanied by generalised tissue irritation. It has been suggested that the experimentally determined decrease in acute toxicity of QACs with chain lengths above C16 is due to decreased water solubility. In general it appears that QACs with a single long-chain alkyl groups are more toxic and irritating than those with two such substitutions, The straight chain aliphatic QACs have been shown to release histamine from minced guinea pig lung tissue However, studies with benzalkonium chloride have shown that the effect on histamine release depends on the concentration of the solution. When cell suspensions (11% mast cells) from rats were exposed to low concentrations, a decrease in histamine release was seen.

11. TOXICOLOGICAL INFORMATION (CONT)

When exposed to high concentrations the opposite result was obtained. In addition, QACs may show curare-like properties (specifically benzalkonium and cetylpyridinium derivatives, a muscular paralysis with no involvement of the central nervous system. This is most often associated with lethal doses Parenteral injections in rats, rabbits and dogs have resulted in prompt but transient limb paralysis and sometimes fatal paresis of the respiratory muscles. This effect seems to be transient. From human testing of different QACs the generalised conclusion is obtained that all the compounds investigated to date exhibit similar toxicological properties.
Long term/repeated exposure:
Inhalation: A group of 196 farmers (with or without respiratory symptoms) were evaluated for the relationship between exposure to QACs (unspecified, exposure levels not given) and respiratory disorders by testing for lung function and bronchial responsiveness to histamine. After histamine provocation statistically significant associations were found between the prevalence of mild bronchial responsiveness (including asthma-like symptoms) and the use of QACs as disinfectant. The association seems even stronger in people without respiratory symptoms.

ISOTHIAZOLINONES, MIXED:

The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

MIDDLE DISTILLATE & ISOTHIAZOLINONES, MIXED:

No significant acute toxicological data identified in literature search.

ETHYLENE GLYCOL MONOBUTYL ETHER & NONYLPHENOL, ETHOXYLATED:

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

ETHYLENE GLYCOL MONOBUTYL ETHER & NONYLPHENOL, ETHOXYLATED & ISOTHIAZOLINONES, MIXED:

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.

11. TOXICOLOGICAL INFORMATION (CONT)

AROMATIC 150 & BENZYL-C12-16-ALKYLDIMETHYLAMMONIUM CHLORIDE & ISOTHIAZOLINONES, MIXED:

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.

12. ECOLOGICAL INFORMATION

TOXICITY:

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment. **DO NOT** discharge into sewer or waterways.

Ingredient	Endpoint Test	Duration (hr)	Species	Value	Source
Kerosene, (petroleum), hydrodesulfurised	NOEC	3072	Fish	=1mg/L	1
Ethylene glycol monobutyl ether	LC50	96	Fish	222.042mg/L	3
	EC50	48	Crustacea	>1000mg/L	4
	EC50	96	Algae or other aquatic plants	1081.644mg/L	3
	EC50	384	Crustacea	51.539mg/L	3
	NOEC	96	Crustacea	1000mg/L	4
Aromatic 150	LC50	96	Fish	0.58mg/L	2
	EC50	48	Crustacea	0.76mg/L	2
	EC50	72	Algae or other aquatic plants	<1mg/L	1
	EC50	48	Crustacea	=0.95mg/L	1
	NOEC	72	Algae or other aquatic plants	0.3mg/L	2
	EC50	48	Crustacea	=6.14mg/L	1
	EC50	72	Algae or other aquatic plants	3.29mg/L	1
	EC10	72	Algae or other aquatic plants	1.13mg/L	1
	NOEC	72	Algae or other aquatic plants	=1mg/L	1

12. ECOLOGICAL INFORMATION (CONT)

Nonylphenol, ethoxylated	LC50	96	Fish	1.3mg/L	4
	EC50	48	Crustacea	12.2mg/L	4
	EC50	96	Algae or other aquatic plants	12.0mg/L	4

Ingredient	Endpoint Test	Duration (hr)	Species	Value	Source
	EC50	120	Crustacea	0.15mg/L	4
	NOEC	2400	Fish	0.035mg/L	4
Benzyl-C12-16-alkyldimethylammonium chloride	LC50	96	Fish	0.28mg/L	4
	EC50	48	Crustacea	0.0059mg/L	4
	EC50	96	Algae or other aquatic plants	0.67mg/L	4
	BCF	1440	Fish	0.25mg/L	4
	EC50	48	Crustacea	0.037mg/L	4

LEGEND: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data.

PERSISTENCE & DEGRADABILITY

Ingredient	Persistence: Water/Soil	Persistence: Air
Ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
Nonylphenol, ethoxylated	LOW	LOW

BIOACCUMULATIVE POTENTIAL

Ingredient	Bioaccumulation
Kerosene, (petroleum), hydrodesulfurised	LOW (BCF = 159)
Ethylene glycol monobutyl ether	LOW (BCF = 2.51)
Aromatic 150	LOW (BCF = 159)
Nonylphenol, ethoxylated	LOW (BCF = 16)

MOBILITY IN SOIL

Ingredient	Mobility
Ethylene glycol monobutyl ether	HIGH (KOC = 1)
Nonylphenol, ethoxylated	LOW (KOC = 940)

13. DISPOSAL CONSIDERATIONS

PRODUCT/PACKAGING DISPOSAL: Consult manufacturer for recycling options and recycle where possible . Consult State Land Waste Management Authority for disposal. Incinerate residue at an approved site. Recycle containers if possible, or dispose of in an authorised landfill.

14. TRANSPORT INFORMATION

LABELS REQUIRED:

MARINE POLLUTANT:

HAZCHEM: •3Z

LAND TRANSPORT (ADG)

UN NUMBER: 3082

UN PROPER SHIPPING NAME: ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains kerosene, (petroleum), hydrodesulfurised and middle distillate)

TRANSPORT HAZARD CLASS(ES): Class: 9
Subrisk: Not Applicable

PACKING GROUP: III

ENVIRONMENTAL HAZARD: Not Applicable

SPECIAL PRECAUTIONS FOR USER: Special provisions: 274 331 335 375 AU01
Limited quantity: 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in:
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

AIR TRANSPORT (ICAO-IATA/DGR):

UN NUMBER:	3082	
UN PROPER SHIPPING NAME:	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. * (contains kerosene, (petroleum), hydrodesulfurised and middle distillate)	
TRANSPORT HAZARD CLASS(ES):	ICAO/IATA Class:	9
	ICAO / IATA Subrisk:	Not Applicable
	ERG Code:	9L
PACKING GROUP:	III	
ENVIRONMENTAL HAZARD:	Not Applicable	
SPECIAL PRECAUTIONS FOR USER:	Special provisions	A97 A158 A197
	Cargo Only Packing Instructions	964
	Cargo Only Maximum Qty / Pack	450 L
	Passenger and Cargo Packing Instructions	964
	Passenger and Cargo Maximum Qty / Pack	450 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y964
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

SEA TRANSPORT (IMDG-CODE / GGVSEE)

UN NUMBER:	3082	
UN PROPER SHIPPING NAME:	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains kerosene, (petroleum), hydrodesulfurised and middle distillate)	
TRANSPORT HAZARD CLASS(ES):	IMDG Class:	9
	IMDG Subrisk:	Not Applicable
PACKING GROUP:	III	
ENVIRONMENTAL HAZARD:	Marine Pollutant	
SPECIAL PRECAUTIONS FOR USER:	EMS Number:	F-A, S-F
	Special provisions:	274 335 969
	Limited Quantities:	5 L
Transport in bulk according to Annex II of MARPOL and the IBC code	Not Applicable	

15. REGULATORY INFORMATION

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

MIDDLE DISTILLATE(68476-34-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

KEROSENE, (PETROLEUM), HYDRODESULFURISED(64742-81-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Exposure Standards
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

AROMATIC 150(64742-95-6.) IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Hazardous Substances Information System - Consolidated Lists
Australia Inventory of Chemical Substances (AICS)

NONYLPHENOL, ETHOXYLATED(9016-45-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Inventory of Chemical Substances (AICS)

BENZYL-C12-16-ALKYLDIMETHYLAMMONIUM CHLORIDE(68424-85-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Inventory of Chemical Substances (AICS)

ISOTHIAZOLINONES, MIXED(55965-84-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS
Australia Hazardous Substances Information System - Consolidated Lists

National Inventory	**Status**
Australia - AICS	N (isothiazolinones, mixed)
Canada - DSL	Y
Canada – NDSL	N (middle distillate; isothiazolinones, mixed; aromatic 150; benzyl-C12-16-alkyldimethylammonium chloride; kerosene, (petroleum), hydrodesulfurised; ethylene glycol monobutyl ether)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (isothiazolinones, mixed)
Japan - ENCS	N (middle distillate)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	N (isothiazolinones, mixed)

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)

16. OTHER INFORMATION

OTHER INFORMATION

Ingredients with multiple cas numbers:

Name	**CAS No**
Middle distillate	68476-34-6, 68334-30-5
Aromatic 150	64742-95-6., 64742-94-5
Nonylphenol, ethoxylated	9016-45-9, 26027-38-3, 26571-11-9, 14409-72-4
Isothiazolinones, mixed	55965-84-9, 96118-96-6

CONTACT PERSON/POINT: General Manager 1300 796 009

This information was prepared in good faith from the best information available at the time of issue. It is based on the present level of research and to this extent we believe it is accurate. However, no guarantee of accuracy is made or implied and since conditions of use are beyond our control, all information relevant to usage is offered without warranty. The manufacturer will not be held responsible for any unauthorised use of this information or for any modified or altered versions.

If you are an employer it is your duty to tell your employees, and any others that may be affected, of any hazards described in this sheet and of any precautions that should be taken.

Safety Data Sheets are updated frequently. Please ensure you have a current copy.

LITERATURE REFERENCES:

* NOHSC: 2011 National Code of Practice for the preparation of Safety Data Sheets.
* Safe Work Australia: 2016 Preparation of Safety Data Sheets for Hazardous Chemicals.
* NOHSC: 1008 Approved Criteria for Classifying Hazardous Substances.
* NOHSC: 10005 List of Designated Hazardous Substances.
* NOHSC: 1005 Control of Workplace Hazardous Substances, National Code of Practice.
* NOHSC: 2007 Control of Workplace Hazardous Substances, National Code of Practice.
* NOHSC: 1003 Exposure Standards for Atmospheric Contaminants in the Occupational Environment, National Exposure Standards.
* NOHSC: 3008 Exposure Standards for Atmospheric Contaminants in the Occupational Environment, Guidance Note.
* NOHSC: 1015 Storage and Handling of Workplace Dangerous Goods, National Standard.
* NOHSC: 2017 Storage and Handling of Workplace Dangerous Goods, National Code of Practice.
* SUSDP: Standard for the Uniform Scheduling of Drugs and Poisons.
* ADG: Australian Dangerous Goods Code.
* SDS of component materials.

LAST CHANGE: Supercedes document issued: 3 August 2017.
Reason/s for revision: Minor editorial changes to comply with GHS requirements.

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END OF SDS