



Nulon Lifter Free and Tune Up

Nulon Products

Chemwatch: 4799-30

Version No: 2.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: 01/16/2013

Print Date: 08/04/2015

Initial Date: Not Available

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

Product Identifier

Product name	Nulon Lifter Free and Tune Up
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions. Additive to engine oil for the removal of gum, carbon and varnish from internal parts.
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Details of the manufacturer/importer

Registered company name	Nulon Products
Address	17 Yulong Close Moorebank 2170 NSW Australia
Telephone	+61 2 9608 7800
Fax	+61 2 9601 4700
Website	Not Available
Email	msds@nulon.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	Not Available
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

COMBUSTIBLE LIQUID, regulated for storage purposes only

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	1	2
Toxicity	2	3
Body Contact	2	3
Reactivity	2	3
Chronic	2	3

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

Poisons Schedule	S5
GHS Classification ^[1]	Flammable Liquid Category 4, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, STOT - SE (Resp. Irr.) Category 3, STOT - SE (Narcosis) Category 3, Aspiration Hazard Category 1, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements



SIGNAL WORD

DANGER**Hazard statement(s)**

H227	Combustible liquid
H302	Harmful if swallowed
H312	Harmful in contact with skin
H332	Harmful if inhaled
H315	Causes skin irritation
H319	Causes serious eye irritation
H335	May cause respiratory irritation
H336	May cause drowsiness or dizziness
H304	May be fatal if swallowed and enters airways
H412	Harmful to aquatic life with long lasting effects
AUH019	May form explosive peroxides

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider
P331	Do NOT induce vomiting.
P362	Take off contaminated clothing.
P363	Wash contaminated clothing before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam for extinction.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
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111-76-2	10-30	<u>ethylene glycol monobutyl ether</u>
64742-94-5	10-30	<u>solvent naphtha petroleum, heavy aromatic</u>
112-80-1	1-10	<u>oleic acid</u>
7664-41-7	1-10	<u>ammonia anhydrous liquefied</u>
71-23-8	1-10	<u>n-propanol</u>
64742-53-6.	NotSpec.	<u>naphthenic distillate, light, hydrotreated (severe)</u>
9003-27-4	NotSpec.	<u>isobutylene homopolymer</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol.

Indication of any immediate medical attention and special treatment needed

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]

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

Followed acute or short term repeated exposures to ethylene glycol monoalkyl ethers and their acetates:

- ▶ Hepatic metabolism produces ethylene glycol as a metabolite.
- ▶ Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.
- ▶ Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

	<ul style="list-style-type: none"> ▶ Water spray or fog. ▶ Alcohol stable foam. ▶ Dry chemical powder. ▶ Carbon dioxide.
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Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ 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. <p>Combustion products include; carbon dioxide (CO₂) other pyrolysis products typical of burning organic material May emit poisonous fumes.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	<ul style="list-style-type: none"> ▶ Remove all ignition sources. ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal. 																																																																											
Major Spills	<p>Chemical Class: ester and ethers For release onto land: recommended sorbents listed in order of priority.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 30%;">SORBENT TYPE</th> <th style="width: 10%;">RANK</th> <th style="width: 30%;">APPLICATION</th> <th style="width: 10%;">COLLECTION</th> <th style="width: 20%;">LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>shovel</td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>wood fiber - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, W, P, DGC</td> </tr> <tr> <td>wood fiber - pillow</td> <td>3</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>treated wood fiber - pillow</td> <td>3</td> <td>throw</td> <td>pitchfork</td> <td>DGC, RT</td> </tr> <tr> <td colspan="5">LAND SPILL - MEDIUM</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>2</td> <td>throw</td> <td>skiploader</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>W, SS, DGC</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> <tr> <td>wood fiber - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, W, P, DGC</td> </tr> </tbody> </table> <p>Legend DGC: Not effective where ground cover is dense R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT: Not effective where terrain is rugged SS: Not for use within environmentally sensitive sites W: Effectiveness reduced when windy Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control; R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988 Moderate hazard.</p> <ul style="list-style-type: none"> ▶ 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. 	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					cross-linked polymer - particulate	1	shovel	shovel	R, W, SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	2	shovel	shovel	R, I, P	wood fiber - particulate	3	shovel	shovel	R, W, P, DGC	wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT	treated wood fiber - pillow	3	throw	pitchfork	DGC, RT	LAND SPILL - MEDIUM					cross-linked polymer - particulate	1	blower	skiploader	R, W, SS	cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT	sorbent clay - particulate	3	blower	skiploader	R, I, P	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC	wood fiber - particulate	4	blower	skiploader	R, W, P, DGC
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- ▶ 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.

Personal Protective Equipment advice is contained in Section 8 of the MSDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Containers, even those that have been emptied, may contain explosive vapours. ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Electrostatic discharge may be generated during pumping - this may result in fire. ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (≤ 1 m/sec until fill pipe submerged to twice its diameter, then ≤ 7 m/sec). ▶ Avoid splash filling. ▶ Do NOT use compressed air for filling discharging or handling operations. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this MSDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this MSDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ DO NOT use aluminium or galvanised containers ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	96.9 mg/m ³ / 20 ppm	242 mg/m ³ / 50 ppm	Not Available	Sk
Australia Exposure Standards	ammonia anhydrous liquefied	Ammonia	17 mg/m ³ / 25 ppm	24 mg/m ³ / 35 ppm	Not Available	Not Available
Australia Exposure Standards	n-propanol	Propyl alcohol	492 mg/m ³ / 200 ppm	614 mg/m ³ / 250 ppm	Not Available	Sk
Australia Exposure Standards	naphthenic distillate, light, hydrotreated (severe)	Oil mist, refined mineral	5 mg/m ³	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	20 ppm	20 ppm	700 ppm
oleic acid	Octadecenoic acid, 9-; (Oleic acid)	220 mg/m ³	2400 mg/m ³	15000 mg/m ³
ammonia anhydrous liquefied	Ammonia	Not Available	Not Available	Not Available
n-propanol	Propyl alcohol, n-; (n-Propanol)	250 ppm	250 ppm	4000 ppm
naphthenic distillate, light, hydrotreated (severe)	Transformer oil; (Mineral oil, petroleum distillates, hydrotreated (mild) light naphthenic)	0.35 mg/m ³	3.8 mg/m ³	990 mg/m ³

Ingredient	Original IDLH	Revised IDLH
ethylene glycol monobutyl ether	700 ppm	700 [Unch] ppm
solvent naphtha petroleum, heavy aromatic	Not Available	Not Available
oleic acid	Not Available	Not Available
ammonia anhydrous liquefied	500 ppm	300 ppm
n-propanol	4,000 ppm	800 ppm
naphthenic distillate, light, hydrotreated (severe)	Not Available	Not Available
isobutylene homopolymer	Not Available	Not Available

MATERIAL DATA

For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL)

For n-propanol:

IDLH Level: 800 ppm

Odour threshold level: 0.13-30 ppm.

Because n-propanol has the same biological effects as isopropanol but is less toxic, the TLV-TWA is intermediate between the limits for isopropyl alcohol and the butanols. Exposure at or below this limit is thought to protect the worker against the significant risk of narcosis and irritation.

Odour Safety Factor(OSF)

OSF=77 (n-PROPANOL)

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 L: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 3% DMSO extract as measured by IP 346.

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

Exposure controls

Appropriate engineering controls	<p>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.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and</p>
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enclosed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

Personal protection



Eye and face protection

- ▶ Safety glasses with side shields.
- ▶ Chemical goggles.
- ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- ▶ Wear chemical protective gloves, e.g. PVC.
 - ▶ Wear safety footwear or safety gumboots, e.g. Rubber
- The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
- The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
- Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
- ▶ frequency and duration of contact,
 - ▶ chemical resistance of glove material,
 - ▶ glove thickness and
 - ▶ dexterity
- Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
- ▶ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - ▶ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - ▶ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
 - ▶ Contaminated gloves should be replaced.
- Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Body protection

See Other protection below

Other protection

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

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

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

Nulon Lifter Free and Tune Up

Material	CPI
NEOPRENE	B
NATURAL RUBBER	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 P2	-	AK-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	AK-2 P2	AK-PAPR-2 P2
up to 50 x ES	-	AK-3 P2	-
50+ x ES	-	Air-line**	-

^ - Full-face

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES**Information on basic physical and chemical properties**

Appearance	Colourless to pale yellow liquid with a characteristic odour; partially miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	<1
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	62	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Combustible.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Partly Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.</p> <p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.</p> <p>Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>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.</p>
Eye	<p>Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.</p>

	<p>Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.</p> <p>Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</p> <p>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.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Studies with some ethylene glycol ethers and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of the glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decrease significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats. This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Ethylene glycol ethers and acetates are mainly metabolised to alkoxyacetic acids but there is also a minor pathway through ethylene glycol to oxalic acid. The main pathway of ethylene glycol ethers is associated with significant clinical or experimental health effects, but the minor pathway is also interesting because formation of urinary stones depends principally upon urinary concentration of oxalate and calcium. In one study (1) the tendency to form urinary stones was 2.4 times higher amongst silk-screen printers exposed to ethylene glycol ethers, than among office workers. (1) Laitinen J., et al: Occupational Environmental Medicine 1996, 53 595-600</p>

Nulon Lifter Free and Tune Up	TOXICITY	IRRITATION
	Not Available	Not Available
ethylene glycol monobutyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	* [Union Carbide]
	Inhalation (rat) LC50: 450 ppm/4H ^[2]	Eye (rabbit): 100 mg SEVERE
	Oral (rat) LD50: 250 mg/kg ^[2]	Eye (rabbit): 100 mg/24h-moderate Skin (rabbit): 500 mg, open; mild
solvent naphtha petroleum, heavy aromatic	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	[PETROFIN]
	Inhalation (rat) LC50: >0.59 mg/L/4H ^[2]	Eye (rabbit): Irritating
	Oral (rat) LD50: >2000 mg/kg ^[1]	
oleic acid	TOXICITY	IRRITATION
	Oral (rat) LD50: 25000 mg/kg ^[2]	Skin (human):15 mg/3d-l- moderate Skin (rabbit):500 mg mild
ammonia anhydrous liquefied	TOXICITY	IRRITATION
	dermal (rat) LD50: 4.84 mg/L/60M ^[2]	Nil reported
	Inhalation (rat) LC50: 2000 ppm/4H ^[2]	
	Inhalation (rat) LC50: 9500 ppm/1H ^[2]	
	Oral (rat) LD50: 350 mg/kg ^[1]	

n-propanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 4032 mg/kg ^[1]	Eye (rabbit): 20 mg/24h moderate
	Oral (rat) LD50: 1870 mg/kg ^[2]	Eye (rabbit): 4 mg open SEVERE
		Skin (rabbit): 20 mg/24h moderate
naphthenic distillate, light, hydrotreated (severe)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	* [MORTON]
	Inhalation (rat) LC50: >3.9 mg/l4 h ^[1]	
	Inhalation (rat) LC50: >4.7 mg/l4 h ^[1]	
	Inhalation (rat) LC50: >5 mg/l4 h ^[1]	
	Inhalation (rat) LC50: >5.2 mg/l4 h ^[1]	
	Inhalation (rat) LC50: >5.3 mg/l4 h ^[1]	
	Inhalation (rat) LC50: 10.5 mg/l4 h ^[1]	
	Inhalation (rat) LC50: 5.7 mg/l4 h ^[1]	
	Inhalation (rat) LC50: 9.6 mg/l4 h ^[1]	
Oral (rat) LD50: >2000 mg/kg ^[1]		
isobutylene homopolymer	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[1]	
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's msds. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

Nulon Lifter Free and Tune Up	<p>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.</p> <p>No significant acute toxicological data identified in literature search.</p>
ETHYLENE GLYCOL MONOBUTYL ETHER	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):</p> <p>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.</p> <p>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.</p> <p>Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red</p>

blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

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

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

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

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m³ and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m³), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m³), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m³) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m³ (rabbit-EGPE), 100 ppm or 425 mg/m³ (rat-EGPE), 50 ppm or 241 mg/m³ (rat EGBE) and 100 ppm or 483 mg/m³ (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m³ (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats.

Teratogenic effects were not observed in other species.

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

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning. The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning. Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embryotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

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

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. 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

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC

	<p>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.</p>
OLEIC ACID	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
AMMONIA ANHYDROUS LIQUEFIED	<p>No significant acute toxicological data identified in literature search.</p> <p>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.</p>
N-PROPANOL	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
NAPHTHENIC DISTILLATE, LIGHT, HYDROTREATED (SEVERE)	<p>The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives;</p> <p>The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:</p> <ul style="list-style-type: none"> ▶ The adverse effects of these materials are associated with undesirable components, and ▶ The levels of the undesirable components are inversely related to the degree of processing; ▶ Distillate base oils receiving the same degree or extent of processing will have similar toxicities; ▶ The potential toxicity of <i>residual base oils</i> is independent of the degree of processing the oil receives. ▶ The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing. <p>Unrefined & mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size. Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing</p> <p>Highly and Severely Refined Distillate Base Oils</p> <p>Acute toxicity: Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to > 4 mg/l.</p> <p>When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating"</p> <p>Testing in guinea pigs for sensitization has been negative</p> <p>Repeat dose toxicity: Several studies have been conducted with these oils. The weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil's toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.</p> <ul style="list-style-type: none"> ▶ The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive, ▶ The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and ▶ The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials. <p>Reproductive and developmental toxicity: A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study's authors.</p> <p>A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage,</p>

on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed fetuses were found among three litters. The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.

Genotoxicity:

In vitro (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay. Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.

In vivo (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.

Carcinogenicity: Highly & severely refined base oils are not carcinogens, when given either orally or dermally.

The substance is classified by IARC as Group 3:

NOT classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

ISOBUTYLENE HOMOPOLYMER	No significant acute toxicological data identified in literature search.
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Acute Toxicity	✓	Carcinogenicity	⊘
Skin Irritation/Corrosion	✓	Reproductivity	⊘
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	⊘	STOT - Repeated Exposure	⊘
Mutagenicity	⊘	Aspiration Hazard	✓

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

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
ethylene glycol monobutyl ether	Not Available					
solvent naphtha petroleum, heavy aromatic	Not Available					
oleic acid	Not Available					
ammonia anhydrous liquefied	Not Available					
n-propanol	Not Available					
naphthenic distillate, light, hydrotreated (severe)	Not Available					
isobutylene homopolymer	Not Available					

Harmful to aquatic organisms.

May cause long-term adverse effects in the aquatic environment.

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

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

When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the oxygen transfer between the air and the water.

Oils of any kind can cause:

- ▶ drowning of water-fowl due to lack of buoyancy, loss of insulating capacity of feathers, starvation and vulnerability to predators due to lack of mobility
- ▶ lethal effects on fish by coating gill surfaces, preventing respiration
- ▶ asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom and
- ▶ adverse aesthetic effects of fouled shoreline and beaches

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.

For ethylene glycol monoalkyl ethers and their acetates:

Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE)

Environmental fate:

The ethers, like other simple glycol ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates

possess an ester group that hydrolyses in neutral ambient water under abiotic conditions.

Level III fugacity modeling indicates that category members, when released to air and water, will partition predominately to water and, to a lesser extent, to air and soil. Estimates of soil and sediment partition coefficients (Kocs ranging from 1- 10) suggest that category members would exhibit high soil mobility. Estimated bioconcentration factors (log BCF) range from 0.463 to 0.732. Biodegradation studies indicate that all category members are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment.

Ecotoxicity:

Glycol ether acetates do not hydrolyse rapidly into their corresponding glycol ethers in water under environmental conditions. The LC50 or EC50 values for EGHE are lower than those for EGPE and EGBE (which have shorter chain lengths and lower log Kow values). Overall, the LC50 values for the glycol ethers in aquatic species range from 94 to > 5000 mg/L. For EGHE, the 96-hour LC50 for *Brachydanio rerio* (zebra fish) is between 94 and mg/L, the 48-hour EC50 for *Daphnia magna* was 145 mg/L and the 72-hour EC50 values for biomass and growth rate of algae (*Scenedesmus subspicatus*) were 98 and 198 mg/L, respectively. LC50/EC50 values for EGPE and EGBE in aquatic species are 835 mg/l or greater.

Aquatic toxicity data for EGBEA show a 96-hour LC50 of 28.3 mg/L for rainbow trout (*Oncorhynchus mykiss*), a 48-hour LC50 of 37-143 mg/L for *Daphnia magna*, a 72-hour EC50 of greater than 500 mg/L for biomass or growth rate of algae (*Scenedesmus subspicatus* and *Pseudokirchneriella subcapitata*, respectively), and a 7-day EC10 of 30.4 mg/L and a NOEC of 16.4 mg/L for reproduction in *Ceriodaphnia dubia*.

For glycol ethers:

Environmental fate:

Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity:

Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.

Glycols exert a high oxygen demand for decomposition and once released to the environments cause the death of aquatic organisms if dissolved oxygen is depleted.

For hydrocarbons:

Environmental fate:

The lower molecular weight hydrocarbons are expected to form a "slick" on the surface of waters after release in calm sea conditions. This is expected to evaporate and enter the atmosphere where it will be degraded through reaction with hydroxy radicals.

Some hydrocarbon will become associated with benthic sediments, and it is likely to be spread over a fairly wide area of sea floor. Marine sediments may be either aerobic or anaerobic. The material, in probability, is biodegradable, under aerobic conditions (isomerised olefins and alkenes show variable results). Evidence also suggests that the hydrocarbons may be degradable under anaerobic conditions although such degradation in benthic sediments may be a relatively slow process.

Under aerobic conditions hydrocarbons degrade to water and carbon dioxide, while under anaerobic processes they produce water, methane and carbon dioxide.

Alkenes have low log octanol/water partition coefficients (Kow) of about 1 and estimated bioconcentration factors (BCF) of about 10; aromatics have intermediate values (log Kow values of 2-3 and BCF values of 20-200), while C5 and greater alkanes have fairly high values (log Kow values of about 3-4.5 and BCF values of 100-1,500

The estimated volatilisation half-lives for alkanes and benzene, toluene, ethylbenzene, xylene (BTEX) components were predicted as 7 days in ponds, 1.5 days in rivers, and 6 days in lakes. The volatilisation rate of naphthalene and its substituted derivatives were estimated to be slower.

Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds.

Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media.

The final products of microbial degradation are carbon dioxide, water, and microbial biomass. The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics, and the aromatics in the C10-C22 range are the most readily biodegradable; n-alkanes, n-alkyl aromatics, and aromatics in the C5-C9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilisation and thus are unavailable in most environments; n-alkanes in the C1-C4 ranges are biodegradable only by a narrow range of specialised hydrocarbon degraders; and n-alkanes, n-alkyl aromatics, and aromatics above C22 are generally not available to degrading microorganisms. Hydrocarbons with condensed ring structures, such as PAHs with four or more rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is, greater than 7.

All biological transformations are affected by temperature. Generally, as the temperature increases, biological activity tends to increase up to a temperature where enzyme denaturation occurs.

Atmospheric fate: Alkanes, isoalkanes, and cycloalkanes have half-lives on the order of 1-10 days, whereas alkenes, cycloalkenes, and substituted benzenes have half-lives of 1 day or less. Photochemical oxidation products include aldehydes, hydroxy compounds, nitro compounds, and peroxyacyl nitrates. Alkenes, certain substituted aromatics, and naphthalene are potentially susceptible to direct photolysis.

Ecotoxicity:

Hydrocarbons are hydrophobic (high log Kow and low water solubility). Such substances produce toxicity in aquatic organisms by a mechanism referred to as "non-polar narcosis" or "baseline" toxicity. The hydrophobicity increases and water solubility decreases with increasing carbon number for a particular class of hydrocarbon. Substances with the same carbon number show increased hydrophobicity and decreased solubility with increasing saturation. Quantitative structure activity relationships (QSAR), relating both solubility and toxicity to Kow predict that the water solubility of single chemical substances decreases more rapidly with increasing Kow than does the acute toxicity.

Based on test results, as well as theoretical considerations, the potential for bioaccumulation may be high. Toxic effects are often observed in species such as blue mussel, daphnia, freshwater green algae, marine copepods and amphipods.

The values of log Kow for individual hydrocarbons increase with increasing carbon number within homologous series of generic types. Quantitative structure activity relationships (QSAR), relating log Kow values of single hydrocarbons to toxicity, show that water solubility decreases more rapidly with increasing Kow than does the concentration causing effects. This relationship varies somewhat with species of hydrocarbon, but it follows that there is a log Kow limit for hydrocarbons, above which, they will not exhibit acute toxicity; this limit is at a log Kow value of about 4 to 5. It has been confirmed experimentally that for fish and invertebrates, paraffinic hydrocarbons with a carbon number of 10 or higher (log Kow >5) show no acute toxicity and that alkylbenzenes with a carbon number of 14 or greater (log Kow >5) similarly show no acute toxicity.

QSAR equations for chronic toxicity also suggest that there should be a point where hydrocarbons with high log Kow values become so insoluble in water that they will not cause chronic toxicity, that is, that there is also a solubility cut-off for chronic toxicity. Thus, paraffinic hydrocarbons with carbon numbers of greater than 14 (log Kow >7.3) should show no measurable chronic toxicity. Experimental support for this cut-off is demonstrated by chronic toxicity studies on lubricant base oils and one "heavy" solvent grade (substances composed of paraffins of C20 and greater) which show no effects

after exposures to concentrations well above solubility.

The initial criteria for classification of substances as dangerous to the aquatic environment are based upon acute toxicity data in fish, daphnids and algae. However, for substances that have low solubility and show no acute toxicity, the possibility of a long-term or chronic hazard to the environment is recognised in the R53 phrase or so-called "safety net". The R53 assignment for possible long-term harm is a surrogate for chronic toxicity test results and is triggered by substances that are both bioaccumulative and persistent. The indicators of bioaccumulation and persistence are taken as a BCF > 100 (or log Kow > 3 if no BCF data) and lack of ready biodegradability. For low solubility substances which have direct chronic toxicity data demonstrating no chronic toxicity at 1 mg/L or higher, these data take precedence such that no classification for long term toxicity is required.

Drinking Water Standards: hydrocarbon total: 10 ug/l (UK max.).

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
oleic acid	LOW	LOW
ammonia anhydrous liquefied	LOW	LOW
n-propanol	LOW	LOW
isobutylene homopolymer	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)
oleic acid	LOW (LogKOW = 7.7294)
ammonia anhydrous liquefied	LOW (LogKOW = 0.229)
n-propanol	LOW (LogKOW = 0.25)
isobutylene homopolymer	LOW (LogKOW = 2.2256)

Mobility in soil

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (KOC = 1)
oleic acid	LOW (KOC = 11670)
ammonia anhydrous liquefied	LOW (KOC = 14.3)
n-propanol	HIGH (KOC = 1.325)
isobutylene homopolymer	LOW (KOC = 35.04)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and MSDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal.
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- ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- ▶ Where in doubt contact the responsible authority.
- ▶ Recycle wherever possible or consult manufacturer for recycling options.
- ▶ Consult State Land Waste Authority for disposal.
- ▶ Bury or incinerate residue at an approved site.
- ▶ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Labels Required

COMBUSTIBLE LIQUID	COMBUSTIBLE LIQUID, regulated for storage purposes only
Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	oleic acid	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	n-propanol	Y
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	isobutylene homopolymer	X

SECTION 15 REGULATORY INFORMATION

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

ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC(64742-94-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists	Australia Inventory of Chemical Substances (AICS)
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OLEIC ACID(112-80-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

AMMONIA ANHYDROUS LIQUEFIED(7664-41-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft

N-PROPANOL(71-23-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

NAPHTHENIC DISTILLATE, LIGHT, HYDROTREATED (SEVERE)(64742-53-6.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

ISOBUTYLENE HOMOPOLYMER(9003-27-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (n-propanol; isobutylene homopolymer; naphthenic distillate, light, hydrotreated (severe); ammonia anhydrous liquefied; solvent naphtha petroleum, heavy aromatic; ethylene glycol monobutyl ether; oleic acid)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Y
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
isobutylene homopolymer	9003-27-4, 9003-29-6

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

The (M)SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

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