

Apparent Paraffinic Oil Adjuvant

AIRR Apparent Pty Ltd.

Chemwatch: 6977762

Version No: 2.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 3

Initial Date: 16/06/2026

Revision Date: 16/06/2026

Print Date: 17/06/2026

L.GHS.AUS.EN.E

SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Apparent Paraffinic Oil Adjuvant
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains nonylphenol ethoxylates, solvent naphtha petroleum, heavy aromatic, lauryl alcohol, ethoxylated and white mineral oil (petroleum))
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Agricultural spray oil adjuvant to enhance the effectiveness of herbicides. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	AIRR Apparent Pty Ltd.
Address	15/16 Princes Street, Newport NSW 2106 Australia
Telephone	+61 3 5820 8400
Fax	Not Available
Website	www.apparent.com.au
Email	enquiries@apparentag.com.au

Emergency telephone number

Association / Organisation	AIRR Apparent Pty Ltd.
Emergency telephone number(s)	1800 033 111 (24 Hours)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S5
Classification ^[1]	Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

Apparent Paraffinic Oil Adjuvant

H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P271	Use only a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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 hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
8042-47-5	58.2	<u>white mineral oil (petroleum)</u>
9016-45-9	12	<u>nonylphenol ethoxylates</u>
9002-92-0	12	<u>lauryl alcohol, ethoxylated</u>
64742-94-5	<7	<u>solvent naphtha petroleum, heavy aromatic</u>
Not Available	balance	Ingredients determined not to be hazardous
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

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"> ▶ Immediately hold eyelids apart and flush the eye continuously with 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. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ 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.

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Apparent Paraffinic Oil Adjuvant

Ingestion

- ▶ If swallowed do **NOT** induce vomiting.
- ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- ▶ Observe the patient carefully.
- ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- ▶ Seek medical advice.
- ▶ Avoid giving milk or oils.
- ▶ Avoid giving alcohol.
- ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

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

For poisons (where specific treatment regime is absent):

BASIC TREATMENT

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

ADVANCED TREATMENT

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

BRONSTEIN, A.C. and CURRRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

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. Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility

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

Advice for firefighters

Fire Fighting

- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear full body protective clothing with breathing apparatus.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Use water delivered as a fine spray to control fire and cool adjacent area.
- ▶ Avoid spraying water onto liquid pools.
- ▶ **DO NOT** approach containers suspected to be hot.
- ▶ Cool fire exposed containers with water spray from a protected location.
- ▶ If safe to do so, remove containers from path of fire.

Fire/Explosion Hazard

- ▶ Combustible.
 - ▶ Slight fire hazard when exposed to heat or flame.
 - ▶ Heating may cause expansion or decomposition leading to violent rupture of containers.
 - ▶ On combustion, may emit toxic fumes of carbon monoxide (CO).
 - ▶ May emit acrid smoke.
 - ▶ Mists containing combustible materials may be explosive.
- Combustion products include:
- ▶ carbon dioxide (CO₂)
 - ▶ other pyrolysis products typical of burning organic material.

HAZCHEM

- 3Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Apparent Paraffinic Oil Adjuvant

Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ 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>Environmental hazard - contain spillage. 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. ▶ 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 SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Overheating of ethoxylates/ alkoxyates in air should be avoided. When some ethoxylates are heated vigorously in the presence of air or oxygen, at temperatures exceeding 160 C, they may undergo exothermic oxidative degeneration resulting in self-heating and autoignition. ▶ Nitrogen blanketing will minimise the potential for ethoxylate oxidation. Prolonged storage in the presence of air or oxygen may cause product degradation. Oxidation is not expected when stored under a nitrogen atmosphere. Inert gas blanket and breathing system needed to maintain color stability. Use dry inert gas having at least -40 C dew point. ▶ Trace quantities of ethylene oxide may be present in the material. Although these may accumulate in the headspace of storage and transport vessels, concentrations are not expected to exceed levels which might produce a flammability or worker exposure hazard. ▶ Avoid skin contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<p>For alkyl aromatics: The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.</p> <ul style="list-style-type: none"> ▶ Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen ▶ Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids. ▶ Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides. ▶ Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily. ▶ Alkali metals accelerate the oxidation while CO₂ as co-oxidant enhances the selectivity. ▶ Microwave conditions give improved yields of the oxidation products. ▶ Photo-oxidation products may occur following reaction with hydroxyl radicals and NO_x - these may be components of photochemical smogs. <p>Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007</p> <ul style="list-style-type: none"> ▶ Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents. ▶ Aromatics can react exothermically with bases and with diazo compounds. <p>· CARE: Water in contact with heated material may cause foaming or a steam explosion with possible severe burns from wide scattering of hot material. Resultant overflow of containers may result in fire.</p> <p>· Oil leaks in a pressurized circuit may result in a fine flammable spray (the lower flammability limit for oil mist is reached for a concentration of about 45 g/m³)</p>

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Apparent Paraffinic Oil Adjuvant

· Autoignition temperatures may be significantly lower under particular conditions (slow oxidation on finely divided materials..

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	white mineral oil (petroleum)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available

MATERIAL DATA

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

OSHA (USA) concluded that exposure to sensory irritants can:

- ▶ cause inflammation
- ▶ cause increased susceptibility to other irritants and infectious agents
- ▶ lead to permanent injury or dysfunction
- ▶ permit greater absorption of hazardous substances and
- ▶ acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

for mineral oils (excluding metal working fluids), pure, highly and severely refined:

Human exposure to oil mist alone has not been demonstrated to cause health effects except at levels above 5 mg/m3 (this applies to particulates sampled by a method that does not collect vapour). It is not advisable to apply this standard to oils containing unknown concentrations and types of additive.

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. 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>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. 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.</p>										
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<p>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.</p>											

Individual protection measures, such as personal protective equipment	
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Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ 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].
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Apparent Paraffinic Oil Adjuvant

Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Respiratory protection

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - 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)

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	Clear pale yellow liquid.		
Physical state	Liquid	Relative density (Water = 1)	0.9
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 (°C)	Not Available

Continued...

Apparent Paraffinic Oil Adjuvant

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)	>180	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	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	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	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

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	There is sufficient evidence to classify this material as an aspiration hazard

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Nonionic surfactants may produce localised irritation of the oral or gastrointestinal mucosa and induce vomiting and mild diarrhoea.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants

Apparent Paraffinic Oil Adjuvant

	<p>The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives .</p> <p>Irritation and skin reactions are possible with sensitive skin</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 style="text-align: center;">Eye</p>	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p> <p>Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation.</p>																																		
<p style="text-align: center;">Chronic</p>	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion 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>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.</p> <p>The alkyl phenolics (which may occur as breakdown products of some polyethoxylated surfactants) have been implicated in a phenomenon which has apparently occurred since the mid 1960s, namely lower sperm counts and reduced fertility in males. Nonyl phenol acts like an oestrogen hormone which stimulates breast cells to divide in vitro. When pregnant rats are fed nonylphenols at doses comparable to that at which humans might be exposed, male offspring had significantly smaller testicles and lower sperm counts. Although the human foetus is "bathed" in naturally occurring oestrogens during pregnancy it is suggested that it has developed a protective mechanism against natural oestrogens but is not safe from synthetic variants. These tend to accumulate in body fats which sets them apart from the natural product. During early pregnancy, fats are broken down and may flood the body with concentrated pollutants. Drinking water may be one source of exposure to alkyl phenols as many polyethoxylated surfactants are discharged to water treatment systems where they undergo degradation. Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.</p> <p>Principal route of exposure is by skin contact; lesser exposures include inhalation of fumes from hot oils, oil mists or droplets. Prolonged contact with mineral oils carries with it the risk of skin conditions such as oil folliculitis, eczematous dermatitis, pigmentation of the face (melanosis) and warts on the sole of the foot (plantar warts). With highly refined mineral oils no appreciable systemic effects appear to result through skin absorption.</p> <p>Exposure to oil mists frequently elicits respiratory conditions, such as asthma; the provoking agent is probably an additive. High oil mist concentrations may produce lipoid pneumonia although clinical evidence is equivocal. In animals exposed to concentrations of 100 mg/m3 oil mist, for periods of 12 to 26 months, the activity of lung and serum alkaline phosphatase enzyme was raised; 5 mg/m3 oil mist did not produce this response. These enzyme changes are sensitive early indicators of lung damage. Workers exposed to vapours of mineral oil and kerosene for 5 to 35 years showed an increased prevalence of slight basal lung fibrosis.</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p>																																		
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Apparent Paraffinic Oil Adjuvant

	TOXICITY	IRRITATION
lauryl alcohol, ethoxylated	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye (Rodent - rabbit): 100mg
	Oral (Rat) LD50: 1000 mg/kg ^[2]	Eye (Rodent - rabbit): 100mg/24H - Moderate
		Eye (Rodent - rabbit): 10mg
		Eye (Rodent - rabbit): 750ug/24H - Severe
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Human - woman): 1%/72H
		Skin (Human): 6mg/3D - Moderate
		Skin (Rodent - rabbit): 100mg
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Moderate
	Skin (Rodent - rabbit): 75mg/24H - Mild	
	Skin: no adverse effect observed (not irritating) ^[1]	
solvent naphtha petroleum, heavy aromatic	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye (Rodent - rabbit): 100uL/24H - Moderate
	Inhalation (Rat) LC50: >0.003 mg/L4h ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (Rodent - rabbit): 500uL/24H - Mild
		Skin (Rodent - rabbit): 500uL/24H - Moderate
		Skin: adverse effect observed (irritating) ^[1]
	Skin: no adverse effect observed (not irritating) ^[1]	

Legend: 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

Apparent Paraffinic Oil Adjuvant	<p>Data demonstrate that during inhalation exposure, aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure.</p> <p>Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism.</p> <p>The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.</p>
WHITE MINERAL OIL (PETROLEUM)	Oral (rat) TCLo: 92000 mg/kg/92D-Cont. Generally the toxicity and irritation is of low order. White oils and highly/solvent refined oils have not shown the long term risk of skin cancer that follows persistent skin contamination with some other mineral oils, due in all probability to refining that produces low content of both polyaromatics (PAH) and benz-alpha-pyrenes (BaP)
NONYLPHENOL ETHOXYLATES	<p>Oral (rat) TDLo: 150 mg/kg/3D-I Skin (rabbit): 500 mg mild</p> <p>For nonylphenol and its compounds:</p> <p>Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor). Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogenous hormone for binding with the estrogen receptors ERalpha and ERbeta.</p> <p>Effects in pregnant women.</p> <p>Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These "low doses" ranged from 10-13-10-9 M, which is lower than what is generally found in the environment.</p> <p>Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications.</p> <p>Effects on metabolism</p> <p>Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an</p>

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Apparent Paraffinic Oil Adjuvant

	<p>estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats.</p> <p>Cancer Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease for nonylphenol:</p> <p>Nonylphenol was studied for oral toxicity in rats in a 28-day repeat dose toxicity test at doses of 0, 4, 15, 60 and 250 mg/kg/day. Changes suggesting renal dysfunction were mainly noted in both sexes given 250 mg/kg. Liver weights were increased in males given 60 mg/kg and in both sexes given 250 mg/kg group. Histopathologically, hypertrophy of the centrilobular hepatocytes was noted in both sexes given 250 mg/kg. Kidney weights were increased in males given 250 mg/kg and macroscopically, disseminated white spots, enlargement and pelvic dilatation were noted in females given 250 mg/kg. Histopathologically, the following lesions were noted in the 250 mg/kg group: basophilic change of the proximal tubules in both sexes, single cell necrosis of the proximal tubules, inflammatory cell infiltration in the interstitium and casts in females, basophilic change and dilatation of the collecting tubules in both sexes, simple hyperplasia of the pelvic mucosa and pelvic dilatation in females. In the urinary bladder, simple hyperplasia was noted in both sexes given 250 mg/kg. In the caecum, macroscopic dilatation was noted in both sexes given 250 mg/kg. Almost all changes except those in the kidney disappeared after a 14-day recovery period. The NOELs for males and females are considered to be 15 mg/kg/day and 60 mg/kg/day, respectively, under the conditions of the present study.</p> <p>Nonylphenol was not mutagenic to Salmonella typhimurium, TA100, TA1535, TA98, TA1537 and Escherichia coli WP2 uvrA, with or without an exogenous metabolic activation system.</p> <p>Nonylphenol induced neither structural chromosomal aberrations nor polyploidy in CHL/IU cells, in the absence or presence of an exogenous metabolic activation system.</p>
<p>LAURYL ALCOHOL, ETHOXYLATED</p>	<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 on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.</p>
<p>SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC</p>	<p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.</p> <p>For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation.</p> <p>Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans.</p> <p>Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants).</p> <p>Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus.</p> <p>Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials.</p> <p>Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.</p>
<p>Apparent Paraffinic Oil Adjuvant & NONYLPHENOL ETHOXYLATES & LAURYL ALCOHOL, ETHOXYLATED</p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p> <p>Polyethers (such as ethoxylated surfactants and polyethylene glycols) are highly susceptible to being oxidized in the air. They then form complex mixtures of oxidation products.</p> <p>Animal testing reveals that whole the pure, non-oxidised surfactant is non-sensitizing, many of the oxidation products are sensitizers. The oxidation products also cause irritation.</p>
<p>Apparent Paraffinic Oil Adjuvant & WHITE MINERAL OIL (PETROLEUM)</p>	<p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans.</p> <p>Evidence of carcinogenicity may be inadequate or limited in animal testing.</p> <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>The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential.</p> <p>Unrefined and 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.</p> <p>Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil is 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>Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method.</p> <p>Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class (Other Lubricant Base Oils).</p>

Apparent Paraffinic Oil Adjuvant

Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class (Other Lubricant Base Oils))

Germ cell mutagenicity: The tests performed within the "in vivo" studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class (Other Lubricant Base Oils)).

Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction.

STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m³ and for systemic effects NOAEL > 980 mg/m³.

Sub-chronic toxicity

90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies).

Repeat dose toxicity:

Oral

NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.

Inhalation

The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m³. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m³.

Dermal

In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.

Toxicity to reproduction:

Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.

Developmental toxicity, teratogenicity:

Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.

The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic

Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.

Highly and Severely Refined Distillate Base Oils

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.

When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating"

Testing in guinea pigs for sensitization has been negative

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.

- ▶ 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.

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.

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, 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.

NONYLPHENOL ETHOXYLATES & LAURYL ALCOHOL, ETHOXYLATED

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Apparent Paraffinic Oil Adjuvant

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose allergic contact dermatitis (ACD) to these compounds by patch testing.

Overall, alcohol alkoxylates (AAs) are not expected to be systemically toxic, although some short chain ethylene glycol ethers, e.g. methyl and ethyl homologues are of concern for a range of adverse health effects. They include skin and eye irritation, liver and kidney damage, bone marrow and central nervous system (CNS) depression, testicular atrophy, developmental toxicity, and immunotoxicity. For higher propyl and butyl homologues, the toxicity involves haemolysis (anaemia) with secondary effects relating to haemosiderin accumulation in the spleen, liver and kidney, and compensatory haematopoiesis in the bone marrow. Systemic toxicity was shown to decrease with increasing alkyl chain lengths and/or alkoxylation degrees (ECETOC, 2005; US EPA, 2010). The chemicals ethylene glycol hexyl ether (with a longer alkyl chain length, CAS No. 112-25-4) and diethylene glycol butyl ether (with a higher ethoxylation degree, CAS No. 112-34-5) have no evidence of systemic effects including haemolysis.

Commercially available AAs are mixtures of homologues of varying carbon chain lengths and it is possible that some of the chemicals with an average alkyl chain length $C \geq 6$ may also contain shorter alkyl chains $C < 6$. It is not practical to quantify the proportion of shorter $C < 6$ chain lengths present in such chemicals, or these shorter chain lengths may not be present at all. The available data suggest a lack of systemic toxicity for the AE chemicals with potential short alkyl chain presence (NICNASa); therefore, the toxicity of the chemicals in this assessment is unlikely to be significantly affected by the presence of shorter chain alkyl groups.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22-41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).

AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been established that there is a connection between the potential of surfactants to denature protein in vitro and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information in vivo and in vitro demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm²/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm²/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur.

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity.

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME.

Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

Apparent Paraffinic Oil Adjuvant

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats. In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity.

Mutagenicity: Mutagenicity studies have been conducted for several category members. All in vitro and in vivo studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

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

Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

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 either not available or does not fill the criteria for classification
 ✓ – Data available to make classification

SECTION 12 Ecological information

Toxicity					
Apparent Paraffinic Oil Adjuvant	Endpoint	Test Duration (hr)	Species	Value	Source
		Not Available	Not Available	Not Available	Not Available
white mineral oil (petroleum)	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	>1000mg/L	2
nonylphenol ethoxylates	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<0.2	7
	EC50	48h	Crustacea	12.2mg/L	4
	EC50	96h	Algae or other aquatic plants	12mg/l	4
	NOEC(ECx)	2400h	Fish	0.035mg/L	4
LC50	96h	Fish	1-1.8mg/L	4	
lauryl alcohol, ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	1.225mg/l	2
	EC50	48h	Crustacea	1.2mg/L	5
	NOEC(ECx)	672h	Fish	0.139mg/l	2
LC50	96h	Fish	1.5mg/L	4	
solvent naphtha petroleum, heavy aromatic	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	<1mg/l	1
	EC50	48h	Crustacea	0.95mg/l	1
	EC50	96h	Algae or other aquatic plants	11.7mg/l	2
	EC50(ECx)	48h	Crustacea	0.95mg/l	1
LC50	96h	Fish	0.58mg/l	2	
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data				

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

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

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

Continued...

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.

Surfactants are in general toxic to aquatic organisms due to their surface-active properties. Historically, synthetic surfactants were often composed of branched alkyl chains resulting in poor biodegradability which led to concerns about their environmental effects. Today however, many of them, for example those used in large amounts, globally, as detergents, are linear and therefore readily biodegradable and considered to be of rather low risk to the environment. A linear structure of the hydrophobic chain facilitates the approach of microorganism while branching, in particular at the terminal position, inhibits biodegradation. Also, the bioaccumulation potential of surfactants is usually low due to the hydrophilic units. Linear surfactants are not always preferred however, as some branching (that ideally does not hinder ready biodegradability) is often preferable from a performance point of view. The reduction in waste water of organic contaminants such as surfactants can either be a consequence of adsorption onto sludge or aerobic biodegradation in the biological step. Similar sorption and degradation processes occur in the environment as a consequence of direct release of surfactants into the environment from product use, or through effluent discharge from sewage treatment plants in surface waters or the application of sewage sludge on land. However, a major part of surfactants in waste water will be efficiently eliminated in the sewage treatment plant. Although toxic to various organisms, surfactants in general only have a limited effect on the bacteria in the biological step. There are occasions however, where adverse effects have been noticed due to e.g. large accidental releases of softeners from laundry companies.

for lubricating oil base stocks:

Vapor Pressure Vapor pressures of lubricating base oils are reported to be negligible. In one study, the experimentally measured vapour pressure of a solvent-dewaxed heavy paraffinic distillate base oil was $1.7 \times 10^{\text{exp-4}}$ Pa. Since base oils are mixtures of C15 to C50 paraffinic, naphthenic, and aromatic hydrocarbon isomers, representative components of those structures were selected to calculate a range of vapor pressures. The estimated vapor pressure values for these selected components of base oils ranged from $4.5 \times 10^{\text{exp-1}}$ Pa to $2 \times 10^{\text{exp-13}}$ Pa. Based on Dalton's Law the expected total vapour pressure for base oils would fall well below minimum levels ($10^{\text{exp-5}}$ Pa) of recommended experimental procedures.

Partition Coefficient (log Kow): In mixtures such as the base oils, the percent distribution of the hydrocarbon groups (i.e., paraffins, naphthenes, and aromatics) and the carbon chain lengths determines in-part the partitioning characteristics of the mixture. Generally, hydrocarbon chains with fewer carbon atoms tend to have lower partition coefficients than those with higher carbon numbers. However, due to their complex composition, unequivocal determination of the log Kow of these hydrocarbon mixtures cannot be made. Rather, partition coefficients of selected C15 chain-length hydrocarbon structures representing paraffinic, naphthenic, and aromatic constituents in base oil lubricants were modelled. Results showed typical log Kow values from 4.9 to 7.7, which were consistent with values of >4 for lubricating oil basestocks

Water Solubility: When released to water, base oils will float and spread at a rate that is viscosity dependent. While water solubility of base oils is typically very low, individual hydrocarbons exhibit a wide range of solubility depending on molecular weight and degree of unsaturation. Decreasing molecular weight (i.e., carbon number) and increasing levels of unsaturation increases the water solubility of these materials. As noted for partition coefficient, the water solubility of lubricating base oils cannot be determined due to their complex mixture characteristics. Therefore, the water solubility of individual C15 hydrocarbons representing the different groups making up base oils (i.e., linear and branched paraffins, naphthenes, and aromatics) was modelled. Based on water solubility modelling of those groups, aqueous solubilities are typically much less than 1 ppm. (0.003-0.63 mg/l)

Environmental Fate:

Photodegradation: Chemicals having potential to photolyse have UV/visible absorption maxima in the range of 290 to 800 nm. Some chemicals have absorption maxima significantly below 290 nm and consequently cannot undergo direct photolysis in sunlight (e.g. chemicals such as alkanes, alkenes, alkynes, saturated alcohols, and saturated acids). Most hydrocarbon constituents of the materials in this category are not expected to photolyse since they do not show absorbance within the 290-800 nm range. However, photodegradation of polyaromatic hydrocarbons (PAHs) can occur and may be a significant degradation pathway for these constituents of lubricating base oils. The degree and rate at which PAHs may photodegrade depend upon whether conditions allow penetration of light with sufficient energy to effect a change. For example, polycyclic aromatic compounds (PAC) compounds bound to sediments may persist due to a lack of sufficient light penetration

Atmospheric gas-phase reactions can occur between organic chemicals and reactive molecules such as photochemically produced hydroxyl radicals, ozone and nitrogen oxides. Atmospheric oxidation as a result of radical attack is not direct photochemical degradation, but indirect degradation. In general, lubricating base oils have low vapour pressures and volatilisation is not expected to be a significant removal mechanism for the majority of the hydrocarbon components. However, some components (e.g., C15 branched paraffins and naphthenes) appear to have the potential to volatilise Atmospheric half-lives of 0.10 to 0.66 days have been calculated for representative C15 hydrocarbon components of lubricating base oils

Stability in Water: Chemicals that have a potential to hydrolyse include alkyl halides, amides, carbamates, carboxylic acid esters and lactones, epoxides, phosphate esters, and sulfonic acid esters. Because lubricating base oils do not contain significant levels of these functional groups, materials in the lubricating base oils category are not subject to hydrolysis

Chemical Transport and Distribution in the Environment : Based on the physical-chemical characteristics of component hydrocarbons in lubricating base oils, the lower molecular weight components are expected to have the highest vapour pressures and water solubilities, and the lowest partition coefficients. These factors enhance the potential for widespread distribution in the environment. To gain an understanding of the potential transport and distribution of lubricating base oil components, the EQC (Equilibrium Criterion) model was used to characterize the environmental distribution of different C15 compounds representing different structures found in lube oils (e.g., paraffins, naphthenes, and aromatics). The modelling found partitioning to soil or air is the ultimate fate of these C15 compounds. Aromatic compounds partition principally to soil. Linear paraffins partition mostly to soil, while branching appears to allow greater distribution to air. Naphthenes distribute to both soil and air, with increasing proportions in soil for components with the greater number of ring structures. Because the modelling does not take into account degradation factors, levels modelled in the atmosphere are likely overstated in light of the tendency for indirect photodegradation to occur.

Biodegradation: The extent of biodegradation measured for a particular lubricating oil basestock is dependent not only on the procedure used but also on how the sample is presented in the biodegradation test. Lubricant base oils typically are not readily biodegradable in standard 28-day tests. However, since the oils consist primarily of hydrocarbons that are ultimately assimilated by microorganisms, and therefore inherently biodegradable. Twenty-eight biodegradability studies have been reported for a variety of lubricating base oils. Based on the results of ultimate biodegradability tests using modified Sturm and manometric respirometry testing the base oils are expected to be, for the most part, inherently biodegradable. Biodegradation rates found using the modified Sturm procedure ranged from 1.5 to 29%. Results from the manometric respirometry tests on similar materials showed biodegradation rates from 31 to 50%. Biodegradation rates measured in 21-day CEC tests for similar materials ranged from 13 to 79%.

Ecotoxicity:

Numerous acute studies covering fish, invertebrates, and algae have been conducted to assess the ecotoxicity of various lubricating base oils. None of these studies have shown evidence of acute toxicity to aquatic organisms. Eight, 7-day exposure studies using rainbow trout failed to demonstrate toxicity when tested up to the maximum concentration of 1000 mg/L applied as dispersions. Three, 96-hour tests with rainbow trout also failed to show any toxic effects when tested up to 1000 mg/L applied as dispersions. Similarly, three 96-hour tests with fathead minnows at a maximum test concentration of 100 mg/L water accommodated fractions (WAF) showed no adverse effects. Two species of aquatic invertebrates (*Daphnia magna* and *Gammarus* sp.) were exposed to WAF solutions up to 10,000 mg/L for 48 and 96-hours, respectively, with no adverse effects being observed. Four-day exposures of the freshwater green alga (*Scenedesmus subspicatus*) to 500 mg/L WAF solutions failed to show adverse effects on growth rate and algal cell densities in four studies

Multiple chronic ecotoxicity studies have shown no adverse effects to daphnid survival or reproduction. In 10 of 11 chronic studies, daphnids were exposed for 21 days to WAF preparations of lubricating base oils with no ill effects on survival or reproduction at the maximum concentration of 1000 mg/L. One test detected a reduction in reproduction at 1000 mg/L. Additional data support findings of no chronic toxicity to aquatic invertebrates and fish. No observed effect levels ranged from 550 to 5,000 mg/L when tested as either dispersions or WAFs.

The data described above are supported by studies on a homologous series of alkanes. The author concluded that the water solubility of carbon chains C10 is too limited to elicit acute toxicity. This also was shown for alkylbenzene compounds having carbon numbers C15. Since base oils consist of carbon compounds of C15 to C50, component hydrocarbons that are of acute toxicological concern are, for the most part, absent in these materials. Similarly, due to their low solubility, the alkylated two to three ring polyaromatic components in base oils are not expected to cause acute or chronic toxicity. This lack of toxicity is borne out in the results of the reported studies.

The effects of crude and refined oils on organisms found in fresh and sea water has been extensively reviewed.

sea water. Where spillages occur the non-mobile species suffer the greatest mortality, whereas fish species can often escape from the affected region. The extent of the initial mortality depends on the chemical nature of the oil, the location, and the physical conditions, particularly the temperature and wind velocity. Most affected freshwater and marine communities recover from the effects of an oil spill within a year. The occurrence of biogenic hydrocarbons in the world's oceans is well recorded. They have the characteristic isoprenoid structure, and measurements made in water columns indicate a background concentration of 1.0 to 10 $\mu\text{l/l}$. The higher molecular weight materials are dispersed as particles, with the highest concentrations of about 20 $\mu\text{l/l}$ occurring in the top 3 mm layer of water.

A wide variation in the response of organisms to oil exposures has been noted. The larvae of fish and crustaceans appear to be most susceptible to the water-soluble fraction of crude oil. Exposures of plankton and algae have indicated that certain species of diatoms and green algae are inhibited, whereas microflagellates are not.

For the most part, molluscs and most intertidal worm species appear to be tolerant of oil contamination.

For Surfactants: Kow cannot be easily determined due to hydrophilic/hydrophobic properties of the molecules in surfactants. BCF value: 1-350.

Aquatic Fate: Surfactants tend to accumulate at the interface of the air with water and are not extracted into one or the other liquid phases.

Terrestrial Fate: Anionic surfactants are not appreciably sorbed by inorganic solids. Cationic surfactants are strongly sorbed by solids, particularly clays. Significant sorption of anionic and non-ionic surfactants has been observed in activated sludge and organic river sediments. Surfactants have been shown to improve water infiltration into soils with moderate to severe hydrophobic or water-repellent properties.

Ecotoxicity: Some surfactants are known to be toxic to animals, ecosystems and humans, and can increase the diffusion of other environmental contaminants. The acute aquatic toxicity generally is considered to be related to the effects of the surfactant properties on the organism and not to direct chemical toxicity. Surfactants should be considered to be toxic to aquatic species under conditions that allow contact of the chemicals with the organisms. Surfactants are expected to transfer slowly from water into the flesh of fish.

During this process, readily biodegradable surfactants are expected to be metabolized rapidly during the process of bioaccumulation. Surfactants are not to be considered to show bioaccumulation potential if they are readily biodegradable.

for alkylphenols and their ethoxylates, or propoxylates:

Environmental fate: Alkylphenols are ubiquitous in the environment after the introduction, generally as wastes, of their alkoxyated forms (ethoxylates and propoxylates, for example); these are extensively used throughout industry and in the home.

Alkylphenol ethoxylates are widely used surfactants in domestic and industrial products, which are commonly found in wastewater discharges and in sewage treatment plant (STP) effluent's. Degradation of APEs in wastewater treatment plants or in the environment generates more persistent shorter-chain APEs and alkylphenols (APs) such as nonylphenol (NP), octylphenol (OP) and AP mono- to triethoxylates (NPE1, NPE2 and NPE3). There is concern that APE metabolites (NP, OP, NPE1-3) can mimic natural hormones and that the levels present in the environment may be sufficient to disrupt endocrine function in wildlife and humans. The physicochemical properties of the APE metabolites (NP, NPE1-4, OP, OPE1-4), in particular the high Kow values, indicate that they will partition effectively into sediments following discharge from STPs. The aqueous solubility data for the APE metabolites indicate that the concentration in water combined with the high partition coefficients will provide a significant reservoir (load) in various environmental compartments. Data from studies conducted in many regions across the world have shown significant levels in samples of every environmental compartment examined. In the US, levels of NP in air ranged from 0.01 to 81 ng/m³, with seasonal trends observed. Concentrations of APE metabolites in treated wastewater effluents in the US ranged from < 0.1 to 369 ug/l, in Spain they were between 6 and 343 ug/l and concentrations up to 330 ug/l were found in the UK. Levels in sediments reflected the high partition coefficients with concentrations reported ranging from < 0.1 to 13,700 ug/kg for sediments in the US. Fish in the UK were found to contain up to 0.8 ug/kg NP in muscle tissue. APEs degraded faster in the water column than in sediment. Aerobic conditions facilitate easier further biotransformation of APE metabolites than anaerobic conditions. Nonylphenols are susceptible to photochemical degradation. Using natural, filtered, lake water it was found that nonylphenol had a half-life of approximately 10-15 h under continuous, noon, summer sun in the surface water layer, with a rate approximately 1.5 times slower at depths 20-25 cm. Photolysis was much slower with ethoxylated nonylphenol, and so it is unlikely to be a significant event in removal of the ethoxylates.

Air: Alkylphenols released to the atmosphere will exist in the vapour phase and is thought to be degraded by reaction with photochemically produced hydroxyl radicals, with a calculated half-life, for nonylphenol, of 0.3 days.

Water: Abiotic degradation of alkylphenol is negligible. Biodegradation does not readily take place. The half-life in surface water may be around 30 days.

Degradation: Alkylphenol ethoxylates (APES) may abiotically degrade into the equivalent alkylphenol. During degradation ethylene oxide units are cleaved off the ethylene oxide chain until only short-chain alkylphenol ethoxylates remain, typically mono- and diethylene oxides. Oxidation of these oligomers creates the corresponding carboxylic acids. This leaves several degradation products: short-chain ethoxylates, their carboxylic acids, and alkylphenols.

Biodegradation: Alkylphenols are not readily biodegradable. Several mechanisms of microbial aromatic ring degradation have been reported, the most common being formation of catechol from phenol, followed by ring scission between or adjacent to the two hydroxyl groups.

The full breakdown pathway for APES has not yet been determined, and all studies have so far focused on identification of intermediates in bacterial culture media, rather than studying cell-free systems or purified enzymes. It is, however, likely that microbial metabolism usually starts by an attack on the ethoxylate chain, rather than on the ring or the hydrophobic chain. The ethoxylate groups are progressively removed, either by ether cleavage, or by terminal alcohol oxidation followed by cleavage of the resulting carboxylic acid.

Biodegradation of APES produces less biodegradable products: alkylphenol mono- and di-ethoxylates, alkylphenoxy acetic and alkylphenoxy polyethoxy acetic acids, and alkylphenols. These metabolites frequently persist through sewage treatment and in rivers. Anaerobic conditions generally lead to the accumulation of alkylphenols. The rate of biodegradation seems to decrease with increasing length of the ethylene oxide chain.

Bioaccumulation: Metabolites of APES accumulate in organisms, with bioconcentration factors varying from ten to several thousand, depending on species, metabolite and organ.

The metabolites of APES are generally more toxic than the original compounds. APES have LC50s above about 1.5 mg/l, whereas alkylphenols, such as nonylphenol, have LC50s are generally around 0.1 mg/l.

Oestrogenic activity: The role of alkyl chain length and branching, substituent position, number of alkylated groups, and the requirement of a phenolic ring structure was assessed in fish. The results showed that most alkylphenols were oestrogenic, although with 3-300 thousand times lower potency than the endogenous estrogen 17β-estradiol. Mono-substituted tertiary alkylphenols with moderate (C4-C5) and long alkyl chain length (C8-C9) in the para position exhibited the highest oestrogenic potency. Substitution with multiple alkyl groups, presence of substituents in the ortho- and meta-position and lack of a hydroxyl group on the benzene ring reduced the oestrogenic activity, although several oestrogenic alkylated non-phenolics were identified.

Human exposure: Alkylphenols were first found to be oestrogenic (oestrogen-mimicking) in the 1930s, but more recent research has highlighted the implications of these effects. The growth of cultured human breast cancer cells is affected by nonylphenol at concentrations as low as 1 μM (220 ug/l) or concentrations of octylphenol as low as 0.1 μM (20 ug/l). Oestrogenic effects have also been shown on rainbow trout hepatocytes, chicken embryo fibroblasts and a mouse oestrogen receptor.

The insecticide chlordecone (Kepone) shows similar behaviour to alkylphenols, accumulating in liver and adipose tissue, and eliciting oestrogenic activity. Workers exposed to this insecticide can suffer reproductive effects such as low sperm counts and sterility. In addition, the oestrogenic effects of chlordecone on MCF7 cells occur at similar concentrations to those of alkylphenols, suggesting that alkylphenols will be a similar health hazard if target cells are exposed to μM levels of these compounds.

By comparing environmental concentrations, bioconcentration factors and *in vitro* oestrogenic effect levels, current environmental levels of alkylphenolic compounds are probably high enough to affect the hormonal control systems of some organisms. It is also possible that human health could be being affected.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
lauryl alcohol, ethoxylated	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
white mineral oil (petroleum)	HIGH (LogKOW = 5.18)
nonylphenol ethoxylates	LOW (BCF = 1.4)
lauryl alcohol, ethoxylated	LOW (LogKOW = 3.4)
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)

Mobility in soil

Ingredient	Mobility
lauryl alcohol, ethoxylated	LOW (Log KOC = 10)

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. Otherwise: <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.

Continued...

Apparent Paraffinic Oil Adjuvant

Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.

A Hierarchy of Controls seems to be common - the user should investigate:



- ▶ Reduction
- ▶ Reuse
- ▶ Recycling
- ▶ Disposal (if all else fails)

This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.

- ▶ **DO NOT allow wash water from cleaning or process equipment to enter drains.**
- ▶ It may be necessary to collect all wash water for treatment before disposal.
- ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- ▶ Where in doubt contact the responsible authority.
- ▶ Recycle wherever possible or consult manufacturer for recycling options.
- ▶ Consult State Land Waste Authority for disposal.
- ▶ Bury or incinerate residue at an approved site.
- ▶ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	•3Z

Land transport (ADG)

14.1. UN number or ID number	3082	
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains nonylphenol ethoxylates, solvent naphtha petroleum, heavy aromatic, lauryl alcohol, ethoxylated and white mineral oil (petroleum))	
14.3. Transport hazard class(es)	Class	9
	Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	274 331 335 375 AU01
	Limited quantity	5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in;

(a) packagings;

(b) IBCs; or

(c) any other receptacle not exceeding 500 kg(L).

- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

14.1. UN number	3082	
14.2. UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. (contains nonylphenol ethoxylates, solvent naphtha petroleum, heavy aromatic, lauryl alcohol, ethoxylated and white mineral oil (petroleum))	
14.3. Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	9L
14.4. Packing group	III	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	A97 A158 A197 A215
	Cargo Only Packing Instructions	964
	Cargo Only Maximum Qty / Pack	450 L
	Passenger and Cargo Packing Instructions	964
	Passenger and Cargo Maximum Qty / Pack	450 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y964
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

Apparent Paraffinic Oil Adjuvant

14.1. UN number	3082	
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains nonylphenol ethoxylates, solvent naphtha petroleum, heavy aromatic, lauryl alcohol, ethoxylated and white mineral oil (petroleum))	
14.3. Transport hazard class(es)	IMDG Class	9
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5 Environmental hazard	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-A, S-F
	Special provisions	274 335 375 969
	Limited Quantities	5 L

14.7. Maritime transport in bulk according to IMO instruments

14.7.1. Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

14.7.2. Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
white mineral oil (petroleum)	Not Applicable
nonylphenol ethoxylates	Not Applicable
lauryl alcohol, ethoxylated	Not Applicable
solvent naphtha petroleum, heavy aromatic	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
white mineral oil (petroleum)	Not Applicable
nonylphenol ethoxylates	Not Applicable
lauryl alcohol, ethoxylated	Not Applicable
solvent naphtha petroleum, heavy aromatic	Not Applicable

SECTION 15 Regulatory information

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

white mineral oil (petroleum) is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

nonylphenol ethoxylates is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

lauryl alcohol, ethoxylated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Industrial Chemicals Introduction Scheme (AICIS) Comparable Chemicals Table

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australian Inventory of Industrial Chemicals (AIIC)

solvent naphtha petroleum, heavy aromatic is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (white mineral oil (petroleum); nonylphenol ethoxylates; lauryl alcohol, ethoxylated; solvent naphtha petroleum, heavy aromatic)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes

Apparent Paraffinic Oil Adjuvant

National Inventory	Status
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (white mineral oil (petroleum); nonylphenol ethoxylates; lauryl alcohol, ethoxylated; solvent naphtha petroleum, heavy aromatic)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	16/06/2026
Initial Date	16/06/2026

Other information

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.

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

Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit,
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration
- ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
- ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
- ▶ IGC: International Gas Carrier Code
- ▶ IBC: International Bulk Chemical Code

- ▶ AIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European Inventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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