

HIGHTEC LHM-PLUS

Motor Active

Chemwatch: 5445-71
Version No: 2.1.1.1
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: 20/01/2021
Print Date: 01/02/2021
L.GHS.AUS.EN

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

Product Identifier

Product name	HIGHTEC LHM-PLUS
Chemical Name	Not Applicable
Synonyms	30501-0010-99 (1 Litre)
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	Central hydraulic motor.
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Details of the supplier of the safety data sheet

Registered company name	Motor Active
Address	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia
Telephone	+61 2 9737 9422 1800 350 622
Fax	+61 2 9737 9414
Website	www.motoractive.com.au
Email	andrew.spira@motoractive.com.au

Emergency telephone number

Association / Organisation	MotorActive
Emergency telephone numbers	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
Other emergency telephone numbers	13 11 26 (In Case of Emergency contact: Poison Information Hotline)

SECTION 2 Hazards identification

Classification of the substance or mixture

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

ChemWatch Hazard Ratings

	Min	Max	
Flammability	1	1	
Toxicity	1	1	
Body Contact	2	2	
Reactivity	1	1	
Chronic	0	0	

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

Poisons Schedule	S5
Classification [1]	Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Chronic Aquatic Hazard Category 3
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)

H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.

HIGHTEC LHM-PLUS

H319	Causes serious eye irritation.
H336	May cause drowsiness or dizziness.
H412	Harmful to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P321	Specific treatment (see advice on this label).
P331	Do NOT induce vomiting.
P362	Take off contaminated clothing and wash before reuse.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.

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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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64742-53-6.	30-<60	<u>naphthenic distillate, light, hydrotreated (severe)</u>
64742-55-8.	15-<30	<u>paraffinic distillate, light, hydrotreated (severe)</u>
64742-46-7.	15-<30	<u>C14-20 aliphatics (<=2% aromatics)</u>
63150-07-2	5-<15	<u>alkyl methacrylate copolymer</u>
128-39-2	0.3-<1	<u>2,6-di-tert-butylphenol</u>
268567-32-4	0.1-<0.3	<u>3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid</u>
94270-86-7	0.1-<0.3	<u>N-alkylated benzotriazole</u>
68478-81-9	0.1-<0.3	<u>oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product</u>

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.

HIGHTEC LHM-PLUS

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.
Ingestion	<ul style="list-style-type: none"> ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ For advice, contact a Poisons Information Centre or a doctor at once. ▶ Urgent hospital treatment is likely to be needed. ▶ 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. ▶ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

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

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
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include: carbon dioxide (CO₂) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</p> <p>CARE: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.</p>
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Slippery when spilt.</p> <ul style="list-style-type: none"> ▶ Remove all ignition sources. ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
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HIGHTEC LHM-PLUS

Major Spills	<p>Slippery when spilt. 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.
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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"> ▶ Containers, even those that have been emptied, may contain explosive vapours. ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Electrostatic discharge may be generated during pumping - this may result in fire. ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). ▶ Avoid splash filling. ▶ Do NOT use compressed air for filling discharging or handling operations. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ 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. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

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>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> <ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	naphthenic distillate, light, hydrotreated (severe)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	paraffinic distillate, light, hydrotreated (severe)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	C14-20 aliphatics (<=2% aromatics)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
naphthenic distillate, light, hydrotreated (severe)	Petroleum distillates; petroleum ether; includes clay-treated light naphthenic [64742-45-6]; low boiling [68477-31-6]; petroleum extracts [64742-06-9]; petroleum base oil [64742-46-7]; petroleum 50 thinner, petroleum spirits [64475-85-0], Soltrol, VM&P naphtha [8032-32-4]; Ligroine, and paint solvent; petroleum paraffins C5-C20 [64771-72-8]; hydrotreated light naphthenic [64742-53-6]; solvent refined	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3

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Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
	light naphthenic [64741-97-5]; and machine coolant 1			
paraffinic distillate, light, hydrotreated (severe)	Mineral oil, heavy or light; (paraffin oil; Deobase, deodorized; heavy paraffinic; heavy naphthenic); distillates; includes 64741-53-3, 64741-88-4, 8042-47-5, 8012-95-1; 64742-54-7	140 mg/m3	1,500 mg/m3	8,900 mg/m3
C14-20 aliphatics (<=2% aromatics)	Petroleum distillates; petroleum ether; includes clay-treated light naphthenic [64742-45-6]; low boiling [68477-31-6]; petroleum extracts [64742-06-9]; petroleum base oil [64742-46-7]; petroleum 50 thinner, petroleum spirits [64475-85-0], Soltrol, VM&P naphtha [8032-32-4]; Ligroine, and paint solvent; petroleum paraffins C5-C20 [64771-72-8]; hydrotreated light naphthenic [64742-53-6]; solvent refined light naphthenic [64741-97-5]; and machine coolant 1	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
naphthenic distillate, light, hydrotreated (severe)	2,500 mg/m3	Not Available
paraffinic distillate, light, hydrotreated (severe)	2,500 mg/m3	Not Available
C14-20 aliphatics (<=2% aromatics)	2,500 mg/m3	Not Available
alkyl methacrylate copolymer	Not Available	Not Available
2,6-di-tert-butylphenol	Not Available	Not Available
3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid	Not Available	Not Available
N-alkylated benzotriazole	Not Available	Not Available
oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
alkyl methacrylate copolymer	E	≤ 0.01 mg/m ³
2,6-di-tert-butylphenol	E	≤ 0.01 mg/m ³
3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid	D	> 0.1 to ≤ 1 ppm
N-alkylated benzotriazole	E	≤ 0.1 ppm
oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product	E	≤ 0.01 mg/m ³

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

NOTE N: The classification as a carcinogen need not apply if the full refining history is known and it can be shown that the substance from which it is produced is not a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

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

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.	
	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. 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.	
	Type of Contaminant:	Air Speed:
	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)
	Within each range the appropriate value depends on:	
	Lower end of the range	Upper end of the range
	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	
3: Intermittent, low production.	3: High production, heavy use	
4: Large hood or large air mass in motion	4: Small hood-local control only	

	Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Personal protection	
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<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> <ul style="list-style-type: none"> ▶ Neoprene rubber gloves
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 ^
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^ - 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	Green golden coloured liquid with characteristic odour; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	-0.87 @ 15C
Odour	Characteristic	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	~-51 (pour pt.)	Viscosity (cSt)	-6.6 @ 100C
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	~126	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)	Negligible	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce</p>
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	<p>functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitizers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
Ingestion	<p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p>
Skin Contact	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</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>Limited evidence suggests that repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
Chronic	<p>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>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses.</p> <p>Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.</p> <p>Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties</p> <p>Animal studies: No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of an alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.</p> <p>Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, on the basis that similar materials tested in appropriate animal studies provide some 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.</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 lipid 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>

HIGHTEC LHM-PLUS	TOXICITY	IRRITATION
	Not Available	Not Available
naphthenic distillate, light, hydrotreated (severe)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; =2.18 mg/4hrs ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >5000 mg/kg ^[2]	
paraffinic distillate, light, hydrotreated (severe)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation(Rat) LC50; =2.18 mg/4hrs ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >5000 mg/kg ^[2]	
C14-20 aliphatics (<=2% aromatics)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye : Not irritating (OECD 405) *
	Inhalation(Rat) LC50; =4.6 mg/4hrs ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; =7400 mg/kg ^[2]	Skin : Not irritating (OECD 404)*
		Skin: adverse effect observed (irritating) ^[1]
alkyl methacrylate copolymer	TOXICITY	IRRITATION
	Not Available	Not Available
2,6-di-tert-butylphenol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >0.01 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral(Rat) LD50; >5000 mg/kg ^[1]	Skin: adverse effect observed (irritating) ^[1]
3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): slight *
	Oral(Rat) LD50; >2000 mg/kg ^[1]	Skin (rabbit): non-irritating *
N-alkylated benzotriazole	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Not Available
	Oral(Rat) LD50; 3300 mg/kg ^[2]	
oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product	TOXICITY	IRRITATION
	Not Available	Not Available

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

PARAFFINIC DISTILLATE, LIGHT, HYDROTREATED (SEVERE)	* Q8 MSDS
C14-20 ALIPHATICS (<=2% AROMATICS)	<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>*Exxsol D 100 SDS</p>
2,6-DI-TERT-BUTYLPHENOL	<p>For hindered phenols:</p> <p>Available data shows that acute toxicity of these substances is low.</p> <p>Mutagenicity. Data from bacterial reverse mutation assays and <i>in vitro</i> and <i>in vivo</i> chromosome aberration studies were reviewed. All assays, with and without metabolic activation, were negative. The weight of evidence for mutagenic potential for this category indicates these substances are not mutagenic.</p> <p>In Vitro Chromosome Aberration Studies. In vitro chromosome aberration studies are available for several members. All except 2,6-di-tert-butyl-p-cresol were negative</p> <p>In Vivo Chromosome Aberration Studies. In vivo studies evaluating chromosome damage are available for six of the hindered phenols. All in vivo evaluations were negative.</p> <p>Repeated Dose Toxicity. Repeated dose toxicity data of approximately three months (90-day, 12- and</p>

13-week) are available for some of the substances in this group. The liver was the target organ in rats for almost all of the substances with subchronic toxicity data in that species. Other target organs included thyroid and kidney and mesenteric lymph nodes. NOAELs in rats ranged from 100 ppm (approximately 5 mg/kg/day) to 10,000 ppm (500 mg/kg/day)

Carcinogenicity: Data is available for 2,6-di-tert-butyl-p-cresol (128-37-0); and 4,4'-thiobis-6-(t-butyl-m-cresol) (96-69-5). Liver adenomas were reported for 2,6-di-tert-butyl-p-cresol (128-37-0) and a NOAEL was established for the study at 25 mg/kg/day. 4,4'-Thiobis-6-(t-butyl-m-cresol) (96-69-5) was not carcinogenic in rats or mice, but the kidney was identified as a target organ in female rats

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

for alkylphenolics category:

The alkylphenolics may be divided into three groups.

Group I: ortho-substituted mono-alkylphenols:

Group II para-substituted mono-alkylphenols

Group III: di- and tri-substituted mixed alkyl phenols

The subdivision of the category alkylphenols into *ortho*, *para* and the *di/tri*-substituted mixed members is supported by several published investigations. In assessing antimicrobial and antifouling activity of twenty-three alkylphenols, a significant difference was noted between *para* and *ortho*-substituted materials. In particular, biological activity was found to vary parabolically with increasing hydrophobicity of the *para*-substituent while introduction of a bulky substituent at the *ortho*-position resulted in a very significant decrease in antimicrobial, antifouling, and membrane-perturbation potency. Several alkylphenolic analogs of butylated hydroxytoluene (BHT) were examined for hepatotoxicity in mice depleted of hepatic glutathione. The structural requirement of both hepatic and pulmonary toxicity was a phenol ring having benzylic hydrogen atoms at the *para* position and an *ortho*-alkyl group(s) that moderately hinders the phenolic hydroxyl group. It is noteworthy that in this model, neither of the Group III members TTBP (2,4,6-tri-tert-butylphenol) nor 2,6-DTBP (2,6-di-tert-butylphenol) showed either hepatic or pulmonary toxicity. Lastly, important differences were observed in gene activation (recombinant yeast cell assay – Lac-Z reporter gene) between *ortho*-substituted and *para*-substituted alkylphenol

Acute toxicity: The acute (single-dose) toxicity of alkylphenols examined to date shows consistency, with LD50 values ranging from approximately 1000 mg/kg to over 2000 mg/kg. These data demonstrate a very low level of acute systemic toxicity and do not suggest any unique structural specificity, despite the general tendency for the chemicals to be, at least, irritants to skin

Repeat dose toxicity: The available studies for members drawn from the three groups range from 28-day and 90-day general toxicity studies, through developmental toxicity and reproductive/developmental screening, to multigeneration reproductive studies are available for some category members

For the overall category of alkylphenols, the dosage at which the relatively mild general toxicity appears tends only to fall below 100 mg/kg/day with extended treatment, with an overall NOAEL for the category of approximately 20 mg/kg/day. No unusual and no apparent structurally unique toxicity is evident Repeat dose studies on OTBP (o-tert-butylphenol; Group I) and PTBP (p-tert-butylphenol; Group II) suggest the forestomach to be the main organ affected. OTBP also appears to have a mild (though statistically significant) protective effect against benzo[a]pyrene induced forestomach tumors. Long-term treatment with high dietary dose levels of PTBP caused hyperplastic changes in the forestomach epithelium of rats and hamsters, a likely consequence of the irritancy of the material. The relevance of this for human hazard is doubtful, particularly since there is no analogous structure in humans to the forestomach of rodents.

There was no evidence of an effect on reproductive function at dosages up to 150 mg/kg. One reproductive screening study reported increased 'breeding loss' and also reduced pup weight gain and survival in early lactation at 750 mg/kg/day. It is reasonable to assume that these effects were secondary to "severe toxic symptoms" reported in the dams at this dosage. Other than an indication of a very mildly oestrogenic effect of PNP (p-nonylphenol; Group II) at a high dose levels (200-300 mg/kg/day) no effect on development was seen in a multigeneration study.

By means of the classification method of Verhaar * all the alkylphenols would be classified as Type 2 compounds (polar narcotics). Narcosis, a non-specific mode of toxicity is caused by disruption (perturbation) of the cell membrane. The ability to induce narcosis is dependent on the hydrophobicity of the substance with biochemical activation or reaction involved. Such narcotic effects are also referred to as minimum or base-line toxicity. Polar narcotics such as the category phenols are usually characterised by having hydrogen bond donor activity and are thought to act by a similar mechanism to the inert, narcotic compounds but exhibit above base-line toxicity. In fact, a large number of alkylphenols have been evaluated as intravenous anesthetic agents. While the structure-activity relationships were found to be complex, the anesthetic potency and kinetics appeared to be a function of both the lipophilic character and the degree of steric hindrance exerted by *ortho* substituents. Less steric hindrance resulted in lower potency, while greater crowding led to complete loss of anesthetic activity and greater lipophilicity resulted in slower kinetics. These data support the notion that the alkylphenols behave as polar narcotics. In addition, the anaesthetic activity/potency differences seen with varying structure and placement of substituents strongly supports the division of alkylphenols category into the *ortho*, *para*, and *di/tri*-substituted groups (i.e. Group I, II and III, respectively).

Genotoxicity: It is reasonable to consider the mutagenic potential of all the alkylphenols together because only functional group is the phenolic, which is not a structural alert for mutagenicity. The data support this, since the results of genotoxicity testing are uniformly negative for all category substances examined

* Verhaar, H.J.M. van Leeuwen, C.J. and Hermens, J.L.M., Classifying Environmental Pollutants. 1: Structure-Activity Relationships for Prediction of Aquatic Toxicity, Chemosphere (25), pp 471 – 491 (1992).

3-(DIISOBUTOXYTHIOPHOSPHORYLSULFANYL)METHYLPROPIONIC ACID

Evidence of sensitisation in guinea pig skin assay * Oral repeat dose toxicity (28 days): NOEL 20 mg/kg/day bw * Non-mutagenic in bacterial reverse mutation assay * Non-genotoxic in vivo micronucleus test * The chemical was of low acute oral and dermal toxicity in rats, was not a skin irritant but was a slight eye irritant in rabbits, and was neither mutagenic in bacteria nor clastogenic in mouse bone marrow cells. It was a skin sensitizer in guinea pigs and exhibited systemic effects at a dose of 500 mg/kg/day bw in a 28-day oral repeated dose study in rats although some of these effects may have been adaptive in nature Clinical Chemistry Elevated triglyceride and phospholipid levels were noted in high dose animals of both sexes and elevated cholesterol levels in high dose females. These changes were considered to be an adaptive response and were reversed during the recovery period. All changes were within the 95% confidence limits of the historical control data. Higher albumin levels and albumin/globulin ratios were found in high dose animals of both sexes, higher total protein in high dose females. After recovery the elevated albumin levels persisted in males. All changes were within the 95% confidence limits of the historical control data. High dose males exhibited higher urea and lower creatinine levels which were within the 95% confidence limits of the historical control data. Haematology Plasma haemoglobin concentration and haematocrit were lower in high dose animals and red blood cell count was lower in high dose females. The toxicological significance of these findings is uncertain. High dose animals exhibited a prolonged activated partial thromboplastin time, females had a shorter thromboplastin time and males had a higher platelet count. All changes were within the 95% confidence limits of the historical control data. Prolonged activated PTT and platelet counts reached statistical significance in high dose recovery group males. Urinalysis Ketone was present in mid dose females and high dose animals of both sexes and was considered to be related to the adaptive changes in lipid metabolism. High dose females exhibited higher specific gravity and high dose animals of both sexes had slightly lower urine production. These were within the 95% confidence limits of historical control data and may indicate a possible change in the ability to concentrate urine. Effects in Organs High dose animals exhibited higher absolute and relative liver weights. These effects persisted in males at the end of the recovery period and were considered to be treatment related yet adaptive. High dose animals exhibited higher kidney/body weight ratios, higher absolute kidney weights and kidney to brain weight ratio. Mid dose males also exhibited an elevated kidney/body weight ratio. CONCLUSION The No Observed Effect Level (NOEL) was established as 20 mg/kg bw/day in this study, based on elevated kidney/body weight ratio in males NICNAS Full Public Report 11 September 2002

N-ALKYLATED BENZOTRIAZOLE

*RT Vanderbilt MSDS Repeat dose toxicity: A combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD 422) revealed parental toxicity at 150 mg/kg bw (clinical signs, reduced body weight gains with lower food consumption, slightly reduced thymus organ weight, and microscopic findings in the thymus and spleen). The NOAEL was considered to be 45 mg/kg body weight per day Genetic toxicity: The test compound did not cause mutations in bacteria and in mammalian cell culture Data obtained with a structural analogue did not reveal any potential for clastogenic effects in mammalian cells ** REACH Dossier

For benzotriazoles

There are several indications that the effects of phenolic benzotriazoles described in the literature might be caused by endocrine disruption, e.g. reduced concentrations of testosterone, higher concentrations of CYP 450, or higher activity of ethoxyresorufin-O-deethylase (EROD-activity). As in these cases there are also indications for toxic effects on the liver reported, the effects might actually be only secondary effects. With the present knowledge it is not possible to attribute them unambiguously as endocrine adverse effects of an equivalent level of concern.

Several benzotriazole UV stabilisers showed significant human aryl hydrocarbon receptor (AhR) ligand activity. The AhR has roles in regulating immunity, stem cell maintenance, and cellular differentiation A study indicated that certain benzotriazole UV stabilisers have the potential to accumulate and exert potent physiological effects in humans, analogous to polycyclic aromatic hydrocarbons and dioxins, which are known stable and toxic ligands. The polycyclic aromatic hydrocarbon the polycyclic aromatic hydrocarbon, benzo[a]pyrene (BaP), a ligand for AhR, induces its own metabolism and bioactivation to a toxic metabolites.

Benzotriazole is the core structure present within the phenolic benzotriazole class. In vitro metabolism with rat liver microsomes yielded formation of 5- and 4-hydroxybenzotriazole (1.6 and 0.32% of the amount added, respectively). Overall metabolism was low (<5% of the total amount added) Oral acute studies in rats and mice yielded LD50 values that ranged from 560 to 909 mg/kg. Intraperitoneal LD50 values in mice and rats ranged from 400-1000 and 500-900 mg/kg, respectively. A mouse intravenous LD50 of 238 mg/kg was identified. Dermal LD50 values were =1000 mg/kg in rats and rabbits, and inhalation LC50 values in rats were 1.5 mg/L and 1.91 mg/L/3 hours). Subchronic and short-term studies showed that oral administration to mice produced minimal effects on body weight while dose-dependent decreases in body weight were observed in rats. Endocrine effects, normocytic anemia, and leukopenia were noted in rats dosed for 26 weeks. The TDLo was 109 mg/kg. No effects on deaths and no clinical symptoms were noted in mice or rats orally administered (in food) benzotriazole =78 weeks. Additionally, no dose-related effects on reproductive organs were noted in either sex. Neoplastic liver nodules were observed in male Fischer rats fed 12,100 ppm benzotriazole for 78 weeks. However, historic laboratory controls incidences varied from 0 to 11% so the treatment-related effects could not be determined. Brain tumors occurred in three males and one female rat. Incidence of endometrial stromal polyps was increased significantly in female rats fed 6700 ppm for 78 weeks (22%), but not in female rats fed 12,100 ppm (16%). Significant increase in alveolar/bronchiolar carcinomas (18%) was observed female B6C3F1 fed 11,700 ppm benzotriazole for 104 weeks. Comparatively, a similar increase was not observed in female mice fed 23,500 ppm benzotriazole for the same period of time (6% increase). Historical laboratory control incidences varied from 0 to 7%. Genotoxicity studies indicate that the compound was not mutagenic to *S. typhimurium* strains TA97, TA98, or TA100 in the presence or absence of S9, or Chinese hamster ovary cells. Benzotriazole was also not mutagenic to *S. typhimurium* strain TA1535 in the absence of S9, but was mutagenic in the presence of S9. Conflicting results were obtained for effects in *S. typhimurium* strains TA1537 and TA1538 and *E. coli* WP2 uvrA. It did not produce DNA damage in *E. coli* PQ37. In Chinese hamster ovary cells, benzotriazole induced chromosomal aberrations in the presence of S9 and sister chromatid exchange in the absence of S9. Benzotriazole was not genotoxic in the mouse micronucleus assay at 800 mg/kg. Benzotriazole was identified as a non-sensitizer in the guinea pig maximization test. Benzotriazole was identified as irritating to rabbit eyes and minimally irritating to rabbit and guinea pig skin

For phenolic benzotriazoles

Overall, oral exposure (either through gavage or in feed) of the tested chemicals to rats led to liver effects. Increased absolute and/or relative liver weights were observed in several studies. Body weight and body weight gain changes were observed after administration of several test substances. Histopathological changes (e.g., foci, hypertrophy, and cytoplasmic vacuolization) and altered liver enzyme content and activities were also noted after treatment with different phenolic benzotriazoles.

Haematological effects (e.g., altered white and red blood cell counts, altered albumin levels, and packed cell volume) were observed. For those studies that calculated no observed adverse effect levels (NOAELs), the values ranged from <0.5 to ~5685 mg/kg/day

Reproductive and teratology effects: The chemicals tested produced a variety of effects. Some chemicals were shown to affect reproductive organ weights, but no direct studies in reproduction and development were located.

Genotoxicity None of the tested compounds were identified as mutagenic in vitro in the absence or presence of a metabolic system (S9) or in vivo

Chemical Information Review Document for Phenolic Benzotriazoles: Supporting Nomination for Toxicological Evaluation by the National Toxicology Program October 2011

http://ntp.niehs.nih.gov/ntp/noms/support_docs/phenolicbenzotriazoles_cird_oct2011_508.pdf

Fatty acid amides (FAA) are ubiquitous in household and commercial environments. The most common of these are based on coconut oil fatty acids alkanolamides. These are the most widely studied in terms of human exposure.

Fatty acid diethanolamides (C8-C18) are classified by Comité Européen des Agents de Surface et de leurs Intermediaires Organiques (CESIO) as Irritating (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes). Fatty acid monoethanolamides are classified as Irritant (Xi) with the risk phrases R41

Several studies of the sensitization potential of cocoamide diethanolamide (DEA) indicate that this FAA induces occupational allergic contact dermatitis and a number of reports on skin allergy patch testing of cocoamide DEA have been published. These tests indicate that allergy to cocoamide DEA is becoming more common.

Alkanolamides are manufactured by condensation of diethanolamine and the methylester of long chain fatty acids. Several alkanolamides (especially secondary alkanolamides) are susceptible to nitrosamine formation which constitutes a potential health problem. Nitrosamine contamination is possible either from pre-existing contamination of the diethanolamine used to manufacture cocoamide DEA, or from nitrosamine formation by nitrosating agents in formulations containing cocoamide DEA. According to the Cosmetic Directive (2000) cocoamide DEA must not be used in products with nitrosating agents because of the risk of formation of N-nitrosamines. The maximum content allowed in cosmetics is 5% fatty acid dialkanolamides, and the maximum content of N-nitrosodialkanolamines is 50 mg/kg. The preservative 2-bromo-2-nitropropane-1,3-diol is a known nitrosating agent for secondary and tertiary amines or amides. Model assays have indicated that 2-bromo-2-nitropropane-1,3-diol may lead to the N-nitrosation of diethanolamine forming the carcinogenic compound, N-nitrosodiethanolamine which is a potent liver carcinogen in rats (IARC 1978).

Several FAAs have been tested in short-term genotoxicity assays. No indication of any potential to cause genetic damage was seen. Lauramide DEA was tested in mutagenicity assays and did not show mutagenic activity in *Salmonella typhimurium* strains or in hamster embryo cells. Cocoamide DEA was not mutagenic in strains of *Salmonella typhimurium* when tested with or without metabolic activation

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Miljøministeriet (Danish Environmental Protection Agency)

OLEIC ACID, TRIETHYLENETETRAMINE, DODECENYLSUCCINIC ANHYDRIDE REACTION PRODUCT

For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides)

The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented.

The Fatty nitrogen-derived amides (FND amides) comprise four categories:

Subcategory I: Substituted Amides

Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components)

Subcategory III: Imidazole Derivatives

Subcategory IV: FND Amphoteric

Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all

Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies.

Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II.

Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories.

Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the *Salmonella* reverse mutation assay exist for all of the subcategories.

Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II.

In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30

chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.

Some typical applications of FND Amides are:

masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers.

The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health.

The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals.

The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives;

The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:

- 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 *residual base oils* 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.

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. 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.

Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing. 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).

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).

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

NAPHTHENIC DISTILLATE, LIGHT, HYDROTREATED (SEVERE) & PARAFFINIC DISTILLATE, LIGHT, HYDROTREATED (SEVERE)

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.

The substance is classified by IARC as Group 3:

NOT classifiable as to its carcinogenicity to humans.

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

PARAFFINIC DISTILLATE, LIGHT, HYDROTREATED (SEVERE) & ALKYL METHACRYLATE COPOLYMER & N-ALKYLATED BENZOTRIAZOLE & OLEIC ACID, TRIETHYLENETETRAMINE, DODECENYLSUCCINIC ANHYDRIDE REACTION PRODUCT

No significant acute toxicological data identified in literature search.

2,6-DI-TERT-BUTYLPHENOL & 3-(DIISOBUTOXYTHIOPHOSPHORYLSULFANYL)METHYLPROPIONIC ACID & N-ALKYLATED BENZOTRIAZOLE

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

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

	Endpoint	Test Duration (hr)	Species	Value	Source	
	HIGHTEC LHM-PLUS	Not Available	Not Available	Not Available	Not Available	Not Available
naphthenic distillate, light, hydrotreated (severe)	Endpoint	Test Duration (hr)	Species	Value	Source	
	LC50	96	Fish	>100mg/L	2	
	EC50	48	Crustacea	>1000mg/L	1	
	EC50	96	Algae or other aquatic plants	>1000mg/L	1	
paraffinic distillate, light, hydrotreated (severe)	Endpoint	Test Duration (hr)	Species	Value	Source	
	LC50	96	Fish	>100mg/L	2	
	EC50	48	Crustacea	>1000mg/L	1	
	NOEC	504	Crustacea	>1mg/L	1	
C14-20 aliphatics (<=2% aromatics)	Endpoint	Test Duration (hr)	Species	Value	Source	
	LC50	96	Fish	1.13mg/L	2	
	EC50	48	Crustacea	2mg/L	2	
	EC50	72	Algae or other aquatic plants	1.714mg/L	2	
	NOEL	504	Crustacea	0.163mg/L	2	
	LC50	96	Fish	2.2-mg/L	4	
	EC50	48	Crustacea	1.4mg/L	2	
	EC50	72	Algae or other aquatic plants	3.7mg/L	2	
alkyl methacrylate copolymer	Endpoint	Test Duration (hr)	Species	Value	Source	
	Not Available	Not Available	Not Available	Not Available	Not Available	
	2,6-di-tert-butylphenol	Endpoint	Test Duration (hr)	Species	Value	Source
		LC50	96	Fish	>0.1mg/L	2
		EC50	48	Crustacea	0.45mg/L	2
		EC50	72	Algae or other aquatic plants	0.51mg/L	2
		BCF	24	Algae or other aquatic plants	0.05-mg/L	4
		EC10	48	Algae or other aquatic plants	0.16mg/L	2
NOEC	504	Crustacea	0.035mg/L	2		
3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid	Endpoint	Test Duration (hr)	Species	Value	Source	
	LC50	96	Fish	38mg/L	2	
	EC50	72	Algae or other aquatic plants	15mg/L	2	
	EC10	72	Algae or other aquatic plants	51mg/L	2	
NOEC	504	Crustacea	3.6mg/L	2		
	N-alkylated benzotriazole	Endpoint	Test Duration (hr)	Species	Value	Source
		Not Available	Not Available	Not Available	Not Available	Not Available
	oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product	Endpoint	Test Duration (hr)	Species	Value	Source
Not Available		Not Available	Not Available	Not Available	Not Available	

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

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

Continued...

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
2,6-di-tert-butylphenol	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
C14-20 aliphatics (<=2% aromatics)	LOW (BCF = 159)
2,6-di-tert-butylphenol	HIGH (LogKOW = 4.92)

Mobility in soil

Ingredient	Mobility
2,6-di-tert-butylphenol	LOW (KOC = 14220)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▶ 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.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

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

Product name	Group
naphthenic distillate, light, hydrotreated (severe)	Not Available
paraffinic distillate, light, hydrotreated (severe)	Not Available
C14-20 aliphatics (<=2% aromatics)	Not Available
alkyl methacrylate copolymer	Not Available
2,6-di-tert-butylphenol	Not Available
3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid	Not Available
N-alkylated benzotriazole	Not Available
oleic acid, triethylenetetramine, dodeceny succinic anhydride reaction product	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
naphthenic distillate, light, hydrotreated (severe)	Not Available
paraffinic distillate, light, hydrotreated (severe)	Not Available
C14-20 aliphatics (<=2% aromatics)	Not Available
alkyl methacrylate copolymer	Not Available
2,6-di-tert-butylphenol	Not Available
3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid	Not Available

Product name	Ship Type
N-alkylated benzotriazole	Not Available
oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product	Not Available

SECTION 15 Regulatory information

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

naphthenic distillate, light, hydrotreated (severe) 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
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

paraffinic distillate, light, hydrotreated (severe) 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
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

C14-20 aliphatics (<=2% aromatics) 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
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

alkyl methacrylate copolymer is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

2,6-di-tert-butylphenol is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
 Australian Inventory of Industrial Chemicals (AIIC)

3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

N-alkylated benzotriazole is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (naphthenic distillate, light, hydrotreated (severe); paraffinic distillate, light, hydrotreated (severe); C14-20 aliphatics (<=2% aromatics); alkyl methacrylate copolymer; 2,6-di-tert-butylphenol; 3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid; N-alkylated benzotriazole; oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (alkyl methacrylate copolymer; 3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid; N-alkylated benzotriazole)
Japan - ENCS	No (alkyl methacrylate copolymer; 3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid; N-alkylated benzotriazole; oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (naphthenic distillate, light, hydrotreated (severe); paraffinic distillate, light, hydrotreated (severe); alkyl methacrylate copolymer; 3-(diisobutoxythiophosphorylsulfanyl)methylpropionic acid; N-alkylated benzotriazole; oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product)
Vietnam - NCI	No (alkyl methacrylate copolymer; oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product)
Russia - ARIPS	No (alkyl methacrylate copolymer; oleic acid, triethylenetetramine, dodecenylsuccinic anhydride reaction product)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 Other information

Revision Date	20/01/2021
Initial Date	20/01/2021

SDS Version Summary

Version	Issue Date	Sections Updated
2.1.1.1	20/01/2021	Chronic Health, Classification

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
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

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