



PX24 #4955049 (AUS)

RS Components

Chemwatch: 7984-64

Version No: 2.1

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

Chemwatch Hazard Alert Code: 4

Initial Date: 24/10/2025

Revision Date: 24/10/2025

Print Date: 27/10/2025

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

Product Identifier

Product name	PX24 #4955049 (AUS)
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	AEROSOLS
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	Professional use, lubricant. Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	RS Components
Address	25 Pavesi Street Smithfield NSW 2164 Australia
Telephone	+1 300 656 636
Fax	+1 300 656 696
Website	www.au.rs-online.com
Email	SupportAU@rs.rsgroup.com

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	+61 1800 951 288 (ID#: 7984-64)
Other emergency telephone number(s)	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

	Min	Max
Flammability	4	
Toxicity	2	
Body Contact	2	
Reactivity	1	
Chronic	0	

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

Poisons Schedule	Not Applicable
Classification ^[1]	Aerosols, Hazard Category 1, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term 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)

H222+H229	Extremely flammable aerosol. Pressurized container: may burst if heated.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H336	May cause drowsiness or dizziness.
H412	Harmful to aquatic life with long lasting effects.
AUH044	Risk of explosion if heated under confinement.

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
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/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1174522-15-6	>75	<u>hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%)</u>
64742-55-8.	1-5	<u>paraffinic distillate, light, hydrotreated (severe)</u>
Not Available	1-5	<u>barium mono-C10-13-alkylbenzene, sulfonate salts</u>
123-42-2	<1	<u>diacetone alcohol</u>
95-38-5	<1	<u>2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol</u>
110-25-8	<1	<u>oleoylsarcosine</u>
124-38-9	1-5	<u>carbon dioxide</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

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SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If aerosols come in contact with the eyes: ▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes 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. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If solids or aerosol mists are deposited upon the skin: ▶ Flush skin and hair with running water (and soap if available). ▶ Remove any adhering solids with industrial skin cleansing cream. ▶ DO NOT use solvents. ▶ Seek medical attention in the event of irritation.
Inhalation	If aerosols, fumes or combustion products are inhaled: ▶ Remove to fresh air. ▶ 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. ▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

For petroleum distillates

- In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.
- Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.
- Positive pressure ventilation may be necessary.
- Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.
- After the initial episode, individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment. Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.
- Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.
- Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur. Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.

Treat symptomatically.

- ▶ After ingestion of barium acid salts, severe gastro-intestinal irritation followed by muscle twitching, progressive flaccid paralysis and severe hypokalaemia and hypertension, occurs.
- ▶ Respiratory failure, renal failure and occasional cardiac dysrhythmias may result from an acute ingestion.
- ▶ Use sodium sulfate as a cathartic. Add 5-10 gm of sodium sulfate to lavage solution or as fluid supplement to Ipecac syrup (the sulfate salt is not absorbed)
- ▶ Monitor cardiac rhythm and serum potassium closely to establish the trend over the first 24 hours. Large doses of potassium may be needed to correct the hypokalaemia.
- ▶ Administer generous amounts of fluid replacement but monitor the urine and serum for evidence of renal failure. [Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

SMALL FIRE:

- ▶ Water spray, dry chemical or CO2

LARGE FIRE:

- ▶ Water spray or fog.

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	▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▶ Liquid and vapour are highly flammable. ▶ Severe fire hazard when exposed to heat or flame. ▶ Vapour forms an explosive mixture with air. ▶ Severe explosion hazard, in the form of vapour, when exposed to flame or spark. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition with violent container rupture. ▶ Aerosol cans may explode on exposure to naked flames. ▶ Rupturing containers may rocket and scatter burning materials. ▶ Hazards may not be restricted to pressure effects. ▶ May emit acrid, poisonous or corrosive fumes. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). Combustion products include: carbon monoxide (CO)

	Combustible. Will burn if ignited. carbon dioxide (CO2) nitrogen oxides (NOx) sulfur oxides (SOx) metal oxides other pyrolysis products typical of burning organic material. Decomposes at high temperatures to produce barium oxide. Barium oxide is strongly alkaline and, upon contact with water, is exothermic. When barium oxide reacts with oxygen to give a peroxide, there is a fire and explosion risk. 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.
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	▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Wear protective clothing, impervious gloves and safety glasses. ▶ Shut off all possible sources of ignition and increase ventilation. ▶ Wipe up. ▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely. Slippery when spilt.
Major Spills	▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse / absorb vapour. ▶ Absorb or cover spill with sand, earth, inert materials or vermiculite. ▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely. ▶ Collect residues and seal in labelled drums for disposal. Slippery when spilt.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ DO NOT incinerate or puncture aerosol cans. ▶ DO NOT spray directly on humans, exposed food or food utensils. ▶ 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 are maintained.
Other information	▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can ▶ Store in original containers in approved flammable liquid storage area. ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped. ▶ No smoking, naked lights, heat or ignition sources. ▶ Keep containers securely sealed. Contents under pressure. ▶ Store away from incompatible materials. ▶ Store in a cool, dry, well ventilated area. ▶ Avoid storage at temperatures higher than 40 deg C. ▶ Store in an upright position. ▶ Protect containers against physical damage. ▶ Check regularly for spills and leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Aerosol dispenser. ▶ Check that containers are clearly labelled.
Storage incompatibility	▶ 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	paraffinic distillate, light, hydrotreated (severe)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	diacetone alcohol	Diacetone alcohol	50 ppm / 238 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	carbon dioxide	Carbon dioxide	5000 ppm / 9000 mg/m3	54000 mg/m3 / 30000 ppm	Not Available	Not Available
Australia Exposure Standards	carbon dioxide	Carbon dioxide in coal mines	12500 ppm / 22500 mg/m3	54000 mg/m3 / 30000 ppm	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%)	Not Available	Not Available
paraffinic distillate, light, hydrotreated (severe)	2,500 mg/m3	Not Available
barium mono-C10-13-alkylbenzene, sulfonate salts	Not Available	Not Available
diacetone alcohol	Not Available	Not Available
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	Not Available	Not Available
oleoylsarcosine	Not Available	Not Available
carbon dioxide	40,000 ppm	Not Available

MATERIAL DATA

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. General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.	
	Type of Contaminant:	Speed:
	aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s
	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
	Within each range the appropriate value depends on:	
Individual protection measures, such as personal protective equipment	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
Eye and face protection	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.	
		
Skin protection	▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59]. ▶ Close fitting gas tight goggles	
	See Hand protection below	

Hands/feet protection	NOTE: ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. ▶ No special equipment needed when handling small quantities. ▶ OTHERWISE: ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear.
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. OTHERWISE: ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces.

Recommended material(s)

GLOVE SELECTION INDEX
Glove selection is based on a modified presentation of the: **"Forsberg Clothing Performance Index"**.
The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:
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Material	CPI
NEOPRENE	A
NITRILE	A
PVA	B
NATURAL RUBBER	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.
Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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

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

▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used
Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Amber coloured highly flammable liquid with characteristic odour; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	0.84 @20C
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>200
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	<20.5
Initial boiling point and boiling range (°C)	192	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	7	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	0.6	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable

Vapour density (Air = 1)	Not Available	VOC g/L	673
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	► Elevated temperatures. ► Presence of open flame. ► Product is considered stable. ► Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

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

Inhaled	Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation hazard is increased at higher temperatures. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Spray mist may produce discomfort Open cuts, abraded or irritated skin should not be exposed to this material 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.
Eye	This material causes serious eye irritation.
Chronic	Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Continued...

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

There is some evidence to provide a presumption that human exposure to the material may result in impaired fertility on the basis of: some evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.

There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

Implantation studies in rats show that paraffin oils may be tumourigen. As a general rule the highly refined paraffins contain a lower level of suspect polyaromatic hydrocarbons than less refined grades and also less than waxes derived from naphthenic base-stocks.

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

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

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 a 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.

Workers exposed to barium compounds have been reported to show an increased incidence of hypertension, irritation of the respiratory system, and damage to the spleen, liver and bone marrow. Long term exposure to some barium compounds (especially inorganic species) may produce a condition known as baritosis, a form of benign pneumoconiosis. X-ray may show this when no other abnormal signs are present.

Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses the cough produces a stringy mucous, vital capacity decreases further and shortness of breath becomes more severe. Pneumoconiosis is the accumulation of dusts in the lungs and the tissue reaction in its presence. Barium sulfate produces noncollagenous pneumoconiosis identified by minimal stromal reaction, consisting mainly of reticulin fibres, an intact alveolar architecture and is potentially reversible. Miners of ores containing barium sulfate do not show symptoms, abnormal physical signs, an incapacity to work, diminished lung function, an increased likelihood of developing pulmonary or other bronchial infections or other thoracic disease despite the fact that particulate matter may have been retained in the lungs for many years.

No changes in mortality were observed in rats chronically exposed to doses as high as 60 mg barium/kg/day as barium chloride in the drinking water. An increase in mortality, attributable to nephropathy, was observed in mice chronically exposed to 160 mg barium/kg/day as barium chloride in drinking water; the number of deaths was similar to controls in mice exposed to 75 mg barium/kg/day. In male mice exposed to 0.95 mg barium/kg/day as barium acetate in drinking water, a significant decrease in longevity (defined as average lifespan of the last five surviving animals) was observed; however, no significant differences in mean lifespan were observed. Similarly, lifespan was not significantly altered in female mice exposed to 0.95 mg barium/kg/day or male or female rats exposed to 0.7 mg barium/kg/day as barium acetate in drinking water.

The potential for barium to induce reproductive and developmental effects has not been well investigated. Decreases in the number of sperm and sperm quality and a shortened estrous cycle and morphological alterations in the ovaries were observed in rats exposed to 2.2 mg barium/m3 and higher in air for an intermediate duration. Interpretation of these data is limited by the poor reporting of the study design and results, in particular, whether the incidence was significantly different from controls. In general, oral exposure studies have not found morphological alterations in reproductive tissues of rats or mice exposed to 180 or 450 mg barium/kg/day, respectively, as barium chloride in drinking water for an intermediate duration. Additionally, no significant alterations in reproductive performance was observed in rats or mice exposed to 200 mg barium/kg/day as barium chloride in drinking water. Decreased pup birth weight and a nonsignificant decrease in litter size have been observed in the offspring of rats exposed to 180/200 mg barium/kg/day as barium chloride in drinking water prior to mating. Several studies have examined the carcinogenic potential of barium following oral exposure and did not find significant increases in the tumour incidence.

WARNING: Aerosol containers may present pressure related hazards.

PX24 #4955049 (AUS)	TOXICITY	IRRITATION
	Not Available	Not Available
hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%)	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >=6.1 mg/L4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
paraffinic distillate, light, hydrotreated (severe)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: 2.18 mg/L4h ^[2]	Skin: no adverse effect observed (not irritating) ^[1]

	Oral (Rat) LD50: >5000 mg/kg ^[2]	
barium mono-C10-13-alkylbenzene, sulfonate salts	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (Rat) LC50: >1.9 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >5000 mg/kg ^[1]	
diacetone alcohol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 13500 mg/kg ^[2]	Eye (Human): 100ppm/15M
	Oral (Rat) LD50: 2520 mg/kg ^[2]	Eye (Rodent - rabbit): 100uL/24H - Severe
		Eye (Rodent - rabbit): 20mg - Severe
		Eye: adverse effect observed (irritating) ^[1]
		Skin (Rodent - rabbit): 10mg/24H
		Skin (Rodent - rabbit): 500mg - Mild
		Skin: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
2-(8-heptadeceny)-4,5-dihydro-1H-imidazole-1-ethanol	TOXICITY	IRRITATION
	Oral (Rat) LD50: 870 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
		Skin: adverse effect observed (corrosive) ^[1]
oleoylsarcosine	TOXICITY	IRRITATION
	Inhalation (Rat) LC50: 0.575 mg/l4h ^[1]	Not Available
	Oral (Rat) LD50: >5000 mg/kg ^[2]	
carbon dioxide	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

HYDROCARBONS, C11-14 N-ALKANES, ISOALKANES, CYCLICS, AROMATIC (2-25%)	May cause mild, short-lasting discomfort to eyes. Based on test data for structurally similar materials. Test(s) equivalent or similar to OECD Guideline 405 Not expected to be a respiratory sensitizer. Not expected to be a skin sensitizer. Based on test data for structurally similar materials. Test(s) equivalent or similar to OECD Guideline 406 May be fatal if swallowed and enters airways. Based on physico-chemical properties of the material. Not expected to be a germ cell mutagen. Based on test data for structurally similar materials. Test(s) equivalent or similar to OECD Guideline 471 473 474 476 478 479 Not expected to cause cancer. Based on test data for structurally similar materials. Test(s) equivalent or similar to OECD Guideline 453 Not expected to be a reproductive toxicant. Based on test data for structurally similar materials. Test(s) equivalent or similar to OECD Guideline 413 414 415 Not expected to cause harm to breast-fed children. Not expected to cause organ damage from a single exposure. Not expected to cause organ damage from prolonged or repeated exposure. Based on test data for structurally similar materials. Test(s) equivalent or similar to OECD Guideline 408 413 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. 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. For alkanes: Exposure to the commercial hexane (a representative of the ECHA group of hydrocarbons, C5-C7, n-alkanes, isoalkanes, n-hexane rich) had no effect on the behavior of rats. Rats were tested monthly throughout the exposure for hindlimb splay and grip strength. The NOAEC for sub-chronic neurological effects is 9000 ppm in rats. In a 13 week subchronic inhalation study, the neurotoxicity of light alkylate naphtha distillate (LAND-2; carbon range C5-C8) was examined in male and female rats and aside from acute CNS effects, no treatment related neurotoxic effects found in any of the treatment groups. The NOAEC was determined to be > 24.3 g/m3 (6646 ppm). Additionally, no neurological effects were reported in the NTP 2 year carcinogenicity study on Stoddard solvent. For hydrocarbons, C5-C7, n-alkanes, isoalkanes, n-hexane rich n-Hexane was metabolized and excreted within 168 h of iv bolus administration, inhalation exposure or dermal application. Exhaled breath and urine were the two primary routes for the excretion and its metabolites. n-Hexane was widely distributed to the body tissues but were not concentrated significantly by any of those tissues. It was extensively metabolized and a number of radio labeled metabolites were excreted in the urine. n-Hexane and its radio labeled metabolites disappeared from the blood of rats with a half-life of approximately 9-10 h. Repeated inhalation exposure had no apparent effect on the rates or routes of excretion of either of the test compounds or their metabolites. The absorption rates into the skin, normalised for exposure concentration, was determined to be 0.013 cm/h. The maximum absorption rate into the blood was determined to be 0.005 nmol/h. A comparison of the estimated whole-body skin uptake with the inhalatory uptake from the same atmosphere, revealed that the dermal uptake contributed 0.1% to the total uptake. C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are absorbed, they are typically metabolized by side chain oxidation to alcohol and carboxylic acid derivatives. These metabolites can be glucuronidated and excreted in the urine or further metabolized before being excreted. The majority of the metabolites are excreted in the urine and to a lower extent, in the faeces. Excretion is rapid with the majority of the elimination occurring within the first 24 hours of exposure. As a result of the lack of systemic toxicity and the ability of the parent material to undergo metabolism and rapid excretion, bioaccumulation of the test substance in the tissues is not likely to occur. C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are poorly absorbed dermally with an estimated overall percutaneous absorption rate of approximately 2ug/cm2/hr or 1% of the total applied fluid. Regardless of exposure route, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are rapidly metabolized and eliminated has been fully evaluated. All of the animal studies were performed in a manner similar or equivalent
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to currently established OECD guidelines. Based on these data, C9-C14 aliphatic, <2% aromatic hydrocarbons have a low order of acute toxicity by the oral, dermal, and inhalation routes of exposure.

In a study examining the oral toxicity of commercial hexane. 6 male rats were given doses of up to 25 ml/kg of test substance by oral gavage. The animals were then observed for 14 days for mortality. No mortality was observed at any of the doses. The oral LD50 is therefore > 25 ml/kg (16.75 g/kg; density of 0.67).

C9-C14 aliphatic, <2% aromatic hydrocarbons is minimally toxic via ingestion where the LD50 is >5000 mg/kg, via dermal exposure where the LD50 is >5000 mg/kg, and by inhalation where the LC50 > 5000 mg/m3. These findings do not warrant classification of C9-C14 aliphatic, <2% aromatic hydrocarbons under the Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP) do not warrant classification under the Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparations (DSD/DPD). C9-C14 aliphatic, <2% aromatic hydrocarbons are classified under EU CLP guidelines as a Category 1 aspiration hazard based on its physical and chemical properties (hydrocarbon fluid, viscosity = 20.5 mm2/s) and as an R65 aspiration hazard under the EU DSD/DPD.

One study examined that acute inhalation toxicity of hexane to male rats. Groups of 10 male rats exposed to various large concentrations of hexane vapour for 4 hrs. Animals were then observed for clinical signs and mortality for at least the next 6 days. Several animals died during the exposure period. The LC50 was determined to be 73,680 ppm (259354 mg/m3). Due to the high concentration of the LC50, the test substance would not be classified as toxic by inhalation according to OECD GHS guidelines. Surviving animals experienced severe toxicological effects during the exposure.

Skin irritation:

For isoparaffinic, normal paraffinic, and mixed C9-C14 aliphatic, <2% aromatic hydrocarbon fluids, the weight of evidence indicates that the erythema and oedema scores (24, 48, and 72 average) are below the classification threshold requirements: 2.0, Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparation; 2.3, the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).

For cycloparaffinic C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids, erythema and oedema scores (24, 48, and 72 average) are above the classification threshold requirements: 2.0, Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparation; 2.3, the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP). This finding warrants classification of the test material as a skin irritant (R38) under Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparations. This finding warrants classification of the test material as a Category 2 dermal irritant under the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).

Eye irritation

Ocular lesion scores (24, 48, and 72 average) are below the classification threshold requirements.

Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparation: 0, cornea opacity; 0, iris lesion; >2.5, redness of the conjunctivae; >2.0, oedema of the conjunctivae (chemosis). Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP): 0, cornea opacity; 0, iris lesion; >2.0, redness of the conjunctivae; >2.0, oedema of the conjunctivae (chemosis).

Respiratory irritation

There are no studies that warrant classification as a respiratory irritant under either the Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC or under the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).

Sensitisation:

A study was performed to determine the concentration of hexane that would be expected to cause sensitization in humans.

Results of previous LLNA experiments were used to calculate the EC3 value, the concentration at which the test substance would produce a 3-fold increase in the proliferative activity of lymph nodes in the LLNA test. The 3-fold increase is considered a positive response for sensitization in the LLNA test. The EC3 value for hexane was determined to be > 100% concentration. The test substance is therefore not sensitizing.

There are no reports of respiratory sensitization from C9-C14 aliphatic, <2% aromatic hydrocarbons fluids in laboratory animals or humans. However, skin sensitization studies utilizing C9-C14 aliphatic, <2% aromatic hydrocarbons fluids found no indication of skin sensitization in guinea pigs. Additional studies in humans also found no indication of skin sensitization. With these observations, it is presumed that C9-C14 aliphatic, <2% aromatic hydrocarbons fluids will not be a respiratory sensitizing agent.

Repeat dose toxicity,

In a study involving n-hexane, neurological effects were only seen at the highest dose level after an average of 101.3 days of exposure. The LOAEL for neurological effects is 46.2 mmol/kg bw (37973 mg/kg), and the NOAEL is 13.2 mmol/kg bw (1135 mg/kg). Reduced body weight gain was seen at all three dose levels, however was only considered treatment related in the 13.2 and 46.2 mmol/kg bw groups. The NOAEL is therefore 6.60 mmol/kg bw.

In a study involving n-hexane The NOAEC for male rats exposed via inhalation was 2984 ppm based on liver and kidney effects. The LOAEC for male rats was 8992 ppm. The NOAEC for female rats was 8992 ppm

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are expected to have a low order of repeated dose toxicity by the oral route of exposure.

All tests were performed in a manner similar or equivalent to currently established OECD guidelines. In a repeated dose study where C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered via oral gavage, no signs of toxicity were observed at the maximum experimental dose tested, 5000 mg/kg/day.

In a repeated dose study where C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered via inhalation, no signs of toxicity were observed at 10400 mg/m3. Based on these observations, the repeat inhalation concentration NOAEL is =10400 mg/m3 (10.4 mg/L) for C9-C14 aliphatic, <2% aromatic hydrocarbon fluid

Genetic toxicity:

A study examined the in vitro mutagenicity of vapours of the test substance commercial hexane. Plates of *S. typhimurium* were exposed for 7-8 hrs to test atmospheres of 0, 600, 1000, 3000, 6000, or 9000 ppm of test substance. The test substance did not produce a positive response in any of the test strains. The test substance is not mutagenic.

In a study to determine the in vivo effect of inhalation exposure of commercial hexane on rat bone marrow. Groups of 5 male and 5 female rats were exposed to 0, 900, 3000, and 9000 ppm of test substance vapour for 6 hrs/day for 5 days. There was no statistically significant increase in cell aberrations in any treatment group. The test substance is not mutagenic.

C9-C14 aliphatic, <2% aromatic hydrocarbons fluids are not mutagenic using in vitro or in vivo genotoxicity assays. In bacterial tests, C9-C14 aliphatic, <2% aromatic hydrocarbons fluids were not mutagenic in *Salmonella* strains tested in the presence or absence of metabolic activation. C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were negative in a in vitro mammalian cell gene mutation assay. In sister chromatid exchange and in chromosomal aberration studies, C9-C14 aliphatic, <2% aromatic hydrocarbons fluids did not produce an effect. C9-C14 aliphatic, <2% aromatic hydrocarbons fluids were also non-mutagenic when tested in an in vivo mouse bone marrow micronucleus assay and when tested in dominant lethal studies utilizing an inhalation route of exposure. All studies were conducted in a manner similar or equivalent to currently established OECD guidelines. C9-C14 aliphatic, <2% aromatic hydrocarbons fluids are a non-genotoxic agent and classification is not warranted under the Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP) or under the Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparations.

Toxicity to reproduction.

In a study examining the effects of commercial hexane the NOAEC for both male and female rats (adults and offspring) was 3000 ppm (10560 mg/m3). The LOAEC for these groups was 9000 ppm based on reduced body weight. There were no adverse effects to reproduction, therefore the NOAEC for reproduction is 9000 ppm (31680 mg/m3).

A study to examine the developmental toxicity of commercial hexane in mice, found the maternal NOAEC was 900 ppm, and the maternal LOAEC was 3000 ppm (10560 mg/m3) based on colour changes in the lungs. The developmental NOAEC was 3000 ppm and the LOAEC was 9000 ppm (31680 mg/m3) in mice.

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are not developmental toxicants. In two developmental studies (OECD TG 414), pregnant dams were dosed by inhalation with 0, 300, or 900 ppm C9-C14 aliphatic, <2% aromatic hydrocarbon fluids during gestational days 6 through 15. No adverse maternal or fetal effects were noted at any dose level. Thus, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids did not produce any maternal or fetal toxicity or any developmental effects in rats. Based on the study results, the maternal and developmental toxicity NOAEC is >= 900 ppm (5220 mg/m3). Based on this study and the lack of systemic toxicity, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids, are not expected to be developmental toxicants.

For high molecular weight aliphatic hydrocarbons:

Acute toxicity:

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Four studies were available for acute oral toxicity, dealing with the toxicity of C5-C20 normal paraffins, C14-C17 n-alkanes, C14-C16 paraffins and isohexadecane. All studies were conducted similarly to OECD guideline 401 without GLP compliance. All studies show no mortality at concentrations up to 5000 mg/kg bw.

Three studies were available for acute dermal toxicity, dealing with the toxicity of C5-C20 normal paraffins, C14-C17 n-alkanes and C14-C16 paraffins. All studies were conducted similarly to OECD guideline 402 without GLP compliance. All studies show no mortality at concentrations equal to or higher than 2000 mg/kg bw.

A reliable study and a non-reliable study were available for acute inhalation, dealing with the toxicity of hydrocarbons, C10-C12, isoalkanes, < 2% aromatics and C14-C16 n-paraffins, respectively. All studies were conducted similarly to OECD guideline 403. They all show no mortality at concentrations equal to or higher than 5000 mg/m3.

Sensitisation:

C9-C14 aliphatic, < 2% aromatic hydrocarbons fluids were determined not to be skin sensitizers using Magnusson and Kligman Guinea-Pig Maximization tests (OECD TG 406). C9-C14 aliphatic, <2% aromatic hydrocarbons fluids were determined not to be skin sensitizers in Human Repeated Insult Patch Tests (HRIPT)

C10-12 isoalkanes (<2% aromatics), C11-C14 n-alkanes (<2% aromatics) and C10-C13 (<2% aromatics) were not dermal sensitizers using a Magnusson and Kligman Guinea-Pig Maximization test (OECD TG 406).

However, skin sensitization studies utilizing C9-14 aliphatics (< 2% aromatics) found no indication of skin sensitization in guinea pigs.

Additional studies on C14-C20 aliphatics (<2% aromatics) in humans also found no indication of skin sensitization.

There are no reports of respiratory sensitization from C14-20 aliphatics (< 2% aromatics) in laboratory animals or humans.

Repeat dose toxicity:

Oral: C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids are expected to have a low order of repeated dose toxicity by the oral route of exposure. All tests were performed in a manner similar or equivalent to currently established OECD guidelines. In a repeated dose study where C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids were administered via oral gavage, no signs of toxicity were observed at the maximum experimental dose tested, 5000 mg/kg/day.

Inhalation: In a repeated dose study where C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids were administered via inhalation, no signs of toxicity were observed at 10400 mg/m3. Based on these observations, the repeat inhalation concentration NOAEL is ≥ 10400 mg/m3 (10.4 mg/L) for C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids.

Two read-across studies from the structurally analogous test materials "hydrocarbons C12-C16, n-alkanes, isoalkanes, cyclics, <2% aromatics" and "hydrocarbons, C10 -C13, n-alkanes, isoalkanes, cyclics, < 2% aromatics" were analysed. All studies were performed in a manner similar or equivalent to currently established OECD guidelines. The systemic NOAEL were determined to be higher than 1000 and 5000 mg/kg/day, respectively.

Inhalation: a repeated inhalation toxicity study was performed with "Hydrocarbons, C10 – C12, isoalkanes, < 2% aromatics" similarly to OECD guideline 413. Albino rats were exposed for 6 hours/day, 5 days/week for 13 weeks at nominal vapour concentrations of 10400 mg/m3, 5200 mg/m3, and 2600 mg/m3 to assess inhalation toxicity. As there were no pathologic changes, changes in organ weights were judged to have been compensatory rather than toxic effects. Based on these results, the No Observed Adverse Effect Concentration (NOAEC) was greater than or equal to 10400 mg/m3.

Oral: C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids are expected to have a low order of repeated dose toxicity by the oral route of exposure. All tests were performed in a manner similar or equivalent to currently established OECD guidelines. In a repeated dose study where C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids were administered via oral gavage, no signs of toxicity were observed at the maximum experimental dose tested, 5000 mg/kg/day.

Inhalation: In a repeated dose study where C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids were administered via inhalation, no signs of toxicity were observed at 10400 mg/m3. Based on these observations, the repeat inhalation concentration NOAEL is ≥ 10400 mg/m3 (10.4 mg/L) for C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids.

Genetic toxicity:

C9-C14 aliphatic, < 2% aromatic hydrocarbons fluids are not mutagenic using in vitro or in vivo genotoxicity assays. In bacterial tests, C9-C14 aliphatic, < 2% aromatic hydrocarbons fluids were not mutagenic in Salmonella strains tested in the presence or absence of metabolic activation. C9 -C14 aliphatic, <2% aromatic hydrocarbon fluids were negative in a in vitro mammalian cell gene mutation assay. In sister chromatid exchange and in chromosomal aberration studies, C9-C14 aliphatic, < 2% aromatic hydrocarbons fluids did not produce an effect.

C9-C14 aliphatic, < 2% aromatic hydrocarbons fluids were also non-mutagenic when tested in an in vivo mouse bone marrow micronucleus assay and when tested in dominant lethal studies utilizing an inhalation route of exposure. All studies were conducted in a manner similar or equivalent to currently established OECD guidelines. C9-C14 aliphatic, < 2% aromatic hydrocarbons fluids are a non-genotoxic agent and classification is not warranted under the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP) or under the Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparations.

All Ames tests on "hydrocarbons, C14-C20, n-alkanes, isoalkanes, cyclics, <2% aromatics" showed no mutagenic effect with and without metabolic activation. The chromosome aberration study in CHO cells on "hydrocarbons, C12-C16, n-alkanes, isoalkanes, cyclics, <2% aromatics" also showed no signs of mutagenicity. A mouse lymphoma forward mutation assay performed with hydrodesulfurised kerosene also showed no mutagenic properties.

The weight of evidence is derived from study records reported for the C9-C14 aliphatic, <2% aromatics. C9-C14 aliphatic, <2% aromatics are not genotoxic and are not classifiable as mutagens based upon the results of reliable in vitro and in vivo studies. In bacterial reverse mutation studies, the C9-C14 aliphatic, <2% aromatics were not mutagenic in the presence or absence of metabolic activation (IUCILID section 7.6.1). In mammalian cells in vitro, and in rats in vivo there were no mutagenic, clastogenic or aneugenic effects reported in read-across from studies on C9-C14 aliphatic, <2% aromatics: a negative chromosome aberration (Human Peripheral Lymphocyte Chromosomal Aberration Test, Chinese Hamster Ovary Sister Chromatid Exchange Assay); and an in vivo inhalation exposure bone marrow chromosomal aberration study and micronucleus test (IUCILID sections 7.6.1 and 7.6.2).

Endpoint Conclusion: No adverse effect observed (negative)

Toxicity to reproduction:

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were examined for reproductive toxicity in a 28 day combined repeated dose toxicity study with the reproduction / developmental toxicity screening test (OECD TG 422). C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered oral gavage at a dose of 0, 25, 150, or 1000 mg/kg/day to groups of Sprague-Dawley rats. It was concluded that C9-C14 aliphatic, <2% aromatic hydrocarbon fluids did not induce reproductive toxicity in the parental animals and no effects on the endocrine system were observed. Therefore, the NOAEL was determined to be ≥ 1000 mg/kg bw/day.

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were examined in a reproduction / developmental toxicity screening test (OECD TG 421). C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered by oral gavage at a dose of 0 (vehicle), 100, 300, 1000 mg/kg/day to groups of Sprague-Dawley rats. It was concluded that C9-C14 aliphatic, <2% aromatic hydrocarbon fluids did not induce reproductive toxicity in the parental animals and no effects on the endocrine system were observed. Therefore, the NOAEL was determined to be ≥ 1000 mg/kg bw/day.

Based on this study and the lack of systemic toxicity, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids, are not expected to be reproductive toxicants.

In bacterial reverse mutation studies, the C14-C20 aliphatic, <2% aromatics were not mutagenic in the presence or absence of metabolic activation (IUCILID section 7.6.1). In mammalian cells in vitro, and in rats in vivo there were no mutagenic, clastogenic or aneugenic effects reported in read-across from studies on hydrodesulfurized kerosene kerosene, and jet fuels that included: a negative chromosome aberration (Human Peripheral Lymphocyte Chromosomal Aberration Test, Chinese Hamster Ovary Sister Chromatid Exchange Assay); and an in vivo inhalation exposure bone marrow chromosomal aberration study and micronucleus test in rats and mice (IUCILID sections 7.6.1 and 7.6.2).

C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids were examined for reproductive toxicity in a 28 day combined repeated dose toxicity study with the reproduction / developmental toxicity screening test (OECD TG 422). C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids were administered oral gavage at a dose of 0, 25, 150, or 1000 mg/kg/day to groups of Sprague-Dawley rats. It was concluded that C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids did not induce reproductive toxicity in the parental animals and no effects on the endocrine system were observed. Therefore, the NOAEL was determined to be ≥ 1000 mg/kg bw/day.

Exposure in humans:

Seven studies were available on the irritation and/or sensitisation potential of several types of hydrocarbon solvents in volunteers. Clinical tests were conducted with populations ranging from 29 to 112 patients. None of the test substances elicited any sensitisation and/or irritation effects except C5-C20 paraffin, which showed a cumulative irritation effect at 75% . However, this substance was tested under occlusive patch, a condition which exacerbates the irritancy of hydrocarbon solvents.

Toxicokinetics:

Continued...

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If C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are absorbed, they are typically metabolized by side chain oxidation to alcohol and carboxylic acid derivatives. These metabolites can be glucuronidated and excreted in the urine or further metabolized before being excreted. The majority of the metabolites are excreted in the urine and to a lower extent, in the faeces. Excretion is rapid with the majority of the elimination occurring within the first 24 hours of exposure. As a result of the lack of systemic toxicity and the ability of the parent material to undergo metabolism and rapid excretion, bioaccumulation of the test substance in the tissues is not likely to occur.

C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are poorly absorbed dermally with an estimated overall percutaneous absorption rate of approximately 2µg/cm²/hr or 1% of the total applied fluid. Regardless of exposure route, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are rapidly metabolized and eliminated.

C14-C20 aliphatic, <2% aromatic hydrocarbon fluids are typically metabolized by side chain oxidation to alcohol and carboxylic acid derivatives. These metabolites can be glucuronidated and excreted in the urine or further metabolized before being excreted. The majority of the metabolites are excreted in the urine and to a lower extent, in the faeces. Excretion is rapid with the majority of the elimination occurring within the first 24 hours of exposure. As a result of the lack of systemic toxicity and the ability of the parent material to undergo metabolism and rapid excretion, bioaccumulation of the test substance in the tissues is not likely to occur.

Short description of key information on absorption rate:

C14-C20 aliphatic, <2% aromatic hydrocarbon fluids can be dermally absorbed, although they tend to partition into the stratum corneum.

When dermally absorbed, C14-C20 aliphatic, <2% aromatic hydrocarbon fluids are rapidly metabolized and eliminated.

Approximately 34% of C14-C20 aliphatic, <2% aromatic hydrocarbon fluids are absorbed when ingested. C14-C20 aliphatic, <2% aromatic hydrocarbon fluids is poorly dermally absorbed. Absorption following inhalation is assumed to be similar to ingestion since exposures will be to aerosol. Regardless of exposure route, C14-C20 aliphatic, <2% aromatic hydrocarbon fluids are rapidly metabolized. Bioaccumulation of C14-C20 aliphatic, <2% aromatic hydrocarbon fluids is not expected.

C14-C20 aliphatic, <2% aromatic hydrocarbon fluids are absorbed if ingested. C14-C20 aliphatic, <2% aromatic hydrocarbon fluids undergo metabolism and rapid excretion and low deposition, bioaccumulation of the test substance in the tissues is not likely to occur.

The fate of pristane (2,6,10,14-tetramethylpentadecane) was studied in rats after a single per os administration of 3H-labeled pristane. The balance study showed extensive fecal excretion (66%) mainly as unchanged hydrocarbon, whereas about 14% of ingested pristane was excreted in urine as pristane metabolites and tritiated water. After one week, 8.3% of the ingested 3H still was stored in the carcass and the radioactive distribution in tissues and organs showed a preferential incorporation into adipose tissue and liver. Over 75% of the radioactivity stored in the carcass was associated with pristane metabolites and tritiated water. Tissue metabolites were characterized by thin layer chromatography, gas chromatography, and mass spectrometric analyses. Four metabolites were identified: pristan-1-ol, pristan-2-ol, pristanic acid and 4,8,12-trimethyltridecanoic acid. These results demonstrated that pristane undergoes subterminal hydroxylation or terminal oxidation followed by the classical beta-oxidation process.

Labeled paraffins with 8-18 C atoms prepared from unsaturated hydrocarbons by addition of deuterium have been added in oily solution to normal rats food. After six days an increase of deuterium content in the body fluid of all the rats was observed indicating that the labeled compounds had been metabolized. Deuterium was found in the fatty acids of the body fats and the liver lipids especially after feeding octadecane and hexadecane. Isolating oleic, stearic, and palmitic acids containing deuterium, indicated that methyl- and beta-oxidation of these hydrocarbons has occurred. Fatty acids resulting from the metabolism of hydrocarbons with shorter chains were not deposited but in these cases the urine contained fatty acids with higher deuterium content than after administration of octadecane and hexadecane. According to the deuterium content of the neutral fractions from the liver and body lipids all the hydrocarbons tested were deposited only to a small extent, the largest depots occurring mainly after feeding with octadecane and hexadecane.

Discussion on absorption rate:

There have not been any in vivo dermal absorption studies of C14 – C20 aliphatic, <2% aromatic hydrocarbon fluids, but there have been in vitro studies of similar constituents, particularly hexadecane.

The percutaneous absorption and cutaneous disposition of topically applied neat Jet-A, JP-8, and JP-8(100) jet fuels (25 µL/5 cm²) was examined by monitoring the absorptive flux of the marker components 14C naphthalene and 4H dodecane simultaneously applied non-occluded to isolated perfused porcine skin flaps (a = 4). Absorption of 14C hexadecane was estimated from JP-8 fuel. Absorption and disposition of naphthalene and dodecane were also monitored using a nonvolatile JP-8 fraction reflecting exposure to residual fuel that might occur 24 h after a jet fuel spill. In all studies, perfusate, stratum corneum, and skin concentrations were measured over 5 h. Naphthalene absorption had a clear peak absorptive flux at less than 1 h, while dodecane and hexadecane had prolonged, albeit significantly lower, absorption flux profiles. Within JP-8, absorption was (mean +/- SEM; % dose) hexadecane (0.18 +/- 0.08). The area under the curve (AUC) was determined to be (mean +/- SEM; % dose-h/mL): hexadecane (0.0017 +/- 0.0003).

The flux, permeability coefficient (Kp), and binding of hexadecane for porcine skin was determined to be 8.80 +/- 0.00 (nmol/cm²/h) x 10E-3.

The permeability coefficient (Kp), and binding of hexadecane for human skin were determined to be 7.02 +/- 0.00 (nmol/cm²/h) x 10E-3.

Factor of difference (FOD) in the permeability of pig and human skin was 1.28 for hexadecane. The FOD in binding of hexadecane to pig and human skin was found to be 0.76.

Over view of percutaneous absorption of hydrocarbon solvents

There are no studies of repeated dose toxicity of hydrocarbon solvents using the dermal route of administration. Accordingly, where it is necessary to calculate dermal DNELs, systemic data from studies utilizing other routes of administration, normally inhalation but also oral data, can be used in some situations. In accordance with ECHA guidance, read across from oral or inhalation data to dermal should account for differences in absorption where these exist. In fact, hydrocarbon solvents are poorly absorbed in most situations, in part because some are volatile and do not remain in contact with the skin for long periods of time and also because, due to their hydrophobic natures, do not partition well into aqueous environments and are poorly absorbed into the blood.

If these differences in relative absorption are introduced into the DNEL calculations to calculate external doses, the DNELs based on systemic effects are highly inflated. This seems potentially misleading as it implies that substances have different intrinsic hazards when encountered by different routes whereas in fact the differences are due ultimately to differences in absorbed dose.

Several authors have assessed the percutaneous absorption of higher molecular weight aliphatic constituents. Using porcine skin models the percutaneous absorption values for aliphatic constituents ranging from nonane to tetradecane were well below 1 µg/cm²/hr. Rat and human skin are considered to be more permeable than human skin (so these numbers can be considered conservative.

Results of percutaneous absorption studies with human skin under in vivo conditions produced values ranging from 1 – 2 µg/kg/day for decane, undecane and dodecane.

With respect to aromatic hydrocarbons, most of the reported percutaneous absorption values) are less than 2 µg/cm²/day.

After considering all of the above, it seems reasonable to assume apparent that across the entire range of hydrocarbon solvent constituents, percutaneous absorption values are less than 2 µg/cm²/day. Accordingly, when systemic dermal DNELs are calculated using route to route extrapolations, the values will not be corrected for differences in absorption. Rather, 2 µg/cm²/hr should be used as a common percutaneous absorption rate for all hydrocarbon solvents for which dermal exposure estimates are provided.

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PARAFFINIC DISTILLATE, LIGHT, HYDROTREATED (SEVERE)

* Q8 MSDS

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

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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 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.

**BARIUM MONO-C10-13-
ALKYLBENZENE,
SULFONATE SALTS**

mono-C10-13-Alkyl derivs., distr. residues, sulfonated, barium salts. The C10 -13 barium sulfonate test substance has a low total base number (TBN = 300), for which the specific concentration limit of 10 % or lower applies as well.

for alkaryl sulfonate petroleum additives:

Mammalian Toxicology - Acute. Existing data on acute mammalian toxicity indicates a low concern for acute toxicity.

Acute oral toxicity: In all but one studies, there were no deaths that could be attributed to treatment with the test material when administered at the limit dose of 2000 or 5000 mg/kg. In some studies, the primary clinical observations were diarrhea and reduced food consumption

Continued...

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(without a change in body weight). These effects are consistent with the gastrointestinal irritant properties of detergents in an oil-based vehicle. In other studies, decreased body weight gain or ruffled fur was observed. In one study where deaths occurred, animals were administered dose levels well above the 2000 mg/kg limit dose. Overall, the acute oral LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.

Acute dermal toxicity: No mortality was observed for any tested substance when administered at the limit dose of 2000 or 5000 mg/kg. The principal clinical observation was erythema and/or edema at the site of dermal application. In some cases, the cutaneous findings included dry, flaky skin, desquamation and hyperkeratosis. Overall, the acute dermal LD50 for these substances was greater than the 2000 mg/kg limit dose indicating a relatively low order of toxicity.

Acute inhalation toxicity: One member of the petroleum additive alkaryl sulfonate category (CAS RN: 6878396-0) was tested for acute inhalation toxicity (OECD Guideline 403, Acute Inhalation Toxicity). Rats were exposed whole-body to an aerosol of the substance at a nominal atmospheric concentration of 1.9 mg/L for four hours. This was the maximum attainable concentration due to the low volatility and high viscosity of the test material. No mortality was noted, and all animals fully recovered following depuration. Clinical signs of toxicity during exposure included reduced activity, matted coat, and closed eyes. Clinical signs of toxicity observed post exposure included lacrimation, nasal discharge, salivation rates, matted coat, hunched appearance, soft stools and closed eyes. No treatment-related macroscopic findings were noted. The lack of mortality at a concentration just below the limit dose of 2.0 mg/L indicates a relatively low order of toxicity for this substance.

Mammalian Toxicology - Subchronic Toxicity. Existing data from repeated-dose toxicity studies indicates minimal signs of toxicity following repeated oral exposure. Adverse effects at the site of contact were observed following repeated dermal exposure (injury to the skin) and repeated inhalation (injury to the lungs).

NOAELs range from 49.5 mg/m3 to 1000 mg/kg/day

Mammalian Toxicology - Reproductive and Developmental Toxicity. A one-generation reproductive toxicity test was conducted on one member of the category (CAS # 115733-09-0). Exposure to the alkaryl sulfonate did not significantly impact reproduction or development and these results were bridged to the remainder of the category.

Mammalian Toxicology - Mutagenicity. Existing data from bacterial reverse mutation assays and in vitro and in vivo chromosome aberration studies indicate a low concern for mutagenicity.

Animal Irritation

An acute eye irritation study indicates that calcium dodecylbenzenesulfonate caused irritation.

Result: irritating at 0.1 ml

An acute skin study indicate that calcium dodecylbenzenesulfonate is irritant to skin 0.5 ml according to OECD GHS guidelines.

Respiratory irritation was not observed. There were no treatment-related changes in the haematological or urinalysis values in any of the animals. No signs of irritation of respiratory tract and nasal effects were observed.

Animal studies show that calcium sulfonates with a TBN greater than 300 are not skin sensitizers while the results in animals at a TBN (Total Base Number) of 300 exhibit a mixed skin sensitisation response. However, human repeat insult patch tests clearly show that high TBN overbased calcium sulfonates (TBN = 300) are not sensitizers and that low TBN calcium sulfonates do not cause sensitisation in a substantial number of persons at concentrations of 10% or lower within the definition of sensitisation under EU Regulation (EC) No. 1272/2008.

The weight-of-evidence indicates that low TBN sodium and calcium sulfonates (TBN < 300) are skin sensitizers with a specific concentration limit (SCL) of 10% and that high TBN sodium and calcium sulfonates (TBN = 300) are not skin sensitizers. Studies in guinea pigs show that low TBN benzenesulfonic acid, mono-C20-24 (even)-sec-alkyl derivs., para-, sodium salts (EC No. None; CAS No. None; TBN = 3) is a skin sensitizer while benzenesulfonic acid, mono-C20-24 (even)-sec-alkyl derivs., para-, sodium salts TBN = 448) is not a skin sensitizer. Studies in guinea pigs and human volunteers show that low TBN benzenesulfonic acid, 4-(mono-C15 -36 branched alkyl derivs., C24 rich) and benzenesulfonic acid, 4-octadecyl, calcium salts (EC 939-141-9; TBN = 13) are skin sensitizers. Numerous well-conducted, reliable, controlled human (HRIPT) studies with benzene, polypropene derivs., sulfonated, calcium salts (EC 616-278-7; TBN values ranging from 13 to 85), sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 30 to 100), and benzenesulfonic acid, 4-(mono-C15-36 branched alkyl derivs., C24 rich) and benzenesulfonic acid, 4-octadecyl, calcium salts (EC 939-141-6; TBN = 13) show that low TBN calcium sulfonates do not cause sensitisation in a substantial number of subjects at 10% and lower. High TBN calcium sulfonates, sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 375 and 400) do not cause skin sensitisation in guinea pigs. Results of guinea pigs studies at TBN = 300 are mixed; two studies of sulfonic acids, petroleum, calcium salts, (EC 263-093-9) report no skin sensitisation while one study of sulfonic acids, petroleum, calcium salts (EC 263-093-9) and one study of benzene, polypropene derivs., sulfonated, calcium salts (EC 616-278-7) report skin sensitisation. However, numerous well-conducted, reliable, controlled human (HRIPT) studies with benzene, polypropene derivs., sulfonated, calcium salts (EC 616-278-7; TBN = 300) and sulfonic acids, petroleum, calcium salts (EC 263-093-9; TBN = 300) also show that high TBN (TBN = 300) do not cause skin sensitisation. In accordance with EU CLP Regulation (EC) No. 1272/2008, classification is required for low TBN sodium and calcium sulfonates (TBN < 300) with a specific concentration limit of 10% and classification is not required for high TBN calcium sulfonates (TBN = 300).

* REACH Dossier

DIACETONE ALCOHOL

Inhalation (human) TClO: 400 ppm resp.effect

For diacetone alcohol (DAA):

Acute toxicity: Oral LD50 of diacetone alcohol is more than 4,000 mg/kg. The lowest reported toxic concentration for human is 0.475 g/m3, although the reliability is not sure because of too old study and no detailed information. This chemical is moderately irritating to skin and irritating to eyes but there is no available data for sensitisation.

Repeat dose toxicity: In oral rat study by an OECD combined repeated dose and reproductive/developmental toxicity screening test [TG 422] at doses of 0, 30, 100, 300 and 1,000 mg/kg/day for at least 44 days, decreased locomotor activity and less response to stimulation by knocking sounds or palpation were observed in males and females of the 300 and 1,000 mg/kg groups. Histopathological examination revealed increases of deposition of hyaline droplets in the proximal tubular epithelium at doses of 100 mg/kg or more, basophilic tubules at doses of 300 and 1,000 mg/kg and dilatation of the distal tubules at dose of 1,000 mg/kg in male kidneys. Slight but no significant increases of dilated distal tubules and fatty degeneration of the proximal tubular epithelium were observed in female kidneys at doses of 300 and 1,000 mg/kg. Furthermore, hepatocellular hypertrophy was evident in both sexes of the 1,000 mg/kg group, and vacuolization of the cells of the zona fasciculata in the adrenals of males receiving 1,000 mg/kg. Based on renal toxicity in male, NOAEL by oral administration was considered 30 mg/kg/day.

An inhalation rat study conducted for 6 hr/day, 6 day/week, 6 weeks at doses of 0.232, 1.035 and 4.94 g/m3 demonstrated the histologic changes in the proximal tubules of the kidneys toxicity in males at the highest dose. As only liver weight was increased at mid dose, NOAEL was considered at 1.035 g/m3 for 6 hr/day, 6 day/week. The daily intake is roughly calculated as 156 mg/kg/day.

Reproductive and developmental toxicity: In reproductive /developmental toxicity study [OECD TG 422], there were no statistically significant adverse effects noted at any dose. However, the composite of data at the 1,000 mg/kg suggest there may be chemically related adverse effects such as decreased tendency in the fertility index, number of implantations, implantation index and birth index with two mothers ability not to normally carry the litter. Therefore, a NOAEL for reproductive/developmental toxicity was considered to be 300 mg/kg/day.

Genotoxicity: Evidence of malformations was not observed at any dose. This chemical was not genotoxic in bacterial test and chromosomal aberration test *in vitro* [OECD TG 471 & 473].

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

2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL

For imidazoline surfactants (amidoamine/ imidazoline - AAI)

All substances within the AAI group show the same reactive groups, show similar composition of amide, imidazoline, and some dimer structures of both, with the length of original EA amines used for production as biggest difference. Inherent reactivity and toxicity is not expected to differ much between these substances.

All in vivo skin irritation/corrosion studies performed on AAI substances all indicate them to be corrosive following 4 hour exposure. There do not seem to be big differences in response with the variation on EA length used for the production of the AAI.

The available data available for AAI substances indicate that for AAI based on shorter polyethyleneamines (EA), higher toxicity is observed compared to AAI based on longer EA. The forming of imidazoline itself does not seem to play a significant role. For cross-reading in general Fatty acid reaction product with diethylenetriamine (AAI-DETA) therefore represents the worst case. In series of 28-day and combined repeated dose/reproduction screening toxicity studies (OECD 422) AAI-DETA has shown the highest level of toxicity

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Acute oral exposure of tall oil + triethylenepentamine (TEPA) show limited acute toxicity, with a LD50 above 2000 mg/kg bw. Hence no classification is required.

Acute dermal testing with corrosive materials is not justified. As a consequence no classification can be made for acute dermal toxicity.

Effects will be characterised by local tissue damage. Systemic uptake via skin is likely to be very limited. The low acute oral toxicity indicate a low systemic toxicity.

For dermal exposure no good overall NOAEL can be established as effects are rather characterized by local corrosive effects that are related to duration, quantity and concentration, than by systemic toxicity due to dermal uptake. The mode of action for AAI follows from its structure, consisting of an apolar fatty acid chain and a polar end of a primary amine from the polyethyleneamine. The structure can disrupt the cytoplasmic membrane, leading to lyses of the cell content and consequently the death of the cell.

The AAI are protonated under environmental conditions which causes them to strongly adsorb to organic matter. This leads to a low dermal absorption.

No classification for acute dermal toxicity is therefore indicated.

Also for acute inhalation toxicity information for classification is lacking, and is testing not justified. Due to very low vapour pressure is the likelihood of exposure low.

AAI do not contain containing aliphatic, alicyclic and aromatic hydrocarbons and have a relatively high viscosity and so do not indicate an immediate concern for aspiration hazard.

Various studies with different AAI indicate that these substances can cause dermal sensitisation.

All substances within the AAI group show the same reactive groups, show similar composition of amide, imidazoline, and some dimer structures of both, with the length of original EA amines used for production as biggest difference. Inherent reactivity and toxicity is not expected to differ much between these substances, aspects which determine sensitization.

The actual risk of sensitisation is probably low, as AAI are corrosive to skin and consequently exposure will be low due to necessary protective measures to limit dermal exposure.

The likelihood for exposure via inhalation and thus experience respiratory irritation or becoming sensitised to AAI, is very low considering the high boiling point (> 300 deg C) and very low vapour pressure (0.00017 mPa at 25 deg C for diethylenetriamine (DETA) based AAI). In case of high exposure by inhalation, local effects will be more prominent than possible systemic effects considering the low systemic toxicity seen in acute oral toxicity testing

However, some calculations can be made for systemic effects following short-term inhalation exposure by extrapolating information from an OECD 422 study on "tall oil reaction products with tetraethylenepentamine showing a NOAEL of 300 mg/kg/day. This would certainly be protective for levels of acute inhalation expected to lead to similar systemic exposure levels.

The corrected 8 hr inhalation NOAEC for workers is NOAEL (300 mg/kg) * 1.76 mg/m³ = 529 mg/m³ (assuming no difference in absorption following oral and inhalation exposure). Assessment factors further applied: No interspecies factor is needed due to allometric scaling applied in calculation of corrected NOAEC. Further combined inter-/intra-species for workers AF = 3 (ECETOC concept). As this involves acute exposures, no extrapolation for duration is needed.

This results in a DNEL of 529/3 = 176 mg/m³. A short term/acute exposure at this level can be assumed not to lead to systemic toxicity.

Repeat dose toxicity:

A combined repeated dose/reproduction screening toxicity study according to OECD 422 with Fatty acid reaction products with tetraethylene-pentamine resulted to a NOAEL of 300 mg/kg bw/day, the highest dose tested. Also available data from the group of Amidoamine/Imidazoline (AAI) substances, including 90-day studies in rat and dogs on a similar substance, indicate very low toxicity. Consequently, serious toxicity is not observed at levels requiring consideration classification for STOTS-RE

Genotoxicity:

Tall oil, reaction products with tetraethylenepentamine is not mutagenic in the Salmonella typhimurium reverse mutation assay (based on test with Fatty acids C16-18, C18 unsaturated reaction products with tetraethylenepentamine), is not clastogenic in human lymphocytes, and not mutagenic in the TK mutation test with L5178Y mouse lymphoma cells.

It can therefore be concluded that tall oil, reaction products with tetraethylenepentamine not genotoxic.

Toxicity to reproduction:

The database of relevant studies available for the group of amidoamine/ imidazolines (AAI) include various OECD 422 studies and an OECD 414 study, that all show no concerns regarding reproduction or developmental toxicity. Also all already available data from the group of AAI substances, including a 90-day study in dogs on a similar substance, indicate low toxicity and no adverse effects on reproductive organs.

REACH Dossier

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.

Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

For amphoteric imidazoline derivatives:

Generally these amphoteric surfactants do not seem to be irritant to the skin and only to a small extent irritating to the eye. Some variation in test results have been reported.

Cocoamphodipropionate was found to be non-irritating as a concentration of 7.5-70%, whereas cocoamphopropionate was slightly irritating to rabbit skin at a concentration of 15-16%. Cocoamphodiacetate was non-irritating to slightly irritating at a concentration of 10-12%.

A Draize test has shown that cocoamphodipropionate was practically non-irritating to the eye at a concentration of 7.5%, whereas cocoamphopropionate was non-irritating to slightly irritating at 5% and 16%. Cocoamphodiacetate was moderately to severely irritating to the eye at a concentration of 10-12%. Cocoamphoacetate was slightly to severely irritating at 16 to 50%.

Sensitisation: Cocoamphoacetate and cocoamphopropionate were non-irritating and non-sensitising in a repeated insult patch test (non-occlusive) involving 141 subjects. The concentration of the surfactants was 10% in distilled water. During induction, each chemical was applied to the back three times per week for three weeks. The challenge phase was initiated 10 to 15 days after application of the final induction patch. Cocoamphoacetate and cocoamphopropionate did not induce sensitization in any of the subjects. Cocoamphoacetate was non-sensitising in guinea pigs when tested in the Magnusson-Kligman maximization test. The tested concentrations for induction and challenge were 25, 50 and 100%

Mutagenicity: Cocoamphodiacetate, cocoamphopropionate, and cocoamphodipropionate were non-mutagenic, when evaluated in the Ames Salmonella/microsome assay using different strains of *Salmonella typhimurium*.

No tests on reproductive toxicity and carcinogenicity were available.

Classification The amphoteric surfactants described in this category are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC.

OLEOYLSARCOSINE

The amino acids alkyl amides most likely dissociate into amino acids and fatty acids in the presence of water. Because most of these amino acids and fatty acids are found in the foods we consume daily, oral toxicity is not expected.

In turn, dermal toxicity would not be expected to be different from oral exposure.

Data from the previous safety assessments on alpha-amino acids and fatty acids support that these ingredients would not likely be irritants or sensitizers.

No irritation was observed in in vitro studies with disodium capryloyl glutamate. Acetyl proline was a mild irritant in another in vitro study. In human studies, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate were not dermal irritants.

No ocular irritation was observed in in vitro studies of acetyl tyrosinamide, disodium capryloyl glutamate, and sodium lauroyl glutamate. No adverse effects were observed during in-use studies of acetyl hydroxyproline and acetyl tyrosinamide in human subject.

Severe irritation was observed in 1 study of sodium cocoyl glutamate at 5%, but was not irritating in another study with an unknown concentration. Sodium cocoyl glutamate and glycinate (fatty acids, C8-14 -even numbered are generally classified as R41/ H318 - Causes

	severe eye damage - by their suppliers, in spite of contrasting evidence. No sensitisation was observed in human studies with acetyl hydroxyproline, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate. Acetyl tyrosinamide, sodium cocoyl glutamate and sodium lauroyl glutamate were not phototoxic in human studies In in vitro studies, acetyl glutamic acid, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate were negative for genotoxicity. Acetyl glutamic acid was negative in an in vivo study. The analogue chemicals, acyl sarcosines, raised concern about the possible formation of potentially carcinogenic nitrosated derivatives. For the analogue, the reactive material is likely to be the precursor sarcosine. This chemical varies in that the precursor amine glycine is a primary amine, whereas the precursor amine sarcosine in the analogue material is a secondary amine. Secondary amines are of more concern for nitrosamine formation than primary or tertiary amines. Whereas the nitrogen in chemicals fatty acid glycines and glutamates is secondary, its functional group is an amide rather than an amine and has different chemical properties. Free amine is not present therefore the possibility of nitrosamine formation is considered to be low. For sarcosine: Motor impairment and respiratory issues have been observed in rats at 10mg/kg. This equates to 800 mg for an 80 kg person. Sarcosine was reported to activate prostate cancer cells and to indicate the malignancy of prostate cancer cells when measured in urine Sarcosine was identified as a differential metabolite that was greatly increased during prostate cancer progression to metastasis and could be detected in urine. Sarcosine levels seemed to control the invasiveness of the cancer. This conclusion has been disputed Sarcosine has been investigated in relation to schizophrenia. Early evidence suggests that intake of 2 g/day sarcosine as add-on therapy to certain antipsychotics (not clozapine) in schizophrenia, sarcosine gives significant additional reductions in both positive and negative symptomatology as well as the neurocognitive and general psychopathological symptoms that are common to the illness. Sarcosine had been tolerated well. It is also under investigation for the possible prevention of schizophrenic illness during the prodromal stage of the disease. It acts as a type 1 glycine transporter inhibitor and a glycine agonist. It increases glycine concentrations in the brain thus causing increased NMDA receptor activation and a reduction in symptoms. A 2011 meta-analysis found adjunctive sarcosine to have a medium effect size for negative and total symptoms. Major depressive disorder is a complex disease and most currently available antidepressants aiming at monoamine neurotransmission exhibit limited efficacy and cognitive effects. N-methyl-D-aspartate receptors (NMDARs), one subtype of glutamate receptor, play an important role in learning and memory, and NMDAR enhancing agents, such as sarcosine (N-methylglycine), have been used as adjunctive therapy of schizophrenia. Preliminary clinic trials indicated that intake of sarcosine improved not only psychotic but also depressive symptoms in patients with schizophrenia, and so may also be a useful supplement for treating depressive type schizoaffective disorders where rapid-acting glutamatergic antidepressants, particularly NMDA antagonists such as esketamine, can promote worsening of psychotic features. Toxicological data are available and well documented for representative toluenesulfonates, xylenesulfonates and cumenesulfonates (including sodium, potassium, ammonium and calcium salts). These data demonstrate that hydrotropes have a low order of acute toxicity by all relevant routes (LC50s range from 100s to 1000s mg/kg), are not genotoxic <i>in vitro</i> or <i>in vivo</i>, show no evidence of a carcinogenic response (or any other systemic toxicity) in 2-year dermal exposure studies, and failed to induce developmental, teratogenic or fertility (sex organ) effects. Adverse effects after repeated long term dosing of hydrotropes to animals included epidermal hyperplasia at the site of application in dermal studies, and decreased relative spleen weight in females in oral studies. The critical adverse effect and corresponding systemic NOAEL is 763 mg a.i./kg bw based upon decreased relative spleen weight in female rats in a 90-day oral study. The NOAEL for local effects, based on epidermal hyperplasia at the site of application, was 440 mg a.i./kg bw for mice in 90-day dermal studies. Hydrotropes can be classified as a negligible-to-slight irritant to skin and a slight-to-moderate irritant to eyes. The irritation potential of aqueous solutions of hydrotropes depends on concentration, and the irritation is lessened with rinsing. Hydrotropes are not considered to be skin sensitisers. HERA Report (Hydrotropes) September 2005 Hydrotropes in this category were assessed for mutagen/ genotoxic potential in a variety of assays including the mouse micronucleus, Ames, mouse lymphoma, sister chromatid exchange and chromosome aberration assays. No positive results were seen in vitro or in vivo in any of the studies. For both mice and rats exposed dermally for two years, there was no evidence of carcinogenic potential. Examination of the sex organs (such as prostate, testes or ovaries) from animals in 90-day feeding studies and 90-day and two year dermal studies yielded no evidence to suggest that these chemicals have an adverse effect on the reproductive organs.		
HYDROCARBONS, C11-14 N-ALKANES, ISOALKANES, CYCLICS, AROMATIC (2-25%) & 2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL & OLEOYL SARCOSINE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.		
PARAFFINIC DISTILLATE, LIGHT, HYDROTREATED (SEVERE) & DIACETONE ALCOHOL & 2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL	No significant acute toxicological data identified in literature search.		
BARIUM MONO-C10-13-ALKYLBENZENE, SULFONATE SALTS & 2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL	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.		
DIACETONE ALCOHOL & 2-(8-HEPTADECENYL)-4,5-DIHYDRO-1H-IMIDAZOLE-1-ETHANOL	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.		
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

Continued...

SECTION 12 Ecological information

Toxicity					
PX24 #4955049 (AUS)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%)	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.53mg/l	2
	EC50	48h	Crustacea	>0.002mg/l	2
	EC50(ECx)	48h	Crustacea	>0.002mg/l	2
	EC50	96h	Algae or other aquatic plants	0.58mg/l	2
paraffinic distillate, light, hydrotreated (severe)	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>1000mg/l	1
	NOEC(ECx)	504h	Crustacea	>1mg/l	1
barium mono-C10-13-alkylbenzene, sulfonate salts	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	1000mg/l	2
diacetone alcohol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>1000mg/l	2
	EC50	48h	Crustacea	>1000mg/l	2
	NOEC(ECx)	504h	Crustacea	100mg/l	2
	LC50	96h	Fish	>100mg/l	2
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.017mg/l	2
	EC50	48h	Crustacea	0.163mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	0.011mg/l	2
oleoysarcosine	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.43mg/l	2
	NOEC(ECx)	504h	Crustacea	0.102mg/L	2
	LC50	96h	Fish	>0.43mg/l	2
carbon dioxide	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	35mg/l	1
Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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 allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
diacetone alcohol	HIGH	HIGH
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	LOW	LOW
oleoysarcosine	LOW	LOW
carbon dioxide	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
diacetone alcohol	LOW (LogKOW = -0.14)
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	LOW (LogKOW = 7.51)
oleoysarcosine	HIGH (LogKOW = 6.83)
carbon dioxide	LOW (LogKOW = 0.83)

Mobility in soil

Ingredient	Mobility
diacetone alcohol	HIGH (Log KOC = 1)
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	LOW (Log KOC = 206300)

Ingredient	Mobility
oleoylsarcosine	LOW (Log KOC = 17090)
carbon dioxide	HIGH (Log KOC = 1.498)

SECTION 13 Disposal considerations

Waste treatment methods	
Product / Packaging disposal	▶ 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. ▶ Consult State Land Waste Management Authority for disposal. ▶ Discharge contents of damaged aerosol cans at an approved site. ▶ Allow small quantities to evaporate. ▶ DO NOT incinerate or puncture aerosol cans. ▶ Bury residues and emptied aerosol cans at an approved site.

SECTION 14 Transport information

Labels Required	
Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG)		
14.1. UN number or ID number	1950	
14.2. UN proper shipping name	AEROSOLS	
14.3. Transport hazard class(es)	Class	2.1
	Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	63 190 277 327 344 381
	Limited quantity	1000ml

Air transport (ICAO-IATA / DGR)		
14.1. UN number	1950	
14.2. UN proper shipping name	Aerosols, flammable	
14.3. Transport hazard class(es)	ICAO/IATA Class	2.1
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	10L
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	A145 A167 A802
	Cargo Only Packing Instructions	203
	Cargo Only Maximum Qty / Pack	150 kg
	Passenger and Cargo Packing Instructions	203
	Passenger and Cargo Maximum Qty / Pack	75 kg
	Passenger and Cargo Limited Quantity Packing Instructions	Y203
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)		
14.1. UN number	1950	
14.2. UN proper shipping name	AEROSOLS	
14.3. Transport hazard class(es)	IMDG Class	2.1
	IMDG Subsidiary Hazard	Not Applicable

14.4. Packing group	Not Applicable	
14.5 Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	F-D, S-U
	Special provisions	63 190 277 327 344 381 959
	Limited Quantities	1000 ml

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%)	Not Applicable
paraffinic distillate, light, hydrotreated (severe)	Not Applicable
barium mono-C10-13-alkylbenzene, sulfonate salts	Not Applicable
diacetone alcohol	Not Applicable
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	Not Applicable
oleoylsarcosine	Not Applicable
carbon dioxide	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%)	Not Applicable
paraffinic distillate, light, hydrotreated (severe)	Not Applicable
barium mono-C10-13-alkylbenzene, sulfonate salts	Not Applicable
diacetone alcohol	Not Applicable
2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol	Not Applicable
oleoylsarcosine	Not Applicable
carbon dioxide	Not Applicable

SECTION 15 Regulatory information

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

hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%) is found on the following regulatory lists

Not Applicable

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

barium mono-C10-13-alkylbenzene, sulfonate salts is found on the following regulatory lists

Not Applicable

diacetone alcohol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

oleoylsarcosine is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

carbon dioxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

FEI Equine Prohibited Substances List - Controlled Medication

FEI Equine Prohibited Substances List (EPSL)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
Canada - DSL	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
Canada - NDSL	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%); paraffinic distillate, light, hydrotreated (severe); diacetone alcohol; 2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol; oleoylsarcosine; carbon dioxide)
China - IECSC	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
Europe - EINEC / ELINCS / NLP	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
Japan - ENCS	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
Korea - KECI	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
New Zealand - NZIoC	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
Philippines - PICCS	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
USA - TSCA	TSCA Inventory 'Active' substance(s) (paraffinic distillate, light, hydrotreated (severe); diacetone alcohol; 2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol; oleoylsarcosine; carbon dioxide); No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
Taiwan - TCSI	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%))
Mexico - INSQ	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%); paraffinic distillate, light, hydrotreated (severe))
Vietnam - NCI	Yes
Russia - FBEPH	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%); oleoylsarcosine)
UAE - Control List (Banned/Restricted Substances)	No (hydrocarbons, C11-14 n-alkanes, isoalkanes, cyclics, aromatic (2-25%); paraffinic distillate, light, hydrotreated (severe); diacetone alcohol; 2-(8-heptadecenyl)-4,5-dihydro-1H-imidazole-1-ethanol; oleoylsarcosine; carbon dioxide)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	24/10/2025
Initial Date	24/10/2025

Other information

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

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

Definitions and abbreviations

- PC - TWA: Permissible Concentration-Time Weighted Average
- PC - STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit,
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code
- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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