

# Pebeo Vitrail

## Jasco Pty Limited

Chemwatch Hazard Alert Code: 3

Chemwatch: 5423-19

Issue Date: 10/03/2023

Version No: 7.2

Print Date: 18/08/2024

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

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

#### Product Identifier

<b>Product name</b>	Pebeo Vitrail
<b>Chemical Name</b>	Not Applicable
<b>Synonyms</b>	EN-FDS097 Vitrail Lightening Medium; EN-FDS015 Vitrail Opale; EN-FDS243 Vitrail Transparent 3 Colours; EN-FDS245 Transparent 1 Colour; EN-FDS246 Vitrail Transparent 2 Colours; EN-FDS247 Vitrail Transparent 1 Colour
<b>Proper shipping name</b>	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
<b>Chemical formula</b>	Not Applicable
<b>Other means of identification</b>	Not Available

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

<b>Relevant identified uses</b>	Paints & Varnishes for artists. Use according to manufacturer's directions.
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#### Details of the manufacturer or supplier of the safety data sheet

<b>Registered company name</b>	Jasco Pty Limited
<b>Address</b>	1-5 Commercial Road Kingsgrove NSW 2208 Australia
<b>Telephone</b>	+61 2 9807 1555
<b>Fax</b>	Not Available
<b>Website</b>	<a href="http://www.jasco.com.au">www.jasco.com.au</a>
<b>Email</b>	quickinfo@jasco.com.au

#### Emergency telephone number

<b>Association / Organisation</b>	Australian Poisons Centre	<b>CHEMWATCH EMERGENCY RESPONSE (24/7)</b>
<b>Emergency telephone numbers</b>	13 11 26 (24/7)	+61 1800 951 288
<b>Other emergency telephone numbers</b>	Not Available	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

<b>Poisons Schedule</b>	S5
<b>Classification [1]</b>	Flammable Liquids Category 3, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Germ Cell Mutagenicity Category 1A, Carcinogenicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
<b>Legend:</b>	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)	
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Signal word	<b>Danger</b>
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**Hazard statement(s)**

<b>H226</b>	Flammable liquid and vapour.
<b>H304</b>	May be fatal if swallowed and enters airways.
<b>H315</b>	Causes skin irritation.
<b>H317</b>	May cause an allergic skin reaction.
<b>H319</b>	Causes serious eye irritation.
<b>H336</b>	May cause drowsiness or dizziness.
<b>H340</b>	May cause genetic defects.
<b>H350</b>	May cause cancer.
<b>H373</b>	May cause damage to organs through prolonged or repeated exposure.
<b>H411</b>	Toxic to aquatic life with long lasting effects.

**Precautionary statement(s) Prevention**

<b>P201</b>	Obtain special instructions before use.
<b>P210</b>	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
<b>P260</b>	Do not breathe mist/vapours/spray.
<b>P271</b>	Use only outdoors or in a well-ventilated area.
<b>P280</b>	Wear protective gloves, protective clothing, eye protection and face protection.
<b>P240</b>	Ground and bond container and receiving equipment.
<b>P241</b>	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
<b>P242</b>	Use non-sparking tools.
<b>P243</b>	Take action to prevent static discharges.
<b>P273</b>	Avoid release to the environment.
<b>P264</b>	Wash all exposed external body areas thoroughly after handling.
<b>P272</b>	Contaminated work clothing should not be allowed out of the workplace.

**Precautionary statement(s) Response**

<b>P301+P310</b>	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
<b>P331</b>	Do NOT induce vomiting.
<b>P308+P313</b>	IF exposed or concerned: Get medical advice/ attention.
<b>P370+P378</b>	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
<b>P302+P352</b>	IF ON SKIN: Wash with plenty of water and soap.
<b>P305+P351+P338</b>	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
<b>P312</b>	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
<b>P333+P313</b>	If skin irritation or rash occurs: Get medical advice/attention.
<b>P337+P313</b>	If eye irritation persists: Get medical advice/attention.
<b>P362+P364</b>	Take off contaminated clothing and wash it before reuse.
<b>P391</b>	Collect spillage.
<b>P303+P361+P353</b>	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
<b>P304+P340</b>	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

**Precautionary statement(s) Storage**

<b>P403+P235</b>	Store in a well-ventilated place. Keep cool.
<b>P405</b>	Store locked up.

**Precautionary statement(s) Disposal**

<b>P501</b>	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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## SECTION 3 Composition / information on ingredients

### Substances

See section below for composition of Mixtures

### Mixtures

CAS No	%[weight]	Name
64742-48-9.	<50	<u>naphtha petroleum, heavy, hydrotreated</u>
64742-48-9	<25	<u>hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, &lt;2% aromatics</u>
64742-82-1.	<10	<u>naphtha, petroleum, hydrodesulfurised heavy</u>
107-98-2	<10	<u>propylene glycol monomethyl ether - alpha isomer</u>
64216-15-5	<2.5	<u>calcium isononanoate</u>
96-29-7	<2.5	<u>methyl ethyl ketoxime</u>
22464-99-9	<2.5	<u>zirconium 2-ethylhexanoate</u>
85203-81-2	<2.5	<u>2-ethylhexanoic acid, zinc salt, basic</u>
136-52-7	<2.5	<u>cobalt 2-ethylhexanoate</u>
85029-59-0	<2.5	<u>C.I. Solvent Orange 54</u>
73297-15-1	<2.5	<u>C.I. Solvent Red 122</u>
85408-46-4	<2.5	<u>C.I. Solvent Yellow 88</u>
7631-86-9	NotSpec	<u>silica amorphous</u>
108-94-1	NotSpec	<u>cyclohexanone</u>
13463-67-7	NotSpec	<u>titanium dioxide</u>
91-20-3	NotSpec	<u>naphthalene</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

## SECTION 4 First aid measures

### Description of first aid measures

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

### Indication of any immediate medical attention and special treatment needed

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

## SECTION 5 Firefighting measures

### Extinguishing media

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

### Special hazards arising from the substrate or mixture

<b>Fire Incompatibility</b>	▶ 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

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▶ Use water delivered as a fine spray to control fire and cool adjacent area.</li> <li>▶ Avoid spraying water onto liquid pools.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Liquid and vapour are flammable.</li> <li>▶ Moderate fire hazard when exposed to heat or flame.</li> <li>▶ Vapour forms an explosive mixture with air.</li> <li>▶ Moderate explosion hazard when exposed to heat or flame.</li> <li>▶ Vapour may travel a considerable distance to source of ignition.</li> <li>▶ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▶ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> </ul> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) nitrogen oxides (NO<sub>x</sub>) metal oxides other pyrolysis products typical of burning organic material.</p>
<b>HAZCHEM</b>	•3Y

## 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

<b>Minor Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Remove all ignition sources.</li> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb small quantities with vermiculite or other absorbent material.</li> </ul>
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	<ul style="list-style-type: none"> <li>▶ Wipe up.</li> <li>▶ Collect residues in a flammable waste container.</li> </ul>
<b>Major Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ May be violently or explosively reactive.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Consider evacuation (or protect in place).</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Increase ventilation.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Water spray or fog may be used to disperse /absorb vapour.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Use only spark-free shovels and explosion proof equipment.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Absorb remaining product with sand, earth or vermiculite.</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

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

## SECTION 7 Handling and storage

### Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of overexposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Prevent concentration in hollows and sumps.</li> <li>▶ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▶ Avoid smoking, naked lights or ignition sources.</li> <li>▶ Avoid generation of static electricity.</li> <li>▶ <b>DO NOT use plastic buckets.</b></li> <li>▶ Earth all lines and equipment.</li> <li>▶ Use spark-free tools when handling.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ Store in original containers in approved flammable liquid storage area.</li> <li>▶ Store away from incompatible materials in a cool, dry, well-ventilated area.</li> <li>▶ <b>DO NOT store in pits, depressions, basements or areas where vapours may be trapped.</b></li> <li>▶ No smoking, naked lights, heat or ignition sources.</li> <li>▶ Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access.</li> <li>▶ Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.</li> <li>▶ Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.</li> <li>▶ Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors.</li> <li>▶ Keep adsorbents for leaks and spills readily available.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul> <p>In addition, for tank storages (where appropriate):</p> <ul style="list-style-type: none"> <li>▶ Store in grounded, properly designed and approved vessels and away from incompatible materials.</li> <li>▶ For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up.</li> <li>▶ Storage tanks should be above ground and diked to hold entire contents.</li> </ul>

### Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<ul style="list-style-type: none"> <li>▶ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure.</li> <li>▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C)</li> <li>▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C)</li> <li>▶ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used.</li> </ul>
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	<ul style="list-style-type: none"> <li>▶ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages</li> <li>▶ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.</li> <li>▶ Polyethylene or polypropylene container.</li> <li>▶ Packing as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid reaction with oxidising agents, bases and strong reducing agents.</li> <li>▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.</li> </ul>

## 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	naphtha petroleum, heavy, hydrotreated	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, <2% aromatics	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	naphtha, petroleum, hydrodesulfurised heavy	White spirits	790 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether - alpha isomer	Propylene glycol monomethyl ether	100 ppm / 369 mg/m3	553 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	zirconium 2-ethylhexanoate	Zirconium compounds (as Zr)	5 mg/m3	10 mg/m3	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Fume (thermally generated)(respirable dust)	2 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Fumed silica (respirable dust)	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Diatomaceous earth (uncalcined)	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica gel	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica amorphous	Silica - Amorphous: Precipitated silica	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	cyclohexanone	Cyclohexanone	25 ppm / 100 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
						no asbestos and < 1% crystalline silica.
Australia Exposure Standards	naphthalene	Naphthalene	10 ppm / 52 mg/m3	79 mg/m3 / 15 ppm	Not Available	Not Available

## Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
naphtha petroleum, heavy, hydrotreated	350 mg/m3	1,800 mg/m3	40,000 mg/m3
hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, <2% aromatics	350 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	350 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	300 mg/m3	1,800 mg/m3	29500** mg/m3
propylene glycol monomethyl ether - alpha isomer	100 ppm	160 ppm	660 ppm
methyl ethyl ketoxime	30 ppm	56 ppm	250 ppm
silica amorphous	18 mg/m3	200 mg/m3	1,200 mg/m3
silica amorphous	18 mg/m3	100 mg/m3	630 mg/m3
silica amorphous	120 mg/m3	1,300 mg/m3	7,900 mg/m3
silica amorphous	45 mg/m3	500 mg/m3	3,000 mg/m3
silica amorphous	18 mg/m3	740 mg/m3	4,500 mg/m3
cyclohexanone	60 ppm	830 ppm	5000* ppm
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3
naphthalene	15 ppm	83 ppm	500 ppm

Ingredient	Original IDLH	Revised IDLH
naphtha petroleum, heavy, hydrotreated	2,500 mg/m3	Not Available
hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, <2% aromatics	2,500 mg/m3	Not Available
naphtha, petroleum, hydrodesulfurised heavy	20,000 mg/m3	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available	Not Available
calcium isononanoate	Not Available	Not Available
methyl ethyl ketoxime	Not Available	Not Available
zirconium 2-ethylhexanoate	25 mg/m3	Not Available
2-ethylhexanoic acid, zinc salt, basic	Not Available	Not Available
cobalt 2-ethylhexanoate	Not Available	Not Available
C.I. Solvent Orange 54	Not Available	Not Available
C.I. Solvent Red 122	Not Available	Not Available
C.I. Solvent Yellow 88	Not Available	Not Available
silica amorphous	3,000 mg/m3	Not Available
cyclohexanone	700 ppm	Not Available

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Ingredient	Original IDLH	Revised IDLH
titanium dioxide	5,000 mg/m <sup>3</sup>	Not Available
naphthalene	250 ppm	Not Available

#### Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
calcium isononanoate	E	≤ 0.01 mg/m <sup>3</sup>
methyl ethyl ketoxime	D	> 0.1 to ≤ 1 ppm
2-ethylhexanoic acid, zinc salt, basic	E	≤ 0.1 ppm
cobalt 2-ethylhexanoate	E	≤ 0.01 mg/m <sup>3</sup>
<b>Notes:</b>	<i>Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.</i>	

#### MATERIAL DATA

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7). Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP


#### 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. For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant. 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.										
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	<p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p> <ul style="list-style-type: none"> <li>· Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance.</li> <li>· Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures.</li> </ul>										

Continued...



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	<ul style="list-style-type: none"> <li>Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)</li> </ul>
<b>Individual protection measures, such as personal protective equipment</b>	
<b>Eye and face protection</b>	<ul style="list-style-type: none"> <li>Safety glasses with side shields.</li> <li>Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]</li> <li>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].</li> </ul>
<b>Skin protection</b>	See Hand protection below
<b>Hands/feet protection</b>	<ul style="list-style-type: none"> <li>Wear chemical protective gloves, e.g. PVC.</li> <li>Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>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.</li> <li>Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>frequency and duration of contact,</li> <li>chemical resistance of glove material,</li> <li>glove thickness and</li> <li>dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>Excellent when breakthrough time &gt; 480 min</li> <li>Good when breakthrough time &gt; 20 min</li> <li>Fair when breakthrough time &lt; 20 min</li> <li>Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.</li> <li>Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential</li> </ul> <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>
<b>Body protection</b>	See Other protection below
<b>Other protection</b>	<ul style="list-style-type: none"> <li>Overalls.</li> <li>PVC Apron.</li> <li>PVC protective suit may be required if exposure severe.</li> <li>Eyewash unit.</li> <li>Ensure there is ready access to a safety shower.</li> <li>Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.</li> <li>For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).</li> <li>Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground</li> </ul>

the foot shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

## 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
BUTYL	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
PE/EVAL/PE	C
PVC	C
TEFLON	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 A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

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

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

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

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

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

<b>Appearance</b>	Coloured liquid; does not mix with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	0.91-0.98
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Available
<b>pH (as supplied)</b>	Not Available	<b>Decomposition temperature (°C)</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	210-250	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	24-37.90	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Flammable.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Available	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Available	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	<300	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Available

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Vapour density (Air = 1)	Not Available	VOC g/L	388-486
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## SECTION 10 Stability and reactivity

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

<b>Inhaled</b>	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>Some aliphatic hydrocarbons produce axonal neuropathies. Isoparaffinic hydrocarbons produce injury to the kidneys of male rats. When albino rats were exposed to isoparaffins at 21.4 mg/l for 4 hours, all animals experienced weakness, tremors, salivation, mild to moderate convulsions, chromodacryorrhoea and ataxia within the first 24 hours. Symptoms disappeared after 24 hours.</p> <p>Several studies have evaluated sensory irritation in laboratory animals or odor or sensory response in humans. When evaluated by a standard procedure to assess upper airway irritation, isoparaffins did not produce sensory irritation in mice exposed to up to 400 ppm isoparaffin in air. Human volunteers were exposed for six hours to 100 ppm isoparaffin. The subjects were given a self-administered questionnaire to evaluate symptoms, which included dryness of the mucous membranes, loss of appetite, nausea, vomiting, diarrhea, fatigue, headache, dizziness, feeling of inebriation, visual disturbances, tremor, muscular weakness, impairment of coordination or paresthesia. No symptoms associated with solvent exposure were observed. With a human expert panel, odour from liquid imaging copier emissions became weakly discernible at approximately 50 ppm.</p> <p>Numerous long-term exposures have been conducted in animals with only one major finding observed. Renal tubular damage has been found in kidneys of male rats upon repeated exposures to isoparaffins. It does not occur in mice or in female rats. This male rat nephropathy has been observed with a number of hydrocarbons, including wholly vaporized unleaded gasoline. The phenomenon has been attributed to reversible binding of hydrocarbon to alpha2-globulin. Since humans do not synthesize alpha2-globulin or a similar protein, the finding is not considered to be of biological significance to man. No clinically significant renal abnormalities have been found in refinery workers exposed to hydrocarbons.</p> <p>When evaluated for developmental toxicity in rats, isoparaffins were neither embryotoxic nor teratogenic. Isoparaffins were consistently negative on standard bacterial genotoxicity assays. They were also non-genotoxic in <i>in vivo</i> mammalian testing for somatic or germ cell mutations (mouse micronucleus test and rat dominant lethal assay, respectively).</p> <p>Mullin et al: Jnl Applied Toxicology 10, pp 136-142, 2006</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p>
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<b>Ingestion</b>	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Many aliphatic hydrocarbons create a burning sensation because they are irritating to the GI mucosa. Vomiting has been reported in up to one third of all hydrocarbon exposures. While most aliphatic hydrocarbons have little GI absorption, aspiration frequently occurs, either initially or in a semi-delayed fashion as the patient coughs or vomits, thereby resulting in pulmonary effects. Once aspirated, the hydrocarbons can create a severe pneumonitis.</p> <p>Rats given isoparaffinic hydrocarbons - isoalkanes- (after 18-24 hours fasting) showed lethargy and/or general weakness, ataxia and diarrhoea. Symptoms disappeared within 24-28 hours.</p> <p>Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.</p>
<b>Skin Contact</b>	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period.</p> <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Dermally, isoparaffins have produced slight to moderate irritation in animals and humans under occluded patch conditions where evaporation cannot freely occur. However, they are not irritating in non-occluded tests, which are a more realistic simulation of human exposure. They have not been found to be sensitisers in guinea pig or human patch testing. However, occasional rare idiosyncratic sensitisation reactions in humans have been reported.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
<b>Eye</b>	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>Instillation of isoparaffins into rabbit eyes produces only slight irritation.</p> <p>Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.</p>
<b>Chronic</b>	<p>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.</p> <p>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.</p> <p>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</p> <p>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.</p> <p>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.</p> <p>On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of</p> <ul style="list-style-type: none"> <li>- appropriate animal studies,</li> <li>- other relevant information</li> </ul> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p>

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Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.

Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

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

Studies with some glycol ethers (principally the monoethylene glycols) and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decreases significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats (which produces haemolytic anaemia). This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure.

Glycol ethers based on propylene oxides, propylene glycol ethers, dipropylene glycol ethers and tripropylene glycol ethers are mainly available, commercially, as alpha-isomers (because of thermodynamic considerations); these are incapable of forming alkoxyacetic or alkoxypropionic acids as metabolites and therefore do not produce erythrocyte fragility unless contaminated by ethylene glycol ethers or to a significant degree by the beta-isomer. Beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic 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.

Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

Pebeo Vitrail	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
naphtha petroleum, heavy, hydrotreated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation (Rat) LC50: >4.42 mg/L4h <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >4500 mg/kg <sup>[1]</sup>	
hydrocarbons, C9-11, n-alkanes, isoalkanes,	<b>TOXICITY</b>	<b>IRRITATION</b>

Continued...

## Pebeo Vitrail

cyclics, <2% aromatics	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation (Rat) LC50: >5.266 mg/L4h <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >5000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
naphtha, petroleum, hydrodesulfurised heavy	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation (Rat) LC50: >1.58 mg/l4h <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
propylene glycol monomethyl ether - alpha isomer	Oral (Rat) LD50: >4500 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit) 230 mg mild
	Inhalation (Rat) LC50: >6 mg/l4h <sup>[2]</sup>	Eye (rabbit) 500 mg/24 h. - mild
	Oral (Rat) LD50: 3739 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg SEVERE
		Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin (rabbit) 500 mg open - mild
calcium isononanoate	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: 2000 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50: 1160 mg/kg <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
methyl ethyl ketoxime	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >184<1840 mg/kg <sup>[1]</sup>	Eye (rabbit): 0.1 ml - SEVERE
	Inhalation (Rat) LC50: >4.83 mg/l4h <sup>[1]</sup>	Eye: adverse effect observed (irreversible damage) <sup>[1]</sup>
zirconium 2-ethylhexanoate	Oral (Rat) LD50: >900 mg/kg <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >870 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
2-ethylhexanoic acid, zinc salt, basic	Inhalation (Rat) LC50: >4.3 mg/l4h <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >=2000 mg/kg <sup>[1]</sup>	
	<b>TOXICITY</b>	<b>IRRITATION</b>
cobalt 2-ethylhexanoate	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Inhalation (Rat) LC50: >2.5 mg/L4h <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: 3129 mg/kg <sup>[1]</sup>	
C.I. Solvent Orange 54	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >5000 mg/kg <sup>[2]</sup>	Eye (rabbit): non-irritant
		Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin (rabbit): non-irritant
C.I. Solvent Red 122		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >5000 mg/kg <sup>[2]</sup>	Eye (rabbit) : Not irritating *
C.I. Solvent Yellow 88		Skin (rabbit) : Not irritating *
	<b>TOXICITY</b>	<b>IRRITATION</b>
	Oral (Rat) LD50: >5000 mg/kg <sup>[2]</sup>	Eye (rabbit): non-irritating *



## Pebeo Vitrail

		Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin (rabbit): non-irritating *
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>silica amorphous</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2000 mg/kg <sup>[1]</sup>	Eye (rabbit): non-irritating ** [Grace]
	Inhalation (Rat) LC50: >0.09<0.84 mg/l4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >1000 mg/kg <sup>[1]</sup>	Skin (rabbit): non-irritating *
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>cyclohexanone</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 948 mg/kg <sup>[2]</sup>	Eye (human): 75 ppm
	Inhalation (Rat) LC50: 8000 ppm4h <sup>[2]</sup>	Eye (rabbit): 0.25 mg/24h SEVERE
	Oral (Rat) LD50: 1535 mg/kg <sup>[2]</sup>	Eye (rabbit): 4.74 mg SEVERE
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit): 500 mg(open) mild
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
<b>titanium dioxide</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (hamster) LD50: >=10000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation (Rat) LC50: >2.28 mg/l4h <sup>[1]</sup>	Skin (human): 0.3 mg /3D (int)-mild *
	Oral (Rat) LD50: >=2000 mg/kg <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>naphthalene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (rat) LD50: >2500 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg - mild
	Inhalation (Rat) LC50: >0.4 mg/l4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: 490 mg/kg <sup>[2]</sup>	Skin (rabbit):495 mg (open) - mild
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>

**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

<b>NAPHTHA PETROLEUM, HEAVY, HYDROTREATED</b>	<p>For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation.</p> <p>Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans.</p> <p>Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants).</p> <p>Reproductive toxicity: Animal studies show that high concentrations of toluene (&gt;0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus.</p> <p>Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials.</p> <p>Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.</p>
<b>HYDROCARBONS, C9-11, N-ALKANES, ISOALKANES, CYCLICS, &lt;2% AROMATICS</b>	<p>For alkanes:</p> <p>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.</p> <p>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 &gt; 24.3 g/m3 (6646 ppm). Additionally, no neurological effects were reported in the NTP 2 year carcinogenicity study on Stoddard solvent.</p> <p>For hydrocarbons, C5-C7, n-alkanes, isoalkanes, n-hexane rich</p> <p>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</p>

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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 2µg/cm<sup>2</sup>/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 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 LD<sub>50</sub> 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 LD<sub>50</sub> is >5000 mg/kg, via dermal exposure where the LD<sub>50</sub> is >5000 mg/kg, and by inhalation where the LC<sub>50</sub> > 5000 mg/m<sup>3</sup>. 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 mm<sup>2</sup>/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 LC<sub>50</sub> was determined to be 73,680 ppm (259354 mg/m<sup>3</sup>). Due to the high concentration of the LC<sub>50</sub>, 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 EC<sub>3</sub> 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 EC<sub>3</sub> 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/m<sup>3</sup>. Based on these observations, the repeat inhalation concentration NOAEL is =10400 mg/m<sup>3</sup> (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/m<sup>3</sup>). 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/m<sup>3</sup>).

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/m<sup>3</sup>) based on colour changes in the lungs. The developmental NOAEC was 3000 ppm and the LOAEC was 9000 ppm (31680 mg/m<sup>3</sup>) 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/m<sup>3</sup>). 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:**

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/m<sup>3</sup>.

**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/m<sup>3</sup>. Based on these observations, the repeat inhalation concentration NOAEL is >=10400 mg/m<sup>3</sup> (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 tests 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/m<sup>3</sup>, 5200 mg/m<sup>3</sup>, and 2600 mg/m<sup>3</sup> to assess inhalation toxicity. As there were no pathological 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/m<sup>3</sup>.

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/m<sup>3</sup>. Based on these observations, the repeat inhalation concentration NOAEL is >=10400 mg/m<sup>3</sup> (10.4 mg/L) for C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids.

**Genetic toxicity:**

C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids are not mutagenic using in vitro or in vivo genotoxicity assays. In bacterial tests, C9-C14 aliphatic, < 2% aromatic hydrocarbon 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 hydrocarbon fluids did not produce an effect. C9-C14 aliphatic, < 2% aromatic hydrocarbon 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 hydrocarbon 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  $\geq 1000$  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  $\geq 1000$  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  $\geq 1000$  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:**

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 2ug/cm<sup>2</sup>/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, pristane-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 cm<sup>2</sup>) 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/cm<sup>2</sup>/h) x 10E-3. The permeability coefficient (Kp), and binding of hexadecane for human skin were determined to be 7.02 +/- 0.00 (nmol/cm<sup>2</sup>/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/cm<sup>2</sup>/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/cm<sup>2</sup>/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/cm<sup>2</sup>/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/cm<sup>2</sup>/hr should be used as a common percutaneous absorption rate for all hydrocarbon solvents for which dermal exposure estimates are provided.

#### PROPYLENE GLYCOL MONOMETHYL ETHER - ALPHA ISOMER

NOTE: For PGE - mixed isomers: Exposure of pregnant rats and rabbits to the substance did not give rise to teratogenic effects at concentrations up to 3000 ppm. Foetotoxic effects were seen in rats but not in rabbits at this concentration; maternal toxicity was noted in both species.

for propylene glycol ethers (PGEs):

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).

Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.

Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).

This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.

Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.

As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.

As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m<sup>3</sup> for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m<sup>3</sup>. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m<sup>3</sup>), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating.

None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology).

LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m<sup>3</sup> (600 ppm) for PnB and 2,010 mg/m<sup>3</sup> (260 ppm) for DPnB.

TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m<sup>3</sup> (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m<sup>3</sup> (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m<sup>3</sup>) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m<sup>3</sup>). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m<sup>3</sup>), with decreased body weights occurring at 3000 ppm (11058 mg/m<sup>3</sup>). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported.

Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

#### CALCIUM ISONONANOATE

Oral (rat) NOAEL 50 mg/kg bw/day \* REACH Dossier

Pelargonic acid and its Esters - Cosmetic Ingredient Review: CIR Expert Panel Meeting, June 2010

For pelargonic acid esters (branched and straight-chain):

The CIR Expert Panel concluded that these esters are safe as cosmetic ingredients in the present practices of use and concentration.

Pelargonic acid and nonanoate esters are cosmetic ingredients that function as skin conditioning agents in cosmetics.

Pelargonic acid, a fatty acid, is absorbed and then metabolized by beta-oxidation in the body. The percutaneous absorption of isononyl alcohol was reported in an acute dermal toxicity study. Iso-fatty acids and straight-chain fatty acids both are metabolized at the beta carbon to yield two-carbon fractions by mitochondrial and microsomal fractions of rat liver homogenate.

The CIR Expert Panel recognizes that the pelargonic acid branched esters included in a safety assessment are not pure substances, but are always mixtures. Thus, the international nomenclature cosmetic ingredient (INCI) name for each pelargonic acid branched ester actually refers to a mixture with general properties that, in many instances, cannot be fully characterized.

Regardless, the material, as supplied to the industry for use in cosmetics, is the same material that is used in safety testing.

The Expert Panel noted the availability of acute inhalation toxicity data on pelargonic acid, isononanoic acid, and isononyl alcohol, but not on any other of the ingredients that are being reviewed in this safety assessment. However, in the absence of these data, the Panel determined that the ingredients included in this review can be used safely in hair sprays, because the product particle size is not respirable. The Panel reasoned that the particle size of aerosol hair sprays (around 38 µm) and pump hair sprays (>80 µm) is large compared to respirable particle sizes (= 10 µm). Because animal sources of cetearyl isononanoate, cetyl isononanoate, cholesteryl nonanoate, dihydrocholesteryl nonanoate, and isostearyl isononanoate have been reported, the Panel was also concerned with the dangers inherent in using animal-derived ingredients, namely the transmission of infectious agents.



	<p>The Expert Panel discussed that this group of ingredients contains both iso (branched) fatty acid esters and straight-chain fatty acid esters. While the metabolic fate of branched esters, alcohols, and fatty acids is still unknown, limited data on metabolism of iso forms versus straight-chain forms in rat liver homogenates indicated that both are oxidized in a similar fashion, but that the iso form additionally may be oxidized at an additional hydrolysis site.</p> <p>Panel deliberations on the safety of pelargonic acid esters focused on current ingredient use concentration data from industry in relation to human RIPT data on cosmetic products containing these ingredients. Current ingredient use concentrations ranged from 0.01% (cholesteryl nonanoate) to 74% (ethylhexyl isononanoate), and ethylhexyl isononanoate was a mild skin irritant, but a nonsensitizer, when tested undiluted. Similarly, cetearyl nonanoate and neopentyl glycol diisononanoate, both undiluted, did not induce sensitization in human RIPTs. Other data indicate that the highest ester concentration evaluated in a human RIPT was 51.35% isodecyl isononanoate in a makeup product (nonirritant and nonsensitizer), which approaches the maximum use concentration of 59% for this ingredient. Based on negative results for sensitization at high ingredient test concentrations, the Expert Panel reasoned that is not likely that the pelargonic acid esters reviewed in this safety assessment would induce sensitization in the present practices of use</p> <p><a href="https://www.cir-safety.org/sites/default/files/115_final_pelarg_rev2.pdf">https://www.cir-safety.org/sites/default/files/115_final_pelarg_rev2.pdf</a></p>
<p><b>METHYL ETHYL KETOXIME</b></p>	<p>Mammalian lymphocyte mutagen *Huls Canada ** Merck For methyl ethyl ketoxime (MEKO)</p> <p><b>Carcinogenicity:</b> Increased incidences of liver tumours were observed in rat and mouse lifetime studies and there was also an increased incidence of mammary gland tumours in female rats, however, this was only seen at mid- and/or high concentrations of MEKO. Consideration of the available information regarding genotoxicity indicate that MEKO is not likely to be genotoxic. Accordingly, although the mode of induction of tumours is not fully elucidated, the tumours observed are not considered to have resulted from direct interaction with genetic material.</p> <p>The European Commission (2000) considered that a possible mechanism for the increased incidences of liver tumours in male rats and mice was the metabolism of MEKO to a carcinogenic agent, mediated by sulfotransferase. The sex and organ specificity of tumour formation correlated with the typically higher activity of this enzyme in male rodents.</p> <p><b>Genotoxicity:</b> The <i>in vitro</i> and <i>in vivo</i> genotoxicity results for MEKO were mostly negative, including an <i>in vivo</i> study that utilized inhalation exposure and was found to be negative for DNA adducts in rat liver cells. Therefore, based on the available data, MEKO appears to lack mutagenic potential.</p> <p><b>Repeat dose toxicity:</b> Non-neoplastic effects were also observed in the nasal cavity of rats and/or mice in inhalation studies of short-term through to chronic exposure. Also, repeated dose studies based on oral exposure showed effects in the spleen, liver and kidney of rats as well as haematological effects in both rats and rabbits.</p> <p><b>Reproductive toxicity:</b> In a one-generation oral rat study, the LOAEL for reproductive toxicity was 100 mg/kg-bw per day, the highest dose, based on a statistically significant decrease in female delivery index (%), whereas no treatment-related effects on reproductive parameters were observed in a two-generation study in which rats were dosed by gavage at 0-200 mg/kg-bw per day. In both the one-generation and two-generation rat studies, a parental LOAEL of 10 mg/kg-bw per day, the lowest dose tested, was established, based on histopathological effects in the spleen and liver (and in the kidney in the one-generation study).</p> <p><b>Developmental toxicity:</b> Teratogenicity was not observed in pregnant rats and rabbits dosed orally with MEKO during gestation. The lowest oral LOAEL for developmental toxicity was 40 mg/kg-bw per day, the highest dose, based on abortions in 3 of 10 adult females in pregnant rabbits dosed by gavage during gestation. The lowest oral LOAEL for maternal toxicity was 10 mg/kg-bw per day, based on signs of anemia (increased reticulocytes and methaemoglobin) in rabbits dosed at 0-80 mg/kg-bw per day in a range-finding developmental study</p>
<p><b>C.I. SOLVENT ORANGE 54</b></p>	<p>Primary skin irritation rabbit: non-irritant (OECD Guideline 404) * Primary irritations of the mucous membrane rabbit: non-irritant (OECD Guideline 405) * * Kelly ** BASF Neozapon Orange 251</p>
<p><b>C.I. SOLVENT YELLOW 88</b></p>	<p>Not mutagenic in Ames assay * * Ciba Speciality Chemicals MSDS</p>
<p><b>SILICA AMORPHOUS</b></p>	<p>Reports indicate high/prolonged exposures to amorphous silicas induced lung fibrosis in experimental animals; in some experiments these effects were reversible. [PATTYS]</p> <p>For silica amorphous: Derived No Adverse Effects Level (NOAEL) in the range of 1000 mg/kg/d.</p> <p>In humans, synthetic amorphous silica (SAS) is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.</p> <p>When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.</p> <p>After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.</p> <p>Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.</p> <p>Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact. Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure.</p> <p>Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m<sup>3</sup> to 150 mg/m<sup>3</sup>. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m<sup>3</sup>. When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m<sup>3</sup>. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL.</p> <p>Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic <i>in vitro</i>. No genotoxicity was</p>

	<p>detected in in vivo assays. SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.</p> <p>For Synthetic Amorphous Silica (SAS)</p> <p>Repeated dose toxicity</p> <p>Oral (rat), 2 weeks to 6 months, no significant treatment-related adverse effects at doses of up to 8% silica in the diet.</p> <p>Inhalation (rat), 13 weeks, Lowest Observed Effect Level (LOEL) = 1.3 mg/m<sup>3</sup> based on mild reversible effects in the lungs.</p> <p>Inhalation (rat), 90 days, LOEL = 1 mg/m<sup>3</sup> based on reversible effects in the lungs and effects in the nasal cavity.</p> <p>For silane treated synthetic amorphous silica:</p> <p>Repeated dose toxicity: oral (rat), 28-d, diet, no significant treatment-related adverse effects at the doses tested.</p> <p>There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS. Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.</p>
CYCLOHEXANONE	<p>Cyclohexanone:</p> <p><b>Acute toxicity:</b> Cyclohexanone exhibits low to slight acute toxicity by the oral and inhalation routes and is moderately toxic by the dermal route. It is an eye and skin irritant; however, it did not induce skin sensitisation.</p> <p>There has been no consistent indication that cyclohexanone causes neurotoxicity, although signs of CNS depression were noted at doses near the LD50. Therefore, this material could not be classified regarding its potential neurotoxicity to humans.</p> <p><b>Repeat dose toxicity:</b> Upon repeated administration to rats in drinking water, the NOAEL was 4700 ppm after 25 weeks and the LOAEL was 3300 ppm after 2 years. Effects at higher concentrations were primarily body weight decreases. The NOAEL in published repeated dose inhalation studies was 100-190 ppm. Those values were based on either gray mottling of the lungs or ocular irritation and degenerative changes in the liver and kidney at higher concentrations. However, the NOAEL in those studies was not confirmed in more conclusive and GLP inhalation studies for reproductive and developmental effects (NOAEL = 650-1000 ppm).</p> <p><b>Genotoxicity:</b> The majority of the experimental evidence indicates that cyclohexanone is not genotoxic, and this material was not considered to be carcinogenic in mice or rats following two years of exposure via the drinking water.</p> <p><b>Reproductive toxicity:</b> In a two-generation reproduction study, decreased fertility was observed in rats exposed via inhalation at 1400 ppm but not at 500 ppm; however, the effect was found to be reversible following a post-exposure recovery period. The NOAEL of 500 ppm for this reproductive effect is 1000 times greater than the highest occupational personal monitoring value (0.5 ppm) reported.</p> <p><b>Developmental toxicity:</b> Developmental studies indicate that foetal toxicity was present only at concentrations which were maternally toxic, and no malformations were detected.</p> <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
TITANIUM DIOXIDE	<p>* IUCLID</p> <p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p> <p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p> <p>For titanium dioxide:</p> <p>Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.</p> <p>Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.</p> <p>Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide</p>

	<p>particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium. Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium oxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.</p> <p><b>Animal carcinogenicity data</b></p> <p>Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.</p> <p>In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative. Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.</p> <p>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p><b>NAPHTHALENE</b></p>	<p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<p><b>NAPHTHA PETROLEUM, HEAVY, HYDROTREATED &amp; HYDROCARBONS, C9-11, N-ALKANES, ISOALKANES, CYCLICS, &lt;2% AROMATICS &amp; NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY</b></p>	<p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.</p>
<p><b>HYDROCARBONS, C9-11, N-ALKANES, ISOALKANES, CYCLICS, &lt;2% AROMATICS &amp; NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY &amp; ZIRCONIUM 2-ETHYLHEXANOATE &amp; 2-ETHYLHEXANOIC ACID, ZINC SALT, BASIC &amp; COBALT 2-ETHYLHEXANOATE &amp; TITANIUM DIOXIDE</b></p>	<p>No significant acute toxicological data identified in literature search.</p>
<p><b>CALCIUM ISONONANOATE &amp; ZIRCONIUM 2-ETHYLHEXANOATE</b></p>	<p>For aliphatic fatty acids (and salts)</p> <p>Acute oral (gavage) toxicity:</p> <p>The acute oral LD50 values in rats for both were greater than &gt;2000 mg/kg bw. Clinical signs were generally associated with poor condition following administration of high doses (salivation, diarrhoea, staining, piloerection and lethargy). There were no adverse effects on body weight in any study. In some studies, excess test substance and/or irritation in the gastrointestinal tract was observed at necropsy.</p> <p>Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length.</p> <p>According to several OECD test regimes the animal skin irritation studies indicate that the C6-10 aliphatic acids are severely irritating or corrosive, while the C12 aliphatic acid is irritating, and the C14-22 aliphatic acids generally are not irritating or mildly irritating.</p> <p>Human skin irritation studies using more realistic exposures (30-minute, 1-hour or 24-hours) indicate that the aliphatic acids have sufficient, good or very good skin compatibility.</p> <p>Animal eye irritation studies indicate that among the aliphatic acids, the C8-12 aliphatic acids are irritating to the eye while the C14-22 aliphatic acids are not irritating.</p> <p>Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes.</p> <p>Dermal absorption:</p> <p>The in vitro penetration of C10, C12, C14, C16 and C18 fatty acids (as sodium salt solutions) through rat skin decreases with increasing chain length. At 86.73 µg C16/cm<sup>2</sup> and 91.84 µg C18/cm<sup>2</sup>, about 0.23% and less than 0.1% of the C16 and C18 soap solutions is absorbed after 24 h exposure, respectively.</p>

**Sensitisation:**

No sensitisation data were located.

**Repeat dose toxicity:**

Repeated dose oral (gavage or diet) exposure to aliphatic acids did not result in systemic toxicity with NOELs greater than the limit dose of 1000 mg/kg bw. .

**Mutagenicity**

Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo

**Carcinogenicity**

No data were located for carcinogenicity of aliphatic fatty acids.

**Reproductive toxicity**

No effects on fertility or on reproductive organs, or developmental effects were observed in studies on aliphatic acids and the NOELs correspond to the maximum dose tested. The weight of evidence supports the lack of reproductive and developmental toxicity potential of the aliphatic acids category.

Given the large number of substances in this category, their closely related chemical structure, expected trends in physical chemical properties, and similarity of toxicokinetic properties, both mammalian and aquatic endpoints were filled using read-across to the closest structural analogue, and selecting the most conservative supporting substance effect level.

Structure-activity relationships are not evident for the mammalian toxicity endpoints. That is, the low mammalian toxicity of this category of substances limits the ability to discern structural effects on biological activity. Regardless, the closest structural analogue with the most conservative effect value was selected for read across. Irritation is observed for chain lengths up to a cut-off\* at or near 12 carbons).

**Metabolism:**

The aliphatic acids share a common degradation pathway in which they are metabolized to acetyl-CoA or other key metabolites in all living systems. Common biological pathways result in structurally similar breakdown products, and are, together with the physico-chemical properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

Differences in metabolism or biodegradability of even and odd numbered carbon chain compounds or saturated/ unsaturated compounds are not expected; even- and odd-numbered carbon chain compounds, and the saturated and unsaturated compounds are naturally occurring and are expected to be metabolized and biodegraded in the same manner.

The acid and alkali salt forms of the homologous aliphatic acid are expected to have many similar physicochemical and toxicological properties when they become bioavailable; therefore, data read across is used for those instances where data are available for the acid form but not the salt, and vice versa. In the gastrointestinal tract, acids and bases are absorbed in the undissociated (non-ionised) form by simple diffusion or by facilitated diffusion. It is expected that both the acids and the salts will be present in (or converted to) the acid form in the stomach. This means that for both aliphatic acid or aliphatic acid salt, the same compounds eventually enter the small intestine, where equilibrium, as a result of increased pH, will shift towards dissociation (ionised form).

Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated. Note that the saturation or unsaturation level is not a factor in the toxicity of these substances and is not a critical component of the read across process..

**Toxicokinetics:**

The turnover of the [14C] surfactants in the rat showed that there was no significant difference in the rate or route of excretion of 14C given by intraperitoneal or subcutaneous administration. The main route of excretion was as 14CO<sub>2</sub> in the expired air at 6 h after administration. The remaining material was incorporated in the body. Longer fatty acid chains are more readily incorporated than shorter chains. At ca. 1.55 and 1.64 mg/kg bw, 71% of the C16:0 and 56% of the C18:0 was incorporated and 21% and 38% was excreted as 14CO<sub>2</sub>, respectively.

Glycidyl fatty acid esters (GEs), one of the main contaminants in processed oils, are mainly formed during the deodorisation step in the refining process of edible oils and therefore occur in almost all refined edible oils. GEs are potential carcinogens, due to the fact that they readily hydrolyze into the free form glycidol in the gastrointestinal tract, which has been found to induce tumours in various rat tissues. Therefore, significant effort has been devoted to inhibit and eliminate the formation of GEs. GEs contain a common terminal epoxide group but exhibit different fatty acid compositions. This class of compounds has been reported in edible oils after overestimation of 3-monochloropropane-1,2-diol (3-MCPD) fatty acid esters analysed by an indirect method. 3-MCPD esters have been studied as food processing contaminants and are found in various food types and food ingredients, particularly in refined edible oils. 3-Monochloropropane-1,2-diol (3-MCPD) and 2-monochloropropane-1,3-diol (2-MCPD) are chlorinated derivatives of glycerol (1,2,3-propanetriol). 3- and 2-MCPD and their fatty acid esters are among non-volatile chloropropanols. Glycidol is associated with the formation and decomposition of 3- and 2-MCPD. It forms monoesters with fatty acids (GE) during the refining of vegetable oils. Chloropropanols are formed in HVP during the hydrochloric acid-mediated hydrolysis step of the manufacturing process. In food production, chloropropanols form from the reaction of endogenous or added chloride with glycerol or acylglycerol.

Although harmful effects on humans and animals have not been demonstrated, the corresponding hydrolysates, 3-MCPD and glycidol, have been identified as rodent genotoxic carcinogens, ultimately resulting in the formation of kidney tumours (3-MCPD) and tumours at other tissue sites (glycidol). Therefore, 3-MCPD and glycidol have been categorised as "possible human carcinogens (group 2B) and "probably carcinogenic to humans (group 2A), respectively, by the International Agency for Research on Cancer (IARC).

Diaclylglyceride (DAG) based oils produced by one company were banned from the global market due to "high levels" of GEs. Several reports have also suggested that a bidirectional transformation process may occur not only between glycidol and 3-MCPD but also their esterified forms in the presence of chloride ions. The transformation rate of glycidol to 3-MCPD was higher than that of 3-MCPD to glycidol under acidic conditions in the presence of chloride ion.

Precursors of GEs in refined oils have been identified as partial acylglycerols, that is, DAGs and monoacylglycerides (MAGs); however, whether they also originate from triacylglycerides (TAGs) is still a topic of controversial debates. Several authors noted that pure TAGs were stable during heat treatment (such as 235 deg C) for 3 h and were therefore not involved in the formation of GEs. However, experimental results have shown that small amounts of GEs are present in a heat-treated oil model consisting of almost 100% TAGs. The formation of GEs from TAGs can be attributed to the pyrolysis of TAGs to DAGs and MAGs. In contrast, 3-MCPD esters in refined oils can be obtained from TAG. Presently, the mechanism for the formation of GE intermediates and the relationship between GEs and 3-MCPD esters are still unknown.



	Fatty acid salts are of low acute toxicity. Their skin and eye irritation potential is chain length dependent and decreases with increasing chain length - they are poorly absorbed through the skin nor are they skin sensitisers. The available repeated dose toxicity data demonstrate the low toxicity of the fatty acids and their salts. Also, they are not considered to be mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. Accidental ingestion of fatty acid salt containing detergent products is not expected to result in any significant adverse health effects. This assessment is based on toxicological data demonstrating the low acute oral toxicity of fatty acid salts and the fact that not a single fatality has been reported in the UK following accidental ingestion of detergents containing fatty acid salts. Also in a report published by the German Federal Institute for Health Protection of Consumers and Veterinary Medicine, detergent products were not mentioned as dangerous products with a high incidence of poisoning. The estimated total human exposure to fatty acid salts, from the different exposure scenarios for the handling and use of detergent products containing fatty acid salts, showed a margin of exposure (MOE) of 258,620. This extremely large MOE is large enough to be reassuring with regard to the relatively small variability of the hazard data on which it is based. Also, in the UK, the recommended dietary fatty acid intake by the Department of Health is about 100 g of fatty acids per day or 1.7 g (1700 mg) of fatty acids per kilogram body weight per day. This exposure is several orders of magnitude above that resulting from exposure to fatty acid salts in household cleaning products. Based on the available data, the use of fatty acid salts in household detergent and cleaning products does not raise any safety concerns with regard to consumer
<b>METHYL ETHYL KETOXIME &amp; COBALT 2-ETHYLHEXANOATE</b>	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.
<b>SILICA AMORPHOUS &amp; CYCLOHEXANONE</b>	The substance is classified by IARC as Group 3: <b>NOT</b> classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
<b>CYCLOHEXANONE &amp; TITANIUM DIOXIDE &amp; NAPHTHALENE</b>	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
<b>TITANIUM DIOXIDE &amp; NAPHTHALENE</b>	<b>WARNING:</b> This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

<b>Acute Toxicity</b>	✗	<b>Carcinogenicity</b>	✓
<b>Skin Irritation/Corrosion</b>	✓	<b>Reproductivity</b>	✗
<b>Serious Eye Damage/Irritation</b>	✓	<b>STOT - Single Exposure</b>	✓
<b>Respiratory or Skin sensitisation</b>	✓	<b>STOT - Repeated Exposure</b>	✓
<b>Mutagenicity</b>	✓	<b>Aspiration Hazard</b>	✓

**Legend:** ✗ – Data either not available or does not fill the criteria for classification  
 ✓ – Data available to make classification

## SECTION 12 Ecological information

### Toxicity

Pebeo Vitrail	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
naphtha petroleum, heavy, hydrotreated	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>0.002mg/l	2
	EC50(ECx)	48h	Crustacea	>0.002mg/l	2
	EC50	96h	Algae or other aquatic plants	64mg/l	2
hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, <2% aromatics	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	NOEC(ECx)	504h	Crustacea	0.011mg/l	2
naphtha, petroleum, hydrodesulfurised heavy	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	13mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	0.1mg/l	1
	EC50	48h	Crustacea	>0.002mg/l	2
	EC50(ECx)	48h	Crustacea	>0.002mg/l	2

Continued...

## Pebeo Vitrail

	EC50	96h	Algae or other aquatic plants	64mg/l	2
	EC50	72h	Algae or other aquatic plants	0.53mg/l	2
	NOEC(ECx)	504h	Crustacea	0.097mg/l	2
	EC50	96h	Algae or other aquatic plants	0.58mg/l	2
	EC50	48h	Crustacea	>100mg/l	1
	EC50(ECx)	48h	Crustacea	>100mg/l	1
	EC50	96h	Algae or other aquatic plants	450mg/l	1
	EC50	72h	Algae or other aquatic plants	6.5mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	<0.1mg/l	1
	LC50	96h	Fish	>100000mg/L	4
	EC50	96h	Algae or other aquatic plants	64mg/l	2
	EC50(ECx)	24h	Crustacea	36mg/l	1
	LC50	96h	Fish	0.007mg/L	4
	EC50	72h	Algae or other aquatic plants	6.5mg/l	1
	EC50	48h	Crustacea	2.7-5.1mg/L	4
	EC50	96h	Algae or other aquatic plants	64mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	<0.1mg/l	1
	LC50	96h	Fish	8.8mg/l	4
	EC50	72h	Algae or other aquatic plants	6.5mg/l	1
	EC50	96h	Algae or other aquatic plants	64mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	<0.1mg/l	1
	EC50	96h	Algae or other aquatic plants	0.277mg/l	2
	NOEC(ECx)	720h	Fish	0.02mg/l	2
	LC50	96h	Fish	0.14mg/l	2
propylene glycol monomethyl ether - alpha isomer	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	>500mg/l	2
	EC50(ECx)	168h	Algae or other aquatic plants	>1000mg/l	1
	EC50	48h	Crustacea	23300mg/l	1
	LC50	96h	Fish	>2000mg/l	Not Available
	EC50	96h	Algae or other aquatic plants	>1000mg/l	2
calcium isononanoate	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	51mg/l	Not Available
	LC50	96h	Fish	122mg/l	Not Available
	EC50(ECx)	72h	Algae or other aquatic plants	51mg/l	Not Available
methyl ethyl ketoxime	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	BCF	1008h	Fish	0.5-0.6	7
	EC50	72h	Algae or other aquatic plants	~6.09mg/l	2
	EC50	48h	Crustacea	~201mg/l	2
	LC50	96h	Fish	>100mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	~1.02mg/l	2
zirconium 2-ethylhexanoate	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>0.042mg/L	2
	EC50	48h	Crustacea	>0.17mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	0.004mg/L	2
2-ethylhexanoic acid, zinc salt, basic	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	2.72mg/l	2
	EC50	48h	Crustacea	0.105mg/L	2
	EC10(ECx)	168h	Algae or other aquatic plants	0.003mg/L	2

Continued...

## Pebeo Vitrail

	LC50	96h	Fish	0.112mg/L	2
cobalt 2-ethylhexanoate	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	0.029mg/L	2
	ErC50	72h	Algae or other aquatic plants	0.654mg/L	2
	EC50	48h	Crustacea	0.241mg/L	2
	LC50	96h	Fish	0.8mg/l	2
	EC10(ECx)	168h	Crustacea	0.001mg/L	2
	EC50	96h	Algae or other aquatic plants	10.8mg/l	2
C.I. Solvent Orange 54	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	>100mg/l	2
	EC0(ECx)	48h	Crustacea	>100mg/l	2
C.I. Solvent Red 122	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	Not Available	Not Available	Not Available	Not Available	Not Available
C.I. Solvent Yellow 88	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	LC50	96h	Fish	10mg/l	Not Available
silica amorphous	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	14.1mg/l	2
	EC50	48h	Crustacea	>86mg/l	2
	LC50	96h	Fish	1033.016mg/l	2
	EC50	96h	Algae or other aquatic plants	217.576mg/l	2
	EC0(ECx)	24h	Crustacea	>=10000mg/l	1
cyclohexanone	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	17.7-85.6mg/l	4
	EC10(ECx)	72h	Algae or other aquatic plants	0.4-7.93mg/l	4
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	481-578mg/L	4
titanium dioxide	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	BCF	1008h	Fish	<1.1-9.6	7
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
	LC50	96h	Fish	1.85-3.06mg/l	4
	NOEC(ECx)	672h	Fish	>=0.004mg/L	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2
naphthalene	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	BCF	1344h	Fish	23-146	7
	EC50	72h	Algae or other aquatic plants	ca.0.4mg/L	1
	EC50	48h	Crustacea	1.09-3.4mg/l	4
	LC50	96h	Fish	0.213mg/L	4
	EC50(ECx)	0.05h	Crustacea	<0.001mg/L	4
<b>Legend:</b>	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				

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

Continued...

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.

For petroleum distillates:

Environmental fate:

When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.

As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.

Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus, when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants. The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes. The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.

Biodegradation:

Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons. Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:

- (1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
- (2) isoalkanes;
- (3) alkenes;
- (4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
- (5) monoaromatics;
- (6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
- (7) higher molecular weight cycloalkanes (which may degrade very slowly).

Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble, volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.

When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile this is referred to as residual NAPL. Above the retention capacity, the NAPL becomes mobile and will move within the soil.

Bioaccumulation:

Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances. Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5.

In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.

Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs.

These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,

one study suggests that some alkyl-PAHs may biomagnify. While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.

In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.

Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.

This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal.

Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish.

Ecotoxicity:

Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and *Daphnia magna*, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. *Daphnia magna* had a 24-hour LC50 of 1.8 mg/L. The values varied greatly for aquatic species such as rainbow trout and *Daphnia magna*, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. *Daphnia magna* had a 24-hour LC50 of 1.8 mg/L.

The tropical mysid *Metamysidopsis insularis* was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L. This species has been shown to be as sensitive as temperate mysids to toxicants. However, this study used nominal concentrations, and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (*Crangon crangon*) a 96-hour LC50 of 22 mg/L was determined. A "gas oil" was also tested and a 96-hour LC50 of 12 mg/L was determined.

The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species. The diatom *Phaeodactylum tricorutum* showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga *Isochrysis galbana* was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L.

Finally, the green algae *Chlorella salina* was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L. All populations of phytoplankton returned to a steady state within 5 days of exposure

In sandy soils, earthworm (*Eisenia fetida*) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight loss was recorded

Nephrotoxic effects of diesel fuel have been documented in several animal and human studies. Some species of birds (mallard ducks in particular) are generally resistant to the toxic effects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality

**DO NOT** discharge into sewer or waterways.

### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene glycol monomethyl ether - alpha isomer	LOW (Half-life = 56 days)	LOW (Half-life = 1.7 days)
methyl ethyl ketoxime	LOW	LOW
silica amorphous	LOW	LOW
cyclohexanone	LOW	LOW
titanium dioxide	HIGH	HIGH
naphthalene	HIGH (Half-life = 258 days)	LOW (Half-life = 1.23 days)

### Bioaccumulative potential

Ingredient	Bioaccumulation
propylene glycol monomethyl ether - alpha isomer	LOW (BCF = 2)
methyl ethyl ketoxime	LOW (BCF = 5.8)
silica amorphous	LOW (LogKOW = 0.5294)
cyclohexanone	LOW (BCF = 2.45)
titanium dioxide	LOW (BCF = 10)
naphthalene	HIGH (BCF = 18000)

### Mobility in soil

Ingredient	Mobility
propylene glycol monomethyl ether - alpha isomer	HIGH (Log KOC = 1)
methyl ethyl ketoxime	LOW (Log KOC = 130.8)
silica amorphous	LOW (Log KOC = 23.74)
cyclohexanone	LOW (Log KOC = 15.15)
titanium dioxide	LOW (Log KOC = 23.74)
naphthalene	LOW (Log KOC = 1837)

## SECTION 13 Disposal considerations

### Waste treatment methods



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

- ▶ Where in doubt contact the responsible authority.
- ▶ Recycle wherever possible.
- ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.
- ▶ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material).
- ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

## SECTION 14 Transport information

### Labels Required

	
Marine Pollutant	
HAZCHEM	•3Y

### Land transport (ADG)

14.1. UN number or ID number	1263				
14.2. UN proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)				
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>Class</td> <td>3</td> </tr> <tr> <td>Subsidiary Hazard</td> <td>Not Applicable</td> </tr> </table>	Class	3	Subsidiary Hazard	Not Applicable
Class	3				
Subsidiary Hazard	Not Applicable				
14.4. Packing group	III				
14.5. Environmental hazard	Environmentally hazardous				
14.6. Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>163 223 367</td> </tr> <tr> <td>Limited quantity</td> <td>5 L</td> </tr> </table>	Special provisions	163 223 367	Limited quantity	5 L
Special provisions	163 223 367				
Limited quantity	5 L				

### Air transport (ICAO-IATA / DGR)

14.1. UN number	1263														
14.2. UN proper shipping name	Paint related material (including paint thinning or reducing compounds)														
14.3. Transport hazard class(es)	<table border="1"> <tr> <td>ICAO/IATA Class</td> <td>3</td> </tr> <tr> <td>ICAO / IATA Subsidiary Hazard</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>3L</td> </tr> </table>	ICAO/IATA Class	3	ICAO / IATA Subsidiary Hazard	Not Applicable	ERG Code	3L								
ICAO/IATA Class	3														
ICAO / IATA Subsidiary Hazard	Not Applicable														
ERG Code	3L														
14.4. Packing group	III														
14.5. Environmental hazard	Environmentally hazardous														
14.6. Special precautions for user	<table border="1"> <tr> <td>Special provisions</td> <td>A3 A72 A192</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>366</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>220 L</td> </tr> <tr> <td>Passenger and Cargo Packing Instructions</td> <td>355</td> </tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td> <td>60 L</td> </tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td> <td>Y344</td> </tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td> <td>10 L</td> </tr> </table>	Special provisions	A3 A72 A192	Cargo Only Packing Instructions	366	Cargo Only Maximum Qty / Pack	220 L	Passenger and Cargo Packing Instructions	355	Passenger and Cargo Maximum Qty / Pack	60 L	Passenger and Cargo Limited Quantity Packing Instructions	Y344	Passenger and Cargo Limited Maximum Qty / Pack	10 L
Special provisions	A3 A72 A192														
Cargo Only Packing Instructions	366														
Cargo Only Maximum Qty / Pack	220 L														
Passenger and Cargo Packing Instructions	355														
Passenger and Cargo Maximum Qty / Pack	60 L														
Passenger and Cargo Limited Quantity Packing Instructions	Y344														
Passenger and Cargo Limited Maximum Qty / Pack	10 L														

### Sea transport (IMDG-Code / GGVSee)

14.1. UN number	1263
14.2. UN proper shipping name	PAINT RELATED MATERIAL (including paint thinning or reducing compound)

Continued...

14.3. Transport hazard class(es)	IMDG Class	3
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	III	
14.5. Environmental hazard	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-E , S-E
	Special provisions	163 223 367 955
	Limited Quantities	5 L

**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
naphtha petroleum, heavy, hydrotreated	Not Available
hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, <2% aromatics	Not Available
naphtha, petroleum, hydrodesulfurised heavy	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available
calcium isononanoate	Not Available
methyl ethyl ketoxime	Not Available
zirconium 2-ethylhexanoate	Not Available
2-ethylhexanoic acid, zinc salt, basic	Not Available
cobalt 2-ethylhexanoate	Not Available
C.I. Solvent Orange 54	Not Available
C.I. Solvent Red 122	Not Available
C.I. Solvent Yellow 88	Not Available
silica amorphous	Not Available
cyclohexanone	Not Available
titanium dioxide	Not Available
naphthalene	Not Available

**14.7.3. Transport in bulk in accordance with the IGC Code**

Product name	Ship Type
naphtha petroleum, heavy, hydrotreated	Not Available
hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, <2% aromatics	Not Available
naphtha, petroleum, hydrodesulfurised heavy	Not Available
propylene glycol monomethyl ether - alpha isomer	Not Available
calcium isononanoate	Not Available
methyl ethyl ketoxime	Not Available
zirconium 2-ethylhexanoate	Not Available
2-ethylhexanoic acid, zinc salt, basic	Not Available
cobalt 2-ethylhexanoate	Not Available
C.I. Solvent Orange 54	Not Available
C.I. Solvent Red 122	Not Available
C.I. Solvent Yellow 88	Not Available
silica amorphous	Not Available

Continued...

Product name	Ship Type
cyclohexanone	Not Available
titanium dioxide	Not Available
naphthalene	Not Available

## SECTION 15 Regulatory information

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

#### naphtha petroleum, heavy, hydrotreated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australian Inventory of Industrial Chemicals (AIIC)  
 Chemical Footprint Project - Chemicals of High Concern List  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

#### hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, <2% aromatics is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australian Inventory of Industrial Chemicals (AIIC)  
 Chemical Footprint Project - Chemicals of High Concern List  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

#### naphtha, petroleum, hydrodesulfurised heavy is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australian Inventory of Industrial Chemicals (AIIC)  
 Chemical Footprint Project - Chemicals of High Concern List  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

#### propylene glycol monomethyl ether - alpha isomer is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australian Inventory of Industrial Chemicals (AIIC)

#### calcium isononanoate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

#### methyl ethyl ketoxime is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6  
 Australian Inventory of Industrial Chemicals (AIIC)  
 Chemical Footprint Project - Chemicals of High Concern List

#### zirconium 2-ethylhexanoate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)  
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

#### 2-ethylhexanoic acid, zinc salt, basic is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4  
 Australian Inventory of Industrial Chemicals (AIIC)

#### cobalt 2-ethylhexanoate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
 Australian Inventory of Industrial Chemicals (AIIC)  
 Chemical Footprint Project - Chemicals of High Concern List  
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic  
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

#### C.I. Solvent Orange 54 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

#### C.I. Solvent Red 122 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

#### C.I. Solvent Yellow 88 is found on the following regulatory lists



## Australian Inventory of Industrial Chemicals (AIIC)

**silica amorphous is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

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

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**cyclohexanone is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

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

**titanium dioxide is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**naphthalene is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

**Additional Regulatory Information**

Not Applicable

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (calcium isononanoate)
Canada - NDSL	No (naphtha petroleum, heavy, hydrotreated; hydrocarbons, C9-11, n-alkanes, isoalkanes, cyclics, <2% aromatics; naphtha, petroleum, hydrodesulfurised heavy; propylene glycol monomethyl ether - alpha isomer; methyl ethyl ketoxime; zirconium 2-ethylhexanoate; 2-ethylhexanoic acid, zinc salt, basic; cobalt 2-ethylhexanoate; C.I. Solvent Orange 54; C.I. Solvent Red 122; C.I. Solvent Yellow 88; cyclohexanone; naphthalene)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (C.I. Solvent Red 122)
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (calcium isononanoate)
USA - TSCA	No (2-ethylhexanoic acid, zinc salt, basic)
Taiwan - TCSI	Yes
Mexico - INSQ	No (calcium isononanoate; zirconium 2-ethylhexanoate; 2-ethylhexanoic acid, zinc salt, basic; C.I. Solvent Orange 54; C.I. Solvent Red 122; C.I. Solvent Yellow 88)
Vietnam - NCI	Yes
Russia - FBEPH	No (calcium isononanoate; 2-ethylhexanoic acid, zinc salt, basic; C.I. Solvent Orange 54; C.I. Solvent Red 122; C.I. Solvent Yellow 88)
<b>Legend:</b>	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**

<b>Revision Date</b>	10/03/2023
<b>Initial Date</b>	01/09/2020

### SDS Version Summary

Version	Date of Update	Sections Updated
7.1	10/03/2023	Classification change due to full database hazard calculation/update.
7.2	07/05/2024	Identification of the substance / mixture and of the company / undertaking - Synonyms

### 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
  
- 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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TEL (+61 3) 9572 4700.