

# Syncrude Synthetic Crude Oil - SAN 0930

## Syncrude Canada Ltd

Chemwatch Hazard Alert Code: 4

Chemwatch: 947-5050

Issue Date: 11/02/2022

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Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 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>	Syncrude Synthetic Crude Oil - SAN 0930
<b>Chemical Name</b>	Not Applicable
<b>Synonyms</b>	Syncrude Sweet Blend; Syncrude Sweet Premium; Synthetic Crude Oil; SSB; SSP; Syncrude sample Tag # 200000
<b>Proper shipping name</b>	PETROLEUM DISTILLATES, N.O.S. or PETROLEUM PRODUCTS, N.O.S. (contains petroleum crude oil)
<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>	Refinery feed stock for petroleum and petrochemical refining.
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#### Details of the manufacturer or supplier of the safety data sheet

<b>Registered company name</b>	Syncrude Canada
<b>Address</b>	PO Bag 4009, MD X203 Fort McMurray Alberta T9H 3L1 Canada
<b>Telephone</b>	+1 780 790 5911
<b>Fax</b>	+1 780 790 6186
<b>Website</b>	<a href="http://www.syncrude.ca">http://www.syncrude.ca</a>
<b>Email</b>	askhr@syncrude.com

#### Emergency telephone number

<b>Association / Organisation</b>	CHEMWATCH EMERGENCY RESPONSE
<b>Emergency telephone numbers</b>	+61 1800 951 288
<b>Other emergency telephone numbers</b>	+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>	Not Applicable
<b>Classification [1]</b>	Flammable Liquids Category 1, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Germ Cell Mutagenicity Category 1B, Carcinogenicity Category 1A, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
<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)**

H224	Extremely flammable liquid and vapour.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H336	May cause drowsiness or dizziness.
H340	May cause genetic defects.
H350	May cause cancer.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H410	Very toxic to aquatic life with long lasting effects.

**Precautionary statement(s) Prevention**

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P271	Use only a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.

**Precautionary statement(s) Response**

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

**Precautionary statement(s) Storage**

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

**Precautionary statement(s) Disposal**

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

### Substances

See section below for composition of Mixtures

### Mixtures

CAS No	%[weight]	Name
8002-05-9	<100	<u>petroleum crude oil</u>
64742-46-7.	13-71	<u>distillates, petroleum, middle, hydrotreated</u>
64742-49-0.	15-58	<u>naphtha petroleum, light, hydrotreated.</u>
64742-59-2	17-50	<u>gas oil, petroleum, hydrotreated, vacuum</u>
111-65-9	5-10	<u>n-octane</u>
108-38-3	5-10	<u>m-xylene</u>
106-42-3	5-10	<u>p-xylene</u>
95-47-6	1-5	<u>o-xylene</u>
142-82-5	1-5	<u>heptane</u>
111-84-2	1-5	<u>n-nonane</u>
108-88-3	1-5	<u>toluene</u>
100-41-4	1-5	<u>ethylbenzene</u>
71-43-2	0.2	<u>benzene</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, without delay.</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.

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- Heavy and persistent skin contamination over many years may lead to dysplastic changes. Pre-existing skin disorders may be aggravated by exposure to this product.
- In general, emesis induction is unnecessary with high viscosity, low volatility products, i.e. most oils and greases.
- High pressure accidental injection through the skin should be assessed for possible incision, irrigation and/or debridement.

**NOTE:** Injuries may not seem serious at first, but within a few hours tissue may become swollen, discoloured and extremely painful with extensive subcutaneous necrosis. Product may be forced through considerable distances along tissue planes.

Following acute or short term repeated exposures to n-hexane:

- Large quantities of n-hexane are expired by the lungs after vapour exposure (50-60%). Humans exposed to 100 ppm demonstrate an n-hexane biological half life of 2 hours.
- Initial attention should be directed towards evaluation and support of respiration. Cardiac dysrhythmias are a potential complication.

#### INGESTION:

- Ipecac syrup should be considered for ingestion of pure hexane exceeding 2-3ml/kg. Extreme caution must be taken to avoid aspiration since small amounts of n-hexane intratracheally, produce a severe chemical pneumonitis.

[Ellenhorn and Barceloux: Medical Toxicology]

#### BIOLOGICAL EXPOSURE INDEX - BEI

BEIs represent the levels of determinants which are most likely to be observed in specimens collected in a healthy worker who has been exposed to chemicals to the same extent as a worker with inhalation exposure to the Exposure Standard (ES or TLV).

Determinant	Index	Sampling Time	Comments
1. 2,5-hexanedione in urine	5 mg/gm creatinine	End of shift	NS
2. n-Hexane in end-exhaled air			SQ

NS: Non-specific determinant; Metabolite observed following exposure to other materials.

SQ: Semi-quantitative determinant; Interpretation may be ambiguous - should be used as a screening test or confirmatory test.

## 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>	<ul style="list-style-type: none"> <li>Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result</li> </ul>
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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 in the event of a fire.</li> <li>Prevent, by any means available, spillage from entering drains or water course.</li> <li>Consider evacuation (or protect in place).</li> <li>Fight fire from a safe distance, with adequate cover.</li> <li>If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>Use water delivered as a fine spray to control the fire and cool adjacent area.</li> <li>Avoid spraying water onto liquid pools.</li> <li><b>Do not approach containers suspected to be hot.</b></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 highly flammable.</li> <li>Severe fire hazard when exposed to heat, flame and/or oxidisers.</li> <li>Vapour forms an explosive mixture with air.</li> </ul>

	<ul style="list-style-type: none"> <li>▸ Severe explosion hazard, in the form of vapour, when exposed to flame or spark.</li> <li>▸ Vapour may travel a considerable distance to source of ignition.</li> <li>▸ Heating may cause expansion / decomposition with 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>)  sulfur oxides (SO<sub>x</sub>)  hydrogen sulfide (H<sub>2</sub>S)  other pyrolysis products typical of burning organic material.  May emit clouds of acrid smoke  <b>CARE:</b> Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.</p>
<b>HAZCHEM</b>	3YE

## 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>	<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> <li>▸ Wipe up.</li> <li>▸ Collect residues in a flammable waste container.</li> </ul>																																																																											
<b>Major Spills</b>	<p>Chemical Class: aromatic hydrocarbons  For release onto land: recommended sorbents listed in order of priority.</p> <table border="1"> <thead> <tr> <th>SORBENT TYPE</th> <th>RANK</th> <th>APPLICATION</th> <th>COLLECTION</th> <th>LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5"><b>LAND SPILL - SMALL</b></td> </tr> <tr> <td>Feathers - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>DGC, RT</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R,W,SS</td> </tr> <tr> <td>cross-linked polymer- pillow</td> <td>2</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I, P,</td> </tr> <tr> <td>treated clay/ treated natural organic - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I</td> </tr> <tr> <td>wood fibre - pillow</td> <td>4</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td colspan="5"><b>LAND SPILL - MEDIUM</b></td> </tr> <tr> <td>cross-linked polymer -particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R, W, SS</td> </tr> <tr> <td>treated clay/ treated natural organic - particulate</td> <td>2</td> <td>blower</td> <td>skiploader</td> <td>R, I</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>W, SS, DGC</td> </tr> <tr> <td>feathers - pillow</td> <td>3</td> <td>throw</td> <td>skiploader</td> <td>DGC, RT</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> </tbody> </table> <p>Legend  DGC: Not effective where ground cover is dense  R; Not reusable  I: Not incinerable  P: Effectiveness reduced when rainy  RT:Not effective where terrain is rugged  SS: Not for use within environmentally sensitive sites  W: Effectiveness reduced when windy  Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;  R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988</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> </ul>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	<b>LAND SPILL - SMALL</b>					Feathers - pillow	1	throw	pitchfork	DGC, RT	cross-linked polymer - particulate	2	shovel	shovel	R,W,SS	cross-linked polymer- pillow	2	throw	pitchfork	R, DGC, RT	sorbent clay - particulate	3	shovel	shovel	R, I, P,	treated clay/ treated natural organic - particulate	3	shovel	shovel	R, I	wood fibre - pillow	4	throw	pitchfork	R, P, DGC, RT	<b>LAND SPILL - MEDIUM</b>					cross-linked polymer -particulate	1	blower	skiploader	R, W, SS	treated clay/ treated natural organic - particulate	2	blower	skiploader	R, I	sorbent clay - particulate	3	blower	skiploader	R, I, P	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	feathers - pillow	3	throw	skiploader	DGC, RT	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC
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- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Consider evacuation (or protect in place).
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse /absorb vapour.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Use only spark-free shovels and explosion proof equipment.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Absorb remaining product with sand, earth or vermiculite.
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

## SECTION 7 Handling and storage

### Precautions for safe handling

#### Safe handling

Hydrogen sulfide (H<sub>2</sub>S or Sour Gas) may be present when loading and unloading transport vessels. Stay upwind and away from newly opened hatches and allow to vent thoroughly before handling material. Steam may be used to vent hatches. Keep all sources of ignition away from loading area.

The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid.

- ▶ Containers, even those that have been emptied, may contain explosive vapours.
- ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.
- ▶ **DO NOT allow clothing wet with material to stay in contact with skin**
- Electrostatic discharge may be generated during pumping - this may result in fire.
- Ensure electrical continuity by bonding and grounding (earthing) all equipment.
- Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec).
- Avoid splash filling.
- Do NOT use compressed air for filling discharging or handling operations.
- Wait 2 minutes after tank filling (for tanks such as those on road tanker vehicles) before opening hatches or manholes.
- Wait 30 minutes after tank filling ( for large storage tanks) before opening hatches or manholes. Even with proper grounding and bonding, this material can still accumulate an electrostatic charge. If sufficient charge is allowed to accumulate, electrostatic discharge and ignition of flammable air-vapour mixtures can occur. Be aware of handling operations that may give rise to additional hazards that result from the accumulation of static charges. These include but are not limited to pumping (especially turbulent flow), mixing, filtering, splash filling, cleaning and filling of tanks and containers, sampling, switch loading, gauging, vacuum truck operations, and mechanical movements. These activities may lead to static discharge e.g. spark formation. Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (= 1 m/s until fill pipe submerged to twice its diameter, then = 7 m/s). Avoid splash filling.
- Do NOT use compressed air for filling, discharging, or handling operations
- ▶ Avoid all personal contact, including inhalation.
- ▶ Wear protective clothing when risk of exposure occurs.
- ▶ Use in a well-ventilated area.
- ▶ Prevent concentration in hollows and sumps.
- ▶ **DO NOT enter confined spaces until atmosphere has been checked.**
- ▶ Avoid smoking, naked lights, heat or ignition sources.
- ▶ When handling, **DO NOT eat, drink or smoke.**
- ▶ Vapour may ignite on pumping or pouring due to static electricity.
- ▶ **DO NOT use plastic buckets.**
- ▶ Earth and secure metal containers when dispensing or pouring product.
- ▶ Use spark-free tools when handling.
- ▶ Avoid contact with incompatible materials.
- ▶ Keep containers securely sealed.
- ▶ Avoid physical damage to containers.
- ▶ Always wash hands with soap and water after handling.
- ▶ Work clothes should be laundered separately.

	<ul style="list-style-type: none"> <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>
Other information	<ul style="list-style-type: none"> <li>• Store in original containers in approved flame-proof area.</li> <li>• No smoking, naked lights, heat or ignition sources.</li> <li>• <b>DO NOT store in pits, depression, basement or areas where vapours may be trapped.</b></li> <li>• Keep containers securely sealed.</li> <li>• Store away from incompatible materials in a cool, dry well ventilated area.</li> <li>• Protect containers against physical damage and check regularly for leaks.</li> <li>• Observe manufacturer's storage and handling recommendations contained within this MSDS.</li> <li>• Tank storage: Tanks must be specifically designed for use with this product. Bulk storage tanks should be diked (bundled). Locate tanks away from heat and other sources of ignition. Cleaning, inspection and maintenance of storage tanks is a specialist operation, which requires the implementation of strict procedures and precautions. Keep in a cool place. Electrostatic charges will be generated during pumping. Electrostatic discharge may cause fire. Ensure electrical continuity by bonding and grounding (earthing) all equipment to reduce the risk. The vapours in the head space of the storage vessel may lie in the flammable/explosive range and hence may be flammable.</li> <li>• For containers, or container linings use mild steel, stainless steel., Examples of suitable materials are: high density polyethylene (HDPE), polypropylene (PP), and Viton (FMK), which have been specifically tested for compatibility with this product., For container linings, use amine-adduct cured epoxy paint., For seals and gaskets use: graphite, PTFE, Viton A, Viton B.</li> <li>• Unsuitable material: Some synthetic materials may be unsuitable for containers or container linings depending on the material specification and intended use. Examples of materials to avoid are: natural rubber (NR), nitrile rubber (NBR), ethylene propylene rubber (EPDM), polymethyl methacrylate (PMMA), polystyrene, polyvinyl chloride (PVC), polyisobutylene., However, some may be suitable for glove materials</li> </ul> <p>o not cut, drill, grind, weld or perform similar operations on or near containers. Containers, even those that have been emptied, can contain explosive vapours</p>

### Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> <li>▶ Packing as supplied by manufacturer.</li> <li>▶ Plastic containers may only be used if approved for flammable liquid.</li> <li>▶ Check that containers are clearly labelled and free from leaks.</li> <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> <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> </ul>
Storage incompatibility	<p>Low molecular weight alkanes:</p> <ul style="list-style-type: none"> <li>▶ May react violently with strong oxidisers, chlorine, chlorine dioxide, dioxygenyl tetrafluoroborate.</li> <li>▶ May react with oxidising materials, nickel carbonyl in the presence of oxygen, heat.</li> <li>▶ Are incompatible with nitronium tetrafluoroborate(1-), halogens and interhalogens</li> <li>▶ may generate electrostatic charges, due to low conductivity, on flow or agitation.</li> <li>▶ Avoid flame and ignition sources</li> </ul> <p>Redox reactions of alkanes, in particular with oxygen and the halogens, are possible as the carbon atoms are in a strongly reduced condition. Reaction with oxygen (if present in sufficient quantity to satisfy the reaction stoichiometry) leads to combustion without any smoke, producing carbon dioxide and water. Free radical halogenation reactions occur with halogens, leading to the production of haloalkanes. In addition, alkanes have been shown to interact with, and bind to, certain transition metal complexes</p> <p>Interaction between chlorine and ethane over activated carbon at 350 deg C has caused explosions, but added carbon dioxide reduces the risk. The violent interaction of liquid chlorine injected into ethane at 80 deg C/10 bar becomes very violent if ethylene is also present A mixture prepared at -196 deg C with either methane or ethane exploded when the temp was raised to -78 deg C. Addition of nickel carbonyl to an n-butane-oxygen mixture causes an explosion at 20-40 deg C.</p> <p>Alkanes will react with steam in the presence of a nickel catalyst to give hydrogen.</p> <p>Xylenes:</p> <ul style="list-style-type: none"> <li>▶ may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride</li> <li>▶ attack some plastics, rubber and coatings</li> <li>▶ may generate electrostatic charges on flow or agitation due to low conductivity.</li> </ul> <p>For alkyl aromatics:</p>

The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.

- Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen
- Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.
- Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.
- Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.
- Alkali metals accelerate the oxidation while CO<sub>2</sub> as co-oxidant enhances the selectivity.
- Microwave conditions give improved yields of the oxidation products.
- Photo-oxidation products may occur following reaction with hydroxyl radicals and NO<sub>x</sub> - these may be components of photochemical smogs.

Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007

- Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents.
- Aromatics can react exothermically with bases and with diazo compounds.

Hydrogen sulfide (H<sub>2</sub>S):

- is a highly flammable and reactive gas
- reacts violently with strong oxidisers, metal oxides, metal dusts and powders, bromine pentafluoride, chlorine trifluoride, chromium trioxide, chromyl chloride, dichlorine oxide, nitrogen trichloride, nitryl hypofluorite, oxygen difluoride, perchloryl fluoride, phospham, phosphorus persulfide, silver fulminate, soda-lime, sodium peroxide
- is incompatible with acetaldehyde, chlorine monoxide, chromic acid, chromic anhydride, copper, nitric acid, phenyldiazonium chloride, sodium
- forms explosive material with benzenediazonium salts
- attacks many metals

Flow or agitation of hydrogen sulfide may generate electrostatic charges due to low conductivity

- **CARE:** Water in contact with heated material may cause foaming or a steam explosion with possible severe burns from wide scattering of hot material. Resultant overflow of containers may result in fire.
- Oil leaks in a pressurized circuit may result in a fine flammable spray (the lower flammability limit for oil mist is reached for a concentration of about 45 g/m<sup>3</sup>)
- Autoignition temperatures may be significantly lower under particular conditions (slow oxidation on finely divided materials..
  - Sulfides are incompatible with acids, diazo and azo compounds, halocarbons, isocyanates, aldehydes, alkali metals, nitrides, hydrides, and other strong reducing agents.
  - Many reactions of sulfides with these materials generate heat and in many cases hydrogen gas.
  - Many sulfide compounds may liberate hydrogen sulfide upon reaction with an acid.

n-Octane/ iso-octane:

- reacts violently with strong oxidisers, dinitrogen tetraoxide
- is incompatible with sulfuric acid, nitric acid, caustics, aliphatic amines, isocyanates
- attacks some plastics, rubber and coatings
- may generate electrostatic charges on agitation or flow, due to low conductivity.

## 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	petroleum crude oil	Oil mist, refined mineral	5 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	distillates, petroleum, middle, hydrotreated	Oil mist, refined mineral	5 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	gas oil, petroleum, hydrotreated, vacuum	Oil mist, refined mineral	5 mg/m <sup>3</sup>	Not Available	Not Available	Not Available
Australia Exposure Standards	n-octane	Octane	300 ppm / 1400 mg/m <sup>3</sup>	1750 mg/m <sup>3</sup> / 375 ppm	Not Available	Not Available
Australia Exposure Standards	m-xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m <sup>3</sup>	655 mg/m <sup>3</sup> / 150 ppm	Not Available	Not Available
Australia Exposure Standards	p-xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m <sup>3</sup>	655 mg/m <sup>3</sup> / 150 ppm	Not Available	Not Available
Australia Exposure Standards	o-xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m <sup>3</sup>	655 mg/m <sup>3</sup> / 150 ppm	Not Available	Not Available

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	heptane	Heptane (n-Heptane)	400 ppm / 1640 mg/m3	2050 mg/m3 / 500 ppm	Not Available	Not Available
Australia Exposure Standards	n-nonane	Nonane	200 ppm / 1050 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	toluene	Toluene	50 ppm / 191 mg/m3	574 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	ethylbenzene	Ethyl benzene	100 ppm / 434 mg/m3	543 mg/m3 / 125 ppm	Not Available	Not Available
Australia Exposure Standards	benzene	Benzene	1 ppm / 3.2 mg/m3	Not Available	Not Available	Not Available

### Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
petroleum crude oil	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
distillates, petroleum, middle, hydrotreated	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha petroleum, light, hydrotreated.	1,000 mg/m3	11,000 mg/m3	66,000 mg/m3
gas oil, petroleum, hydrotreated, vacuum	140 mg/m3	1,500 mg/m3	8,900 mg/m3
n-octane	230 ppm	385 ppm	5000** ppm
m-xylene	130 ppm	920 ppm	2500* ppm
p-xylene	130 ppm	920 ppm	2500* ppm
o-xylene	130 ppm	920 ppm	2500* ppm
heptane	500 ppm	830 ppm	5000* ppm
n-nonane	600 ppm	830 ppm	5,000 ppm
toluene	Not Available	Not Available	Not Available
ethylbenzene	Not Available	Not Available	Not Available
benzene	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
petroleum crude oil	1,100 ppm	Not Available
distillates, petroleum, middle, hydrotreated	2,500 mg/m3	Not Available
naphtha petroleum, light, hydrotreated.	Not Available	Not Available
gas oil, petroleum, hydrotreated, vacuum	2,500 mg/m3	Not Available
n-octane	1,000 ppm	Not Available
m-xylene	900 ppm	Not Available
p-xylene	900 ppm	Not Available
o-xylene	900 ppm	Not Available
heptane	750 ppm	Not Available
n-nonane	Not Available	Not Available
toluene	500 ppm	Not Available
ethylbenzene	800 ppm	Not Available
benzene	500 ppm	Not Available

### Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
naphtha petroleum, light, hydrotreated.	E	≤ 0.1 ppm

#### Notes:

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

**MATERIAL DATA**

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

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

NOTE 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

NOTE E: Substances with specific effects on human health that are classified as carcinogenic, mutagenic and/ or toxic for reproduction in categories 1 or 2 are ascribed Note E if they are classified as very toxic (T+), toxic (T) or harmful (Xn). For these substances the risk phrases R20, R21, R22, R23, R24, R25, R26, R27, R28, R39, R68, R48 and R65 and all combinations of these risk phrases shall be preceded by the word "Also".

R45-23: May cause cancer. Also toxic by inhalation

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**

<p><b>Appropriate engineering controls</b></p>	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> <li>▶ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.</li> <li>▶ Work should be undertaken in an isolated system such as a "glove-box". Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.</li> <li>▶ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.</li> <li>▶ Open-vessel systems are prohibited.</li> <li>▶ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.</li> <li>▶ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.</li> <li>▶ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.</li> <li>▶ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).</li> <li>▶ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.</li> <li>▶ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.</li> </ul> <p><b>CARE:</b> Use of a quantity of this material in confined space or poorly ventilated area, where rapid build up of concentrated atmosphere may occur, could require increased ventilation and/or protective gear</p>
<p><b>Personal protection</b></p>	
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</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], [AS/NZS 1336 or national equivalent]</li> </ul>
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>
<p><b>Hands/feet protection</b></p>	<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>

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

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.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

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.

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.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

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.

- Neoprene rubber gloves

#### Body protection

See Other protection below

#### Other protection

- Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent]
- Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent]
- Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely.
- Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood.
- Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Overalls.
- PVC Apron.
- PVC protective suit may be required if exposure severe.
- Eyewash unit.
- Ensure there is ready access to a safety shower.
- Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.
- For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).
- 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 the foot an 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:

Syncrude Synthetic Crude Oil - SAN 0930

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NATURAL RUBBER	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/CHLOROBUTYL	C
VITON/NEOPRENE	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 AB-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	AB-AUS P2	-	AB-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AB-AUS / Class 1 P2	-
up to 100 x ES	-	AB-2 P2	AB-PAPR-2 P2 ^

^ - Full-face

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

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

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

<b>Appearance</b>	Translucent yellow slightly viscous highly flammable liquid with hydrocarbon like odour; does not mix with water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	0.85-0.87
<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>	245
<b>pH (as supplied)</b>	Not Applicable	<b>Decomposition temperature (°C)</b>	Not Available
<b>Melting point / freezing point (°C)</b>	-45 (freezing pt.)	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	-20.0-560	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	<-20 (TCC)	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	0.3 BuAC = 1	<b>Explosive properties</b>	Not Available

<b>Flammability</b>	HIGHLY 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>	20.68-41.36	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (Not Available%)</b>	Not Applicable
<b>Vapour density (Air = 1)</b>	2.7	<b>VOC g/L</b>	Not Available

## SECTION 10 Stability and reactivity

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

## SECTION 11 Toxicological information

### Information on toxicological effects

<b>Inhaled</b>	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>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. 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>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 sensitiser and may cause ventricular fibrillations. Concentrated nonane vapours may cause irritation of the nose and throat, headache, drowsiness, dizziness, confusion, nausea, tremors, incoordination and difficulty in breathing. Very high concentrations may cause unconsciousness and death. Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.</p> <p>Symptoms of hydrogen sulfide (H2S) exposure may include profuse salivation, nausea, vomiting, diarrhoea, giddiness, headache, vertigo, amnesia, palpitations, arrhythmia, weakness, muscle cramps, confusion, sudden collapse, unconsciousness and death due to respiratory paralysis (above 300 ppm). Inhalation of (H2S) at low concentrations causes headache, dizziness and upset stomach. Higher concentrations cause olfactory fatigue, irritation to the respiratory tract, excitement, confusion, and exposure for a prolonged period may cause bronchitis and pulmonary oedema.</p> <p>Although hydrogen sulfide is extremely odourous, the "rotten egg" odour is not a reliable indicator for warning of exposure since odour fatigue readily occurs. Odour sensation is lost immediately at concentrations exceeding 200 ppm. Case reports suggest</p>
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	<p>that toxic amounts can enter the body through a punctured ear drum, even while wearing some sorts of respiratory protection. Hydrogen sulfide is primarily a respiratory toxin which inhibits the cytochrome-oxidase system and is probably more potent than hydrogen cyanide. The lifetime of hydrogen sulfide in oxygenated blood is short and sulfmethaemoglobin is rapidly detoxified by red blood cells and the liver. Most fatalities due to hydrogen sulfide intoxication occur at the scene of exposure and immediate supportive care is imperative. Ensure such contingencies are addressed as part of the site emergency plan and that operators or other employees who may become accidentally exposed, are made aware of the existence of such a plan.</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. 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>The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.</p> <p>Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.</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 style="text-align: center;"><b>Ingestion</b></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>
<p style="text-align: center;"><b>Skin Contact</b></p>	<p>The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> <li>▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▸ produces significant, but moderate, 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.</li> </ul> <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>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</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>The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives .</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with</p>

	<p>harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.</p>
<p style="text-align: center;"><b>Eye</b></p>	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals. Repeated or prolonged eye contact may cause inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Instillation of isoparaffins into rabbit eyes produces only slight irritation. Exposure to H<sub>2</sub>S may produce pain, blurred vision, and irritation. These symptoms are temporary in all but severe cases. Eye irritation may produce conjunctivitis, photophobia, pain, and at higher concentrations blurred vision and corneal blistering.</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. The liquid may produce eye discomfort and is capable of causing temporary impairment of vision and/or transient eye inflammation, ulceration</p>
<p style="text-align: center;"><b>Chronic</b></p>	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</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 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. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility</p> <p>There is sufficient evidence to establish a causal relationship between human exposure to the material and subsequent developmental toxic effects in the off-spring.</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>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.</p> <p>Principal route of exposure is by skin contact; lesser exposures include inhalation of fumes from hot oils, oil mists or droplets. Prolonged contact with mineral oils carries with it the risk of skin conditions such as oil folliculitis, eczematous dermatitis, pigmentation of the face (melanosis) and warts on the sole of the foot (plantar warts). With highly refined mineral oils no appreciable systemic effects appear to result through skin absorption.</p> <p>Exposure to oil mists frequently elicits respiratory conditions, such as asthma; the provoking agent is probably an additive. High oil mist concentrations may produce lipid pneumonia although clinical evidence is equivocal. In animals exposed to concentrations of 100 mg/m<sup>3</sup> oil mist, for periods of 12 to 26 months, the activity of lung and serum alkaline phosphatase enzyme was raised; 5 mg/m<sup>3</sup> oil mist did not produce this response. These enzyme changes are sensitive early indicators of lung damage. Workers exposed to vapours of mineral oil and kerosene for 5 to 35 years showed an increased prevalence of slight basal lung fibrosis.</p> <p>Many studies have linked cancers of the skin and scrotum with mineral oil exposure. Contaminants in the form of additives and the polycyclic aromatic hydrocarbons (PAHs - as in the crude base stock) are probably responsible. PAH levels are higher in aromatic process oils/used/reclaimed motor oils. Subchronic 90-day feeding studies conducted on male and female rats on highly refined white mineral oils and waxes found that higher molecular-weight hydrocarbons (microcrystalline waxes and the higher viscosity oils) were without biological effects. Paraffin waxes and low- to mid viscosity oils produced biological effects that were inversely proportional to molecular weight, viscosity and melting point: oil-type and processing did not appear to be determinants. Biological effects were more pronounced in females than in males. Effects occurred mainly in the liver and mesenteric lymph nodes and included increased organ weights, microscopic inflammatory changes, and evidence for the presence of saturated mineral hydrocarbons in affected tissues. Inflammation of the cardiac mitral valve was also observed at high doses in rats treated with paraffin waxes.</p> <p>Smith J.H., et al: Toxicologic Pathology: 24, 2, 214-230, 1996</p> <p>The so-called polycyclic aromatic hydrocarbons (PAHs) comprise a large family; some members occur in coal tar, tobacco smoke, petroleum and air pollution.</p> <p>PAHs are potent atmospheric pollutants that consist of fused aromatic rings and do not contain heteroatoms or carry substituents. As a pollutant, they are of concern because some compounds have been identified as carcinogenic, mutagenic, and teratogenic. Some substituted derivatives have been identified, in animal studies, as amongst the most highly active carcinogens. Rodent species are sensitive to some PAHs with skin application producing cancerous growths. Injection produces soft tissue tumours (sarcomas) in rats and mice.</p> <p>The toxicity of PAHs is structure-dependent. Isomers (PAHs with the same formula and number of rings) can vary from being nontoxic to extremely toxic. Natural crude oil and coal deposits contain significant amounts of PAHs, arising from chemical conversion of natural product molecules, such as steroids, to aromatic hydrocarbons. They are also found in processed fossil fuels, tar and various edible oils. In a study evaluating the genotoxic and carcinogenic risks associated with the consumption of repeatedly heated coconut oil (RCO), one of the commonly consumed cooking and frying medium, it was concluded that dietary consumption of RCO can cause a genotoxic and preneoplastic change in the liver.</p>

High prenatal exposure to PAH is associated with lower IQ and childhood asthma. Exposure to PAH pollution during pregnancy is related to adverse birth outcomes including low birth weight, premature delivery, and heart malformations. Cord blood of exposed babies shows DNA damage that has been linked to cancer. Follow-up studies show a higher level of developmental delays at age three, lower scores on IQ tests and increased behavioural problems at ages six and eight; one study found that exposure to higher levels of PAH was associated with a 24% higher score of anxiety/depression for children ages 6 to 7 than those with low exposure levels. Infants found to have elevated PAH levels in their umbilical cord blood were 46% more likely to eventually score highly on the anxiety/depression scale than those with low PAH levels in cord blood

Administration of PAHs to Rhesus monkey on the other hand has not yet proved successful in yielding tumours and there is inadequate data to support the proposition that individual PAHs produce cancer in humans. There are however a number of epidemiology and mortality studies that show increased incidence of cancer in humans exposed to mixtures of PAHs. Evidence exists of lung and genito-urinary cancer mortality amongst coke-oven workers and skin tumours in workers exposed to creosote. Exposures to other chemical mixtures containing PAHs such as cigarette smoke, coal tar, coal tar pitch and bitumens, have been associated with increased incidences of lung cancer in humans.

Anthracene, the basic unit on which most PAHs are built, is not carcinogenic whereas benz[a]anthracene appears to have weak carcinogenicity. Additions of other benzene rings to select positions on the benz[a]anthracene skeleton results in agents with powerful carcinogenicity (e.g. dibenz[a,h]anthracene and benz[a]pyrene). Further substitution of methyl groups in positions on the rings enhances carcinogenicity (7,12 dimethylbenz[a]anthracene is one of the most powerful PAH carcinogens known).

Biotransformation to produce soluble metabolites suitable for excretion appears to transform some PAHs to reactive electrophiles (as epoxides) which bind to DNA. Initiation of carcinogenesis is thought to rely upon such interactions.

One study examined the correlation between the weight percentage of various chemical classes of compounds in thirteen refinery streams and the magnitude of various effects produced in rats treated dermally with these substances in repeat-dose and developmental toxicity studies. In general, toxicity is correlated with concentrations of PAH composed of 3, 4, 5, 6, and/or 7 rings (decreased thymus weight, increased liver weight, aberrant haematology and serum chemistry, increased incidence of resorptions, decreased foetal body weight), PAH containing nonbasic nitrogen heteroatoms (increased mortality, decreased body weight, decreased thymus weight, increased liver weight, decreased haemoglobin content, haematocrit level, decreased foetal body weight), and/or PAH containing sulfur heteroatoms (decreased red blood cell and platelet counts, increased sorbitol dehydrogenase). A relationship between 2- ring PAH and skin irritation was demonstrated. Severity of effect was ranked against concentration of component class and statistical significance determined by the rank order correlation of Spearman. For the 13 streams tested, the presence and severity of systemic and developmental toxicity were dependent upon the levels of PAH and nonbasic nitrogen PAH. It is reasonable to assume that refinery streams rich in 3- to 7-ring PAH, S-PAH, and nonbasic N-PAH (e.g., carbazole derivatives) would be toxic, not only to the adult animal, but to the foetus as well.

Certain PAHs have structures resembling steroidal hormones and exhibit weak estrogenic or antiestrogenic activity. PAHs and their metabolites can act in the same manner as hormonal estrogens by binding to the estrogen receptor (ER) or modify estrogen availability by influencing estrogen metabolism by the CYP450 system. Estrogen induced carcinogenesis could therefore also be related to PAH induced carcinogenesis. After bioactivation by rat liver microsomes with induced P4501A1 and P4501A2 activity, metabolites from benz[a]anthracene and chrysene were found to be estrogenic in binding experiments

NOTE: Some jurisdictions require that health surveillance be conducted on workers occupationally exposed to polycyclic aromatic hydrocarbons. Such surveillance should emphasise

- demography, occupational and medical history
- health advice, including recognition of photosensitivity and skin changes
- physical examination if indicated
- records of personal exposure including photosensitivity
- completion of a standardised respiratory questionnaire
- standardised respiratory function tests such as FEV1, FVC and FEV1/FVC
- chest X-ray, full size PA view
- records of personal exposure

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 low level exposures to hydrogen sulfide may produce headache, fatigue, dizziness, irritability and loss of libido. These symptoms may also result from damage produced by isolated or repeated unmeasured peak high level exposures in healthy persons or those suffering from pre-existing neurological diseases. A study on long term effects showed that H2S apparently can cause continuing, sometimes unrecognised olfactory deficits. [Hirsch, A.R. - *Occ. Env. Med.*, 1999, Vol 5, Iss 4, pp 284-287] Repeated application of mildly hydrotreated oils (principally paraffinic), to mouse skin, induced skin tumours; no tumours were induced with severely hydrotreated oils.

Chronic exposure to benzene may cause headache, fatigue, loss of appetite and lassitude with incipient blood effects including anaemia and blood changes. Benzene is a myelotoxicant known to suppress bone- marrow cell proliferation and to induce haematologic disorders in humans and animals. Signs of benzene-induced aplastic anaemia include suppression of leukocytes (leukopenia), red cells (anaemia), platelets (thrombocytopenia) or all three cell types (pancytopenia). Classic symptoms include weakness, purpura, and haemorrhage. The most significant toxic effect is insidious and often reversible injury to the blood forming tissue. Leukaemia may develop. Occupational exposures have shown a relationship between exposure to benzene and production of myelogenous leukaemia. There may also be a relationship between benzene exposure and the production of lymphoma and multiple myeloma. In chronic exposure, workers exhibit signs of central nervous system lesions and impairment of hearing.

Benzene haemotoxicity and leukaemogenicity involve metabolism, growth factor regulation, oxidative stress, DNA damage, cell regulation, and apoptosis. (Yoon et al *Environmental Health Perspectives*, 111, pp 1411-1420, 2003)

Syncrude Synthetic Crude Oil - SAN 0930	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
petroleum crude oil	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[2]</sup> Oral (Rat) LD50; >4300 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg mild Skin (rabbit): 500 mg/24H Mild
distillates, petroleum, middle, hydrotreated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[2]</sup> Inhalation(Rat) LC50; 1.72 mg/14h <sup>[1]</sup> Oral (Rat) LD50; >5000 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: adverse effect observed (irritating) <sup>[1]</sup>
naphtha petroleum, light, hydrotreated.	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup> Inhalation(Rat) LC50; >4.42 mg/L4h <sup>[1]</sup> Oral (Rat) LD50; >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: adverse effect observed (irritating) <sup>[1]</sup>
gas oil, petroleum, hydrotreated, vacuum	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup> Inhalation(Rat) LC50; >0.32 mg/L4h <sup>[1]</sup> Oral (Rat) LD50; 4320 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
n-octane	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup> Inhalation(Rat) LC50; >24.88 mg/14h <sup>[1]</sup> Oral (Rat) LD50; >5000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
m-xylene	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 14100 mg/kg <sup>[2]</sup> Inhalation(Rat) LC50; 5922 ppm4h <sup>[1]</sup> Oral (Rat) LD50; 5000 mg/kg <sup>[2]</sup>	Eye (rabbit): 5 mg/24h - SEVERE Eye: adverse effect observed (irritating) <sup>[1]</sup> Skin (rabbit): 20 mg/24h - mod Skin (rabbit):0.01 mg/24h(open)

		Skin: adverse effect observed (irritating) <sup>[1]</sup>
<b>p-xylene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 12126 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Inhalation(Rat) LC50; 4550 ppm4h <sup>[2]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50; 5000 mg/kg <sup>[2]</sup>	
<b>o-xylene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 12126 mg/kg <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Inhalation(Rat) LC50; 5922 ppm4h <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50; 3523 mg/kg <sup>[1]</sup>	
<b>heptane</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation(Rat) LC50; >29.29 mg/l4h <sup>[1]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50; >5000 mg/kg <sup>[1]</sup>	
<b>n-nonane</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: >2000 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation(Rat) LC50; 3200 ppm4h <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50; >5000 mg/kg <sup>[1]</sup>	
<b>toluene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 12124 mg/kg <sup>[2]</sup>	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation(Rat) LC50; >13350 ppm4h <sup>[2]</sup>	Eye (rabbit):0.87 mg - mild
	Oral (Rat) LD50; 636 mg/kg <sup>[2]</sup>	Eye (rabbit):100 mg/30sec - mild
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
		Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>	
<b>ethylbenzene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 17800 mg/kg <sup>[2]</sup>	Eye (rabbit): 500 mg - SEVERE
	Inhalation(Rat) LC50; 17.2 mg/l4h <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50; 3500 mg/kg <sup>[2]</sup>	Skin (rabbit): 15 mg/24h mild
	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>	
<b>benzene</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	dermal (mouse) LD50: 48 mg/kg <sup>[2]</sup>	Eye (rabbit): 2 mg/24h - SEVERE
	Inhalation(Rat) LC50; 43.767 mg/L4h <sup>[1]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50; 930 mg/kg <sup>[2]</sup>	SKIN (rabbit):20 mg/24h - moderate
	Skin: adverse effect observed (irritating) <sup>[1]</sup>	
<b>Legend:</b>	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>PETROLEUM CRUDE OIL</b>	Tumorigenic - Carcinogenic by RTECS criteria. 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>DISTILLATES, PETROLEUM, MIDDLE, HYDROTREATED</b>	Highly and Severely Refined Distillate Base Oils <b>Acute toxicity:</b> Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to > 4 mg/l. When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating"

Testing in guinea pigs for sensitization has been negative

**Repeat dose toxicity:** Several studies have been conducted with these oils. The weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil s toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.

- The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,
- The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and
- The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.

**Reproductive and developmental toxicity:** A highly refined base oil was used as the vehicle control in a one-generation reproduction study. The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study s authors.

A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported. Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed foetuses were found among three litters The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.

**Genotoxicity:**

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

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

**Carcinogenicity:** Highly & severely refined base oils are not carcinogens, when given either orally or dermally. typical for isoparaffinic hydrocarbons: isoparaffinic hydrocarbon:

For Low Boiling Point Naphthas (LBPNS):

**Acute toxicity:**

LBPNS generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure

Most LBPNS are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices.

**Sensitisation:**

LBPNS do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies

**Repeat dose toxicity:**

The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPNS substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNS in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNS, were considered species- and sex-specific These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.

Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNS. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3

No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats

No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNS and very few non-cancer chronic toxicity studies were identified for other LBPNS. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3

A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.

**Genotoxicity:**

Although few genotoxicity studies were identified for the site-restricted LBPNS, the genotoxicity of several other LBPNS substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results.

For in vivo genotoxicity tests, LBPNS exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes

**NAPHTHA PETROLEUM,  
LIGHT, HYDROTREATED.**

and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells.

For in vitro genotoxicity studies, LBPNS were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNS exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay. Mixed results were observed for UDS and the mouse lymphoma assay.

While the majority of in vivo genotoxicity results for LBPNS substances are negative, the potential for genotoxicity of LBPNS as a group cannot be discounted based on the mixed in vitro genotoxicity results.

#### **Carcinogenicity:**

Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect of human exposure to LBPNS substances.

No inhalation studies assessing the carcinogenicity of the site-restricted LBPNS were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m<sup>3</sup> of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously. Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol. However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPNS group.

Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPNS substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPNS, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans).

Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNS. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light straight-run naphtha and naphtha. Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.

#### **Reproductive/ Developmental toxicity:**

No reproductive or developmental toxicity was observed for the majority of LBPNS substances evaluated. Most of these studies were carried out by inhalation exposure in rodents.

NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m<sup>3</sup> (CAS RN 8052-41-3) to 27 687 mg/m<sup>3</sup> (CAS RN 64741-63-5) for the LBPNS group evaluated, and from 7690 mg/m<sup>3</sup> to 27 059 mg/m<sup>3</sup> for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m<sup>3</sup>, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted. For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13.

For most LBPNS, no treatment-related developmental effects were observed by the different routes of exposure. However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m<sup>3</sup> delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

Low Boiling Point Naphthas [Site-Restricted]

<b>N-OCTANE</b>	Oral (rat) LD50: 5630 mg/kg* [CCINFO] Nil reported
<b>M-XYLENE</b>	The material may produce severe 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) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

	Effects on fertility, specific developmental abnormalities (craniofacial) recorded.
<b>O-XYLENE</b>	Paternal effects recorded.
<b>N-NONANE</b>	<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 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/m<sup>3</sup> (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 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.</p> <p>Repeated inhalation exposure had no apparent effect on the rates or routes of excretion of either of the test compounds or their metabolites.</p> <p>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</p> <p>C9-C14 aliphatic, &lt;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.</p> <p>C9-C14 aliphatic, &lt;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, &lt;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, &lt;2% aromatic hydrocarbons have a low order of acute toxicity by the oral, dermal, and inhalation routes of exposure.</p> <p>In a study examining the oral toxicity of commercial hexane. 6 male rats were given doses of up to 25 ml/kg of test substance by oral gavage. The animals were then observed for 14 days for mortality. No mortality was observed at any of the doses. The oral LD50 is therefore &gt; 25 ml/kg (16.75 g/kg; density of 0.67).</p> <p>C9-C14 aliphatic, &lt;2% aromatic hydrocarbons is minimally toxic via ingestion where the LD50 is &gt;5000 mg/kg, via dermal exposure where the LD50 is &gt;5000 mg/kg, and by inhalation where the LC50 &gt; 5000 mg/m<sup>3</sup>. These findings do not warrant classification of C9-C14 aliphatic, &lt;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, &lt;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.</p> <p>One study examined that acute inhalation toxicity of hexane to male rats. Groups of 10 male rats exposed to various large concentrations of hexane vapour for 4 hrs. Animals were then observed for clinical signs and mortality for at least the next 6 days. Several animals died during the exposure period. The LC50 was determined to be 73,680 ppm (259354 mg/m<sup>3</sup>). Due to the high concentration of the LC50, the test substance would not be classified as toxic by inhalation according to OECD GHS guidelines. Surviving animals experienced severe toxicological effects during the exposure.</p> <p>Skin irritation:</p> <p>For isoparaffinic, normal paraffinic, and mixed C9-C14 aliphatic, &lt;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).</p> <p>For cycloparaffinic C9-C14 aliphatic, &lt; 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).</p> <p>Eye irritation</p> <p>Ocular lesion scores (24, 48, and 72 average) are below the classification threshold requirements.</p> <p>Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparation: 0, cornea opacity; 0, iris lesion; &gt;2.5,</p>

redness of the conjunctivae; >2.0, oedema of the conjunctivae (chemosis). Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP): 0, cornea opacity; 0, iris lesion; >2.0, redness of the conjunctivae; >2.0, oedema of the conjunctivae (chemosis).

#### Respiratory irritation

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

#### Sensitisation:

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

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

#### Repeat dose toxicity,

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

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

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

In a repeated dose study where C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered via inhalation, no signs of toxicity were observed at 10400 mg/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.

## TOLUENE

#### For toluene:

##### Acute Toxicity

Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies.

**Humans** - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy.

Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days.

Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea. Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death

Toluene can also strip the skin of lipids causing dermatitis

**Animals** - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days

**Subchronic/Chronic Effects:**

Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm.

**Humans** - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin.

Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L

**Animals** - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day).

**Developmental/Reproductive Toxicity**

Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.

**Humans** - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy

**Animals** - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m<sup>3</sup> toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m<sup>3</sup> 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m<sup>3</sup> toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m<sup>3</sup>. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.

**Absorption** - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene.

**Distribution** - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues.

**Metabolism** - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites

**Excretion** - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.

**ETHYLBENZENE**

Liver changes, uterine tract, effects on fertility, foetotoxicity, specific developmental abnormalities (musculoskeletal system) recorded.

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.

Ethylbenzene is readily absorbed following inhalation, oral, and dermal exposures, distributed throughout the body, and excreted primarily through urine. There are two different metabolic pathways for ethylbenzene with the primary pathway being the alpha-oxidation of ethylbenzene to 1-phenylethanol, mostly as the R-enantiomer. The pattern of urinary metabolite excretion varies with different mammalian species. In humans, ethylbenzene is excreted in the urine as mandelic acid and phenylglyoxylic acids; whereas rats and rabbits excrete hippuric acid and phenacetic acid as the main metabolites. Ethylbenzene can induce liver enzymes and hence its own metabolism as well as the metabolism of other substances.

Ethylbenzene has a low order of acute toxicity by the oral, dermal or inhalation routes of exposure. Studies in rabbits indicate that ethylbenzene is irritating to the skin and eyes. There are numerous repeat dose studies available in a variety of species, these include: rats, mice, rabbits, guinea pig and rhesus monkeys.

Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (400 ppm and greater) of ethylbenzene

In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncytial alteration, hypertrophy

	<p>and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid gland.</p> <p>In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity</p> <p>Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination.</p> <p><b>NOTE:</b> Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p> <p><b>WARNING:</b> This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
<p style="text-align: center;"><b>BENZENE</b></p>	<p>Inhalation (man) TCLo: 150 ppm/1y - I</p> <p>Data demonstrate that during inhalation exposure, aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure.</p> <p>Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism.</p> <p>The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.</p> <p><b>WARNING:</b> This substance has been classified by the IARC as Group 1: <b>CARCINOGENIC TO HUMANS.</b></p>
<p><b>PETROLEUM CRUDE OIL &amp; DISTILLATES, PETROLEUM, MIDDLE, HYDROTREATED &amp; GAS OIL, PETROLEUM, HYDROTREATED, VACUUM</b></p>	<p>The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives; The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:</p> <ul style="list-style-type: none"> <li>· The adverse effects of these materials are associated with undesirable components, and</li> <li>· The levels of the undesirable components are inversely related to the degree of processing;</li> <li>· Distillate base oils receiving the same degree or extent of processing will have similar toxicities;</li> <li>· The potential toxicity of <i>residual base oils</i> is independent of the degree of processing the oil receives.</li> <li>· The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing.</li> </ul> <p>The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential.</p> <p>Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.</p> <p>Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil s mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing</p> <p>Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).</p> <p>Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class(Other Lubricant Base Oils).</p> <p>Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class(Other Lubricant Base Oils))</p> <p>Germ cell mutagenicity: The tests performed within the 'in vivo' studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class(Other Lubricant Base Oils)).</p> <p>Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction.</p> <p>STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects &gt; 280 mg/m3 and for systemic effects NOAEL &gt; 980 mg/m3.</p>

Sub-chronic toxicity  
90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies).  
Repeat dose toxicity:  
Oral  
NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.  
Inhalation  
The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m<sup>3</sup>. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m<sup>3</sup>.  
Dermal  
In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.  
Toxicity to reproduction:  
Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.  
Developmental toxicity, teratogenicity:  
Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE. The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic  
Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.

for Unrefined and Mildly Refined Distillate Base Oils  
**Acute toxicity:** LD50s of >5000 mg/kg (bw) and >2g/kg (bw) for the oral and dermal routes of exposure, respectively, have been observed in rats dosed with an unrefined light paraffinic distillate. The same material was also reported to be "moderately irritating" to the skin of rabbits. When tested for eye irritation in rabbits, the material produced Draize scores of 3.0 and 4.0 (unwashed/washed eyes) at 24 hours, with the scores returning to zero by 48 hours. The material was reported to be "not sensitising" when tested in guinea pigs  
**Repeat dose toxicity:** 200, 1000 and 2000 mg/kg (bw)/day of an unrefined base oil has been applied undiluted to the skin of male and female rabbit. The test material was applied to the rabbits skins 3 times/week for 4 weeks. To ensure maximum exposure, the applied material was covered with an occlusive dressing for 6 hours. In the high dose group, body weight gains were affected by treatment. These effects were largely due to effects on growth rate during the first week of the study. There were no significant differences between treated and control groups for any of the recorded haematological and clinical chemistry values. Gross and microscopic pathology findings relating to the treated skin were seen in all rabbits in the highest dose group. The findings consisted of "slight" to "moderate" proliferative changes in the treated skin.  
**Reproductive/ developmental toxicity** No reproductive or developmental toxicity studies have been reported for unrefined & mildly refined distillate base oils. However, a developmental toxicity screening study has been reported for heavy vacuum gas oil, a material with a process history similar to the unrefined distillate base oils. As an unrefined vacuum distillate material, heavy vacuum gas oil contains the broadest spectrum of chemical components and highest concentration of bioavailable and/or biologically active components. Because of their lack of or low level of processing, in comparison to other refined base oils, the unrefined lubricating base oils will also have higher concentrations of bioavailable and/or biologically active components. Heavy vacuum gas oil was applied daily to the skin of pregnant rats on days 0-19 of gestation. Dose levels administered included: 30, 125, 500 and 1000 mg/kg (bw)/day. All animals were euthanised on day 20. In the dams, the only dose-related finding at gross necropsy was pale colored lungs in four animals in the highest dose group and in one animal in the 500 mg/kg (bw)/day group. Mean thymus weights of the dams in the highest dose group were approximately half those of the control groups. Although absolute liver weights were unaffected by exposure to the gas oil, mean relative liver weights were increased (approximately 15%) in groups exposed to doses greater than 125 mg/kg (bw)/day. Maternal and foetal body weights were reduced at 500 and 1000 mg/kg (bw)/day. Significant increases in resorptions were also seen in these two dose groups. Soft tissue variations and malformations, and skeletal malformations were also increased at 500 and 1000 mg/kg  
**Genotoxicity:** Modified Ames assays have been carried out on a number of base oils that were either unrefined or poorly refined. The oils were found to be mutagenic, with a strong correlation between mutagenicity and 3-7 ring PAC content.  
**Carcinogenicity:** The general conclusions that can be drawn from the animal carcinogenicity studies are potential skin carcinogens. When applied repeatedly to the skin, carcinogenic base oils are associated only with skin tumours and not with an increase in systemic tumours

**PETROLEUM CRUDE OIL  
& GAS OIL, PETROLEUM,  
HYDROTREATED,  
VACUUM**

<b>PETROLEUM CRUDE OIL &amp; NAPHTHA PETROLEUM, LIGHT, HYDROTREATED. &amp; GAS OIL, PETROLEUM, HYDROTREATED, VACUUM</b>	No significant acute toxicological data identified in literature search.
<b>PETROLEUM CRUDE OIL &amp; DISTILLATES, PETROLEUM, MIDDLE, HYDROTREATED &amp; NAPHTHA PETROLEUM, LIGHT, HYDROTREATED. &amp; N-NONANE</b>	<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>
<b>PETROLEUM CRUDE OIL &amp; NAPHTHA PETROLEUM, LIGHT, 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> <p>The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
<b>PETROLEUM CRUDE OIL &amp; TOLUENE &amp; BENZENE</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 of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
<b>M-XYLENE &amp; ETHYLBENZENE</b>	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

<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

Syncrude Synthetic Crude Oil - SAN 0930	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
petroleum crude oil	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	24h	Crustacea	36mg/l	1
	LC50	96h	Fish	0.628mg/L	4

Continued...

## Syncrude Synthetic Crude Oil - SAN 0930

distillates, petroleum, middle, hydrotreated	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	NOEC(ECx)	72h	Algae or other aquatic plants	<0.03mg/l	1
naphtha petroleum, light, hydrotreated.	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	NOEC(ECx)	504h	Crustacea	0.17mg/l	2
	EC50	48h	Crustacea	0.64mg/l	2
	LC50	96h	Fish	4.26mg/l	2
	EC50	96h	Algae or other aquatic plants	64mg/l	2
gas oil, petroleum, hydrotreated, vacuum	<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
n-octane	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	0.3mg/l	2
	NOEC(ECx)	504h	Crustacea	0.17mg/l	2
m-xylene	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	4.7mg/l	2
	EC50	48h	Crustacea	1.593-3.822mg/L	4
	NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
p-xylene	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	4.7mg/l	2
	EC50	48h	Crustacea	3.2mg/l	2
	NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
o-xylene	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	LC50	96h	Fish	2.6mg/l	2
	EC50	48h	Crustacea	0.78-2.51mg/l	4
	NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
heptane	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	72h	Algae or other aquatic plants	4.7mg/l	2
	EC50	48h	Crustacea	0.64mg/l	2
	LC50	96h	Fish	3446.8mg/L	4
n-nonane	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	0.2mg/l	2
	NOEC(ECx)	504h	Crustacea	0.17mg/l	2
toluene	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	3.78mg/L	5
	NOEC(ECx)	168h	Crustacea	0.74mg/L	5
	LC50	96h	Fish	5-35mg/l	4
ethylbenzene	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	96h	Algae or other aquatic plants	>376.71mg/L	4
	EC50	72h	Algae or other aquatic plants	4.6mg/l	1
	EC50	48h	Crustacea	1.37-4.4mg/l	4
ethylbenzene	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	NOEC(ECx)	720h	Fish	0.381mg/L	4
	LC50	96h	Fish	3.381-4.075mg/L	4

## Syncrude Synthetic Crude Oil - SAN 0930

	EC50	96h	Algae or other aquatic plants	3.6mg/l	2
<b>benzene</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	7.578-13.983mg/L	4
	LC50	96h	Fish	2.54-7.217mg/L	4
	EC50	96h	Algae or other aquatic plants	>1360mg/l	1
	EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4
	ErC50	72h	Algae or other aquatic plants	>1360mg/l	1
	EC50	72h	Algae or other aquatic plants	29mg/l	1
<b>Legend:</b>	<i>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</i>				

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

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

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

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

Oils of any kind can cause:

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

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

for gas oils and distillate fuels:

The gas oils category includes both finished products (distillate fuels) and the refinery streams (gas oils) from which they are blended. The materials in this category, together with those in the Jet Fuel/Kerosene category, constitute a generic class of petroleum substances commonly known as middle distillates. The distillate fuels covered in this category are used in diesel engines and for both industrial and domestic heating. While within the refinery the gas oil streams exist primarily as intermediates in closed systems. Selected gas oil streams may ultimately be blended into distillate fuels, marine bunker fuels and occasionally into lubricants. At ambient temperatures, all the substances in this category are liquids. Gas oil streams and distillate fuels are complex petroleum mixtures, composed primarily of saturated (paraffinic and naphthenic) or aromatic hydrocarbons with carbon numbers ranging from C9 to C30.

Gas Oils are similar from both a process and physical-chemical perspective, being differentiated from each other primarily by their aromatic and saturated hydrocarbon content. The compositions of the gas oil streams range from those that are predominantly saturated hydrocarbons to those that are predominantly aromatic hydrocarbons. Consequently, the category can be considered a continuum, bounded by materials that are compositionally either high in saturated hydrocarbons or aromatic hydrocarbons. While the ratio of the saturated and aromatic hydrocarbons may vary between category members the saturated and aromatic hydrocarbons species that make up the category members are similar. Based on the available data, the physical-chemical properties of an individual category member depend on its compositional makeup, vis a vis saturated and aromatic hydrocarbons. Therefore, gas oil streams that are predominantly saturated hydrocarbons will have similar physical-chemical properties, while those that are composed predominantly of aromatic hydrocarbons will have somewhat different properties. As products that are blended from the gas oil streams, the compositions of the distillate fuels fall within the range of the compositions shown by the gas oil streams and reflect the characteristics of the gas oils streams from which they are blended.

**Boiling Point** Gas oils do not have a single numerical value for boiling point, but rather a boiling or distillation range that reflects the individual components in the hydrocarbon mixture. Distillation ranges for a variety of gas oils have been reported for a number of blended gas oil products and individual gas oil production streams. Typical distillation ranges for blended fuels are 160 to 390 C for an automotive gas oil (diesel fuel), 160 to 400 C for a heating oil, and 170 to 420 C for a distillate marine fuel. Typical low end and high end distillation temperatures for gas oil production streams were 172 and 344 C for a hydrodesulfurised middle distillate (65.6% -79.4% saturated hydrocarbons), 185 and 391 C for a straight-run middle distillate (78.8% saturated hydrocarbons), and 185 and 372 C for a light catalytic cracked distillate (60.8% -79.8% aromatic hydrocarbons). No substantial differences in boiling range were apparent for gas oils with high concentrations of either aromatic or saturated hydrocarbons

**Vapor Pressure** : For mixtures such as petroleum products, the vapor pressure of the mixture is the sum of the partial pressures of the individual components (Dalton's Law of Partial Pressures). Gas oils are expected to have low vapor pressure due to their boiling range (150 to 450 C) and molecular weights of the constituent hydrocarbons (C9 – 30 carbon atoms). Because the physical-chemical characteristics of distillate fuels reflect the gas oil streams from which they were produced, these vapor pressure measurements are expected to approximate the vapor pressures of individual gas oils. Vapour pressure estimates of low molecular weight hydrocarbons of varying isomeric structures fell within a range of 0.01-1.6 kPa, with higher molecular weight hydrocarbons showing very low vapour pressures (e.g., 10-8 to 10-10 kPa).

**Partition Coefficient** The percent distribution of the hydrocarbon groups (i.e., paraffins, olefins, naphthenes, and aromatics) and the carbon chain lengths of hydrocarbon constituents in gas oils largely determines the partitioning characteristics of the mixture. Generally, hydrocarbon chains with fewer carbon atoms tend to have lower partition coefficients than those with higher carbon numbers. Because gas oils are complex mixtures, it is not possible to determine their log Kow values. Rather, partition coefficients have been calculated for individual component hydrocarbons from known hydrocarbon composition. Those calculated Kow values ranged from 3.9 to >6.0 for a hydrodesulfurised middle distillate ((65.6% -79.4% saturated hydrocarbons), straight-run middle distillate (78.8% saturated hydrocarbons), and a light cat-cracked distillate (60.8% -79.8% aromatic hydrocarbons). There are no apparent differences in the range of Kow values determined for gas oils with high concentrations of either aromatic or saturated hydrocarbons. A similar range of partition coefficients would be expected for component hydrocarbons in distillate fuels.

**Environmental fate:**

**Photodegradation** : The direct aqueous photolysis of an organic molecule occurs when it absorbs sufficient light energy to result in a structural transformation.

Continued...

## Syncrude Synthetic Crude Oil - SAN 0930

Only light energy at wavelengths between 290 and 750 nm can result in photochemical transformations in the environment, although absorption is not always sufficient for a chemical to undergo photochemical degradation. Saturated and one-ring aromatic hydrocarbons do not show absorbance in the 290 to 800 nm range and would not be expected to be directly photodegraded. Polyaromatic hydrocarbons, on the other hand, have shown absorbance of the 290 to 800 nm range of light energy and could potentially undergo photolysis reactions. The degree and rate at which these compounds photodegrade depends upon whether conditions allow penetration of light with sufficient energy to effect a change.

Components in gas oils that do not directly photodegrade (e.g., paraffins, naphthenes, and one-ring aromatic compounds) may be subject to indirect photodegradation. Indirect photodegradation is the reaction with photosensitized oxygen in the atmosphere in the form of hydroxyl radicals (OH<sup>•</sup>). Atmospheric oxidation rates and half-lives were calculated for the low and high end of the range of molecular weight constituents of gas oils (e.g., C9 and C30 hydrocarbon structures). Half-life estimates for these compounds ranged from 0.1 (for various C9 to C30 olefinic structures and C30 2+ring aromatic compounds) to 1.5 days (for a C9 one-ring aromatic structure). Based on the calculated half-life values calculated no substantial differences in indirect photodegradation potential is expected between gas oils with high concentrations of either aromatic or saturated hydrocarbons. A similar range of water solubility values would be expected for component hydrocarbons in distillate fuels.

**Water Solubility** : When released to water, gas oils will float and spread at a rate that is viscosity-dependent. Component hydrocarbons in gas oils will partition to water according to their individual solubility values. For individual hydrocarbon constituents in gas oils, water solubility values vary by orders of magnitude.

Molecular weight and chemical structure have a great influence on the ultimate degree of solubility. Calculated water solubility ranged from essentially insoluble (approximately 10-8 mg/L) for the higher molecular weight fractions (e.g., C30) within gas oil to approximately 52 mg/L for a C9 alkylbenzene.

**Hydrolysis**: The materials in the gas oils category do not contain chemical moieties that undergo hydrolysis.

**Transport and Distribution in the Environment (Fugacity)** Models have been used to estimate the percent distribution in environmental media (i.e., air, water, soil, sediment, and fish) of various C9 to C30 compounds representing the different classes of hydrocarbons found in gas oils (e.g., paraffins, olefins, naphthenes, and aromatics). Hydrocarbons having nine carbon atoms showed a tendency to partition to air (up to 98%). As molecular weight increases, partitioning shifts to soil, which accounts for 98% of the distribution of the C30 components. This trend was similar for saturate and aromatic structures alike. Therefore, gas oils with high concentrations of either aromatic or saturated hydrocarbons are expected to partition in the environment in a similar manner

**Biodegradation** : Much of what is known is based on information gained from testing hydrocarbon mixtures of other petroleum products. Under standard biodegradability tests, hydrocarbon compounds representative of those found in gas oils typically do not pass ready biodegradability test conditions. Although those compounds are not recognized as being readily biodegradable, most hydrocarbon species present in gas oils are known to be ultimately degraded by aerobic microorganisms. Lower molecular weight compounds may be expected to be degraded relatively quickly in aerobic conditions, while higher molecular weight compounds, particularly polycyclic aromatics, will degrade slower. Much of this evidence is based on bioremediation studies of contaminated soils, which have shown that hydrocarbon components in gas oils are degraded in the presence of oxygen. Bioremediation of a diesel fuel spill has also been demonstrated under Arctic conditions

Under anaerobic conditions, such as anoxic sediments, rates of biodegradation of gas oils components are negligible and the gas oils may persist under those conditions for some time. Degradation then will be dependent on bioturbation or resuspension to provide microbes with access to oxygen.

**Ecotoxicity:**

Multiple ecotoxicological studies on heating and transportation fuels (e.g., no. 2 fuel oil and diesel fuel) have been conducted. In general, these commercial distillate fuels show moderate toxicity to aquatic life. LC50 values for fish ranged from 3.2 to 65 mg/L, while EC50 values for invertebrates ranged from 2.0 to 210 mg/L

For Aromatic Substances Series:

**Environmental Fate**: Large, molecularly complex polycyclic aromatic hydrocarbons, or PAHs, are persistent in the environment longer than smaller PAHs.

**Atmospheric Fate**: PAHs are "semi-volatile substances" which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. **Terrestrial Fate**: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive.

**Ecotoxicity** - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes > naphthalenes. Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.

**for lubricating oil base stocks:**

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

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

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

**Environmental Fate:**

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

penetration

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

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

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

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

**Ecotoxicity:**

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

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

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

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

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

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

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

For 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  $\log K_{ow} > -4.5$ . 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 tricornutum* 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.

for crude petroleum oil:

#### Environmental fate:

The processes determining the fate of oil in seawater are reasonably well understood.

Initially, the oil spreads out as a film on the sea surface as a result of wind and wave action. The more volatile, lower molecular weight hydrocarbons are lost by evaporation. Polar compounds and the mono-aromatic hydrocarbons have an appreciable water solubility and are taken into solution. A key ancillary process is that of emulsification, since crude oil has a natural tendency to form emulsions in sea water. Such emulsions are usually of the oil-in-water type, but may also be of the water-in-oil type.

The latter are often of the intractable 'chocolate mousse' type. Significant amounts of crude oil, particularly the higher molecular weight compounds, sink naturally, rolling along the ocean bottom picking up sand and shells and forming tarry balls which are resistant to degradation by any method.

Hydrocarbons may also reach the bottom sediments by sorption onto suspended particles which ultimately settle on the sea floor. Spill oil also undergoes

chemical changes, particularly oxidation by free radical mechanisms initiated by sunlight.

The initial products of such reactions are hydroperoxides, and these in turn form compounds such as alcohols, acids and aldehydes, many of which have an appreciable water solubility. Polymerization also occurs to yield intractable tarry materials.

The bulk of spilled crude oil is biodegraded by the micro-organisms present in sea water. Emulsification to form oil-in-water emulsions yields small particles of crude oil that are biodegraded by bacteria, yeasts, fungi and actinomycetes. Many factors influence the rate of biodegradation, in particular temperature, dissolved oxygen concentration and the availability of nitrogen and phosphorus nutrients. Adapted micro-organisms are often found in ocean areas where crude oil spills are common. It has calculated that where an adapted microbial population is available in well-aerated sea water at 20 to 30 °C, the rate of crude oil oxidation ranges from 0.02 to 0.2 g of oil oxidized/m<sup>2</sup>/day. Experimentally it has been determined that complete oxidation of 1.0 mg of hydrocarbon requires between 3 and 4 g of oxygen, i.e. it has a BOD of 3 to 4 mg oxygen/mg. Since the oxygen content of sea water is between 6 and 11 mg/liter, depending on salinity and temperature, this means that about 320 000 litres of sea water is required to oxidise one liter of crude oil. Crude oil contains hydrocarbons of well-defined generic types that are biodegraded at different rates. n-Alkanes are readily degraded in sea water, since many micro-organisms can utilize them. Branched-chain or iso-alkanes are less readily biodegraded but they do ultimately biodegrade. The degradation of cycloalkanes has not been extensively studied, but the ring structure is resistant to biodegradation. Aromatic hydrocarbons are also resistant to biodegradation, but a few micro-organisms are able to utilize them. High molecular weight compounds, the tars and asphaltenes, degrade very slowly.

#### Ecotoxicity:

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

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

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

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

When released in the environment, alkanes don't undergo rapid biodegradation, because they have no functional groups (like hydroxyl or carbonyl) that are needed by most organisms in order to metabolize the compound.

However, some bacteria can metabolise some alkanes (especially those linear and short), by oxidizing the terminal carbon atom. The product is an alcohol, that could be next oxidised to an aldehyde, and finally to a carboxylic acid. The resulting fatty acid could be metabolised through the fatty acid degradation pathway.

For n-heptane:

log Kow : 4.66

Koc : 2400-8100

Half-life (hr) air : 52.8

Half-life (hr) H<sub>2</sub>O surface water : 2.9-312

Henry's atm m<sup>3</sup> /mol: 2.06

BOD 5 if unstated: 1.92

COD : 0.06

BCF : 340-2000

log BCF : 2.53-3.31

#### Environmental fate:

Photolysis or hydrolysis of n-heptane are not expected to be important environmental fate processes. Biodegradation of n-heptane may occur in soil and water, however volatilisation and adsorption are expected to be more important fate processes. A high Koc (2400-8200) indicates n-heptane will be slightly mobile to immobile in soil. In aquatic systems n-heptane may partition from the water column to organic matter in sediments and suspended solids. The bioconcentration of n-heptane may be important in aquatic environments. The Henry's Law constant suggests rapid volatilisation from environmental waters and surface soils. The volatilisation half-lives from a model river and a model pond (the latter considers the effect of adsorption) have been estimated to be 2.9 hr and 13 days, respectively.

n-Heptane is expected to exist entirely in the vapour phase in ambient air. Reactions with photochemically produced hydroxyl radicals in the atmosphere have been shown to be important (estimated half-life of 2.4 days calculated from its rate constant of 7.15x10<sup>-12</sup> cu cm/molecule-sec at 25 deg C). Data also suggests that night-time reactions with nitrate radicals may contribute to the atmospheric transformation of n-heptane, especially in urban environments. n-Heptane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight

An estimated BCF of 2,000 using log Kow suggests the potential for bioconcentration in aquatic organisms is very high. Based on 100% degradation after 4 days in water inoculated with gasoline contaminated soil and 100% degradation after 25 days in water inoculated with activated sewage sludge, biodegradation is expected to be an important fate process for n-heptane in water.

#### Ecotoxicity:

Fish LC50 (48 h): goldfish (*Carrasius auratus*) 4 mg/l; golden orfe (*Idus melanotus*) 2940 mg/l; western mosquitofish (*Gambusia affinis*) 4924 mg/l

Daphnia LC50 (24 h): >10 mg/l

Daphnia EC50 (96 h): 82 mg/l (immobilisation)

Opposum shrimp (*Mysidopsis bahia*) LC50 (96 h): 0.1 mg/l

Snail EC50 (96 h): 472 mg/l

For n-hexane:

log Kow: 3.17-3.94

BOD 5 if unstated: 2.21

COD: 0.04

ThOD: 3.52

#### Environmental fate:

**Transport and Partitioning:** The physical properties of n-hexane that affect its transport and partitioning in the environment are: water solubility of 9.5 mg/L; log[Kow] (octanol/water partition coefficient), estimated as 3.29; Henry's law constant, 1.69 atm-m<sup>3</sup> mol; vapor pressure, 150 mm Hg at 25 °C; and log[Koc] in the range of 2.90 to 3.61. As with many alkanes, experimental methods for the estimation of the Koc parameter are lacking, so that estimates must be made based on theoretical considerations.

The dominant transport process from water is volatilization. Based on mathematical models the half-life for *n*-hexane in bodies of water with any degree of turbulent mixing (e.g., rivers) would be less than 3 hours. For standing bodies of water (e.g. small ponds), a half-life no longer than one week (6.8 days) is estimated. Based on the log octanol/water partition coefficient (i.e.  $\log[K_{oc}]$ ) and the estimated log sorption coefficient (i.e.  $\log[K_{oc}]$ ) *n*-hexane is not expected to become concentrated in biota. A calculated bioconcentration factor (BCF) of 453 for a fathead minnow further suggests a low potential for *n*-hexane to bioconcentrate or bioaccumulate in trophic food chains.

In soil, the dominant transport mechanism for *n*-hexane present near the surface probably is volatilisation (based on its Henry's law constant, water solubility, vapor pressure, and  $K_{oc}$ ). While its estimated  $K_{oc}$  values suggest a moderate ability to sorb to soil particles, *n*-hexane has a density (0.6603 g/mL at 20 C) well below that of water and a very low water solubility of 9.5 mg/L. *n*-Hexane would, therefore, be viewed as a light nonaqueous phase liquid (LNAPL), which would suggest a low potential for leaching into the lower soil depths since the *n*-hexane would tend to float on the top of the saturated zone of the water table. *n*-Hexane would generally stay near the soil surface and, if not appreciably sorbed into the soil matrix, would be expected eventually to volatilise to the atmosphere. Exceptions would involve locations with shallow groundwater tables where there were large spills of hexane products. In such cases, the *n*-hexane could spread out to contaminate a large volume of soil materials.

**Air:** *n*-Hexane does not absorb ultraviolet (UV) light at 290 nm and is thus not expected to undergo direct photolysis reactions. The dominant tropospheric removal mechanism for *n*-hexane is generally regarded to be decomposition by hydroxyl radicals. Calculations assuming typical hydroxyl radical concentrations suggest a half-life of approximately 2.9 days. While *n*-hexane can react with nitrogen oxides to produce ozone precursors under controlled laboratory conditions, the smog-producing potential of *n*-hexane is very low compared to that of other alkanes or chlorinated VOCs. Hydroxyl ion reactions in the upper troposphere, therefore, are probably the primary mechanisms for *n*-hexane degradation in the atmosphere. As with most alkanes, *n*-hexane is resistant to hydrolysis.

**Water:** Although few data are available dealing explicitly with the biodegradation of *n*-hexane in water, neither hydrolysis nor biodegradation in surface waters appears to be rapid compared with volatilization. In surface waters, as in the atmosphere, alkanes such as *n*-hexane would be resistant to hydrolysis. Biodegradation is probably the most significant degradation mechanism in groundwater. The ability of *Pseudomonas mendocina* bacteria to metabolise *n*-hexane in laboratory microcosms simulating groundwater conditions has been documented. Mixed bacterial cultures as well as pure cultures are documented as capable of metabolizing *n*-hexane under aerobic conditions. In general, linear alkanes (such as *n*-hexane) are viewed as the most readily biodegradable fractions in petroleum, particularly when oxygen is present in solution. Once introduced into groundwater, *n*-hexane may be fairly persistent since its degradation by chemical hydrolysis is slow and opportunities for biodegradation may be limited under anoxic conditions or where nutrients such as nitrogen or phosphorus are in limited supply.

**Sediment and Soil:** The most important biodegradation processes involve the conversion of the *n*-hexane to primary alcohols, aldehydes and, ultimately, into fatty acids. Similar processes are encountered with other light hydrocarbons such as heptane. In general, unless the *n*-hexane is buried at some depth within a soil or sediment, volatilisation is generally assumed to occur at a much more rapid rate than chemical or biochemical degradation processes. Once introduced into deeper sediments, *n*-hexane may be fairly persistent.

#### Ecotoxicity:

Fish LC50 (96 h): Oncorhynchus mykiss 4.14 mg/l; Pimephales promelus 2.5 mg/l (flow through); Lepomis macrochirus 4.12 mg/l

Daphnia EC50 (48 h): 3.87 mg/l

For vanadium compounds:

#### Environmental fate:

The global biogeochemical cycling of vanadium is characterized by releases to the atmosphere, water, and land by natural and anthropogenic sources, long-range transportation of particles in both air and water, wet and dry deposition, adsorption, and complexing. Vanadium generally enters the atmosphere as an aerosol. From natural sources, vanadium is probably in the form of mineral particles; it has been suggested that these may frequently be in the less-soluble trivalent form. From man-made sources almost all the vanadium released to the atmosphere is in the form of simple or complex vanadium oxides. The size distribution of vanadium-bearing particles in the atmosphere is substantially altered during long-range transportation.

Natural sources of vanadium, as well as man-made sources such as ore-processing dust, tend to release large particles that are more likely to settle near the source. Smaller particles, such as those emitted from oil-fueled power plants, have a longer residence time in the atmosphere and are more likely to be transported farther away from the site of release. Vanadium transported within the atmosphere is eventually transferred to soil and water on the earth's surface by wet and dry deposition and dissolution in sea water. Eventually, in the course of biogeochemical movement between soil and water, these particulates are adsorbed to hydroxides or associated with organic compounds and are deposited on the sea bed.

The transport and partitioning of vanadium in water and soil is influenced by pH, redox potential, and the presence of particulate. In fresh water, vanadium generally exists in solution as the vanadyl ion (V<sup>4+</sup>) under reducing conditions and the vanadate ion (V<sup>5+</sup>) under oxidizing conditions, or as an integral part of, or adsorbed onto, particulate matter. The chemical formulas of the vanadyl species most commonly reported in fresh water are VO<sup>2+</sup> and VO(OH)<sup>+</sup>, and the vanadate species are H<sub>2</sub>VO<sub>4</sub><sup>-</sup> and HVO<sub>4</sub>. The partitioning of vanadium between water and sediment is strongly influenced by the presence of particulate in the water. Both vanadate and vanadyl species are known to bind strongly to mineral or biogenic surfaces by adsorption or complexing. Thus, vanadium is transported in water in one of two ways: solution or suspension. It has been estimated that only 13% is transported in solution, while the remaining 87% is in suspension. Upon entering the ocean, vanadium in suspension or sorbed onto particulate is deposited upon the sea bed. The fate of the remaining dissolved vanadium is more complex. Only about 0.001% of vanadium entering the oceans is estimated to persist in soluble form. Sorption and biochemical processes are thought to contribute to the extraction of vanadium from sea water. Adsorption to organic matter as well as to manganese oxide and ferric hydroxide, demonstrated by the high particle-water partition coefficient of 5.7x10<sup>5</sup> L/kg for the adsorption of manganese oxide in sea water, results in the precipitation of the dissolved vanadium.

Biochemical processes are also of importance in the partitioning from sea water to sediment. Some marine organisms, in particular the ascidians (sea squirts), bioconcentrate vanadium very efficiently, attaining body concentrations approximately 10,000 times greater than the ambient sea water. Upon the death of the organism, the body burden adds to the accumulation of vanadium in silt. The extent to which either bioconcentration or adsorption dominates is uncertain. In general, marine plants and invertebrates contain higher levels of vanadium than terrestrial plants and animals. In the terrestrial environment bioconcentration is more commonly observed amongst the lower plant phyla than in the higher, seed-producing phyla. The vanadium levels in terrestrial plants are dependent upon the amount of water-soluble vanadium available in the soil, pH, and growing conditions. It has been found that the uptake of vanadium into the above-ground parts of many plants is low, although root concentrations have shown some correlation with levels in the soil. Certain legumes, such as *Astragalus preussi*, have been shown to be vanadium accumulators. Vanadium is believed to replace molybdenum as a specific catalyst in nitrogen fixation and the root nodules of these plants may contain vanadium levels three times greater than those of the surrounding soil. Of the few plants known to actively accumulate vanadium, *Amanita muscaria*, a poisonous mushroom, has been demonstrated to contain levels up to 112 ppm (dry weight). Vanadium appears to be present in all terrestrial animals, but, in vertebrates, tissue concentrations are often so low that detection is difficult. The highest levels of vanadium in terrestrial mammals are generally found in the liver and skeletal tissues. No data are available regarding biomagnification of vanadium within the food chain, but human studies suggest that it is unlikely; most of the 1%-2% vanadium that appears to be absorbed by humans following ingestion is rapidly excreted in the urine with no evidence of long-term accumulation.

The form of vanadium present in the soil is determined largely by the parent rock. Ferric hydroxides and solid bitumens (organic) constitute the main carriers of

vanadium in the sedimentation process. Iron acts as a carrier for trivalent vanadium due to the great affinity between trivalent vanadium and trivalent iron, and is responsible for its diffusion through molten rocks where it becomes trapped during crystallization. The mobility of vanadium in soils is affected by the pH of the soil. Relative to other metals, vanadium is fairly mobile in neutral or alkaline soils, but its mobility decreases in acidic soils. Similarly, under oxidizing, unsaturated conditions some mobility is observed, but under reducing, saturated conditions vanadium is immobile

#### Ecotoxicity:

The available reliable ecotoxicity results selected for the effect of vanadium on aquatic organisms are all based on pentavalent V substances (NaVO<sub>3</sub>, NH<sub>4</sub>VO<sub>3</sub>, Na<sub>3</sub>VO<sub>4</sub>, V<sub>2</sub>O<sub>5</sub> and ammonium polyvanadate).

Reliable short-term toxicity data for freshwater organisms are available for three trophic levels: aquatic invertebrates, fish and algae.

Fish LC<sub>50</sub> (96 h): *Leuciscus idus* 0.7 mg V/l (V<sub>2</sub>O<sub>5</sub> flake)

Algae EC<sub>50</sub> (72 h): *Scenedesmus subspicatus* 2.9 mg V/l (V<sub>2</sub>O<sub>5</sub>)

Mysid shrimp LC<sub>50</sub> (48 h): 13.3 mg V/l

Flagship larvae NOEC (30 d): 76 ug V/l (V<sub>2</sub>O<sub>5</sub>)

Reliable long-term toxicity data are also available for the effect of V<sub>2</sub>O<sub>5</sub> on the development of eggs from two marine organisms (mollusc *Crassostrea gigas* and echinoderm *Paracentrotus lividus*). The NOEC values varied between 25 and 50 ug V/L with the lowest value observed for a 48-h development test of *Crassostrea gigas* eggs.

For Xylenes:

log K<sub>oc</sub> : 2.05-3.08; K<sub>oc</sub> : 25.4-204; Half-life (hr) air : 0.24-42; Half-life (hr) H<sub>2</sub>O surface water : 24-672; Half-life (hr) H<sub>2</sub>O ground : 336-8640; Half-life (hr) soil : 52-672; Henry's Pa m<sup>3</sup>/mol : 637-879; Henry's atm m<sup>3</sup>/mol - 7.68E-03; BOD 5 if unstated - 1.4,1%; COD - 2.56,13% ThOD - 3.125 : BCF : 23; log BCF : 1.17-2.41.

Environmental Fate: Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. Soil - Xylenes are expected to have moderate mobility in soil evaporating rapidly from soil surfaces. The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. Xylene can remain below the soil surface for several days and may travel through the soil profile and enter groundwater. Soil and water microbes may transform it into other, less harmful compounds, although this happens slowly. It is not clear how long xylene remains trapped deep underground in soil or groundwater, but it may be months or years.

Atmospheric Fate: Xylene evaporates quickly into the air from surface soil and water and can remain in the air for several days until it is broken down by sunlight into other less harmful chemicals. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylene may contribute to photochemical smog formation. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzyl nitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

Aquatic Fate: p-xylene may adsorb to suspended solids and sediment in water and is expected to volatilise from water surfaces. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. Measurements taken from goldfish, eels and clams indicate that bioconcentration in aquatic organisms is low. Photo-oxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. p-Xylene is biodegradable and has been observed to degrade in pond water however; it is unclear if it degrades in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high.

Ecotoxicity: Xylenes are slightly toxic to fathead minnow, rainbow trout and bluegill and not acutely toxic to water fleas. For *Photobacterium phosphoreum* EC<sub>50</sub> (24 h): 0.0084 mg/L. and *Gammarus lacustris* LC<sub>50</sub> (48 h): 0.6 mg/L.

Sulfide ion is very toxic to aquatic life, threshold concentration for fresh or saltwater fish is 0.5ppm. The product therefore is very toxic to aquatic life. The major decomposition product, hydrogen sulfide, is damaging to vegetation at 5ppm for 24 hours

For n-octane:

K<sub>oc</sub> : 5500-15600

Half-life (hr) air : 43.44

Half-life (hr) H<sub>2</sub>O surface water : 3.1-715.2

Henry's atm m<sup>3</sup>/mol: 3.21

ThOD : 3.5

BCF : 780-5100

Log BCF : 2.89-3.71

#### Environmental Fate

**Terrestrial fate:** An estimated K<sub>oc</sub> value of 16,000, determined from a log K<sub>ow</sub> of 5.18 indicates that n-octane is expected to be immobile in soil. Volatilisation of n-octane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 3.2 atm-cu m/mole(SRC), derived from its vapor pressure, 14.1 mm Hg, and water solubility, 0.66 mg/l. However, adsorption to soil is expected to attenuate volatilization. The potential for volatilisation of n-octane from dry soil surfaces may exist based upon its vapor pressure. n-Octane is expected to biodegrade in soil under aerobic conditions. One milligram of n-octane and 1 ml of a 1:10 suspension of Hudson-Collamer silt loam soil in mineral salts media was incubated in the dark at 25 deg C. The average theoretical biological oxygen demand (ThBOD) of 2 trials for n-octane was 13, 58, 70 and 69% after 2, 5, 10 and 20 days, respectively.

**Aquatic fate:** The estimated K<sub>oc</sub> value indicates that n-octane is expected to adsorb to suspended solids and sediment(SRC). Volatilisation from water surfaces is expected based upon a Henry's Law constant of Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 1.1 hours and 4.2 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. The estimated volatilization half-life from a model pond is 11 months if adsorption is considered. An estimated BCF of 16,000 based on the log K<sub>ow</sub> suggests the potential for bioconcentration in aquatic organisms is very high. When evaporation rates are low, biodegradation of n-octane under aerobic conditions may be important in water. For example, a 49% loss of n-octane occurred within 5 days and completely disappeared within 15 days when 1 ml of crude oil was added to a 100 ml simulated seawater solution inoculated with sediment samples from Fukae of Kobe harbour, Japan and incubated at 20 deg C. Although complete recovery was reported for the control samples, no account was made of volatilization losses. However, in a similar study using a jet fuel mixture and freshwater at 25 deg C, a 99% loss of n-octane in sample controls was attributed to evaporation.

**Atmospheric fate:** According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, n-octane, which has a vapor pressure of 14.1 mm Hg at 25 deg C, is expected to exist solely as a vapor in the ambient atmosphere. Vapor-phase n-octane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 44 hrs, calculated from its rate constant of 8.68x10<sup>-12</sup> cu cm/molecule-sec at 25 deg C. Experimental data also showed that 33.2% of the n-octane fraction in a dark chamber reacted with nitrate radicals to form the corresponding alkyl nitrate, suggesting nighttime reactions with nitrate radicals may contribute to the atmospheric transformation of n-octane, especially in urban environments.

**Ecotoxicity:**

Daphnia EC50 (48 h): 0.38 mg/l

Fish LC50: 230 mg/l

Photobacterium phosphoreum EC50 (30 m): 890 mg/l

For petroleum derivatives:

Chemical analysis for all individual compounds in a petroleum bulk product released to the environment is generally unrealistic due to the complexity of these mixtures and the laboratory expense. Determining the chemical composition of a petroleum release is further complicated by hydrodynamic, abiotic, and biotic processes that act on the release to change the chemical character.

The longer the release is exposed to the environment, the greater the change in chemical character and the harder it is to obtain accurate analytical results reflecting the identity of the release. After extensive weathering, detailed knowledge of the original bulk product is often less valuable than current site-specific information on a more focused set of hydrocarbon components. Health assessment efforts are frequently frustrated by three primary problems: (1) the inability to identify and quantify the individual compounds released to the environment as a consequence of a petroleum spill; (2) the lack of information characterizing the fate of the individual compounds in petroleum mixtures; and (3) the lack of specific health guidance values for the majority of chemicals present in petroleum products. To define the public health implications associated with exposure to petroleum hydrocarbons, it is necessary to have a basic understanding of petroleum properties, compositions, and the physical, chemical, biological, and toxicological properties of the compounds most often identified as the key chemicals of concern.

**Environmental fate:**

Petroleum products released to the environment migrate through soil via two general pathways: (1) as bulk oil flow infiltrating the soil under the forces of gravity and capillary action, and (2) as individual compounds separating from the bulk petroleum mixture and dissolving in air or water. When bulk oil flow occurs, it results in little or no separation of the individual compounds from the product mixture and the infiltration rate is usually fast relative to the dissolution rate. Many compounds that are insoluble and immobile in water are soluble in bulk oil and will migrate along with the bulk oil flow. Factors affecting the rate of bulk oil infiltration include soil moisture content, vegetation, terrain, climate, rate of release (e.g., catastrophic versus slow leakage), soil particle size (e.g., sand versus clay), and oil viscosity (e.g., gasoline versus motor oil).

As bulk oil migrates through the soil column, a small amount of the product mass is retained by soil particles. The bulk product retained by the soil particles is known as "residual saturation".

Depending upon the persistence of the bulk oil, residual saturation can potentially reside in the soil for years. Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater. Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater. When the amount of product released to the environment is small relative to the volume of available soil, all of the product is converted to residual saturation and downward migration of the bulk product usually ceases prior to affecting groundwater resources. Adverse impacts to groundwater may still occur if rain water infiltrates through soil containing residual saturation and initiates the downward migration of individual compounds. When the amount of product released is large relative to the volume of available soil, the downward migration of bulk product ceases as water-saturated pore spaces are encountered. If the density of the bulk product is less than that of water, the product tends to "float" along the interface between the water saturated and unsaturated zones and spread horizontally in a pancake-like layer, usually in the direction of groundwater flow. Almost all motor and heating oils are less dense than water. If the density of the bulk product is greater than that of water, the product will continue to migrate downward through the water table aquifer under the continued influence of gravity. Downward migration ceases when the product is converted to residual saturation or when an impermeable surface is encountered.

As the bulk product migrates through the soil column, individual compounds may separate from the mixture and migrate independently. Chemical transport properties such as volatility, solubility, and sorption potential are often used to evaluate and predict which compounds will likely separate from the mixture. Since petroleum products are complex mixtures of hundreds of compounds, the compounds characterized by relatively high vapor pressures tend to volatilise and enter the vapor phase. The exact composition of these vapors depends on the composition of the original product. Using gasoline as an example, compounds such as butane, propane, benzene, toluene, ethylbenzene and xylene are preferentially volatilised. Because volatility represents transfer of the compound from the product or liquid phase to the air phase, it is expected that the concentration of that compound in the product or liquid phase will decrease as the concentration in the air phase increases.

In general, compounds having a vapor pressure in excess of 10-2 mm Hg are more likely to be present in the air phase than in the liquid phase. Compounds characterized by vapor pressures less than 10-7 mm Hg are more likely to be associated with the liquid phase. Compounds possessing vapor pressures that are less than 10-2 mm Hg, but greater than 10-7 mm Hg, will have a tendency to exist in both the air and the liquid phases.

Lighter petroleum products such as gasoline contain constituents with higher water solubility and volatility and lower sorption potential than heavier petroleum products such as fuel oil.

Data compiled from gasoline spills and laboratory studies indicate that these light-fraction hydrocarbons tend to migrate readily through soil, potentially threatening or affecting groundwater supplies. In contrast, petroleum products with heavier molecular weight constituents, such as fuel oil, are generally more persistent in soils, due to their relatively low water solubility and volatility and high sorption capacity. Solubility generally decreases with increasing molecular weight of the hydrocarbon compounds. For compounds having similar molecular weights, the aromatic hydrocarbons are more water soluble and mobile in water than the aliphatic hydrocarbons and branched aliphatics are less water-soluble than straight-chained aliphatics. Aromatic compounds in petroleum fuels may comprise as much as 50% by weight; aromatic compounds in the C6-C13, range made up approximately 95% of the compounds dissolved in water.

Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds. Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media.

The final products of microbial degradation are carbon dioxide, water, and microbial biomass. The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds. The n-alkanes, n-alkyl aromatics, and the aromatics in the C10-C22 range are the most readily biodegradable; n-alkanes, n-alkyl aromatics, and aromatics in the C5-C9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilisation and thus are unavailable in most environments; n-alkanes in the C1-C4 ranges are biodegradable only by a narrow range of specialized hydrocarbon degraders; and n-alkanes, n-alkyl aromatics, and aromatics above C22 are generally not available to degrading microorganisms. Hydrocarbons with condensed ring structures, such as PAHs with four or more rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded. A large proportion of the water-soluble fraction of the petroleum product may be degraded as the compounds go into solution. As a result, the remaining product may become enriched in the alicyclics, the highly branched aliphatics, and PAHs with many fused rings.

In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. Anaerobic decomposition of petroleum hydrocarbons leads to extremely low rates of degradation. The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is,

greater than 7. The moisture content of the contaminated soil will affect biodegradation of oils due to dissolution of the residual compounds, dispersive actions, and the need for microbial metabolism to sustain high activity. The moisture content in soil affects microbial locomotion, solute diffusion, substrate supply, and the removal of metabolic by-products. Biodegradation rates in soils are also affected by the volume of product released to the environment. At concentrations of 0.5% of oil by volume, the degradation rate in soil is fairly independent of oil concentrations. However, as oil concentration rises, the first order degradation rate decreases and the oil degradation half-life increases. Ultimately, when the oil reaches saturation conditions in the soil (i.e., 30-50% oil), biodegradation virtually ceases.

Excessive moisture will limit the gaseous supply of oxygen for enhanced decomposition of petroleum hydrocarbons. Most studies indicate that optimum moisture content is within 50-70% of the water holding capacity.

All biological transformations are affected by temperature. Generally, as the temperature increases, biological activity tends to increase up to a temperature where enzyme denaturation occurs. The presence of oil should increase soil temperature, particularly at the surface. The darker color increases the heat capacity by adsorbing more radiation. The optimal temperature for biodegradation to occur ranges from 18 C to 30 C. Minimum rates would be expected at 5 C or lower.

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

### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
n-octane	LOW	LOW
m-xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.08 days)
p-xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.75 days)
o-xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
heptane	LOW	LOW
n-nonane	LOW	LOW
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
benzene	HIGH (Half-life = 720 days)	LOW (Half-life = 20.88 days)

### Bioaccumulative potential

Ingredient	Bioaccumulation
n-octane	HIGH (LogKOW = 5.18)
m-xylene	LOW (BCF = 1.37)
p-xylene	LOW (BCF = 2.2)
o-xylene	LOW (BCF = 219)
heptane	HIGH (LogKOW = 4.66)
n-nonane	HIGH (LogKOW = 4.7613)
toluene	LOW (BCF = 90)
ethylbenzene	LOW (BCF = 79.43)
benzene	HIGH (BCF = 4360)

### Mobility in soil

Ingredient	Mobility
n-octane	LOW (KOC = 506.7)
m-xylene	LOW (KOC = 434)
p-xylene	LOW (KOC = 434)
o-xylene	LOW (KOC = 443.1)
heptane	LOW (KOC = 274.7)
n-nonane	LOW (KOC = 934.6)
toluene	LOW (KOC = 268)
ethylbenzene	LOW (KOC = 517.8)
benzene	LOW (KOC = 165.5)

## SECTION 13 Disposal considerations

### Waste treatment methods

#### Product / Packaging disposal

- Containers may still present a chemical hazard/ danger when empty.
- Return to supplier for reuse/ recycling if possible.

Continued...

	<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> <li>▸ Where in doubt contact the responsible authority.</li> <li>▸ Recycle wherever possible.</li> <li>▸ 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.</li> <li>▸ 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).</li> <li>▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.</li> </ul>
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## SECTION 14 Transport information

### Labels Required

	
Marine Pollutant	
HAZCHEM	3YE

### Land transport (ADG)

UN number	1268	
UN proper shipping name	PETROLEUM DISTILLATES, N.O.S. or PETROLEUM PRODUCTS, N.O.S. (contains petroleum crude oil)	
Transport hazard class(es)	Class	3
	Subrisk	Not Applicable
Packing group	I	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	Not Applicable
	Limited quantity	500 ml

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

UN number	1268	
UN proper shipping name	Petroleum products, n.o.s. (contains petroleum crude oil); Petroleum distillates, n.o.s. (contains petroleum crude oil)	
Transport hazard class(es)	ICAO/IATA Class	3
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	3H
Packing group	I	
Environmental hazard	Environmentally hazardous	

<b>Special precautions for user</b>	Special provisions	A3
	Cargo Only Packing Instructions	361
	Cargo Only Maximum Qty / Pack	30 L
	Passenger and Cargo Packing Instructions	351
	Passenger and Cargo Maximum Qty / Pack	1 L
	Passenger and Cargo Limited Quantity Packing Instructions	Forbidden
	Passenger and Cargo Limited Maximum Qty / Pack	Forbidden

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

<b>UN number</b>	1268	
<b>UN proper shipping name</b>	PETROLEUM DISTILLATES, N.O.S. or PETROLEUM PRODUCTS, N.O.S. (contains petroleum crude oil)	
<b>Transport hazard class(es)</b>	IMDG Class	3
	IMDG Subrisk	Not Applicable
<b>Packing group</b>	I	
<b>Environmental hazard</b>	Marine Pollutant	
<b>Special precautions for user</b>	EMS Number	F-E, S-E
	Special provisions	Not Applicable
	Limited Quantities	500 mL

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

Not Applicable

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

Product name	Group
petroleum crude oil	Not Available
distillates, petroleum, middle, hydrotreated	Not Available
naphtha petroleum, light, hydrotreated.	Not Available
gas oil, petroleum, hydrotreated, vacuum	Not Available
n-octane	Not Available
m-xylene	Not Available
p-xylene	Not Available
o-xylene	Not Available
heptane	Not Available
n-nonane	Not Available
toluene	Not Available
ethylbenzene	Not Available
benzene	Not Available

**Transport in bulk in accordance with the ICG Code**

Product name	Ship Type
petroleum crude oil	Not Available
distillates, petroleum, middle, hydrotreated	Not Available
naphtha petroleum, light, hydrotreated.	Not Available
gas oil, petroleum, hydrotreated, vacuum	Not Available
n-octane	Not Available
m-xylene	Not Available

Product name	Ship Type
p-xylene	Not Available
o-xylene	Not Available
heptane	Not Available
n-nonane	Not Available
toluene	Not Available
ethylbenzene	Not Available
benzene	Not Available

## SECTION 15 Regulatory information

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

#### petroleum crude oil 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

#### distillates, petroleum, middle, 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

#### naphtha petroleum, light, 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

#### gas oil, petroleum, hydrotreated, vacuum 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

#### n-octane is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

#### m-xylene 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 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

#### p-xylene 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)

#### o-xylene 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 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

#### heptane is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

#### n-nonane is found on the following regulatory lists

## Australian Inventory of Industrial Chemicals (AIIC)

**toluene 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 5

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

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

**ethylbenzene 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 5

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

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

Australia - New South Wales Work Health and Safety Regulation - Restricted carcinogens

Australia - Northern Territories Work Health and Safety National Uniform Legislation Regulations- Restricted carcinogens

Australia - Queensland Work Health and Safety Regulation - Restricted Carcinogens

Australia - South Australia - Work Health and Safety Regulations - Restricted carcinogens

Australia - Tasmania - Work Health and Safety Regulations - Restricted carcinogens

Australia - Western Australia Carcinogenic substances to be used only for purposes approved by the Commissioner

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

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

Australia Model Work Health and Safety Regulations - Restricted carcinogens

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7

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

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (petroleum crude oil; distillates, petroleum, middle, hydrotreated; naphtha petroleum, light, hydrotreated.; gas oil, petroleum, hydrotreated, vacuum; n-octane; m-xylene; p-xylene; o-xylene; heptane; n-nonane; toluene; ethylbenzene; benzene)
China - IECSC	No (petroleum crude oil; gas oil, petroleum, hydrotreated, vacuum)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (naphtha petroleum, light, hydrotreated.)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (gas oil, petroleum, hydrotreated, vacuum)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (gas oil, petroleum, hydrotreated, vacuum)
Vietnam - NCI	No (gas oil, petroleum, hydrotreated, vacuum)
Russia - FBEPH	No (gas oil, petroleum, hydrotreated, vacuum)
<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**

Revision Date | 11/02/2022

Continued...

<b>Initial Date</b>	26/01/2022
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## 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  
AIIIC: 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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