Western Canadian Select (WCS)

Husky Oil Marketing

Chemwatch Hazard Alert Code: **4**

Chemwatch: **972-3485**
Version No: **1.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **24/02/2020**
Print Date: **19/10/2022**
L.GHS.AUS.EN

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

Product Identifier

Product name	Western Canadian Select (WCS)
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	PETROLEUM CRUDE OIL
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Chemical feedstock.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Husky Oil Marketing**
Address	Box 6525, Station “D” Calgary Alberta T2P 3G7 Canada
Telephone	+1 403 298 6111
Fax	+1 403 298 7464
Website	http://huskyenergy.com/
Email	investor.relations@cenovus.com

Emergency telephone number

Association / Organisation	**Husky Oil Marketing**
Emergency telephone numbers	+1 877 262 2111 (24 Hour)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Flammable Liquids Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Page 1 continued...

Hazard statement(s)

H224	Extremely flammable liquid and vapour.

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.
P235	Keep cool.
P240	Ground and bond container and receiving equipment.
P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P271	Use only a well-ventilated area.
P280	Wear protective gloves and protective clothing.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
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.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P337+P313	If eye irritation persists: Get medical advice/attention.

Precautionary statement(s) Storage

P403+P233	Store in a well-ventilated place. Keep container tightly closed.
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.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
8002-05-9	91.24	petroleum crude oil
7704-34-9.	2.73-5.47	sulfur
142-82-5	0.91-4.56	heptane
110-54-3	0.91-4.56	n-hexane
111-65-9	0.91-4.56	n-octane
109-66-0	0.91-4.56	n-pentane
111-84-2	0.91-4.56	n-nonane
1330-20-7	0.45-1.36	xylene
108-88-3	0.45-1.36	toluene
100-41-4	0.45-1.36	ethylbenzene

Continued...

CAS No	%[weight]	Name
71-43-2	0.09-0.91	benzene
130498-29-2	NotSpec	polycyclic aromatic hydrocarbons
7783-06-4	Trace	hydrogen sulfide

Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▸ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

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.
BP America Product Safety & Toxicology Department
- 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.

SECTION 5 Firefighting measures

Extinguishing media

- Foam.

Continued...

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	‣ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ May be violently or explosively reactive. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Fight fire from a safe distance, with adequate cover. ‣ If safe, switch off electrical equipment until vapour fire hazard removed. ‣ Use water delivered as a fine spray to control the fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **Do not** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	‣ Liquid and vapour are highly flammable. ‣ Severe fire hazard when exposed to heat, flame and/or oxidisers. ‣ Vapour forms an explosive mixture with air. ‣ Severe explosion hazard, in the form of vapour, when exposed to flame or spark. ‣ Vapour may travel a considerable distance to source of ignition. ‣ Heating may cause expansion / decomposition with violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO) Combustion products include: , carbon dioxide (CO_2) , sulfur oxides (SOx) , hydrogen sulfide (H_2S) , other pyrolysis products typical of burning organic material. **CARE**: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire.
HAZCHEM	3WE

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb small quantities with vermiculite or other absorbent material. ‣ Wipe up. ‣ Collect residues in a flammable waste container.
Major Spills	Chemical Class: aromatic hydrocarbons For release onto land: recommended sorbents listed in order of priority. SORBENT TYPE | RANK | APPLICATION | COLLECTION | LIMITATIONS LAND SPILL - SMALL Feathers - pillow | 1 | throw | pitchfork | DGC, RT cross-linked polymer - particulate | 2 | shovel | shovel | R,W,SS

Continued...

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

LAND SPILL - MEDIUM

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

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

- Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear full body protective clothing with breathing apparatus.
- Prevent, by any means available, spillage from entering drains or water course.
- No smoking, naked lights or ignition sources.
- Increase ventilation.
- Stop leak if safe to do so.
- 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 (H2S 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.
- 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.

Continued...

	‣ Avoid physical damage to containers. ‣ Always wash hands with soap and water after handling. ‣ Work clothes should be laundered separately. ‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	· Store in original containers in approved flame-proof area. · No smoking, naked lights, heat or ignition sources. · **DO NOT** store in pits, depression, basement or areas where vapours may be trapped. · Keep containers securely sealed. · Store away from incompatible materials in a cool, dry well ventilated area. · Protect containers against physical damage and check regularly for leaks. · Observe manufacturer's storage and handling recommendations contained within this MSDS. · Tank storage: Tanks must be specifically designed for use · with this product. Bulk storage tanks should be diked · (bunded). 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. · 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. · 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 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

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Packing as supplied by manufacturer. ‣ Plastic containers may only be used if approved for flammable liquid. ‣ Check that containers are clearly labelled and free from leaks. ‣ 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. ‣ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ‣ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ‣ 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. ‣ 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 ‣ 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.
Storage incompatibility	Sulfur: ‣ is both and oxidising agent and a reducing agent ‣ when finely divided and dry, forms explosive mixtures with air ‣ when molten can generate hydrogen sulfide and carbon disulfide when in contact with some organic materials. ‣ is a flammable substance in both the solid and liquid states; the dust is characterised by a very low ignition point of 190 C compared to other combustible dusts - dust clouds are readily ignited by weak frictional sparks if the oxygen content is above 8%. ‣ vapours reacts violently with lithium carbide ‣ forms explosive and extremely sensitive mixtures with most oxidising substances such as chlorates, nitrates, perchlorates and permanganates; mixtures may be extremely sensitive to friction or vibration. ‣ reacts violently with many substances, including strong oxidisers, aluminium powders, boron, bromine pentafluoride, bromine trifluoride, calcium hypochlorite, carbides, caesium, chlorates, chlorine dioxide, chlorine trifluoride, chromic acid, chromyl chloride, dichlorine oxide, diethylzinc, fluorine, halogen compounds, hexalithium disilicide, lampblack, lead chlorite, lead dioxide, lithium powdered nickel, nickel catalysts, red phosphorus, phosphorus trioxide, potassium, potassium chlorite, potassium iodate, potassium peroxoferrate, rubidium acetylide, ruthenium tetraoxide, sodium, sodium chlorite, sodium peroxide, tin, uranium, zinc, zinc(II) nitrate, hexahydrate ‣ forms friction-, impact- and shock- sensitive explosive or pyrophoric mixtures with ammonia, ammonium nitrate, barium

Continued...

bromate, bromates, calcium carbide, charcoal, hydrocarbons, iodates, iodine pentafluoride, iodine pentoxide, iron, lead chromate, mercurous oxide, mercury nitrate, mercury oxide, nitryl fluoride, nitrogen dioxide, inorganic perchlorates, potassium bromate, potassium nitride, potassium perchlorate, silver nitrate, sodium hydride, sulfur dichloride
- is incompatible with barium carbide, calcium, calcium carbide, calcium phosphide, chromates, chromic acid, chromic anhydride, 1,5-dinitronaphthalene, hafnium, indium, iodates, iodic acid, iodine oxide, lead chlorate, lithium acetylide, mercury oxide, mercuric nitrate, palladium, potassium permanganate, silver bromate, silver chlorate, silver oxide,sodium, strontium carbide, thallium oxide, thorium, thorium dicarbide
- attacks copper, mercury, silver
- when molten reacts with air forming sulfur dioxide, and with hydrogen, forming hydrogen sulfide; explosion may occur
- may accumulate static electrical charges; vapours may ignite

NOTE: Dusts containing 25% or more elemental sulfur may be almost as explosive as pure sulfur.
Sulfur will form sulfides with most metals, including iron, and reacts vigorously with metals in the sodium and magnesium groups on the periodic table. Sulfides of iron will oxidise fairly rapidly in moist air. In the presence of other readily oxidised combustibles (such as some oily materials) under certain conditions, the heat liberated may be sufficient to result in spontaneous ignition. This phenomenon has not been observed with pure sulfur products or disintegrating sulfur in contact with unprotected steel at ordinary ambient temperatures. Inadvertent mixtures of sulfur, iron, and miscellaneous oils should be avoided.. Oxidation is accelerated by higher temperatures. Heat buildup and ignition can be prevented by keeping the sulfides wet until oxidation is complete.
Xylenes:
- may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride
- attack some plastics, rubber and coatings
- may generate electrostatic charges on flow or agitation due to low conductivity.
- 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 (H2S):
- 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

For alkyl aromatics:
The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.
- Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen
- Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.
- Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.
- Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.
- Alkali metals accelerate the oxidation while CO2 as co-oxidant enhances the selectivity.
- Microwave conditions give improved yields of the oxidation products.
- Photo-oxidation products may occur following reaction with hydroxyl radicals and NOx - 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
· **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/m3
· 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.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Continued...

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	petroleum crude oil	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
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-hexane	Hexane (n-Hexane)	20 ppm / 72 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	n-octane	Octane	300 ppm / 1400 mg/m3	1750 mg/m3 / 375 ppm	Not Available	Not Available
Australia Exposure Standards	n-pentane	Pentane	600 ppm / 1770 mg/m3	2210 mg/m3 / 750 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	xylene	Xylene (o-, m-, p-isomers)	80 ppm / 350 mg/m3	655 mg/m3 / 150 ppm	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
Australia Exposure Standards	hydrogen sulfide	Hydrogen sulphide	10 ppm / 14 mg/m3	21 mg/m3 / 15 ppm	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
heptane	500 ppm	830 ppm	5000* ppm
n-hexane	260 ppm	Not Available	Not Available
n-octane	230 ppm	385 ppm	5000** ppm
n-pentane	3000* ppm	33000*** ppm	200000*** ppm
n-nonane	600 ppm	830 ppm	5,000 ppm
xylene	Not Available	Not Available	Not Available
toluene	Not Available	Not Available	Not Available
ethylbenzene	Not Available	Not Available	Not Available
benzene	Not Available	Not Available	Not Available
hydrogen sulfide	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
petroleum crude oil	1,100 ppm	Not Available
sulfur	Not Available	Not Available
heptane	750 ppm	Not Available
n-hexane	1,100 ppm	Not Available
n-octane	1,000 ppm	Not Available
n-pentane	1,500 ppm	Not Available
n-nonane	Not Available	Not Available
xylene	900 ppm	Not Available
toluene	500 ppm	Not Available
ethylbenzene	800 ppm	Not Available
benzene	500 ppm	Not Available
polycyclic aromatic hydrocarbons	Not Available	Not Available
hydrogen sulfide	100 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit

Continued...

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sulfur	E	≤ 0.01 mg/m³
polycyclic aromatic hydrocarbons	C	> 0.1 to ≤ milligrams per cubic meter of air (mg/m³)
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

IFRA Prohibited Fragrance Substance
The International Fragrance Association (IFRA) Standards form the basis for the globally accepted and recognized risk management system for the safe use of fragrance ingredients and are part of the IFRA Code of Practice. This is the self-regulating system of the industry, based on risk assessments carried out by an independent Expert Panel
Toxicity and Irritation data for petroleum-based mineral oils are related to chemical components and vary as does the composition and source of the original crude.
A small but definite risk of occupational skin cancer occurs in workers exposed to persistent skin contamination by oils over a period of years. This risk has been attributed to the presence of certain polycyclic aromatic hydrocarbons (PAH) (typified by benz[a]pyrene).
Petroleum oils which are solvent refined/extracted or severely hydrotreated, contain very low concentrations of both.
for mineral oils, excluding metal working fluids, poorly and mildly refined:
A2; Suspected Human Carcinogen (ACGIH)
A2 is used primarily when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals with relevance to humans
polycyclic aromatic hydrocarbons (as benzene solubles):
TLV TWA: 0.2 mg/m3 (A1)
WARNING : Benzene solubles are classified by ACGIH as A1 - CONFIRMED HUMAN CARCINOGEN.
ES TWA: 0.2 mg/m3 Carcinogen Category 1
WARNING : Benzene solubles are classified by Worksafe as Category 1 - Established Human Carcinogen.
These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:
D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class	OSF	Description
A	550	Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B	26-550	As "A" for 50-90% of persons being distracted
C	1-26	As "A" for less than 50% of persons being distracted
D	0.18-1	10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E	<0.18	As "D" for less than 10% of persons aware of being tested

for benzene
Odour Threshold Value: 34 ppm (detection), 97 ppm (recognition)
NOTE: Detector tubes for benzene, measuring in excess of 0.5 ppm, are commercially available. The relative quality of epidemiological data and quantitative health risk assessments related to documented and theoretical leukaemic deaths constitute the basis of the TLV-recommendation.
One study [Dow Chemical] demonstrates a significant fourfold increase in myelogenous leukaemia for workers exposed to average benzene concentrations of about 5 ppm for an average of 9 years and that 2 out of four individuals in the study who died from leukaemia were characterised as having been exposed to average benzene levels below 2 ppm. Based on such findings the estimated risk of leukaemia in workers exposed at daily benzene concentrations of 10 ppm for 40 years is 155 times that of unexposed workers; at 1 ppm the risk falls to 1.7 times whilst at 0.1 ppm the risk is about the same in the two groups. A revision of the TLV-TWA to 0.1 ppm was proposed in 1990 but this has been revised upwards as result of industry initiatives.
Typical toxicities displayed following inhalation:

- At 25 ppm (8 hours): no effect
- 50-150 ppm: signs of intoxication within 5 hours
- 500-1500 ppm: signs of intoxication within 1 hour
- 7500 ppm: severe intoxication within 30-60 minutes
- 20000 ppm: fatal within 5-10 minutes

Some jurisdictions require that health surveillance be conducted on occupationally exposed workers. Some surveillance should emphasise (i) demography,

Continued...

occupational and medical history and health advice (ii) baseline blood sample for haematological profile (iii) records of personal exposure.
for heptane (all isomers)
The TLV-TWA is protective against narcotic and irritant effects which are greater than those of pentane or n-hexane but less than those of octane. The TLV-TWA applies to all isomers.
Inhalation by humans of 1000 ppm for 6 minutes produced slight dizziness. Higher concentrations for shorter periods produce marked vertigo, incoordination and hilarity. Signs of central nervous system depression occur in the absence of mucous membrane irritation. Brief exposures to high levels (5000 ppm for 4 minutes) produce nausea, loss of appetite and a "gasoline-like" taste in the mouth that persists for many hours after exposure ceases
for: hexane, isomers (excluding n-hexane)
The TLV-TWA is thought to be protective against nausea, headache, upper respiratory tract irritation and CNS depression. The STEL is added to prevent objective depression of the CNS. The lower value ascribed
to n-hexane is due to the neurotoxicity of its metabolites, principally 5-hydroxy-2-hexanone and 2,5-hexanedione. It is considered unlikely that other hexanes follow the same metabolic route. It should be noted however that the n-hexane TLV-TWA also applies to commercial hexane having a concentration of greater than 5% n-hexane.

For n-nonane and isomers:
Odour Threshold: 47 ppm
Inhalation of high concentrations of aliphatic hydrocarbons produces central nervous system depression leading to coma with inhibition of deep tendon reflexes. The TLV-TWA is protective against narcotic effects
produced at higher concentrations.
Odour Safety Factor(OSF)
OSF=4.3 (n-NONANE)

Odour threshold: 0.25 ppm.
The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour. A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m3 is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.
Odour Safety Factor (OSF)
OSF=0.042 (gasoline)
For n-hexane:
Odour Threshold Value: 65 ppm
NOTE: Detector tubes for n-hexane, measuring in excess of 100 ppm, are available commercially.
Occupational polyneuropathy may result from exposures as low as 500 ppm (as hexane), whilst nearly continuous exposures of 250 ppm have caused neurotoxic effects in animals. Many literature reports have failed to distinguish hexane from n-hexane and on the assumption that the commercial hexane contains 30% n-hexane, a worst case recommendation for TLV is assumed to reduce the risk of peripheral neuropathies (due to the metabolites 2,5-heptanedione and 3,6-octanedione) and other adverse neuropathic effects.
Concurrent exposure to chemicals (including MEK) and drugs which induce hepatic liver oxidative metabolism can reduce the time for neuropathy to appear.
Odour Safety Factor(OSF)
OSF=0.15 (n-HEXANE)

For n-octane:
Odour Threshold Value: 152 ppm (detection), 235 ppm (recognition)
The TLV-TWA is thought to be protective against narcotic effects produced at higher concentrations.
Odour Safety Factor(OSF)
OSF=6.3 (n-OCTANE)
For n-pentane
NOTE: Detector tubes for n-pentane, measuring in excess of 100 ppm, are commercially available.
The TLV-TWA is thought to be protective against narcotic effects produced at higher concentrations and the development of axonopathies. Although the possibility exists that chronic exposure to high concentrations may produce polyneuropathy, there is no specific data to support the role of pentane in the pathogenesis of central peripheral distal axonopathy.

for xylenes:
IDLH Level: 900 ppm
Odour Threshold Value: 20 ppm (detection), 40 ppm (recognition)
NOTE: Detector tubes for o-xylene, measuring in excess of 10 ppm, are available commercially. (m-xylene and p-xylene give almost the same response).
Xylene vapour is an irritant to the eyes, mucous membranes and skin and causes narcosis at high concentrations. Exposure to doses sufficiently high to produce intoxication and unconsciousness also produces transient liver and kidney toxicity. Neurologic impairment is NOT evident amongst volunteers inhaling up to 400 ppm though complaints of ocular and upper respiratory tract irritation occur at 200 ppm for 3 to 5 minutes.
Exposure to xylene at or below the recommended TLV-TWA and STEL is thought to minimise the risk of irritant effects and to produce neither significant narcosis or chronic injury. An earlier skin notation was deleted because percutaneous absorption is gradual and protracted and does not substantially contribute to the dose received by inhalation.
Odour Safety Factor(OSF)
OSF=4 (XYLENE)

For toluene:
Odour Threshold Value: 0.16-6.7 (detection), 1.9-69 (recognition)
NOTE: Detector tubes measuring in excess of 5 ppm, are available.
High concentrations of toluene in the air produce depression of the central nervous system (CNS) in humans. Intentional toluene exposure (glue-sniffing) at

Continued...

maternally-intoxicating concentration has also produced birth defects. Foetotoxicity appears at levels associated with CNS narcosis and probably occurs only in those with chronic toluene-induced kidney failure. Exposure at or below the recommended TLV-TWA is thought to prevent transient headache and irritation, to provide a measure of safety for possible disturbances to human reproduction, the prevention of reductions in cognitive responses reported amongst humans inhaling greater than 40 ppm, and the significant risks of hepatotoxic, behavioural and nervous system effects (including impaired reaction time and incoordination). Although toluene/ethanol interactions are well recognised, the degree of protection afforded by the TLV-TWA among drinkers is not known.
Odour Safety Factor(OSF)
OSF=17 (TOLUENE)

for ethyl benzene:
Odour Threshold Value: 0.46-0.60 ppm
NOTE: Detector tubes for ethylbenzene, measuring in excess of 30 ppm, are commercially available.
Ethyl benzene produces irritation of the skin and mucous membranes and appears to produce acute and chronic effects on the central nervous system. Animal experiments also suggest the effects of chronic exposure include damage to the liver, kidneys and testes. In spite of structural similarities to benzene, the material does not appear to cause damage to the haemopoietic system. The TLV-TWA is thought to be protective against skin and eye irritation. Exposure at this concentration probably will not result in systemic effects.
Subjects exposed at 200 ppm experienced transient irritation of the eyes; at 1000 ppm there was eye irritation with profuse lachrymation; at 2000 ppm eye irritation and lachrymation were immediate and severe accompanied by moderate nasal irritation, constriction in the chest and vertigo; at 5000 ppm exposure produced intolerable irritation of the eyes and throat.
Odour Safety Factor(OSF)
OSF=43 (ETHYL BENZENE)

Odour Threshold Value for hydrogen sulfide: 0.0011 ppm (detection), 0.0045 ppm (recognition)
NOTE: Detector tubes for hydrogen sulfide, measuring in excess of 0.5 ppm are available commercially.
The TLV-TWA is protective against sudden death, eye irritation, neurasthenic symptoms such as fatigue, headache, dizziness, and irritability, or permanent central nervous system effects that may result from acute, subchronic, or acute exposure to hydrogen sulfide. The offensive odour of hydrogen sulfide does not give a reliable warning signal because olfactory fatigue occurs at concentrations of 150 to 200 ppm.
Hydrogen sulfide is probably the leading cause of sudden death in the workplace. Lethal hydrogen sulfide toxicity following inhalation of 1000-2000 ppm paralyses the respiratory centre and causes breathing to stop. At concentrations between 500 to 1000 pm, the carotid bodies are stimulated causing hypernea which is followed by apnea. Low concentrations
(50-1500 ppm) produce eye and respiratory tract irritation. Prolonged exposure to concentrations of the order of 250-500 ppm may produce pulmonary oedema although 50 ppm has also reportedly produced this effect.
Concentrations in excess of 50 ppm produce acute conjunctivitis with pain, lachrymation and photophobia. These acute changes may progress to keratoconjunctivitis and vesiculation of the corneal epithelium.
Concentrations between 5 and 30 ppm produce ocular toxicity.
The inherent toxic and olfactory (sense of smell) fatiguing properties of hydrogen sulfide require that air monitoring alarms be used if concentrations are expected to reach harmful levels such as in enclosed spaces, heated transport vessels
and spill or leak situations. If the air concentration exceeds 10 ppm, the area should be evacuated unless respiratory
protection is in use. In areas where hydrogen sulfide vapours may accumulate, a positive-pressure air-supplied respirator is advised.
Odour Safety Factor(OSF)
OSF=1.2E3 (HYDROGEN SULFIDE)
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 proceeded 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

Appropriate engineering controls	Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. ▸ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▸ 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. ▸ 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. ▸ Open-vessel systems are prohibited. ▸ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▸ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless

Continued...

	decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▸ 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. ▸ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▸ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▸ 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.
Personal protection	
Eye and face protection	▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber
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:
Western Canadian Select (WCS)

Respiratory protection

Type A 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.

Continued...

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23 2-PLY	C
SARANEX-23	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.

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

^ - 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)

For concentrations exceeding 10 ppm hydrogen sulfide or for unknown concentrations:

- Respirators should be equipped with pressure demand regulators and operated in pressure demand mode only. If airline units are used, a 5-minute egress bottle must also be carried.
- Gas masks or other air-purifying respirators must never be used for H2S, due to the poor warning properties of the gas.
- When exposure concentrations are unknown and respiratory protection is not used, personal H2S warning devices should be worn.
- These devices should not be relied on to warn of life-threatening concentrations.
- H2S rapidly fatigues the sense of smell; the rotten egg odour disappears quickly even where high concentrations are present.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Brown to black liquid.

Physical state	Liquid	**Relative density (Water = 1)**	0.900 to 0.950 (Water = 1) at 15 °C (59 °F)
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	200-300 @ 15
Initial boiling point and boiling range (°C)	≤ 35 °C (95 °F) (ASTM D86)	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	<-5	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available

Continued...

Vapour pressure (kPa)	< 70 kPa at 37.8 °C (100 °F) (ASTM D6377)	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severely damaging effects to the health of the individual. Relatively small amounts absorbed from the lungs may prove fatal. There is no evidence that systemic poisoning results from inhalation of sulfur dust. Sulfur vapour irritates both the upper and lower respiratory passages and if inhaled may cause coughing, conjunctivitis, nausea,vomiting and chest tightness, bronchitis and in extreme pulmonary oedema (sudden or delayed). Inhalation hazard is increased at higher temperatures. High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. 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. 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. 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 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.

Continued...

	Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue. Xylene is a central nervous system depressant. 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.
Ingestion	Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Ingested sulfur is converted to sulfides in the gastrointestinal tract, and ingestion of 10 to 20 grams has caused irritation of the GI tract and renal injury. Individuals with known allergies to sulfide drugs may also have allergic reactions to elemental sulfur. Swallowing large amounts may cause nausea and vomiting. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. 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. Chronic inhalation or skin exposure to n-hexane may cause peripheral neuropathy, which is damage to nerve ends in extremities, e.g. fingers, with loss of sensation and characteristic thickening. Nerve damage has been documented with chronic exposures of greater than 500 ppm. Improvement in condition does not immediately follow removal from exposure and symptoms may progress for two or three months. Recovery may take a year or more depending on severity of exposure, and may not always be complete. Exposure to n-hexane with methyl ethyl ketone (MEK) will accelerate the appearance of damage, but MEK alone will not cause the nerve damage. Other isomers of hexane do not cause nerve damage. [*Source: Shell Co.*]
Skin Contact	The material may accentuate any pre-existing dermatitis condition 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 . Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. 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. The material may produce moderate skin irritation; limited evidence or practical experience suggests, that the material either: ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact and/or ▸ 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. 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.
Eye	Exposure to H2S 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 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. Limited evidence or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. 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. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development

Continued...

of heritable genetic damage, generally on the basis of
- appropriate animal studies,
- other relevant information

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

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.

Exposure to oil mists frequently elicits respiratory conditions, such as asthma; the provoking agent is probably an additive. High oil mist concentrations may produce lipoid pneumonia although clinical evidence is equivocal. In animals exposed to concentrations of 100 mg/m3 oil mist, for periods of 12 to 26 months, the activity of lung and serum alkaline phosphatase enzyme was raised; 5 mg/m3 oil mist did not produce this response. These enzyme changes are sensitive early indicators of lung damage. Workers exposed to vapours of mineral oil and kerosene for 5 to 35 years showed an increased prevalence of slight basal lung fibrosis.

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.

Smith J.H., et al: Toxicologic Pathology: 24, 2, 214-230, 1996

The so-called polycyclic aromatic hydrocarbons (PAHs) comprise a large family; some members occur in coal tar, tobacco smoke, petroleum and air pollution.

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.

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.

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 date 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.

Continued...

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.
Sensitive persons can experience skin irritation from repeated exposure to the sulfur dust. Allergic responses can occur.

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*]

Chronic inhalation or skin exposure to n-hexane may cause peripheral neuropathy, which is damage to nerve ends in extremities, e.g. fingers, with loss of sensation and characteristic thickening. Nerve damage has been documented with chronic exposures of greater than 500 ppm. Improvement in condition does not immediately follow removal from exposure and symptoms may progress for two or three months. Recovery may take a year or more depending on severity of exposure, and may not always be complete. Exposure to n-hexane with methyl ethyl ketone (MEK) will accelerate the appearance of damage, but MEK alone will not cause the nerve damage. Other isomers of hexane do not cause nerve damage. [*Source: Shell Co.*]
Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mix ed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.
Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers.
Xylene has been classed as a developmental toxin in some jurisdictions.
Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in

Continued...

	the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity. Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex. 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)

Western Canadian Select (WCS)	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
petroleum crude oil	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (rabbit): 100 mg mild
	Oral (Rat) LD50; >4300 mg/kg[2]	Skin (rabbit): 500 mg/24H Mild
sulfur	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (human): 8 ppm irritant
	Inhalation(Rat) LC50; >5.43 mg/L4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >2000 mg/kg[1]	Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
heptane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >29.29 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg[1]	
n-hexane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye(rabbit): 10 mg - mild
	Inhalation(Rat) LC50; 48000 ppm4h[2]	
	Oral (Rat) LD50; 28710 mg/kg[2]	
n-octane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >24.88 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg[1]	
n-pentane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 3000 mg/kg[2]	Not Available
	Inhalation(Rat) LC50; >25.3 mg/l4h[1]	
	Oral (Rat) LD50; >2000 mg/kg[1]	
n-nonane	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; 3200 ppm4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >5000 mg/kg[1]	
xylene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1700 mg/kg[2]	Eye (human): 200 ppm irritant

Continued...

	Inhalation(Rat) LC50; 5000 ppm4h[2]	Eye (rabbit): 5 mg/24h SEVERE
	Oral (Mouse) LD50; 2119 mg/kg[2]	Eye (rabbit): 87 mg mild
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):500 mg/24h moderate
		Skin: adverse effect observed (irritating)[1]

toluene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 12124 mg/kg[2]	Eye (rabbit): 2mg/24h - SEVERE
	Inhalation(Rat) LC50; >13350 ppm4h[2]	Eye (rabbit):0.87 mg - mild
	Oral (Rat) LD50; 636 mg/kg[2]	Eye (rabbit):100 mg/30sec - mild
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):20 mg/24h-moderate
		Skin (rabbit):500 mg - moderate
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]

ethylbenzene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 17800 mg/kg[2]	Eye (rabbit): 500 mg - SEVERE
	Inhalation(Rat) LC50; 17.2 mg/l4h[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; 3500 mg/kg[2]	Skin (rabbit): 15 mg/24h mild
		Skin: no adverse effect observed (not irritating)[1]

benzene	**TOXICITY**	**IRRITATION**
	dermal (mouse) LD50: 48 mg/kg[2]	Eye (rabbit): 2 mg/24h - SEVERE
	Inhalation(Rat) LC50; 43.767 mg/L4h[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50; 930 mg/kg[2]	SKIN (rabbit):20 mg/24h - moderate
		Skin: adverse effect observed (irritating)[1]

polycyclic aromatic hydrocarbons	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

hydrogen sulfide	**TOXICITY**	**IRRITATION**
	Inhalation(Mouse) LC50; 316.028 ppm4h[2]	Not Available

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

Western Canadian Select (WCS)	For polynuclear aromatic hydrocarbons (PAH) such as the benz[a]anthracenes (BA), carcinogenic activity is appreciably influenced by the numbers and positions of methyl and other substituents and hence by the molecular shapes. The planarities and dimensions of methyl-substituted BA and related PAH, including methyl phenanthrenes (MP) which also contain the carcinogenically important bay and K regions, have been compared. BA molecules with substituents well removed from the bay region, including those substituted at 5 or 6 (the K region), are nearly, but not quite, planar, with a mutual inclination of several degrees between A and C rings on each side of the bay region. With one or both bay positions 1 and 12 methyl-substituted, distortion is much greater (A/C up to 29 deg in 1,12-dimethyl BA). For phenanthrenes, the presence of the two methyl substituents in the bay, as in 2,4,5,7-tetra MP, can lead to A/C of 28 deg compared with the very small (2 deg) mutual inclination in 9,10-di MP. In the bay regions of all the PAH studied, the beach C–C bond is typically as long as 1.45 -1.46 angstrom and the beach C–C–C bond angles are enlarged to 122–123 deg. Effect of Methyl Substitution on Molecular Shapes and Dimensions of Phenanthrenes and Benz[a]anthracenes: Owen Johnson et al: Polycyclic Aromatic Compounds Volume 2, 1991 - Issue 2-3
PETROLEUM CRUDE OIL	Tumorigenic - Carcinogenic by RTECS criteria.
N-OCTANE	Oral (rat) LD50: 5630 mg/kg* [CCINFO] Nil reported
N-PENTANE	[GENIUM and CCINFO, V.W.&R.]
N-NONANE	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

Continued...

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.
For alkanes:
Exposure to the commercial hexane (a representative of the ECHA group of hydrocarbons, C5-C7, n-alkanes, isoalkanes, n-hexane rich) had no effect on the behavior of rats. Rats were tested monthly throughout the exposure for hindlimb splay and grip strength. The NOAEC for sub-chronic neurological effects is 9000 ppm in rats.
In a 13 week subchronic inhalation study, the neurotoxicity of light alkylate naphtha distillate (LAND-2; carbon range C5-C8) was examined in male and female rats and aside from acute CNS effects, no treatment related neurotoxic effects found in any of the treatment groups. The NOAEC was determined to be > 24.3 g/m3 (6646 ppm). Additionally, no neurological effects were reported in the NTP 2 year carcinogenicity study on Stoddard solvent.
For hydrocarbons, C5-C7, n-alkanes, isoalkanes, n-hexane rich
n-Hexane was metabolized and excreted within 168 h of iv bolus administration, inhalation exposure or dermal application. Exhaled breath and urine were the two primary routes for the excretion and its metabolites. n-Hexane was widely distributed to the body tissues but were not concentrated significantly by any of those tissues. It was extensively metabolized and a number of radio labeled metabolites were excreted in the urine. n-Hexane and its radio labeled metabolites disappeared from the blood of rats with a half-life of approximately 9-10 h.
Repeated inhalation exposure had no apparent effect on the rates or routes of excretion of either of the test compounds or their metabolites.
The absorption rates into the skin, normalised for exposure concentration, was determined to be 0.013 cm/h The maximum absorption rate into the blood was determined to be 0.005 nmol/h. A comparison of the estimated whole-body skin uptake with the inhalatory uptake from the same atmosphere, revealed that the dermal uptake contributed 0.1% to the total uptake
C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are absorbed, they are typically metabolized by side chain oxidation to alcohol and carboxylic acid derivatives. These metabolites can be glucuronidated and excreted in the urine or further metabolized before being excreted. The majority of the metabolites are excreted in the urine and to a lower extent, in the faeces. Excretion is rapid with the majority of the elimination occurring within the first 24 hours of exposure. As a result of the lack of systemic toxicity and the ability of the parent material to undergo metabolism and rapid excretion, bioaccumulation of the test substance in the tissues is not likely to occur.
C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are poorly absorbed dermally with an estimated overall percutaneous absorption rate of approximately 2ug/cm2/hr or 1% of the total applied fluid. Regardless of exposure route, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are rapidly metabolized and eliminated has been fully evaluated. All of the animal studies were performed in a manner similar or equivalent to currently established OECD guidelines. Based on these data, C9-C14 aliphatic, <2% aromatic hydrocarbons have a low order of acute toxicity by the oral, dermal, and inhalation routes of exposure.
In a study examining the oral toxicity of commercial hexane. 6 male rats were given doses of up to 25 ml/kg of test substance by oral gavage. The animals were then observed for 14 days for mortality. No mortality was observed at any of the doses. The oral LD50 is therefore > 25 ml/kg (16.75 g/kg; density of 0.67).
C9-C14 aliphatic, <2% aromatic hydrocarbons is minimally toxic via ingestion where the LD50 is >5000 mg/kg, via dermal exposure where the LD50 is >5000 mg/kg, and by inhalation where the LC50 > 5000 mg/m3. These findings do not warrant classification of C9-C14 aliphatic, <2% aromatic hydrocarbons under the Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP) do not warrant classification under the Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparations (DSD/DPD). C9-C14 aliphatic, <2% aromatic hydrocarbons are classified under EU CLP guidelines as a Category 1 aspiration hazard based on its physical and chemical properties (hydrocarbon fluid, viscosity = 20.5 mm2/s) and as an R65 aspiration hazard under the EU DSD/DPD.
One study examined that acute inhalation toxicity of hexane to male rats. Groups of 10 male rats exposed to various large concentrations of hexane vapour for 4 hrs. Animals were then observed for clinical signs and mortality for at least the next 6 days. Several animals died during the exposure period. The LC50 was determined to be 73,680 ppm (259354 mg/m3). Due to the high concentration of the LC50, the test substance would not be classified as toxic by inhalation according to OECD GHS guidelines. Surviving animals experienced severe toxicological effects during the exposure.
Skin irritation:
For isoparaffinic, normal paraffinic, and mixed C9-C14 aliphatic, <2% aromatic hydrocarbon fluids, the weight of evidence indicates that the erythema and oedema scores (24, 48, and 72 average) are below the classification threshold requirements: 2.0, Directive 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparation; 2.3, the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).
For cycloparaffinic C9-C14 aliphatic, < 2% aromatic hydrocarbon fluids, erythema and oedema scores (24, 48, and 72 average) are above the classification threshold requirements: 2.0, Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparation; 2.3, the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP). This finding warrants classification of the test material as a skin irritant (R38) under Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparations. This finding warrants classification of the test material as a Category 2 dermal irritant under the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).
Eye irritation
Ocular lesion scores (24, 48, and 72 average) are below the classification threshold requirements.
Drective 67/548/EEC for dangerous substances and Directive 1999/45/EC for preparation: 0, cornea opacity; 0, iris lesion; >2.5, redness of the conjunctivae; >2.0, oedema of the conjunctivae (chemosis). Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP): 0, cornea opacity; 0, iris lesion; >2.0, redness of the conjunctivae; >2.0, oedema of the conjunctivae (chemosis).
Respiratory irritation
There are no studies that warrant classification as a respiratory irritant under either the Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC or under the new Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP).
Sensitisation:

Continued...

	A study was performed to determine the concentration of hexane that would be expected to cause sensitization in humans. Results of previous LLNA experiments were used to calculate the EC3 value, the concentration at which the test substance would produce a 3 -fold increase in the proliferative activity of lymph nodes in the LLNA test. The 3 -fold increase is considered a positive response for sensitization in the LLNA test. The EC3 value for hexane was determined to be > 100% concentration. The test substance is therefore not sensitizing. There are no reports of respiratory sensitization from C9-C14 aliphatic, <2% aromatic hydrocarbons fluids in laboratory animals or humans. However, skin sensitization studies utilizing C9-C14 aliphatic, <2% aromatic hydrocarbons fluids found no indication of skin sensitization in guinea pigs. Additional studies in humans also found no indication of skin sensitization. With these observations, it is presumed that C9-C14 aliphatic, <2% aromatic hydrocarbons fluids will not be a respiratory sensitizing agent. Repeat dose toxicity, In a study involving n-hexane, neurological effects were only seen at the highest dose level after an average of 101.3 days of exposure. The LOAEL for neurological effects is 46.2 mmol/kg bw (37973 mg/kg), and the NOAEL is 13.2 mmol/kg bw (1135 mg/kg). Reduced body weight gain was seen at all three dose levels, however was only considered treatment related in the 13.2 and 46.2 mmol/kg bw groups. The NOAEL is therefore 6.60 mmol/kg bw. In a study involving n-hexane The NOAEC for male rats exposed via inhalation was 2984 ppm based on liver and kidney effects. The LOAEC for male rats was 8992 ppm. The NOAEC for female rats was 8992 ppm C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are expected to have a low order of repeated dose toxicity by the oral route of exposure. All tests were performed in a manner similar or equivalent to currently established OECD guidelines. In a repeated dose study where C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered via oral gavage, no signs of toxicity were observed at the maximum experimental dose tested, 5000 mg/kg/day. In a repeated dose study where C9-C14 aliphatic, <2% aromatic hydrocarbon fluids were administered via inhalation, no signs of toxicity were observed at 10400 mg/m3. Based on these observations, the repeat inhalation concentration NOAEL is =10400 mg/m3 (10.4 mg/L) for C9-C14 aliphatic, <2% aromatic hydrocarbon fluid Genetic toxicity: A study examined the in vitro mutagenicity of vapours of the test substance commercial hexane. Plates of S. typhimurium were exposed for 7 -8 hrs to test atmospheres of 0, 600, 1000, 3000, 6000, or 9000 ppm of test substance. The test substance did not produce a positive response in any of the test strains. The test substance is not mutagenic. In a study to determine the in vivo effect of inhalation exposure of commercial hexane on rat bone marrow. Groups of 5 male and 5 female rats were exposed to 0, 900, 3000, and 9000 ppm of test substance vapour for 6 hrs/day for 5 days. There was no statistically significant increase in cell aberrations in any treatment group. The test substance is not mutagenic. C9-C14 aliphatic, <2% aromatic hydrocarbons fluids are not mutagenic using in vitro or in vivo genotoxicity assays. In bacterial tests, C9-C14 aliphatic, <2% aromatic hydrocarbons fluids were not mutagenic in Salmonella strains tested in the presence or absence of metabolic activation. C9 -C14 aliphatic, <2% aromatic hydrocarbon fluids were negative in a in vitro mammalian cell gene mutation assay. In sister chromatid exchange and in chromosomal aberration studies, C9-C14 aliphatic, <2% aromatic hydrocarbons fluids did not produce an effect. C9-C14 aliphatic, <2% aromatic hydrocarbons fluids were also non-mutagenic when tested in an in vivo mouse bone marrow micronucleus assay and when tested in dominant lethal studies utilizing an inhalation route of exposure. All studies were conducted in a manner similar or equivalent to currently established OECD guidelines. C9-C14 aliphatic, <2% aromatic hydrocarbons fluids are a non-genotoxic agent and classification is not warranted under the Regulation (EC) 1272/2008 on classification, labeling and packaging of substances and mixtures (CLP) or under the Directive 67/518/EEC for dangerous substances and Directive 1999/45/EC for preparations. Toxicity to reproduction. In a study examining the effects of commercial hexane the NOAEC for both male and female rats (adults and offspring) was 3000 ppm (10560 mg/m3). The LOAEC for these groups was 9000 ppm based on reduced body weight. There were no adverse effects to reproduction, therefore the NOAEC for reproduction is 9000 ppm (31680 mg/m3). A study to examine the developmental toxicity of commercial hexane in mice, found the maternal NOAEC was 900 ppm, and the maternal LOAEC was 3000 ppm (10560 mg/m3) based on colour changes in the lungs. The developmental NOAEC was 3000 ppm and the LOAEC was 9000 ppm(31680 mg/m3) in mice. C9-C14 aliphatic, <2% aromatic hydrocarbon fluids are not developmental toxicants. In two developmental studies (OECD TG 414), pregnant dams were dosed by inhalation with 0, 300, or 900 ppm C9-C14 aliphatic, <2% aromatic hydrocarbon fluids during gestational days 6 through 15. No adverse maternal or fetal effects were noted at any dose level. Thus, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids did not produce any maternal or fetal toxicity or any developmental effects in rats. Based on the study results, the maternal and developmental toxicity NOAEC is >= 900 ppm (5220 mg/m3). Based on this study and the lack of systemic toxicity, C9-C14 aliphatic, <2% aromatic hydrocarbon fluids, are not expected to be developmental toxicants.
XYLENE	Reproductive effector in rats
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:**

Continued...

	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/m3 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/m3 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/m3 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/m3. 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, utheral 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 phenylgloxylic acids; whereas rats and rabbits excrete hippuric acid and phenaceturic 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 syncitial alteration, hypertrophy 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. 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

Continued...

	studies indicate that the reproductive organs are not a target for ethylbenzene toxicity Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination. **NOTE:** 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. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
BENZENE	Inhalation (man) TCLo: 150 ppm/1y - I 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. 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. 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. **WARNING:** This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**.
Western Canadian Select (WCS) & PETROLEUM CRUDE OIL	The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives; The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since: · The adverse effects of these materials are associated with undesirable components, and · The levels of the undesirable components are inversely related to the degree of processing; · Distillate base oils receiving the same degree or extent of processing will have similar toxicities; · The potential toxicity of *residual base oils* is independent of the degree of processing the oil receives. · The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing. The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential. Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size. Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil s mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method). Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 “in vivo” tests on 7 CASs from the OLBO class(Other Lubricant Base Oils). Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class(Other Lubricant Base Oils)) Germ cell mutagenicity: The tests performed within the ‘in vivo” studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class(Other Lubricant Base Oils)). Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction. STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m3 and for systemic effects NOAEL > 980 mg/m3. Sub-chronic toxicity 90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies). Repeat dose toxicity: Oral NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally. Inhalation

Continued...

The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m3. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m3.
Dermal
In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals. Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.
Toxicity to reproduction:
Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.
Developmental toxicity, teratogenicity:
Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.
The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic
Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.
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 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
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.
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.
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).
Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as

Continued...

	lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. 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. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.
Western Canadian Select (WCS) & PETROLEUM CRUDE OIL & N-NONANE	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 that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.
PETROLEUM CRUDE OIL & POLYCYCLIC AROMATIC HYDROCARBONS	No significant acute toxicological data identified in literature search.
PETROLEUM CRUDE OIL & N-HEXANE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
PETROLEUM CRUDE OIL & XYLENE & TOLUENE & BENZENE	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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
PETROLEUM CRUDE OIL & XYLENE	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.
XYLENE & ETHYLBENZENE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

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

SECTION 12 Ecological information

Toxicity

Western Canadian Select (WCS)

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

sulfur

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	>100mg/l	2
LC50	96h	Fish	>207mg/L	4

heptane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.64mg/l	2

Continued...

	NOEC(ECx)	504h	Crustacea	0.17mg/l	2
	LC50	96h	Fish	3446.8mg/L	4

n-hexane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	240h	Algae or other aquatic plants	25.023-137.802mg/L	4

n-octane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.3mg/l	2
NOEC(ECx)	504h	Crustacea	0.17mg/l	2

n-pentane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	1.26mg/l	2
EC50	48h	Crustacea	2.7mg/l	2
EC50(ECx)	8h	Algae or other aquatic plants	1mg/l	1
LC50	96h	Fish	4.26mg/l	2

n-nonane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.2mg/l	2
NOEC(ECx)	504h	Crustacea	0.17mg/l	2

xylene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	4.6mg/l	2
EC50	48h	Crustacea	1.8mg/l	2
NOEC(ECx)	73h	Algae or other aquatic plants	0.44mg/l	2
LC50	96h	Fish	2.6mg/l	2

toluene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	3.78mg/L	5
NOEC(ECx)	168h	Crustacea	0.74mg/L	5
LC50	96h	Fish	5-35mg/l	4
EC50	96h	Algae or other aquatic plants	>376.71mg/L	4

ethylbenzene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	4.6mg/l	1
EC50	48h	Crustacea	1.37-4.4mg/l	4
NOEC(ECx)	720h	Fish	0.381mg/L	4
LC50	96h	Fish	3.381-4.075mg/L	4
EC50	96h	Algae or other aquatic plants	3.6mg/l	2

benzene

Endpoint	Test Duration (hr)	Species	Value	Source
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

polycyclic aromatic hydrocarbons

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	192h	Fish	6.3mg/L	4

hydrogen sulfide

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	3960h	Fish	<0.001mg/L	5
EC50	48h	Crustacea	0.12mg/l	2
LC50	96h	Fish	075>0.4mg/l	Not Available

Continued...

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

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 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. Anthrcene 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 petroleum distillates:
Environmental fate:
When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.
As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.
Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus,when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants . The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes. The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.
Biodegradation:
Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:
(1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
(2) isoalkanes;
(3) alkenes;
(4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
(5) monoaromatics;
(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
(7) higher molecular weight cycloalkanes (which may degrade very slowly.
Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble,volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.
When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile this is referred to as residual NAPL . Above the retention capacity, the NAPL becomes mobile and will move within the soil
Bioaccumulation:
Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances. Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5
In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.
Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs
These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,
one study suggests that some alkyl-PAHs may biomagnify.While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.
In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not

Continued...

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/.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,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. Spilt 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 spilt 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/m2/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 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 n-heptane:
log Kow : 4.66
Koc : 2400-8100
Half-life (hr) air : 52.8

Continued...

Half-life (hr) H2O surface water : 2.9-312
Henry's atm m3 /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 7.15x10-12 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-m3 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[Koc]) and the estimated log sorption coefficient (i.e. log[Koc]) *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 Koc). While its estimated Koc 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 contaminant 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): Oncorhyncus 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:

Continued...

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 (V4+) under reducing conditions and the vanadate ion (V5+) 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 VO2+ and VO(OH)+, and the vanadate species are H2VO4 - and HVO4. 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 exp5 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 Astralagus 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 l%-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 (NaVO3, NH4VO3, Na3VO4, V2O5 and ammonium polyvandate).
Reliable short-term toxicity data for freshwater organisms are available for three trophic levels: aquatic invertebrates, fish and algae.
Fish LC50 (96 h): Leuciscus idus 0.7 mg V/l (V2O5 flake)
Algae EC50 (72 h): Scenedesmus subspicatus 2.9 mg V/l (V2O5)
Mysid shrimp LC50 (48 h): 13.3 mg V/l
Flagship larvae NOEC (30 d): 76 ug V/l (V2O5)
Reliable long-term toxicity data are also available for the effect of V2O5 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 Koc : 2.05-3.08; Koc : 25.4-204; Half-life (hr) air : 0.24-42; Half-life (hr) H2O surface water : 24-672; Half-life (hr) H2O ground : 336-8640; Half-life (hr) soil : 52-672; Henry's Pa m3 /mol : 637-879; Henry's atm m3 /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-methylbenzylnitrate, 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

Continued...

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 EC50 (24 h): 0.0084 mg/L. and Gammarus lacustris LC50 (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 hydrogen sulfide:
Environmental fate:
Since hydrogen sulfide exists as a gas at atmospheric pressure, partitioning to the air is likely to occur after environmental releases. However, the compound is also soluble in oil and water, and therefore, may partition as well to surface water, groundwater, or moist soil. In addition, sorption of hydrogen sulfide from air onto soil and plant foliage occurs. Hydrogen sulfide's solubility in pure water varies with temperature from 5.3 g/L at 10 °C to 3.2 g/L at 30 C. Once hydrogen sulfide is dissolved in water, it will dissociate into bisulfide ion (HS-) and sulfide ion (S2-); the ratio of the concentrations of these various ions will depend on the pH of the solution. Hydrogen sulfide can also form insoluble sulfide salts with various metals (i.e., copper, zinc, nickel, and iron) that may be present in soils or environmental waters .
Hydrogen sulfide evaporates easily from water, and the rate of evaporation depends on factors such as temperature, humidity, pKa, pH, and the concentration of certain metal ions. Hydrogen sulfide will cross the air-water interface with kinetics similar to other unreactive gases, such as oxygen (O2), nitrogen (N2), and carbon dioxide (CO2), at pHs <=6. At higher pHs, such as seawater, which has a pH of 8 or greater, hydrogen sulfide escape is enhanced due to an ionic species gradient in the water close to the surface. Complexation of bisulfide and sulfide ions to trace metal ions (i.e., Zn2+, Co2+, and Ni2+) found in seawater will also have an effect on the transport of hydrogen sulfide across the air-water interface.
Clay or organic matter may sorb hydrogen sulfide. Under natural conditions, it is likely that some of the hydrogen sulfide would be oxidized to sulfate, which may be removed by leaching or taken up by plants. This, in turn, may make gas sorption sites available for additional sorption. Several species of soil, aquatic, and marine microorganisms oxidize hydrogen sulfide to elemental sulfur, and its half-time in these environments usually ranges from 1 hour to several hours .Food chain bioconcentration and biomagnification are unlikely.
In the atmosphere, hydrogen sulfide may be oxidized by oxygen (O2) and ozone (O3) to give sulfur dioxide (SO2), and ultimately sulfate compounds. Sulfur dioxide and sulfates are eventually removed from the atmosphere through absorption by plants, deposition on and sorption by soils, or through precipitation. A residence time of approximately 1.7 days at an ozone concentration of 0.05 mg/m3 has been calculated for hydrogen sulfide. The effective life-times for hydrogen sulfide based on summer daytime and yearly average hydroxyl radical concentrations have been estimated to be 0.23 and 2.3 days, respectively, based a measured rate constant of 4.8x10-12 cm3/molecule second. Life-times in air ranging from approximately 1 day in the summer to 42 days in the winter have been estimated for hydrogen sulfide. Hydrogen sulfide is not expected to be decomposed by direct absorption of ultraviolet radiation and the reaction with ozone is not expected to be a significant environmental fate.
In aqueous solution, hydrogen sulfide is a weak acid, exhibiting two acid dissociation constants. The first dissociation yields bisulfide ion (HS–), and the second yields sulfide ion (S2–), with pKa values for each of these dissociations of 7.04 and 11.96, respectively. At a pH of 7.0, the ratio of the concentration of aqueous hydrogen sulfide to bisulfate ion is approximately 1-to-1. As the pH increases above 7.0, the ratio of the concentration of bisulfide ion to aqueous hydrogen sulfide increases. At a pH of 8, the ratio of the concentration of bisulfide ion to the concentration of aqueous hydrogen sulfide is approximately 10-to-1. The relative concentration of sulfide ion does not begin to increase until a pH of 11 is exceeded; only above pH 12 will the concentration of sulfide ion become significant (>50%). Hydrogen sulfide oxidation by O2 readily occurs in surface waters. At 25 °C and pH 8, half-times of 50 and 26 hours were reported for hydrogen sulfide in water and seawater, respectively. Above pH 8, however, the rate of oxidation was independent of pH.
Hydrogen sulfide in waste water may be controlled by addition of oxidizing chemicals, which react to form less toxic byproducts. In warm, damp environments (such as manholes and gravity sewers), hydrogen sulfide may be oxidized by autotrophic bacteria to sulfuric acid . Chemical oxidation of hydrogen sulfide dissolved in sewage water produces sulfur at pH 6–7, while sulfur, polysulfides, thiosulfates, and ultimately sulfate are formed at pHs of 7–9.
Hydrogen sulfide is one of the principal components in the natural sulfur cycle. Bacteria, fungi, and actinomycetes (a fungus-like bacteria) release hydrogen sulfide during the decomposition of sulfur containing proteins and by the direct reduction of sulfate (SO42-). Hydrogen sulfide is also consumed by bacteria found in soil and water that oxidize hydrogen sulfide to elemental sulfur. Photosynthetic bacteria can oxidize hydrogen sulfide to sulfur and sulfate in the presence of light and the absence of oxygen.
A number of microorganisms have been found to degrade hydrogen sulfide to elemental sulfur or sulfate. Among these are a heterotrophic bacterium of the genus *Xanthomonas* isolated from dimethyl disulfide-acclimated peat, heterotrophic fungi, and a marine isopod. Soils may sorb considerable amounts of hydrogen sulfide from the air, retaining most of it in the form of elemental sulfur. Manganese compound found in these soils appeared to catalyze the oxidation of hydrogen sulfide to elemental sulfur
Ecotoxicity:
Fish LC50 (96 h): 075->0.4 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).

Continued...

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
sulfur	LOW	LOW
heptane	LOW	LOW
n-hexane	LOW	LOW
n-octane	LOW	LOW
n-pentane	LOW	LOW

Continued...

Ingredient	Persistence: Water/Soil	Persistence: Air
n-nonane	LOW	LOW
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
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)
hydrogen sulfide	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
sulfur	LOW (LogKOW = 0.229)
heptane	HIGH (LogKOW = 4.66)
n-hexane	MEDIUM (LogKOW = 3.9)
n-octane	HIGH (LogKOW = 5.18)
n-pentane	LOW (BCF = 2.35)
n-nonane	HIGH (LogKOW = 4.7613)
xylene	MEDIUM (BCF = 740)
toluene	LOW (BCF = 90)
ethylbenzene	LOW (BCF = 79.43)
benzene	HIGH (BCF = 4360)
hydrogen sulfide	LOW (LogKOW = 0.229)

Mobility in soil

Ingredient	Mobility
sulfur	LOW (KOC = 14.3)
heptane	LOW (KOC = 274.7)
n-hexane	LOW (KOC = 149)
n-octane	LOW (KOC = 506.7)
n-pentane	LOW (KOC = 80.77)
n-nonane	LOW (KOC = 934.6)
toluene	LOW (KOC = 268)
ethylbenzene	LOW (KOC = 517.8)
benzene	LOW (KOC = 165.5)
hydrogen sulfide	LOW (KOC = 14.3)

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. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

Continued...

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	NO
HAZCHEM	3WE

Land transport (ADG)

UN number	1267
UN proper shipping name	PETROLEUM CRUDE OIL
Transport hazard class(es)	Class: 3 Subrisk: Not Applicable
Packing group	I
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: 357 Limited quantity: 500 ml

Air transport (ICAO-IATA / DGR)

UN number	1267
UN proper shipping name	Petroleum crude oil
Transport hazard class(es)	ICAO/IATA Class: 3 ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Packing group	I
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A3 A177 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)

UN number	1267
UN proper shipping name	PETROLEUM CRUDE OIL
Transport hazard class(es)	IMDG Class: 3 IMDG Subrisk: Not Applicable
Packing group	I
Environmental hazard	Not Applicable
Special precautions for user	EMS Number: F-E, S-E Special provisions: 357 Limited Quantities: 500 mL

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

Continued...

Not Applicable

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

Product name	Group
petroleum crude oil	Not Available
sulfur	Not Available
heptane	Not Available
n-hexane	Not Available
n-octane	Not Available
n-pentane	Not Available
n-nonane	Not Available
xylene	Not Available
toluene	Not Available
ethylbenzene	Not Available
benzene	Not Available
polycyclic aromatic hydrocarbons	Not Available
hydrogen sulfide	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
petroleum crude oil	Not Available
sulfur	Not Available
heptane	Not Available
n-hexane	Not Available
n-octane	Not Available
n-pentane	Not Available
n-nonane	Not Available
xylene	Not Available
toluene	Not Available
ethylbenzene	Not Available
benzene	Not Available
polycyclic aromatic hydrocarbons	Not Available
hydrogen sulfide	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

sulfur is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

heptane is found on the following regulatory lists

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

n-hexane is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

n-octane is found on the following regulatory lists

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

n-pentane 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 4
Australian Inventory of Industrial Chemicals (AIIC)

n-nonane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

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

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

polycyclic aromatic hydrocarbons is found on the following regulatory lists

Australia Model Work Health and Safety Regulations - Hazardous chemicals (other than lead) requiring health monitoring
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

hydrogen sulfide is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7	

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (polycyclic aromatic hydrocarbons)
Canada - DSL	No (polycyclic aromatic hydrocarbons)
Canada - NDSL	No (petroleum crude oil; sulfur; heptane; n-hexane; n-octane; n-pentane; n-nonane; xylene; toluene; ethylbenzene; benzene; polycyclic aromatic hydrocarbons; hydrogen sulfide)
China - IECSC	No (petroleum crude oil; polycyclic aromatic hydrocarbons)
Europe - EINEC / ELINCS / NLP	No (polycyclic aromatic hydrocarbons)
Japan - ENCS	No (sulfur; polycyclic aromatic hydrocarbons)
Korea - KECI	No (polycyclic aromatic hydrocarbons)
New Zealand - NZIoC	No (polycyclic aromatic hydrocarbons)
Philippines - PICCS	No (polycyclic aromatic hydrocarbons)
USA - TSCA	No (polycyclic aromatic hydrocarbons)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	No (polycyclic aromatic hydrocarbons)
Russia - FBEPH	No (polycyclic aromatic hydrocarbons)
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	24/02/2020
Initial Date	06/05/2022

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
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List

Continued...

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