suncor energy Suncor Bhb

suncor energy (Petro-Canada)

Chemwatch Hazard Alert Code: **4**

Chemwatch: **977-1797**
Version No: **2.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **23/02/2022**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	suncor energy Suncor Bhb
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	Refinery feedstock.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**suncor energy (Petro-Canada)**
Address	PO Box 2844, Petro-Canada Centre Calgary Alberta T2P 3E3 Canada
Telephone	+1 403 296 8000
Fax	+1 403 296 3030
Website	http://www.suncor.com/
Email	media@suncor.com

Emergency telephone number

Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE**
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 3 9573 3188

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

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Flammable Liquids Category 1, Acute Toxicity (Oral) Category 4, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Germ Cell Mutagenicity Category 1B, Carcinogenicity Category 1A, Reproductive Toxicity Category 1B, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard 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**

Hazard statement(s)

H224	Extremely flammable liquid and vapour.
H302	Harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H340	May cause genetic defects.
H350	May cause cancer.
H360Fd	May damage fertility. Suspected of damaging the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe mist/vapours/spray.
P271	Use only a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P240	Ground and bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use non-sparking tools.
P243	Take action to prevent static discharges.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P331	Do NOT induce vomiting.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
128683-24-9	40.32-53.77	bitumen (oil sands)
128683-33-0	0-26.88	naphtha (oil sand), hydrotreated
78-78-4	6.72-13.44	isopentane
142-82-5	3.36-6.72	n-heptane
124-18-5	3.36-6.72	n-decane
111-84-2	3.36-6.72	n-nonane
111-65-9	3.36-6.72	n-octane
110-54-3	3.36-6.72	n-hexane
109-66-0	3.36-6.72	n-pentane
75-28-5.	0.67-3.36	iso-butane
1330-20-7	0.67-3.36	xylene
106-97-8.	1.34-3.36	butane
7704-34-9.	0-2.35	sulfur
71-43-2	0.33-1	benzene
108-88-3	0.33-1	toluene
25551-13-7	0.06-0.67	trimethylbenzene (mixed isomers)
106-99-0	0.06-0.67	1,3-butadiene
100-41-4	0.06-0.67	ethylbenzene

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: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	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. ▸ Immediately drench burn area in cold running water. ▸ If hot bitumen adheres to the skin, **DO NOT** attempt to remove it (it acts as a sterile dressing). ▸ For burns to the head and neck and trunk, apply cold wet towels to the burn area, and change frequently to maintain cooling. ▸ Cooling should be maintained for no longer than thirty minutes. ▸ When hot bitumen completely encircles a limb, it may have a tourniquet effect and should be split as it cools. ▸ Transport to hospital or doctor. In case of burns: ▸ Immediately apply cold water to burn either by immersion or wrapping with saturated clean cloth. ▸ **DO NOT** remove or cut away clothing over burnt areas. **DO NOT** pull away clothing which has adhered to the skin as this can cause further injury. ▸ **DO NOT** break blister or remove solidified material.

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	▸ Quickly cover wound with dressing or clean cloth to help prevent infection and to ease pain. ▸ For large burns, sheets, towels or pillow slips are ideal; leave holes for eyes, nose and mouth. ▸ **DO NOT** apply ointments, oils, butter, etc. to a burn under any circumstances. ▸ Water may be given in small quantities if the person is conscious. ▸ Alcohol is not to be given under any circumstances. ▸ Reassure. ▸ Treat for shock by keeping the person warm and in a lying position. ▸ Seek medical aid and advise medical personnel in advance of the cause and extent of the injury and the estimated time of arrival of the patient.
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	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice. ▸ Avoid giving milk or oils. ▸ Avoid giving alcohol. ▸ 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.

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Burns : No attempt should be made to remove the bitumen (it acts as a sterile dressing). Cover the bitumen with tulle gras and leave for two days when any detached bitumen can be removed. Re-dress and leave for a further week. If necessary refer to a burns unit. [Manufacturer]

- 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

- Do NOT direct a solid stream of water or foam into burning molten material; this may cause spattering and spread the fire.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

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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) nitrogen oxides (NOx) sulfur oxides (SOx) sulfur dioxide (SO_2) other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions. May emit clouds of acrid smoke **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
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

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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.

Contains low boiling substance:
Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.

- Check for bulging containers.
- Vent periodically
- Always release caps or seals slowly to ensure slow dissipation of vapours
- **DO NOT** allow clothing wet with material to stay in contact with skin

· Electrostatic discharge may be generated during pumping - this may result in fire.
· Ensure electrical continuity by bonding and grounding (earthing) all equipment.
· Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec).
· Avoid splash filling.
· Do NOT use compressed air for filling discharging or handling operations.
· Wait 2 minutes after tank filling (for tanks such as those on
· road tanker vehicles) before opening hatches or manholes.
· Wait 30 minutes after tank filling (for large storage tanks)
· before opening hatches or manholes. Even with proper
· grounding and bonding, this material can still accumulate an
· electrostatic charge. If sufficient charge is allowed to
· accumulate, electrostatic discharge and ignition of flammable

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	· air-vapour mixtures can occur. Be aware of handling · operations that may give rise to additional hazards that result · from the accumulation of static charges. These include but are · not limited to pumping (especially turbulent flow), mixing, · filtering, splash filling, cleaning and filling of tanks and · containers, sampling, switch loading, gauging, vacuum truck · operations, and mechanical movements. These activities may · lead to static discharge e.g. spark formation. Restrict line · velocity during pumping in order to avoid generation of · electrostatic discharge (= 1 m/s until fill pipe submerged to · twice its diameter, then = 7 m/s). Avoid splash filling. · Do NOT use compressed air for filling, discharging, or handling operations ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ Avoid smoking, naked lights, heat or ignition sources. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Vapour may ignite on pumping or pouring due to static electricity. ▸ **DO NOT** use plastic buckets. ▸ Earth and secure metal containers when dispensing or pouring product. ▸ Use spark-free tools when handling. ▸ Avoid contact with incompatible materials. ▸ Keep containers securely sealed. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ 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)

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	▸ 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	Low molecular weight alkanes: ▸ May react violently with strong oxidisers, chlorine, chlorine dioxide, dioxygenyl tetrafluoroborate. ▸ May react with oxidising materials, nickel carbonyl in the presence of oxygen, heat. ▸ Are incompatible with nitronium tetrafluoroborate(1-), halogens and interhalogens ▸ may generate electrostatic charges, due to low conductivity, on flow or agitation. ▸ Avoid flame and ignition sources Redox reactions of alkanes, in particular with oxygen and the halogens, are possible as the carbon atoms are in a strongly reduced condition. Reaction with oxygen (if present in sufficient quantity to satisfy the reaction stoichiometry) leads to combustion without any smoke, producing carbon dioxide and water. Free radical halogenation reactions occur with halogens, leading to the production of haloalkanes. In addition, alkanes have been shown to interact with, and bind to, certain transition metal complexes Interaction between chlorine and ethane over activated carbon at 350 deg C has caused explosions, but added carbon dioxide reduces the risk. The violent interaction of liquid chlorine injected into ethane at 80 deg C/10 bar becomes very violent if ethylene is also present A mixture prepared at -196 deg C with either methane or ethane exploded when the temp was raised to -78 deg C. Addition of nickel carbonyl to an n-butane-oxygen mixture causes an explosion at 20-40 deg C. Alkanes will react with steam in the presence of a nickel catalyst to give hydrogen. 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. 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 ▸ 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 Butane/ isobutane ▸ reacts violently with strong oxidisers ▸ reacts with acetylene, halogens and nitrous oxides ▸ is incompatible with chlorine dioxide, conc. nitric acid and some plastics ▸ may generate electrostatic charges, due to low conductivity, in flow or when agitated - these may ignite the vapour. Segregate from nickel carbonyl in the presence of oxygen, heat (20-40 C) · **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.. n-Pentane ▸ reacts violently with strong oxidisers ▸ attacks some plastics, rubber and coatings

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- may generate static charges o flow or agitation, due to low conductivity
- 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.

38wbit
n-Octane/ iso-octane:

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

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	bitumen (oil sands)	Bitumen fumes	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	n-heptane	Heptane (n-Heptane)	400 ppm / 1640 mg/m3	2050 mg/m3 / 500 ppm	Not Available	Not Available
Australia Exposure Standards	n-nonane	Nonane	200 ppm / 1050 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	n-octane	Octane	300 ppm / 1400 mg/m3	1750 mg/m3 / 375 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-pentane	Pentane	600 ppm / 1770 mg/m3	2210 mg/m3 / 750 ppm	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	butane	Butane	800 ppm / 1900 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	benzene	Benzene	1 ppm / 3.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	toluene	Toluene	50 ppm / 191 mg/m3	574 mg/m3 / 150 ppm	Not Available	Not Available
Australia Exposure Standards	trimethylbenzene (mixed isomers)	Trimethyl benzene	25 ppm / 123 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	1,3-butadiene	1,3-Butadiene	10 ppm / 22 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ethylbenzene	Ethyl benzene	100 ppm / 434 mg/m3	543 mg/m3 / 125 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
bitumen (oil sands)	30 mg/m3	330 mg/m3	2,000 mg/m3
isopentane	3000* ppm	33000*** ppm	200000*** ppm
n-heptane	500 ppm	830 ppm	5000* ppm
n-decane	6.6 ppm	73 ppm	440 ppm
n-nonane	600 ppm	830 ppm	5,000 ppm
n-octane	230 ppm	385 ppm	5000** ppm
n-hexane	260 ppm	Not Available	Not Available
n-pentane	3000* ppm	33000*** ppm	200000*** ppm
iso-butane	5500* ppm	17000** ppm	53000*** ppm
xylene	Not Available	Not Available	Not Available
butane	Not Available	Not Available	Not Available

Continued...

Ingredient	TEEL-1	TEEL-2	TEEL-3
benzene	Not Available	Not Available	Not Available
toluene	Not Available	Not Available	Not Available
1,3-butadiene	Not Available	Not Available	Not Available
ethylbenzene	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
bitumen (oil sands)	Not Available	Not Available
naphtha (oil sand), hydrotreated	Not Available	Not Available
isopentane	Not Available	Not Available
n-heptane	750 ppm	Not Available
n-decane	Not Available	Not Available
n-nonane	Not Available	Not Available
n-octane	1,000 ppm	Not Available
n-hexane	1,100 ppm	Not Available
n-pentane	1,500 ppm	Not Available
iso-butane	Not Available	Not Available
xylene	900 ppm	Not Available
butane	Not Available	1,600 ppm
sulfur	Not Available	Not Available
benzene	500 ppm	Not Available
toluene	500 ppm	Not Available
trimethylbenzene (mixed isomers)	Not Available	Not Available
1,3-butadiene	2,000 ppm	2,000 ppm
ethylbenzene	800 ppm	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
naphtha (oil sand), hydrotreated	E	≤ 0.1 ppm
sulfur	E	≤ 0.01 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

NOTE D: Certain substances which are susceptible to spontaneous polymerisation or decomposition are generally placed on the market in a stabilised form. It is in this form that they are listed on Annex I
When they are placed on the market in a non-stabilised form, the label must state the name of the substance followed by the words "non-stabilised"
European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP
NOTE E: Substances with specific effects on human health that are classified as carcinogenic, mutagenic and/ or toxic for reproduction in categories 1 or 2 are ascribed Note E if they are classified as very toxic (T+), toxic (T) or harmful (Xn). For these substances the risk phrases R20 ,R21, R22, R23, R24,R25, R26, R27, R28, R39, R68, R48 and R65 and all combinations of these risk phrases shall be 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

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	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 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. For molten materials: Provide mechanical ventilation; in general such ventilation should be provided at compounding/ converting areas and at fabricating/ filling work stations where the material is heated. Local exhaust ventilation should be used over and in the vicinity of machinery involved in handling the molten material. Keep dry!! Processing temperatures may be well above boiling point of water, so wet or damp material may cause a serious steam explosion if used in unvented equipment.
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 **NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for

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	long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
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:
suncor energy Suncor Bhb

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C

Respiratory protection

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

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

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

^ - Full-face

Continued...

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	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON	C
VITON/CHLOROBUTYL	C
VITON/NEOPRENE	C

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

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

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

For molten materials:
76a-p()

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Highly flammable liquid; does not mix with water.

Physical state	Liquid	**Relative density (Water = 1)**	0.915-0.940
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	291
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	63.5
Initial boiling point and boiling range (°C)	< -0.5	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	<-35	**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
Vapour pressure (kPa)	40-50	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (Not Available%)**	Not Applicable
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7

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Chemical stability	▸ Extremely high temperatures. ▸ 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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation hazard is increased at higher temperatures. No health effects were seen in humans exposed at 1,000 ppm isobutane for up to 8 hours or 500 ppm for 8 hours/day for 10 days. Isobutane can have anaesthetic and asphyxiant effects at high concentrations, well above the lower explosion limit of 1.8% (18,000 ppm). Butane is a simple asphyxiant and is mildly anaesthetic at high concentrations (20-25%). 10000 ppm for 10 minutes causes drowsiness. Narcotic effects may be accompanied by exhilaration, dizziness, headache, nausea, confusion, incoordination and unconsciousness in severe cases The paraffin gases C1-4 are practically nontoxic below the lower flammability limit, 18,000 to 50,000 ppm; above this, low to moderate incidental effects such as CNS depression and irritation occur, but are completely reversible upon cessation of the exposure. Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. 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. A significant number of individuals exposed to mixed trimethylbenzenes complained of nervousness, tension, anxiety and asthmatic bronchitis. Peripheral blood showed a tendency to hypochromic anaemia and a deviation from normal in coagulability of the blood. Hydrocarbon concentrations ranged from 10 to 60 ppm. Contamination of the mixture with benzene may have been responsible for the blood dyscrasias. High concentrations of mesitylene vapour (5000 to 9000 ppm) caused central nervous system depression in mice. Similar exposures of pseudocumene also produced evidence of CNS involvement. 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. Exposure to toxic levels of butadiene has also produced chromosome damage. Human volunteers exposed at 2000-8000 ppm 1,3-butadiene for 6-8 hours showed slight smarting of the eyes, difficulty in focusing on instrument scales and a transient objection to butadiene odour. Characteristics of exposure include dry nose/mouth/throat, fatigue, headache, vertigo, nausea, narcosis, respiratory paralysis, and central nervous system depression. Very high concentrations may cause loss of consciousness or death. Repeated and prolonged exposure to 1,3-butadiene vapour may cause kidney and liver damage. Deep anaesthesia was induced in rabbits in 8 to 10 minutes at 200000 to 250000 ppm. Recovery from brief periods of anaesthesia occurred within two minutes of terminating the exposure.

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Inhalation, by humans, of 1000 ppm heptane for 6 minutes was associated with slight dizziness; inhalation of higher concentrations for shorter periods, resulted in marked vertigo, incoordination, and hilarity. Signs of central nervous system system (CNS) involvement occurred in the absence of noticeable mucous membrane irritation and were noticed promptly on entering such atmospheres.
Concentrations of 10,000-15,000 ppm, heptane produced narcosis on mice within 30-50 minutes. Exposure at higher concentrations (15,000-20,000 ppm) for 30-60 minutes caused convulsions and death in mice; inhalation of 48,000 ppm produced respiratory arrest in three of four head-exposed mice within 3 minutes. Brief exposure (4 minutes) to high levels (5000 ppm) produced nausea, loss of appetite and a "gasoline-taste" that persisted for several hours post-exposure.
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.
Some aliphatic hydrocarbons produce axonal neuropathies. Isoparaffinic hydrocarbons produce injury to the kidneys of male rats. When albino rats were exposed to isoparaffins at 21.4 mg/l for 4 hours, all animals experienced weakness, tremors, salivation, mild to moderate convulsions, chromodacryorrhoea and ataxia within the first 24 hours. Symptoms disappeared after 24 hours.
Several studies have evaluated sensory irritation in laboratory animals or odor or sensory response in humans. When evaluated by a standard procedure to assess upper airway irritation, isoparaffins did not produce sensory irritation in mice exposed to up to 400 ppm isoparaffin in air. Human volunteers were exposed for six hours to 100 ppm isoparaffin. The subjects were given a self-administered questionnaire to evaluate symptoms, which included dryness of the mucous membranes, loss of appetite, nausea, vomiting, diarrhea, fatigue, headache, dizziness, feeling of inebriation, visual disturbances, tremor, muscular weakness, impairment of coordination or paresthesia. No symptoms associated with solvent exposure were observed. With a human expert panel, odour from liquid imaging copier emissions became weakly discernible at approximately 50 ppm.
Numerous long-term exposures have been conducted in animals with only one major finding observed. Renal tubular damage has been found in kidneys of male rats upon repeated exposures to isoparaffins. It does not occur in mice or in female rats. This male rat nephropathy has been observed with a number of hydrocarbons, including wholly vaporized unleaded gasoline. The phenomenon has been attributed to reversible binding of hydrocarbon to alpha2-globulin. Since humans do not synthesize alpha2-globulin or a similar protein, the finding is not considered to be of biological significance to man. No clinically significant renal abnormalities have been found in refinery workers exposed to hydrocarbons.
When evaluated for developmental toxicity in rats, isoparaffins were neither embryotoxic nor teratogenic. Isoparaffins were consistently negative on standard bacterial genotoxicity assays. They were also non-genotoxic in *in vivo* mammalian testing for somatic or germ cell mutations (mouse micronucleus test and rat dominant lethal assay, respectively).
Mullin et al: Jnl Applied Toxicology 10, pp 136-142, 2006
Symptoms of pentane inhalation exposure may include, hyperactivity, anaesthesia and a persistent taste of gasoline. Light anaesthesia occurs in mice after 10 minutes exposure to 70000 ppm n-pentane.
Inhalation of high vapour concentrations may result in coughing, headache, mild depression, incoordination, blurred vision, confusion, loss of appetite, nausea, vomiting, irregular heartbeat and unconsciousness.
Concentrations of 270 to 400 mg/l isopentane may cause narcosis and cardiac sensitisation .

Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.
The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.
Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.

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.
Acute exposure to bitumen/ asphalt vapours may cause coughing, chest tightness, headache, muscle weakness, dizziness, tiredness, poor coordination, and even nausea and vomiting.
Workers exposed to hot blown bitumens show bronchitis, rhinitis, oropharyngitis and laryngitis; symptoms include cough, phlegm, burning of the throat and chest, hoarseness, headache and nasal discharge. Guinea pigs, rabbits and mice exposed to blown

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	bitumen fumes, aerosols and smoke, developed patchy regions of emphysema, bronchiolar dilation, pneumonitis, and severe localised bronchitis. Mice, exposed to aerosols of petroleum bitumens and smoke from heated petroleum bitumens, showed congestion, acute bronchitis, pneumonitis, bronchial dilation, abscess formation, epithelial atrophy, and necrosis. In health studies in the workplace, environmental measurement showed concentrations of asphalt, ranging from "non-detectable", where there was good mechanical ventilation, to 40 mg/m3, where there was very poor natural draft. Breathing zone samples, collected during drum-filling operations, ranged from 1.0 (upwind) to 5 mg/m3 (downwind) as means of 4-hour exposures. In the opinion of industrial hygienists conducting these studies, work conditions were satisfactory where asphalt fumes were kept below 10 mg/m3 Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
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). Many aliphatic hydrocarbons create a burning sensation because they are irritating to the GI mucosa. Vomiting has been reported in up to one third of all hydrocarbon exposures. While most aliphatic hydrocarbons have little GI absorption, aspiration frequently occurs, either initially or in a semi-delayed fashion as the patient coughs or vomits, thereby resulting in pulmonary effects. Once aspirated, the hydrocarbons can create a severe pneumonitis. Rats given isoparaffinic hydrocarbons - isoalkanes- (after 18-24 hours fasting) showed lethargy and/or general weakness, ataxia and diarrhoea. Symptoms disappeared within 24-28 hours. Swallowing pieces of bitumen may produce pyloric obstruction due to accumulation in the stomach and the formation of a stony concretion. Ingestion of pentanes may result in diarrhoea, haemorrhage of the mucous membranes, or when the liquid vapourises in the trachea, asphyxiation leading to brain damage or death. Ingestion may also cause nausea, vomiting and abdominal swelling. Large doses (1 ml/kg) may cause central nervous system depression, ventricular fibrillation and kidney, liver and bone marrow damage. 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. Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Dermally, isoparaffins have produced slight to moderate irritation in animals and humans under occluded patch conditions where evaporation cannot freely occur. However, they are not irritating in non-occluded tests, which are a more realistic simulation of human exposure. They have not been found to be sensitisers in guinea pig or human patch testing. However, occasional rare idiosyncratic sensitisation reactions in humans have been reported. 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 . The diepoxide of butadiene (1,2:3,4-diepoxybutane), a probable metabolite, has been reported to be a mild skin tumourigen when applied topically to the skin of mice Symptoms of pentane exposure may include drying, cracking, itching, blistering, redness, pigmentation, swelling, burning and pain. Because pentane boils just below body temperature, absorption is not expected to be a significant route of entry. Toluene by comparison is absorbed through the skin at 20 times the rate of n-pentane Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Instillation of isoparaffins into rabbit eyes produces only slight irritation. Workers exposed to fumes of blown bitumens developed keratoconjunctivitis.

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	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 Eye-contact with the liquid pentanes may result in inflammation of the iris and mucous membranes resulting in pain and lachrymation. Eye contact with liquid or very high vapour concentrations may result in drying, redness, swelling and pain. 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.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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 of heritable genetic damage, generally on the basis of - appropriate animal studies, - other relevant information Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. Implantation studies in rats show that paraffin oils may be tumourigen. As a general rule the highly refined paraffins contain a lower level of suspect polyaromatic hydrocarbons than less refined grades and also less than waxes derived from naphthenic base-stocks. 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 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

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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.
Amongst humans occupationally exposed to 1,3-butadiene several cancer sites with high statistically significant mortality ratios were identified.
These included cancer of the testes, cancers of the digestive system (oesophagus, stomach, large intestine), larynx and Hodgkin's disease.
Exposure by rats to 1,3-butadiene gas at 1000 ppm/6hrs/day, 5 days /week (105 weeks for females and 111 weeks for males) caused significant increases in the incidence of tumours at various sites; mammary gland adenomas and sarcomas; uterine sarcomas; Zymbal gland carcinomas; thyroid adenomas and pancreatic adenomas. A high incidence of malignant lymphoma was found amongst a group of exposed rats in a second study
Chronic exposure to bitumen/ asphalt fumes, over extended periods, may cause central nervous system depression, and liver and kidney changes. Chronic bitumen/ asphalt poisoning may result in a decrease in the number of white and red blood cells. [*ILO Encyclopedia*]
Prolonged contact with bitumens may produce irritation, inflammation, dermatitis, acne-like lesions, keratoses, melanosis and photosensitisation.
Animal inhalation studies do NOT yield sufficient evidence of bitumen/ asphalt-induced lung cancer. It is generally accepted that oxidation of polycyclic aromatic hydrocarbons (PAHs) destroys their carcinogenic potential and the differing character of the polycyclic aromatic fraction of petroleum asphalt fume and those of coal tar pitch volatiles suggested a lessened potential for carcinogenicity.
Inhalation of fumes of heated bitumens by guinea pigs and rats produced chronic fibrosing pneumonitis with peribronchial adenomatosis; rats developed squamous cell metaplasias.
Various extracts of steam-refined and air-refined bitumens and their mixtures, undiluted steam-refined bitumens and cracking residue bitumens, produced skin tumours following application to mouse skin. Subcutaneous injection in mice and rats, of steam- and air- reined bitumens, produced sarcomas at the sites of injection. Application of air-refined bitumens, in toluene, to the skin of mice, produced skin tumours. No tumours were produced by the undiluted bitumen. A pooled mixture of steam- and air-blown petroleum bitumen in benzene, produced tumours at the site of application to mouse skin.
No significant difference was found in the health of asphalt workers and of groups of controls in a study conducted in 25 oil refineries. Other studies have not demonstrated health defects in paving and roofing operations (using asphalt-based products) and interstate trucking over asphalt highways.
NOTE: The term bitumen and asphalt are often used interchangeably and have been used to describe products derived from petroleum and/ or coal. Asphalt is a native mixture of hydrocarbons which occurs as an amorphous, brownish-black solid or semisolid and results from the evaporation of the lighter hydrocarbons from petroleum and partial oxidation of the residue. Petroleum asphalts (bitumens) should therefore be differentiated from coal pitch bitumens which result from the destructive distillation of coal.
The term "asphalt" originally applied to "Trinidad asphalt" which is a mined solid and is closely related to gilsonite.
On occasion there are reports of epidemiological studies which have found an increased cancer mortality in workers exposed to heated bitumens and bitumen fumes. There are reports of significantly increased incidence of cancers of the mouth, oesophagus, rectum and lung. The bitumens, used by this cohort, are likely to have their origin in coal and should be distinguished from materials derived from the petroleum industry (the asphalts).
Hardened bitumens/ asphalts do not normally constitute a health hazard. Mined sources of bitumens/ asphalts may present an additional hazard related to their naturally occurring content of quartz. Chronic inhalation of high levels of quartz dusts may produce silicosis, a disabling form of pneumoconiosis which may lead to scarring of the lining of the air-sacs of the lung.

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 exposure to pentanes may result in chemical pneumonitis, pulmonary oedema or peripheral neuropathy. Prolonged or repeated inhalation may cause dizziness, weakness, weight loss, anaemia, nervousness, pain in the limbs and peripheral

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numbness ("pins and needles")

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 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)

suncor energy Suncor Bhb	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

bitumen (oil sands)	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; >5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]

naphtha (oil sand), hydrotreated	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

isopentane	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >25.3 mg/l4h[1]	Not Available
	Oral (Rat) LD50; >2000 mg/kg[1]	

n-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-decane	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Inhalation(Rat) LC50; >5.266 mg/L4h[1]	
	Oral (Rat) LD50; >5000 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]

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	Oral (Rat) LD50; >5000 mg/kg[1]	
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-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-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]	
iso-butane	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >13023 ppm4h[1]	Not Available
xylene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1700 mg/kg[2]	Eye (human): 200 ppm irritant
	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]
butane	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; 658 mg/l4h[2]	Not Available
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]
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]
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]

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trimethylbenzene (mixed isomers)	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 8970 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h-moderate
1,3-butadiene	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; 128826.955 ppm4h[2]	Not Available
	Oral (Rat) LD50; 5480 mg/kg[2]	
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]
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*	

BITUMEN (OIL SANDS)	* Syncrude SDS A black or dark brown highly viscous liquid. Heated bitumen can cause severe skin burns and eye injury. Eye, skin, gastrointestinal, and respiratory tract irritation can occur. Poisonous hydrogen sulfide gas may accumulate in confined spaces. 55rad
NAPHTHA (OIL SAND), HYDROTREATED	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

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	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 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 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.
N-DECANE	No significant acute toxicological data identified in literature search.
N-OCTANE	Oral (rat) LD50: 5630 mg/kg* [CCINFO] Nil reported
N-PENTANE	[GENIUM and CCINFO, V.W.&R.]
XYLENE	Reproductive effector in rats 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.
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

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	excretion of metabolites is the dominant route of excretion.
TOLUENE	For toluene: **Acute Toxicity** Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies. **Humans -** Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy. Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days. Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death Toluene can also strip the skin of lipids causing dermatitis **Animals -** The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days **Subchronic/Chronic Effects:** Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm. **Humans -** Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin. Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia. Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L **Animals -** The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) . **Developmental/Reproductive Toxicity** Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals. **Humans -** Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy **Animals** - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/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.
TRIMETHYLBENZENE (MIXED ISOMERS)	For trimethylbenzenes: Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion . After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of

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	approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates . The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid . The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates. **Acute Toxicity** Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis . High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness . The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg) . Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels . No effects were reported for rats exposed to a mixture of trimethyl- benzenes at 1700 ppm for 10 to 21 days **Neurotoxicity** 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested. **Subchronic/Chronic Toxicity** Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia . **Genotoxicity:** Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella tymphimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation. **Developmental/Reproductive Toxicity:** A three-generation reproductive study on the C9 fraction was conducted CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established Developmental toxicity, including possible develop- mental neurotoxicity, was evident in rats in a 3-generation reproductive study No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation NOTE: This data is for mixed isomers of unstated proportions.
1,3-BUTADIENE	Hallucinations, distorted perceptions, visual field changes, conjunctive irritation, cough, general anaesthesia, respiratory depression, papilliary dilation, tremors, muscle weakness For 1,3-butadiene: Metabolism of 1,3-butadiene appears to be qualitatively similar across species, although there are quantitative differences in the amounts of putatively toxic metabolites formed; mice appear to oxidise 1,3-butadiene to the monoepoxide, and subsequently the diepoxide, metabolite to a greater extent than do rats or humans. However, there may also be inter-individual variation in metabolic capability for 1,3-butadiene in humans, related to genetic polymorphism for relevant enzymes. 1,3-Butadiene is of low acute toxicity in experimental animals. However, long-term exposure to 1,3- butadiene was associated with the development of ovarian atrophy at all concentrations tested in mice. Other effects in the ovaries have also been observed in shorter-term studies. Atrophy of the testes was also observed in male mice at concentrations greater than those associated with effects in females. Based on limited available data, there is no conclusive evidence that 1,3-butadiene is teratogenic in experimental animals following maternal or paternal exposure or that it induces significant fetal toxicity at concentrations below those that are maternally toxic. 1,3-Butadiene also induced a variety of effects on the blood and bone marrow of mice; although data are limited, similar effects have not been observed in rats. Inhaled 1,3-butadiene is a potent carcinogen in mice, inducing tumours at multiple sites at all concentrations tested in all identified studies. 1,3-Butadiene was also carcinogenic in rats at all exposure levels in the only relevant study available; although only much higher concentrations were tested in rats than in mice, rats appear to be the less sensitive species, based on comparison of tumour incidence data. The greater sensitivity in mice than in rats to induction of these effects by 1,3- butadiene is likely related to species differences in metabolism to the active epoxide metabolites. 1,3-Butadiene is mutagenic in somatic cells of both mice and rats, although the mutagenic potency was greater in mice than in rats. Similarly, 1,3-butadiene induced other genetic damage in somatic cells of mice, but not in those of rats. 1,3-Butadiene was also consistently genotoxic in germ cells of mice, but not in the single assay in rats identified. However, there were no apparent differences in species sensitivity to genetic effects induced by epoxide metabolites of 1,3-butadiene. There is also limited evidence from occupationally exposed populations that 1,3-butadiene is genotoxic in humans, inducing mutagenic and clastogenic damage in somatic cells.

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	An association between exposure to 1,3-butadiene in the occupational environment and leukaemia fulfils several of the traditional criteria for causality. In the largest and most comprehensive study conducted to date, involving a cohort of workers from multiple plants, mortality due to leukaemia increased with estimated cumulative exposure to 1,3-butadiene in the styrene- butadiene rubber industry; this association remained after controlling for exposure to styrene and benzene and was strongest in those subgroups with highest potential exposure. Similarly, an association between exposure to 1,3-butadiene and leukaemia was observed in an independently conducted case-control study of largely the same population of workers. However, there was no increase in mortality due to leukaemia in butadiene monomer production workers who were not concomitantly exposed to some of the other substances present in the styrene-butadiene rubber industry, although there was some limited evidence of an association with mortality due to lymphosarcoma and reticulosarcoma in some subgroups. The available epidemiological and toxicological data provide evidence that 1,3-butadiene is carcinogenic in humans and may also be genotoxic in humans. The carcinogenic potency (the concentration associated with a 1% increase in mortality due to leukaemia) was deter mined to be 1.7 mg/m3, based on the results of the largest well conducted epidemiological investigation in exposed workers. This value is similar to the lower end of the range of tumourigenic concentrations determined on the basis of studies in rodents. 1,3-Butadiene also induced reproductive toxicity in experimental animals. As a measure of its potency to induce reproductive effects, a benchmark concentration of 0.57 mg/m3 was derived for ovarian toxicity in mice. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
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 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.
BITUMEN (OIL SANDS) & ETHYLBENZENE	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
NAPHTHA (OIL SAND), HYDROTREATED & N-DECANE & 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.
N-DECANE & N-NONANE	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

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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:
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

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	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.
N-NONANE & TRIMETHYLBENZENE (MIXED ISOMERS)	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
N-HEXANE & TRIMETHYLBENZENE (MIXED ISOMERS)	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
XYLENE & ETHYLBENZENE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
XYLENE & BENZENE & TOLUENE & TRIMETHYLBENZENE (MIXED ISOMERS)	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.
BENZENE & 1,3-BUTADIENE	**WARNING:** This substance has been classified by the IARC as Group 1: **CARCINOGENIC TO HUMANS**.

Acute Toxicity	✔	**Carcinogenicity**	✔
Skin Irritation/Corrosion	✔	**Reproductivity**	✔
Serious Eye Damage/Irritation	✔	**STOT - Single Exposure**	✔

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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

suncor energy Suncor Bhb

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

bitumen (oil sands)

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

naphtha (oil sand), hydrotreated

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

isopentane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	72h	Algae or other aquatic plants	1.26mg/l	2
EC50	72h	Algae or other aquatic plants	1.26mg/l	2
EC50	48h	Crustacea	2.3mg/l	1
LC50	96h	Fish	4.26mg/l	2
EC50	96h	Algae or other aquatic plants	5.2mg/l	2

n-heptane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.64mg/l	2
NOEC(ECx)	504h	Crustacea	0.17mg/l	2
LC50	96h	Fish	3446.8mg/L	4

n-decane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>0.002mg/l	2
EC50(ECx)	48h	Crustacea	>0.002mg/l	2
LC50	96h	Fish	>365mg/L	4
EC50	96h	Algae or other aquatic plants	89mg/l	4

n-nonane

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

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-hexane

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

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

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iso-butane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
LC50	96h	Fish	24.11mg/l	2
EC50	96h	Algae or other aquatic plants	7.71mg/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

butane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
LC50	96h	Fish	24.11mg/l	2
EC50	96h	Algae or other aquatic plants	7.71mg/l	2

sulfur

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

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

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

trimethylbenzene (mixed isomers)

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

1,3-butadiene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	33mg/l	2
EC50(ECx)	96h	Algae or other aquatic plants	11mg/l	2
EC50	48h	Crustacea	33mg/l	2
LC50	96h	Fish	43mg/l	2
EC50	96h	Algae or other aquatic plants	11mg/l	2

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

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*

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

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Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the
oxygen transfer between the air and the water
Oils of any kind can cause:

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

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.
For 1,2,4 - Trimethylbenzene:
Half-life (hr) air: 0.48-16;
Half-life (hr) H2O surface water: 0.24 -672;
Half-life (hr) H2O ground: 336-1344;
Half-life (hr) soil: 168-672;
Henry's Pa m3 /mol: 385 -627;
Bioaccumulation: not significant. 1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance.
Atmospheric Fate: 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs. Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals. Reaction also occurs with ozone but very slowly (half life 8820 days).
Aquatic Fate: 1,2,4-Trimethylbenzene volatilizes rapidly from surface waters with volatilization half-life from a model river calculated to be 3.4 hours. Biodegradation of 1,2,4-trimethylbenzene has been noted in both seawater and ground water. Various strains of Pseudomonas can biodegrade 1,2,4-trimethylbenzene.
Terrestrial Fate: 1,2,4-Trimethylbenzene also volatilizes from soils however; moderate adsorption to soils and sediments may occur. Volatilization is the major route of removal of 1,2,4- trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.
Ecotoxicity: No significant bioaccumulation has been noted. 1,2,4-Trimethylbenzene is moderately toxic to fathead minnow and slightly toxic to dungeness crab. 1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms. No stress was observed in rainbow trout, sea lamprey and Daphnia magna water fleas. The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.
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 C9 aromatics (typically trimethylbenzene - TMBs)
Chemicals in this category possess properties indicating a hazard for the environment (acute toxicity for fish, invertebrates, and algae from 1 to 10 mg/L). Category members are readily biodegradable, except 1,3,5-trimethylbenzene (CAS RN 108-67-8). Category members are not expected to be bioaccumulative.
Environmental Fate:
In the air, category member constituents have the potential to rapidly degrade through indirect photolytic processes mediated primarily by hydroxyl radicals with calculated degradation half-lives ranging from 0.54 to 2.81 days (based on a 12-hour day and a hydroxyl radical concentration of 5x10+5). Aqueous photolysis and hydrolysis will not contribute to the transformation of category chemical constituents in aquatic environments because they are either poorly reactive or not susceptible to these reactions.
Results of the Mackay Level I environmental distribution model show that chemical constituents of C9 Aromatic Hydrocarbon Solvents Category members have the potential to partition to air (96.8 to 98.9 %), with a negligible amount partitioning to water (0.2 to 0.6%) and soil (0.9 to 2.7%). In comparison, Level III modeling indicates that category members partition primarily to soil (66.3 to 79.6%) and water (17.8 to 25.0%) compartments rather than air (2.4 to 8.4%) when an equal emission rate (1000 kg/hr) is assumed to each of the air, water, and soil compartments. When release (1000 kg/hr) is modeled only to either the air, water, or soil compartment, constituents are indicated in the modeling to partition primarily (>94%) to the compartment to which they are emitted as advection and degradation influence constituent concentration in compartments to which constituents are not released. Solvent naphtha, (pet.), light aromatic (CAS RN 64742-95-6), 1,2,4-trimethylbenzene (CAS RN 95-63-6), and 1-ethyl-3-methylbenzene (CAS RN 620-14-4) were determined to be readily biodegradable based on the studies that used the TG OECD 301F (the latter substance is used to characterize the potential biodegradability of the category member, ethylmethylbenzene (CAS RN 25550-14-5)). These three substances exceed 60%
biodegradation in 28 days and met the 10-day window criterion for ready biodegradation. In comparison 1,3,5-trimethylbenzene (CAS RN 108-67-8) was not readily biodegradable. It achieved 42% biodegradation after 28 days and 60% biodegradation after 39 days. The result for the multi-constituent substance (CAS RN 64742-95-6), a UVCB, characterizes the biodegradability of that substance as a whole, but it does not suggest that each constituent is equally biodegradable. As with all ready biodegradation test guidelines, the test system and study design used with these substances (OECD TG 301F) is not capable of distinguishing the relative contribution of the substances' constituents to the total biodegradation measured.
Based on Henry's Law constants (HLCs) representing a potential to volatilize from water that range from 590 to 1000 Pa-m3/mole, the potential to volatilize from surface waters for chemicals in the C9 Aromatic Hydrocarbon Solvents Category is expected to be high.
Based on the measured bioconcentration factors that range from 23 to 342 for 1,2,4-trimethylbenzene and 1,3,5-trimethylbenzene, the category members are not expected to be bioaccumulative.
Ecotoxicity
Acute toxicity values used to characterize this category for fish (LL50; LC50) and invertebrates (EL50; EC50) range from 3.5 to 9.2 mg/L, based on measured data. For algae, one study for a category member (CAS RN 64742-95-6) resulted in a 72-hr EC50 of 2.4 mg/L (biomass) and 2.7 mg/L (growth rate) based on

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measured concentrations.
The algal 72-hour NOEC (no observed effect concentration) for biomass and growth rate is 1.3 mg/L, based on mean measured concentrations. A 21-day Daphnia magna reproduction study with 1,3,5-trimethylbenzene (CAS RN 108-67-8) resulted in a NOEC value of 0.4 mg/L, based on a minimum measured value.
for lubricating oil base stocks:
Vapor Pressure Vapor pressures of lubricating base oils are reported to be negligible. In one study, the experimentally measured vapour pressure of a solvent-dewaxed heavy paraffinic distillate base oil was 1.7 x 10exp-4 Pa . Since base oils are mixtures of C15 to C50 paraffinic, naphthenic, and aromatic hydrocarbon isomers, representative components of those structures were selected to calculate a range of vapor pressures. The estimated vapor pressure values for these selected components of base oils ranged from 4.5 x 10exp-1 Pa to 2 x 10exp-13Pa. Based on Dalton's Law the expected total vapour pressure for base oils would fall well below minimum levels (10exp-5 Pa) of recommended experimental procedures.
Partition Coefficient (log Kow): In mixtures such as the base oils, the percent distribution of the hydrocarbon groups (i.e., paraffins, naphthenes, and aromatics) and the carbon chain lengths determines in-part the partitioning characteristics of the mixture. Generally, hydrocarbon chains with fewer carbon atoms tend to have lower partition coefficients than those with higher carbon numbers .However, due to their complex composition, unequivocal determination of the log Kow of these hydrocarbon mixtures cannot be made. Rather, partition coefficients of selected C15 chain-length hydrocarbon structures representing paraffinic, naphthenic, and aromatic constituents in base oil lubricants were modelled . Results showed typical log Kow values from 4.9 to 7.7, which were consistent with values of >4 for lubricating oil basestocks
Water Solubility: When released to water, base oils will float and spread at a rate that is viscosity dependent. While water solubility of base oils is typically very low, individual hydrocarbons exhibit a wide range of solubility depending on molecular weight and degree of unsaturation. Decreasing molecular weight (i.e., carbon number) and increasing levels of unsaturation increases the water solubility of these materials. As noted for partition coefficient, the water solubility of lubricating base oils cannot be determined due to their complex mixture characteristics. Therefore, the water solubility of individual C15 hydrocarbons representing the different groups making up base oils (i.e., linear and branched paraffins, naphthenes, and aromatics) was modelled. Based on water solubility modelling of those groups, aqueous solubilities are typically much less than 1 ppm. (0.003-0.63 mg/l)
Environmental Fate:
Photodegradation: Chemicals having potential to photolyse have UV/visible absorption maxima in the range of 290 to 800 nm. Some chemicals have absorption maxima significantly below 290 nm and consequently cannot undergo direct photolysis in sunlight (e.g. chemicals such as alkanes, alkenes, alkynes, saturated alcohols, and saturated acids). Most hydrocarbon constituents of the materials in this category are not expected to photolyse since they do not show absorbance within the 290-800 nm range. However, photodegradation of polyaromatic hydrocarbons (PAHs) can occur and may be a significant degradation pathway for these constituents of lubricating base oils. The degree and rate at which PAHs may photodegrade depend upon whether conditions allow penetration of light with sufficient energy to effect a change. For example,polycyclic aromatic compounds (PAC) compounds bound to sediments may persist due to a lack of sufficient light penetration
Atmospheric gas-phase reactions can occur between organic chemicals and reactive molecules such as photochemically produced hydroxyl radicals, ozone and nitrogen oxides. Atmospheric oxidation as a result of radical attack is not direct photochemical degradation, but indirect degradation. In general, lubricating base oils have low vapour pressures and volatilisation is not expected to be a significant removal mechanism for the majority of the hydrocarbon components. However, some components (e.g., C15 branched paraffins and naphthenes) appear to have the potential to volatilise Atmospheric half-lives of 0.10 to 0.66 days have been calculated for representative C15 hydrocarbon components of lubricating base oils
Stability in Water: Chemicals that have a potential to hydrolyze include alkyl halides, amides, carbamates, carboxylic acid esters and lactones, epoxides, phosphate esters, and sulfonic acid esters. Because lubricating base oils do not contain significant levels of these functional groups, materials in the lubricating base oils category are not subject to hydrolysis
Chemical Transport and Distribution in the Environment : Based on the physical-chemical characteristics of component hydrocarbons in lubricating base oils, the lower molecular weight components are expected to have the highest vapour pressures and water solubilities, and the lowest partition coefficients. These factors enhance the potential for widespread distribution in the environment. To gain an understanding of the potential transport and distribution of lubricating base oil components, the EQC (Equilibrium Criterion) model was used to characterize the environmental distribution of different C15 compounds representing different structures found in lube oils (e.g., paraffins, naphthenes, and aromatics). The modelling found partitioning to soil or air is the ultimate fate of these C15 compounds. Aromatic compounds partition principally to soil. Linear paraffins partition mostly to soil, while branching appears to allow greater distribution to air. Naphthenes distribute to both soil and air, with increasing proportions in soil for components with the greater number of ring structures. Because the modelling does not take into account degradation factors, levels modelled in the atmosphere are likely overstated in light of the tendency for indirect photodegradation to occur.
Biodegradation: The extent of biodegradation measured for a particular lubricating oil basestock is dependent not only on the procedure used but also on how the sample is presented in the biodegradation test. Lubricant base oils typically are not readily biodegradable in standard 28-day tests. However, since the oils consist primarily of hydrocarbons that are ultimately assimilated by microorganisms, and therefore inherently biodegradable. Twenty-eight biodegradability studies have been reported for a variety of lubricating base oils. Based on the results of ultimate biodegradability tests using modified Sturm and manometric respirometry testing the base oils are expected to be, for the most part, inherently biodegradable. Biodegradation rates found using the modified Sturm procedure ranged from 1.5 to 29%. Results from the manometric respirometry tests on similar materials showed biodegradation rates from 31 to 50%. Biodegradation rates measured in 21-day CEC tests for similar materials ranged from 13 to 79%.
Ecotoxicity:
Numerous acute studies covering fish, invertebrates, and algae have been conducted to assess the ecotoxicity of various lubricating base oils. None of these studies have shown evidence of acute toxicity to aquatic organisms. Eight, 7-day exposure studies using rainbow trout failed to demonstrate toxicity when tested up to the maximum concentration of 1000 mg/L applied as dispersions. Three, 96-hour tests with rainbow trout also failed to show any toxic effects when tested up to 1000 mg/L applied as dispersions. Similarly, three 96-hour tests with fathead minnows at a maximum test concentration of 100 mg/L water accommodated fractions (WAF) showed no adverse effects. Two species of aquatic invertebrates (Daphnia magna and Gammarus sp.) were exposed to WAF solutions up to 10,000 mg/L for 48 and 96-hours, respectively, with no adverse effects being observed. Four-day exposures of the freshwater green alga (Scenedesmus subspicatus) to 500 mg/L WAF solutions failed to show adverse effects on growth rate and algal cell densities in four studies
Multiple chronic ecotoxicity studies have shown no adverse effects to daphnid survival or reproduction. In 10 of 11 chronic studies, daphnids were exposed for 21 days to WAF preparations of lubricating base oils with no ill effects on survival or reproduction at the maximum concentration of 1000 mg/L. One test detected a reduction in reproduction at 1000 mg/L. Additional data support findings of no chronic toxicity to aquatic invertebrates and fish. No observed effect levels ranged from 550 to 5,000 mg/L when tested as either dispersions or WAFs.
The data described above are supported by studies on a homologous series of alkanes. The author concluded that the water solubility of carbon chains .C10 is too limited to elicit acute toxicity. This also was shown for alkylbenzene compounds having carbon numbers .C15. Since base oils consist of carbon compounds of C15 to C50, component hydrocarbons that are of acute toxicological concern are, for the most part, absent in these materials. Similarly, due to their low solubility, the alkylated two to three ring polyaromatic components in base oils are not expected to cause acute or chronic toxicity. This lack of toxicity is borne out in the

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results of the reported studies.
The effects of crude and refined oils on organisms found in fresh and sea water ha been extensively reviewed.
sea water. Where spillages occur the non-mobile species suffer the greatest mortality, whereas fish species can often escape from the affected region. The extent of the initial mortality depends on the chemical nature of the oil, the location, and the physical conditions, particularly the temperature and wind velocity. Most affected freshwater and marine communities recover from the effects of an oil spill within a year. The occurrence of biogenic hydrocarbons in the world's oceans is well recorded. They have the characteristic isoprenoid structure, and measurements made in water columns indicate a background concentration of 1.0 to 10 ul/l. The higher molecular weight materials are dispersed as particles, with the highest concentrations of about 20 ul/l occurring in the top 3 mm layer of water.
A wide variation in the response of organisms to oil exposures has been noted. The larvae of fish and crustaceans appear to be most susceptible to the water-soluble fraction of crude oil. Exposures of plankton and algae have indicated that certain species of diatoms and green algae are inhibited, whereas microflagellates are not.
For the most part, molluscs and most intertidal worm species appear to be tolerant of oil contamination.
For petroleum distillates:
Environmental fate:
When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption. These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.
As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.
Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus,when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants . The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes. The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.
Biodegradation:
Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:
(1) n-alkanes, especially in the C10–C25 range, which are degraded readily;
(2) isoalkanes;
(3) alkenes;
(4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);
(5) monoaromatics;
(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and
(7) higher molecular weight cycloalkanes (which may degrade very slowly.
Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble,volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.
When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile this is referred to as residual NAPL . Above the retention capacity, the NAPL becomes mobile and will move within the soil
Bioaccumulation:
Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances. Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5
In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.
Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs
These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,
one study suggests that some alkyl-PAHs may biomagnify.While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.
In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.
Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.
This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal
Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish
Ecotoxicity:
Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and Daphnia magna, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids

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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.
When released in the environment, alkanes don't undergo rapid biodegradation, because they have no functional groups (like hydroxyl or carbonyl) that are needed by most organisms in order to metabolize the compound.
However, some bacteria can metabolise some alkanes (especially those linear and short), by oxidizing the terminal carbon atom. The product is an alcohol, that could be next oxidised to an aldehyde, and finally to a carboxylic acid. The resulting fatty acid could be metabolised through the fatty acid degradation pathway.
For n-heptane:
log Kow : 4.66
Koc : 2400-8100
Half-life (hr) air : 52.8
Half-life (hr) 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,

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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 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.

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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 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 butadiene:
Kow: 1.99
Koc : 72-228
Half-life (hr) air : 4.9
Henry's Pa m3 /mol: 2.57
Henry's atm m3 /mol: 7.24E-02
BCF : 19.1
Environmental fate:
The high volatility of this compound suggests that it will partition predominantly to the atmospheric compartment, where it is not expected to be adsorbed to particulate matter to any significant extent.
Terrestrial Fate: If spilled on land, 1,3-butadiene will predominately volatilise very rapidly due to its very low boiling point. Dissolved in water, it may leach through soil into ground water due to its high water solubility and low estimated soil adsorption coefficient. It will not appreciably hydrolyse but may be subject to biodegradation based on screening tests.
1,3-Butadiene is expected to volatilize rapidly from either moist or dry soil to the atmosphere. This follows from the estimated lack of any appreciable adsorption to soil, and consideration of 1,3-butadiene's calculated Henry's law constant for moist soil or its vapor pressure, 2,100 mm Hg at 25 C,for dry soil. Both values suggest a rapid rate of volatilisation from their respective media. The calculated soil adsorption coefficient of 288 suggests that 1,3-butadiene may display moderate mobility in soil. However, the expected rapid rate of volatilisation and the possibility of rapid degradation in soil suggest that there is little potential for 1,3-butadiene to leach into groundwater. Methane-utilizing bacteria isolated from the soil of an oil refinery epoxidised 1,3-butadiene under aerobic conditions
Aquatic Fate: When released into water, 1,3-butadiene will volatilise rapidly with a half-life estimated to be several hours. It will not hydrolyse appreciably, but may be subject to biodegradation, based on screening tests.
Atmospheric Fate: Butadiene is a reactive, electron-rich chemical that is expected to undergo rapid reactions with the electrophilic oxidants typically present in the atmosphere: ozone, photochemically produced hydroxyl radicals, nitrate radicals, and molecular oxygen. Among these, the most rapid reaction in the atmosphere is with photochemically produced hydroxyl radicals. The atmospheric destruction of 1,3-butadiene by photo-initiated processes has been established empirically by early studies There are four gas-phase pathways that can destroy 1,3-butadiene in the atmosphere. Depending on local conditions, any one or all of these reactions may occur. Destruction of atmospheric 1,3-butadiene by the gas-phase reaction with photochemically produced hydroxyl radicals is expected to be the dominant photo-initiated pathway. Destruction by nitrate radicals is expected to be a significant night-time process in urban areas.
Reaction with hydroxyl radicals is the dominant removal mechanism, with an estimated half-life of several hours. Reaction with ozone and nitrate radicals may also contribute to the degradation of the chemical. Polluted urban atmospheres increase the rate of degradation somewhat during daylight hours as suggested by the detection of the highest atmospheric levels of the chemical in the early morning hours. Acetaldehyde and acrolein have been identified as products of photooxidation. Washout may contribute to removal of 1,3-butadiene from the atmosphere; however, evaporation from the rain may be rapid and the compound returned to the atmosphere relatively quickly unless it leaches into the soil.
Biodegradation: No data concerning the biodegradation of 1,3-butadiene in natural systems could be found in the literature. 1,3-Butadiene was listed in a group of chemicals which should be biodegraded by biological sewage treatment, as long as suitable acclimatization is achieved. Screening tests suggest that 1,3-butadiene may be biodegradable in the environment with 1,2-epoxybutene being a potential product .
Soil Adsorption/Mobility: The range of estimated adsorption coefficients for 1,3-butadiene from the soils and sediments is 72-228 based on its octanol/water partition coefficient or its water solubility and would therefore not be expected to appreciably adsorb in soils and sediments.
Volatilization from Water/Soil: Using the Henry's Law constant, the estimated half-life for evaporation of 1,3-butadiene from a river 1 m deep with a 1 m/sec current and a 3 m/sec wind is 3.8 hours . Due to its low boiling point, 1,3-butadiene would be expected to rapidly evaporate from soils.
Ecotoxicity:
Fish LC50 (24 h): 71.5 mg/L
1,3-Butadiene is moderately toxic to aquatic life in the short term and slightly toxic in the long term. There is not enough information to predict additional short or long-term effects of 1,3-butadiene on plants, birds, or other animals. 1,3-Butadiene is not expected to accumulate in fish. Animal studies have reported development effects such as skeletal abnormalities and decreased foetal weights, and reproductive effects, including an increased incidence of shrinkage of the ovaries and testicles. Animal studies have also reported tumours at a variety of sites from inhalation of 1,3-butadiene.
for bitumens/ asphalts:
This family of hydrocarbon is expected to have similar boiling points, vapor pressures, log Kow values (>10), and water solubilities. Limited environmental fate data also support the grouping of bitumens/ asphalts under one category. Bitumen/ asphalts contain complex hydrocarbon mixtures with molecular weights ranging from 500-2000 and carbon numbers predominantly higher than C25, vapor pressures are negligible. The high molecular weights and similar hydrocarbon distributions among the bitumens/ asphalts support the conclusion that the toxicity of this group, in general, is not expected to vary significantly across members.
Environmental fate:
Upon release to the environment, bitumens/ asphalts are expected to distribute similarly because of their low volatility and limited water solubility. Bitumen/ asphalts are expected to be resistant to biodegradation, and those components that are soluble in water are expected to be resistant to hydrolysis. When bitumen/ asphalts are heated to facilitate paving or roofing applications, the lighter, more volatile components are distilled into the atmosphere. They condense as they cool, forming small droplets of liquid known as bitumen or asphalt fume condensate. The majority of hydrocarbons in bitumen/ asphalts are not susceptible to direct

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photolysis, since they do not have functional groups that absorb sunlight greater than 290 nm. However, certain aromatic and unsaturated compound members have the potential to undergo photolysis because they absorb light in the environmental UV region. Since bitumens/ asphalts contain high molecular weight hydrocarbons, partitioning to the atmosphere is not considered to be important.
When compositionally analysing bitumens/ asphalts for certain toxicity endpoints the percentage of 3- to 7-ring polyaromatic hydrocarbons (PAHs) is important. The levels of 3- to 7-ring PAHs are expected to be low considering the processes used to manufacture these substances.. Fumes generated experimentally at high temperatures are more likely to contain carcinogenic PAHs than fumes generated at the lower temperatures usually seen in field samples. Therefore, generating conditions are expected to significantly affect toxicity. .

Ecotoxicity:

Bitumens/ asphalts by analogy with other high molecular weight hydrocarbons are not likely to show adverse acute or chronic ecological effects in aquatic species.
For isopentane:

Environmental Fate

Terrestrial fate: An estimated Koc value of 520, determined from a water solubility of 48 mg/L indicates that isopentane is expected to have low mobility in soil. Volatilisation of isopentane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 1.4 atm-cu m/mole, derived from its estimated vapor pressure, 689 mm Hg, and water solubility. Isopentane is expected to volatilise from dry soil surfaces based upon its vapor pressure. Following a 6.1 day lag period, isopentane was completely degraded under aerobic conditions using an activated sludge over the course of a 20 day incubation period.

Aquatic fate: :The estimated Koc value indicates that isopentane is expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected to occur rapidly based upon an estimated Henry's Law constant. Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 52 minutes and 3 days, respectively. An estimated BCF of 70 suggests the potential for bioconcentration in aquatic organisms is moderate. The biodegradation half-life of a mixture containing isopentane, pentane, and cyclopentane in seawater was 2.4 days, suggesting isopentane may biodegrade in water.

Atmospheric fate:: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, isopentane, is expected to exist solely as vapor. Vapour-phase isopentane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 4 days, calculated from its rate constant of 3.9x10-12 cu cm/molec-sec at 25 deg C.

Ecotoxicity:

Daphnia magna EC50 948 h): 2.3 mg/l
for n-decane:
log Kow : 5.98
Koc : 22200-42700
Half-life (hr) air : 33.6
Half-life (hr) H2O surface water : 84-3120
Henry's atm m3 /mol: 5.15
Log BCF : 3.52-4.31

Environmental fate:

When released into the soil, decane may biodegrade to a moderate extent.and is not expected to leach into groundwater; quick evaporate is expected.
When released into water, decane may biodegrade to a moderate extent but is. expected to quickly evaporate. n-Decane has an estimated bioconcentration factor (BCF) of greater than 100 and a. log octanol-water partition coefficient (log Kow) greater than 3.0. This material may bioaccumulate to some extent.
When released into the air, n-decane is expected to be readily degraded by reaction with photochemically produced hydroxyl radicals; photolysis is not expected to be an important fate mechanism.. n-Decane is expected to have a half-life between 1 and 10 days

Ecotoxicity:

Fish LC50 (96 h): 350 mg/l
For n-octane:
Koc : 5500-15600
Half-life (hr) air : 43.44
Half-life (hr) H2O surface water : 3.1-715.2
Henry's atm m3 /mol: 3.21
ThOD : 3.5
BCF : 780-5100
Log BCF : 2.89-3.71

Environmental Fate

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

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

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

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corresponding alkyl nitrate, suggesting nighttime reactions with nitrate radicals may contribute to the atmospheric transformation of n-octane, especially in urban environments.

Ecotoxicity:

Daphnia EC50 (48 h): 0.38 mg/l
Fish LC50: 230 mg/l
Photobacterium phosphoreum EC50 (30 m): 890 mg/l
For n-pentane;
Koc : 580-1600
Half-life (hr) air : 72-108
Half-life (hr) H2O surface water : 2.5-168
Henry's atm m3 /mol: 1.26
Log BCF : 1.9-2.35

Environmental fate:

Photolysis, hydrolysis and bioconcentration of n-pentane are not expected to be important environmental fate processes. Biodegradation of n-pentane may occur in soil and water, however volatilisation and to some extent adsorption are expected to be far more important environmental fate processes. A Koc range of 580-1600 indicates a low mobility class in soil for n-pentane. In aquatic systems n-pentane may partition from the water column to organic matter contained in sediments and suspended materials. The Henry's Law constant suggests rapid volatilisation of n-pentane from environmental waters. The volatilisation half-lives from a model river and model pond (the latter considers the effect of absorption) have been estimated to be 2.5 hrs and 3.5 days, respectively.
The log bioconcentration factor (log BCF) has been estimated to be 1.9-2.35 suggesting that n-pentane will not bioconcentrate in aquatic organisms
n-Pentane 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 (average half-life of 4.1 days). Rate constants for n-pentane were measured to be 4.06x10-12, 5.30x10-12 and 3.51x 10-12 cm3/mol-sec at 26, 27 and 27 deg. C respectively, which correspond to atmospheric half-lives of 3.9, 3.0 and 4.5 days at an atmospheric concentration of 5x10+5 hydroxyl radicals per cm3. Data also suggests that night-time reactions with nitrate radicals may contribute to atmospheric transformation of n-pentane especially in urban environments. Experimental data showed that 12.9% of the n-pentane fraction in a dark chamber reacted with NO3 to form the corresponding alkyl nitrate.
n-Pentane does not absorb UV light in the environmentally significant range >290 nm and probably will not undergo direct photolysis in the atmosphere.

Biodegradation: The theoretical oxygen demand (ThOD) of benzene acclimated activated sludge for n-pentane was 0.3, 3.2 after 6, 24 days.

Ecotoxicity:

Fish LC50 (96 h): Oncorhyncus mykiss 9.87 mg/l; Pimephales promelus 11.59 mg/l; Lepomis macrochirus 9.99 mg/l
Daphnia EC50 (48 h): 9.7 mg/l
isopentane:
Daphnia magna EC50 (48 h): 2.3 mg/l

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

Environmental fate:

Petroleum products released to the environment migrate through soil via two general pathways: (1) as bulk oil flow infiltrating the soil under the forces of gravity and capillary action, and (2) as individual compounds separating from the bulk petroleum mixture and dissolving in air or water. When bulk oil flow occurs, it results in little or no separation of the individual compounds from the product mixture and the infiltration rate is usually fast relative to the dissolution rate. Many compounds that are insoluble and immobile in water are soluble in bulk oil and will migrate along with the bulk oil flow. Factors affecting the rate of bulk oil infiltration include soil moisture content, vegetation, terrain, climate, rate of release (e.g., catastrophic versus slow leakage), soil particle size (e.g., sand versus clay), and oil viscosity (e.g., gasoline versus motor oil).
As bulk oil migrates through the soil column, a small amount of the product mass is retained by soil particles. The bulk product retained by the soil particles is known as "residual saturation".
Depending upon the persistence of the bulk oil, residual saturation can potentially reside in the soil for years. Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater. Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater. When the amount of product released to the environment is small relative to the volume of available soil, all of the product is converted to residual saturation and downward migration of the bulk product usually ceases prior to affecting groundwater resources. Adverse impacts to groundwater may still occur if rain water infiltrates through soil containing residual saturation and initiates the downward migration of individual compounds. When the amount of product released is large relative to the volume of available soil, the downward migration of bulk product ceases as water-saturated pore spaces are encountered. If the density of the bulk product is less than that of water, the product tends to "float" along the interface between the water saturated and unsaturated zones and spread horizontally in a pancake-like layer, usually in the direction of groundwater flow. Almost all motor and heating oils are less dense than water. If the density of the bulk product is greater than that of water, the product will continue to migrate downward through the water table aquifer under the continued influence of gravity. Downward migration ceases when the product is converted to residual saturation or when an impermeable surface is encountered.
As the bulk product migrates through the soil column, individual compounds may separate from the mixture and migrate independently. Chemical transport properties such as volatility, solubility, and sorption potential are often used to evaluate and predict which compounds will likely separate from the mixture. Since petroleum products are complex mixtures of hundreds of compounds, the compounds characterized by relatively high vapor pressures tend to volatilise and enter

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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
isopentane	HIGH	HIGH
n-heptane	LOW	LOW
n-decane	LOW	LOW
n-nonane	LOW	LOW
n-octane	LOW	LOW
n-hexane	LOW	LOW
n-pentane	LOW	LOW
iso-butane	HIGH	HIGH
xylene	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
butane	LOW	LOW
sulfur	LOW	LOW
benzene	HIGH (Half-life = 720 days)	LOW (Half-life = 20.88 days)
toluene	LOW (Half-life = 28 days)	LOW (Half-life = 4.33 days)
1,3-butadiene	LOW (Half-life = 56 days)	LOW (Half-life = 0.33 days)
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)

Bioaccumulative potential

Ingredient	Bioaccumulation

Continued...

Ingredient	Bioaccumulation
isopentane	LOW (LogKOW = 2.7234)
n-heptane	HIGH (LogKOW = 4.66)
n-decane	HIGH (BCF = 3636)
n-nonane	HIGH (LogKOW = 4.7613)
n-octane	HIGH (LogKOW = 5.18)
n-hexane	MEDIUM (LogKOW = 3.9)
n-pentane	LOW (BCF = 2.35)
iso-butane	LOW (BCF = 1.97)
xylene	MEDIUM (BCF = 740)
butane	LOW (LogKOW = 2.89)
sulfur	LOW (LogKOW = 0.229)
benzene	HIGH (BCF = 4360)
toluene	LOW (BCF = 90)
1,3-butadiene	LOW (BCF = 19.1)
ethylbenzene	LOW (BCF = 79.43)

Mobility in soil

Ingredient	Mobility
isopentane	LOW (KOC = 67.7)
n-heptane	LOW (KOC = 274.7)
n-decane	LOW (KOC = 1724)
n-nonane	LOW (KOC = 934.6)
n-octane	LOW (KOC = 506.7)
n-hexane	LOW (KOC = 149)
n-pentane	LOW (KOC = 80.77)
iso-butane	LOW (KOC = 35.04)
butane	LOW (KOC = 43.79)
sulfur	LOW (KOC = 14.3)
benzene	LOW (KOC = 165.5)
toluene	LOW (KOC = 268)
1,3-butadiene	LOW (KOC = 43.79)
ethylbenzene	LOW (KOC = 517.8)

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. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **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.

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	▸ 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.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
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	Environmentally hazardous
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	Environmentally hazardous
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

Continued...

Transport hazard class(es)	IMDG Class	3
	IMDG Subrisk	Not Applicable
Packing group	I	
Environmental hazard	Marine Pollutant	
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

Not Applicable

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

Product name	Group
bitumen (oil sands)	Not Available
naphtha (oil sand), hydrotreated	Not Available
isopentane	Not Available
n-heptane	Not Available
n-decane	Not Available
n-nonane	Not Available
n-octane	Not Available
n-hexane	Not Available
n-pentane	Not Available
iso-butane	Not Available
xylene	Not Available
butane	Not Available
sulfur	Not Available
benzene	Not Available
toluene	Not Available
trimethylbenzene (mixed isomers)	Not Available
1,3-butadiene	Not Available
ethylbenzene	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
bitumen (oil sands)	Not Available
naphtha (oil sand), hydrotreated	Not Available
isopentane	Not Available
n-heptane	Not Available
n-decane	Not Available
n-nonane	Not Available
n-octane	Not Available
n-hexane	Not Available
n-pentane	Not Available
iso-butane	Not Available
xylene	Not Available
butane	Not Available
sulfur	Not Available
benzene	Not Available
toluene	Not Available

Continued...

Product name	Ship Type
trimethylbenzene (mixed isomers)	Not Available
1,3-butadiene	Not Available
ethylbenzene	Not Available

SECTION 15 Regulatory information

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

bitumen (oil sands) is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

naphtha (oil sand), hydrotreated is found on the following regulatory lists

Not Applicable

isopentane is found on the following regulatory lists

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 4

n-heptane is found on the following regulatory lists

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

n-decane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

n-nonane is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

n-octane 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

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

n-pentane is found on the following regulatory lists

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 4

iso-butane is found on the following regulatory lists

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

xylene is found on the following regulatory lists

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 5
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

butane 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

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)

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

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

trimethylbenzene (mixed isomers) is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

1,3-butadiene 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

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

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (naphtha (oil sand), hydrotreated)
Canada - DSL	Yes
Canada - NDSL	No (bitumen (oil sands); naphtha (oil sand), hydrotreated; isopentane; n-heptane; n-decane; n-nonane; n-octane; n-hexane; n-pentane; iso-butane; xylene; butane; sulfur; benzene; toluene; trimethylbenzene (mixed isomers); 1,3-butadiene; ethylbenzene)
China - IECSC	No (naphtha (oil sand), hydrotreated)
Europe - EINEC / ELINCS / NLP	No (naphtha (oil sand), hydrotreated)
Japan - ENCS	No (bitumen (oil sands); naphtha (oil sand), hydrotreated; sulfur)
Korea - KECI	No (naphtha (oil sand), hydrotreated)

Continued...

National Inventory	Status
New Zealand - NZIoC	No (naphtha (oil sand), hydrotreated)
Philippines - PICCS	No (naphtha (oil sand), hydrotreated)
USA - TSCA	No (naphtha (oil sand), hydrotreated)
Taiwan - TCSI	No (naphtha (oil sand), hydrotreated)
Mexico - INSQ	No (naphtha (oil sand), hydrotreated)
Vietnam - NCI	No (naphtha (oil sand), hydrotreated)
Russia - FBEPH	No (naphtha (oil sand), hydrotreated)
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	23/02/2022
Initial Date	02/02/2022

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
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