SAFETY DATA SHEET 1258

IKO STANDARD ASPHALT PRIMER

SECTION 1 – SUBSTANCE IDENTITY AND COMPANY CONTACT INFORMATION

PRODUCT NAME	IKO Standard Asphalt Primer
TRADE NAME	Asphalt Primer
PRODUCT NUMBER	7870013
CHEMICAL FAMILY	Mixture
PRODUCT USE	Membrane primer
MANUFACTURER/SUPPLIER	GH International Sealants ULC 2540 Rena Road Mississauga, ON L4T 3C9 Canada +1-905-677-5522 Website: www.icpgroup.com Email: sds@icpgroup.com
WEBSITE	www.iko.com
EMERGENCY NUMBER	CANUTEC: 1-613-996-6666 (24 hours information only)

SECTION 2 – HAZARD IDENTIFICATION

CLASSIFICATION OF THE SUBSTANCE OR MIXTURE

SIGNAL WORD	DANGER
SYMBOL(S)	
CLASSIFICATION	Carcinogenicity —Category 2. Eye Irritation – Category 2A. Skin Irritation — Category 2. Flammable Liquids — Category 3. Aspiration Hazard – Category 1. Hazardous to the Aquatic Environment Long Term – Category 3. Specific Target Organ Toxicity, Single Exposure – Category 3 (narcotic effects). Specific Target Organ Toxicity, Single Exposure – Category 3 (Respiratory Tract Irritation).
HAZARD STATEMENTS	H226 Flammable liquid and vapour. 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.

H351 Suspected of causing cancer.
H412 Harmful to aquatic life with long lasting effects.

PRECAUTIONARY STATEMENTS

P201 Obtain special instructions before use.
P202 Do not handle until all safety precautions have been read and understood.
P210: Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P233 Keep container tightly closed.
P240 Ground and bond container and receiving equipment.
P241 Use explosion-proof electrical, ventilating, lighting equipment.
P242 Use non-sparking tools.
P243 Take action to prevent static discharge.
P261 Avoid breathing fume/mist/vapours/spray.
P264 Wash thoroughly after handling.
P271 Use only outdoors or in a well-ventilated area.
P273 Avoid release to the environment.
P280 Wear protective gloves/protective clothing/eye protection/face protection.
P301+P310 IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352 IF ON SKIN: Wash with plenty of soap and water.
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 victim to fresh air and keep at rest in a position comfortable for breathing.
P305+P351+P338 IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313 IF exposed or concerned: Get medical advice/attention.
P312 Call a POISON CENTER or doctor/physician if you feel unwell.
P331 Do NOT induce vomiting.
P332+P313 If skin irritation occurs: Get medical advice/attention.
P337+P313 If eye irritation persists: Get medical advice/attention.
P362+P364 Take off contaminated clothing and wash before reuse.
P370+P378: In case of fire: Use dry chemical, alcohol foam, carbon dioxide or water spray to extinguish.
P403+P235 Store in a well-ventilated place. Keep cool.
P405 Store locked up.
P501 Dispose of contents/container to authorized hazardous or special waste collection point in accordance with any local regulation.

NFPA

Health: 2
Flammability: 2
Reactivity: 0

HMIS

No information available.

SECTION 3 – CHEMICAL COMPOSITION AND DATA ON COMPONENTS

HAZARDOUS CHEMICAL NAME	% (w/w)	CAS NUMBER
Asphalt Bitumen (petroleum)	15-40	8052-42-4

naphtha petroleum, light aromatic solvent	15-40	64742-95-6
1,2,4-trimethyl benzene	10-30	95-63-6
Xylene (xylene)	0.1-5	1330-20-7
Cumene	0.1-5	98-82-8
trimethylbenzene (mixed isomers)	10-30	25551-13-7
1,3,5-trimethyl benzene	1-10	108-67-8
1,2,3-trimethyl benzene	0.1-5	526-73-8
naphtha petroleum, heavy, hydrotreated	5-10	64742-48-9

SECTION 4 – FIRST AID

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 spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.
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.

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. 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.
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.
ACUTE AND CHRONIC SYMPTOMS	Refer to section 11, Toxicological Information, for additional information.

Indication of any IMMEDIATE MEDICAL ATTENTION and special treatment needed:

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

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

BP America Product Safety & Toxicology Department
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]

For Acute or Short Term Repeated Exposures To Xylene:
Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg, intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
Pulmonary absorption is rapid with about 60-65% retained at rest.
Primary threat to life from ingestion and/or inhalation, is respiratory failure.
Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 < 50 mm Hg or pCO2 > 50 mm Hg) should be intubated.
Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

BIOLOGICAL EXPOSURE INDEX - BEI
These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
Methylhippu-ric acids in urine	1.5 gm/gm creatinine	End of shift	
	2 mg/min	Last 4 hrs of shift	

SECTION 5 – FIRE-FIGHTING 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.
FIRE FIGHTING	Liquid and vapour are flammable. Moderate fire hazard when exposed to heat or flame. Vapour forms an explosive mixture with air. Moderate explosion hazard when exposed to heat or flame. Vapour may travel a considerable distance to source of ignition. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO).
FLAMMABILITY	Combustible.
FIRE/EXPLOSION HAZARD	Combustion products include: carbon dioxide (CO2) carbon monoxide (CO) nitrogen oxides (NOx) sulfur oxides (SOx) sulfur dioxide (SO2) other pyrolysis products typical of burning organic material. May emit clouds of acrid smoke

PROPERTIES:

FLASH POINT	47°C
FLAMMABLE LIMITS IN AIR	Lower flammability limit (% vol): No information available Upper flammability limit (% vol): No information available
AUTO IGNITION TEMPERATURE	> 200°C
SPECIAL PPE FOR FIRE-FIGHTERS	Firefighters should be equipped with self-contained breathing apparatus and turn-out gear.
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

SECTION 6 – ACCIDENTAL RELEASE MEASURES

PERSONAL PRECAUTIONS, PROTECTIVE MEASURES AND EMERGENCY PROCEDURES:	Refer to section 8, Exposure Control and Personal Protection, for additional information
ENVIRONMENTAL PRECAUTIONS:	Refer to section 12, Ecological Information, for additional information.

SPILL MANAGEMENT: 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	Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. Collect recoverable product into labelled containers for recycling. Neutralise/decontaminate residue (see Section 13 for specific agent). Collect solid residues and seal in labelled drums for disposal. Wash area and prevent runoff into drains. After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. If contamination of drains or waterways occurs, advise emergency services.
	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

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

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 100pS/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. Even with proper grounding and bonding, this material can still accumulate an electrostatic charge. If sufficient charge is allowed to accumulate, electrostatic

	discharge and ignition of flammable air-vapour mixtures can occur. 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. 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. Avoid all personal contact, including inhalation. Wear protective clothing when risk of overexposure occurs. Use in a well-ventilated area. Prevent concentration in hollows and sumps. DO NOT enter confined spaces until atmosphere has been checked. Avoid smoking, naked lights or ignition sources. Avoid generation of static electricity. DO NOT use plastic buckets. Earth all lines and equipment. Use spark-free tools when handling. Avoid contact with incompatible materials. When handling, DO NOT eat, drink or smoke. Keep containers securely sealed when not in use. 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. DO NOT allow clothing wet with material to stay in contact with skin.
OTHER INFORMATION	Store in original containers in approved flammable liquid storage area. Store away from incompatible materials in a cool, dry, well-ventilated area. DO NOT store in pits, depressions, basements or areas where vapours may be trapped. No smoking, naked lights, heat or ignition sources. Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access. Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances. Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems. Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors. Keep adsorbents for leaks and spills readily available. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS. In addition, for tank storages (where appropriate): Store in grounded, properly designed and approved vessels and away from incompatible materials. For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ ice build-up. Storage tanks should be above ground and diked to hold entire contents.

CONDITIONS FOR SAFE STORAGE, INCLUDING ANY INCOMPATIBILITIES:

SUITABLE CONTAINER	Packing as supplied by manufacturer. Plastic containers may only be used if approved for flammable liquid. Check that containers are clearly labelled and free from leaks. For low viscosity materials (i): Drums and jerry cans must be of the non-removable head type. (ii): Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages. In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
STORAGE INCOMPATIBILITY	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. 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.

	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 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 Sulfides are incompatible with acids, diazo and azo compounds, halocarbons, isocyanates, aldehydes, alkali metals, nitrides, hydrides, and other strong reducing agents. Many reactions of sulfides with these materials generate heat and in many cases hydrogen gas. Many sulfide compounds may liberate hydrogen sulfide upon reaction with an acid.

SECTION 8 – EXPOSURE CONTROL AND PERSONAL PROTECTION

CONTROL PARAMETERS

OCCUPATIONAL EXPOSURE LIMITS (OEL)

Ingredient Data:

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Canada - Nova Scotia Occupational Exposure Limits	1,2,4-trimethyl benzene	1,2,4-Trimethyl benzene	25 ppm	Not Available	Not Available	TLV Basis: central nervous system impairment; asthma; hematologic effects
Canada - Northwest Territories Occupational Exposure Limits	1,2,4-trimethyl benzene	Trimethyl benzene (mixed isomer)	25 ppm	30 ppm	Not Available	Not Available
Canada - Yukon Permissible Concentrations for Airborne Contaminant Substances	Xylene (xylene)	Dimethylbenzene, see Xylene - Skin	100 ppm / 435 mg/m3	650 mg/m3 / 150 ppm	Not Available	Not Available
Canada - Nova Scotia Occupational Exposure Limits	Xylene (xylene)	Xylene - Mixed isomers	100 ppm	150 ppm	Not Available	TLV Basis: upper respiratory tract & eye irritation; central nervous system impairment. BEI
Canada - Alberta Occupational Exposure Limits	Xylene (xylene)	Dimethylbenzene (Xylene, o,m & p isomers)	100 ppm / 434 mg/m3	651 mg/m3 / 150 ppm	Not Available	Not Available
Canada - Saskatchewan Occupational Health and Safety Regulations - Contamination Limits	Xylene (xylene)	Xylene (o, m-, p-isomers)	100 ppm	150 ppm	Not Available	Not Available
Canada - Manitoba Occupational Exposure Limits	Xylene (xylene)	Not Available	100 ppm	150 ppm	Not Available	TLV® Basis: URT & eye irr; CNS impair; BEI
Canada - British Columbia Occupational Exposure Limits	Xylene (xylene)	Xylene (o, m & p isomers)	100 ppm	150 ppm	Not Available	Not Available
Canada - Prince Edward Island Occupational Exposure Limits	Xylene (xylene)	Xylene (all isomers)	100 ppm	150 ppm	Not Available	TLV® Basis: URT & eye irr; CNS impair; BEI
Canada - Northwest Territories Occupational Exposure Limits	Xylene (xylene)	Xylene (o, m-, p-isomers)	100 ppm	150 ppm	Not Available	Not Available
Canada - Quebec Permissible Exposure Values for Airborne Contaminants	Xylene (xylene)	Xylene (o-,m-,p-isomers)	100 ppm / 434 mg/m3	651 mg/m3 / 150 ppm	Not Available	Not Available
Canada - Yukon Permissible Concentrations for Airborne Contaminant Substances	cumene	Cumene - Skin	50 ppm / 245 mg/m3	365 mg/m3 / 75 ppm	Not Available	Not Available
Canada - Nova Scotia Occupational Exposure Limits	cumene	Cumene	50 ppm	Not Available	Not Available	TLV Basis: eye, skin & upper respiratory tract irritation; central nervous system impairment
Canada - Alberta Occupational Exposure Limits	cumene	Cumene	50 ppm / 246 mg/m3	Not Available	Not Available	Not Available
Canada - Saskatchewan Occupational Health and Safety Regulations - Contamination Limits	cumene	Cumene	50 ppm	74 ppm	Not Available	Not Available
Canada - Manitoba Occupational Exposure Limits	cumene	Not Available	50 ppm	Not Available	Not Available	TLV® Basis: Eye, skin, & URT irr; CNS impair
Canada - British Columbia Occupational Exposure Limits	cumene	Cumene	25 ppm	75 ppm	Not Available	Not Available
Canada - Prince Edward Island Occupational Exposure Limits	cumene	Cumene	50 ppm	Not Available	Not Available	TLV® Basis: Eye, skin, & URT irr; CNS impair
Canada - Northwest Territories Occupational Exposure Limits	cumene	Cumene	50 ppm	74 ppm	Not Available	Not Available
Canada - Quebec Permissible Exposure Values for Airborne Contaminants	cumene	Cumene	50 ppm / 246 mg/m3	Not Available	Not Available	Not Available
Canada - Yukon Permissible Concentrations for Airborne Contaminant Substances	trimethylbenzene (mixed isomers)	Trimethyl benzene	25 ppm / 120 mg/m3	180 mg/m3 / 35 ppm	Not Available	Not Available

Canada - Nova Scotia Occupational Exposure Limits	trimethylbenzene (mixed isomers)	Trimethyl benzene - Mixed isomers	25 ppm	Not Available	Not Available	TLV Basis: central nervous system impairment; asthma; hematologic effects
Canada - Alberta Occupational Exposure Limits	trimethylbenzene (mixed isomers)	Trimethyl benzene (mixed isomers)	25 ppm / 123 mg/m3	Not Available	Not Available	Not Available
Canada - Saskatchewan Occupational Health and Safety Regulations - Contamination Limits	trimethylbenzene (mixed isomers)	Trimethyl benzene (mixed isomer)	25 ppm	30 ppm	Not Available	Not Available
Canada - Manitoba Occupational Exposure Limits	trimethylbenzene (mixed isomers)	Not Available	25 ppm	Not Available	Not Available	TLV® Basis: CNS impair; asthma; hematologic eff
Canada - British Columbia Occupational Exposure Limits	trimethylbenzene (mixed isomers)	Trimethyl benzene (mixed isomers)	25 ppm	Not Available	Not Available	Not Available
Canada - Prince Edward Island Occupational Exposure Limits	trimethylbenzene (mixed isomers)	Trimethyl benzene (mixed isomers)	25 ppm	Not Available	Not Available	TLV® Basis: CNS impair; asthma; hematologic eff
Canada - Northwest Territories Occupational Exposure Limits	trimethylbenzene (mixed isomers)	Trimethyl benzene (mixed isomer)	25 ppm	30 ppm	Not Available	Not Available
Canada - Quebec Permissible Exposure Values for Airborne Contaminants	trimethylbenzene (mixed isomers)	Trimethyl benzene	25 ppm / 123 mg/m3	Not Available	Not Available	Not Available
Canada - Nova Scotia Occupational Exposure Limits	1,3,5-trimethyl benzene	1,3,5-Trimethyl benzene	25 ppm	Not Available	Not Available	aka: Mesitylene. TLV Basis: central nervous system impairment; asthma; hematologic effects
Canada - Northwest Territories Occupational Exposure Limits	1,3,5-trimethyl benzene	Trimethyl benzene (mixed isomer)	25 ppm	30 ppm	Not Available	Not Available
Canada - Nova Scotia Occupational Exposure Limits	1,2,3-trimethyl benzene	1,2,3-Trimethyl benzene	25 ppm	Not Available	Not Available	TLV Basis: central nervous system impairment; asthma; hematologic effects
Canada - Northwest Territories Occupational Exposure Limits	1,2,3-trimethyl benzene	Trimethyl benzene (mixed isomer)	25 ppm	30 ppm	Not Available	Not Available
Canada - Yukon Permissible Concentrations for Airborne Contaminant Substances	bitumen (petroleum)	Asphalt (petroleum) fumes	5 mg/m3	10 mg/m3	Not Available	Not Available
Canada - Nova Scotia Occupational Exposure Limits	bitumen (petroleum)	Asphalt (Bitumen; Petroleum) - Fume	0.5 mg/m3	Not Available	Not Available	TLV Basis: Upper respiratory tract & eye irritation. Measured as benzene-soluble aerosol. BEI-P
Canada - Nova Scotia Occupational Exposure Limits	bitumen (petroleum)	Coal dust - Bituminous	0.9 mg/m3	Not Available	Not Available	TLV Basis: lung damage; pulmonary fibrosis
Canada - Alberta Occupational Exposure Limits	bitumen (petroleum)	Asphalt (Petroleum; Bitumen) fume	5 mg/m3	Not Available	Not Available	Not Available
Canada - Saskatchewan Occupational Health and Safety Regulations - Contamination Limits	bitumen (petroleum)	Coal dust: Bituminous (respirable fraction++)	0.9 mg/m3	2.7 mg/m3	Not Available	Not Available
Canada - Saskatchewan Occupational Health and Safety Regulations - Contamination Limits	bitumen (petroleum)	Asphalt (bitumen) fume, as benzene soluble aerosol (inhalable fraction++)	0.5 mg/m3	1.5 mg/m3	Not Available	Not Available
Canada - Manitoba Occupational Exposure Limits	bitumen (petroleum)	Not Available	0.9 mg/m3	Not Available	Not Available	TLV® Basis: Lung dam; pulm fibrosis
Canada - Manitoba Occupational Exposure Limits	bitumen (petroleum)	Not Available	0.5 mg/m3	Not Available	Not Available	TLV® Basis: URT & eye irr; BElp
Canada - British Columbia Occupational Exposure Limits	bitumen (petroleum)	Asphalt (Bitumen) fume, as benzene-soluble aerosol, Inhalable	0.5 mg/m3	Not Available	Not Available	IARC group 2A carcinogen - Bitumens, occupational exposure to oxidized bitumens and their emissions during road paving IARC group 2B carcinogen - Bitumens, occupational exposure to straight-run bitumens and their emissions during road paving
Canada - Prince Edward Island Occupational Exposure Limits	bitumen (petroleum)	Asphalt (Bitumen) fumes, as benzene-soluble aerosol	0.5 mg/m3	Not Available	Not Available	TLV® Basis: URT & eye irr; BElp
Canada - Prince Edward Island Occupational Exposure Limits	bitumen (petroleum)	Coal dust - Bituminous or Lignite	0.9 mg/m3	Not Available	Not Available	TLV® Basis: Lung dam; pulm fibrosis
Canada - Ontario Occupational Exposure Limits	bitumen (petroleum)	Particles (Insoluble or Poorly Soluble) Not Otherwise Specified (PNOS) (Inhalable fraction)	10 mg/m3	Not Available	Not Available	(I) Inhalable fraction: means that size fraction of the airborne particulate deposited anywhere in the respiratory tract and collected during air sampling with a particle size-selective device that, (a) meets the ACGIH particle size-selective sampling criteria for airborne particulate matter; and (b) has the cut point of 100 µm at 50 per cent collection efficiency.

Canada - Ontario Occupational Exposure Limits	bitumen (petroleum)	Particles (Insoluble or Poorly Soluble) Not Otherwise Specified (PNOS) (Respirable fraction)	3 mg/m3	Not Available	Not Available	(R) Respirable fraction: means that size fraction of the airborne particulate deposited in the gas-exchange region of the respiratory tract and collected during air sampling with a particle size-selective device that, (a) meets the ACGIH particle size-selective sampling criteria for airborne particulate matter; and (b) has the cut point of 4 µm at 50 per cent collection efficiency.
Canada - Northwest Territories Occupational Exposure Limits	bitumen (petroleum)	Asphalt (bitumen) fume, as benzene soluble aerosol (inhalable fraction)	0.5 mg/m3	1.5 mg/m3	Not Available	Not Available
Canada - Quebec Permissible Exposure Values for Airborne Contaminants	bitumen (petroleum)	Asphalt (petroleum) fumes	5 mg/m3	Not Available	Not Available	Not Available
Canada - Yukon Permissible Concentrations for Airborne Contaminant Substances	naphtha petroleum, heavy, hydrotreated	Oil mist, mineral	5 mg/m3	10 mg/m3	Not Available	Not Available
Canada - Nova Scotia Occupational Exposure Limits	naphtha petroleum, heavy, hydrotreated	Oil mist - mineral	5 mg/m3	10 mg/m3	Not Available	TLV Basis: lung. As sampled by method that does not collect vapor.
Canada - Alberta Occupational Exposure Limits	naphtha petroleum, heavy, hydrotreated	Oil mist, mineral	5 mg/m3	10 mg/m3	Not Available	Not Available
Canada - Saskatchewan Occupational Health and Safety Regulations - Contamination Limits	naphtha petroleum, heavy, hydrotreated	Oil mist, mineral	5 mg/m3	10 mg/m3	Not Available	Not Available
Canada - Manitoba Occupational Exposure Limits	naphtha petroleum, heavy, hydrotreated	Not Available	5 mg/m3	Not Available	Not Available	TLV® Basis: URT irr
Canada - British Columbia Occupational Exposure Limits	naphtha petroleum, heavy, hydrotreated	Oil mist - mineral, severely refined	1 mg/m3	Not Available	Not Available	Not Available
Canada - Prince Edward Island Occupational Exposure Limits	naphtha petroleum, heavy, hydrotreated	Mineral oil, excluding metal working fluids - Pure, highly and severely refined	5 mg/m3	Not Available	Not Available	TLV® Basis: URT irr
Canada - Northwest Territories Occupational Exposure Limits	naphtha petroleum, heavy, hydrotreated	Oil mist, mineral	5 mg/m3	10 mg/m3	Not Available	Not Available
Canada - Quebec Permissible Exposure Values for Airborne Contaminants	naphtha petroleum, heavy, hydrotreated	Mineral oil (mist)	5 mg/m3	10 mg/m3	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	TEEL-1	TEEL-2	TEEL-3
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
1,2,4-trimethyl benzene	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Not Available	Not Available	480 ppm
Xylene (xylene)	Not Available	Not Available	Not Available
cumene	Not Available	Not Available	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available	480 ppm
1,2,3-trimethyl benzene	Not Available	Not Available	480 ppm
bitumen (petroleum)	30 mg/m3	330 mg/m3	2,000 mg/m3
naphtha petroleum, heavy, hydrotreated	350 mg/m3	1,800 mg/m3	40,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
naphtha petroleum, light aromatic solvent	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
Xylene (xylene)	900 ppm	Not Available
cumene	900 ppm	Not Available
trimethylbenzene (mixed isomers)	Not Available	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
1,2,3-trimethyl benzene	Not Available	Not Available
bitumen (petroleum)	Not Available	Not Available
naphtha petroleum, heavy, hydrotreated	2,500 mg/m3	Not Available

OCCUPATIONAL EXPOSURE BANDING:

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
naphtha petroleum, light aromatic solvent	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA:

IFRA Prohibited Fragrance Substance
The International Fragrance Association (IFRA) Standards form the basis for the globally accepted and recognized risk management system for the safe use of fragrance ingredients and are part of the IFRA Code of Practice. This is the self-regulating system of the industry, based on risk assessments carried out by an independent Expert Panel
bitumen (asphalt) fumes [8052-42-4]
TLV* TWA: 0.5 mg/m3 A4 asphalt (petroleum, bitumen) fume, as benzene soluble aerosol
ES* TWA: 5 mg/m3 as fumes
OES* TWA: 5 mg/m3; STEL: 10 mg/m3 as fumes
Based on surveys of asphalt workers in oil refineries and in the roofing industry the TLV-TWA is thought to reduce the risk of possible carcinogenicity
For trimethyl benzene as mixed isomers (of unstated proportions)
Odour Threshold Value: 2.4 ppm (detection)
Use care in interpreting effects as a single isomer or other isomer mix. Trimethylbenzene is an eye, nose and respiratory irritant. High concentrations cause central nervous system depression. Exposed workers show CNS changes, asthmatic bronchitis and blood dyscrasias at 60 ppm. The TLV-TWA is thought to be protective against the significant risk of CNS excitation, asthmatic bronchitis and blood dyscrasias associated with exposures above the limit.
Odour Safety Factor (OSF)
OSF=10 (1,2,4-TRIMETHYLBENZENE)

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

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

The Odour Safety Factor (OSF) is defined as:

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

Classification into classes follows:

Class OSF Description

A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities

B - 26-550As "A" for 50-90% of persons being distracted
C - 1-26 As "A" for less than 50% of persons being distracted
D - 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E <0.18 As "D" for less than 10% of persons aware of being tested

FOR XYLENES:

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

FOR CUMENE:

Odour Threshold Value: 0.008-0.132 ppm (detection), 0.047 ppm (recognition)
Exposure at or below the TLV-TWA is thought to prevent induction of narcosis.

EXPOSURE CONTROLS:

APPROPRIATE ENGINEERING CONTROLS	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. Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

PERSONAL PROTECTIVE EQUIPMENT:

RESPIRATORY PROTECTION

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

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

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

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses,
B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(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.
SKIN AND BODY PROTECTION	Wear chemical protective gloves, e.g. PVC. Wear safety footwear or safety gumboots, e.g. Rubber The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: frequency and duration of contact, chemical resistance of glove material, glove thickness and dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: Excellent when breakthrough time > 480 min Good when breakthrough time > 20 min Fair when breakthrough time < 20 min Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

	It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
EYE 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]
OTHER PROTECTION	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:
Asphalt Based Prime

Material	CPI
BUTYL	C
BUTYL/NEOPRENE	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
TEFLON	C
VITON	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.

SECTION 9 – PHYSCAL AND CHEMICAL PROPERTIES

APPEARANCE

Bitumen (known as asphalt in the U.S.) "is the residuum produced from the non-destructive distillation of crude petroleum at atmospheric pressure and/ or under reduced pressures or absence of steam. Bitumens/ asphalts are composed mainly of high-molecular-weight alkylaryl hydrocarbons with high carbon to hydrogen ratios, with carbon numbers > C25, boiling points >400 "C, high viscosity, and negligible water solubility and vapor pressure. These bitumen/ asphalt alkylaryl hydrocarbons are a heterogeneous mixture of linear, branched and cyclic, saturated and unsaturated, and aromatic functional groups. Importantly, polycyclic aromatic hydrocarbons (PAH) such as benzo(a)pyrene, which are toxicologically significant, are only present in bitumen/ asphalt feedstock at very low concentrations.

Bitumens/ asphalts contain much larger proportions of high-molecular-weight paraffinic and naphthenic hydrocarbons that are substituted with alkyl groups and ultimately sulfonated, which reduces their potential to exhibit PAH-like toxicity. In practice, the asphalt alkylaryl feedstocks are chemically characterised by a saturates, aromatics, resins, and asphaltenes.

Saturates consist mainly of long chain saturated hydrocarbons with some

Saturates branching, alkyl aromatics with long side chains, and cyclic paraffins (napthenes), with molecular weight of 500-1000.

Asphaltenes consist mainly of substituted benzene and napthenic-aromatic nuclei with alkyl side chain constituents, with molecular weight of 500-900.

Resins consist mainly of heterogeneous polar aromatic compounds with small amounts of oxygen, nitrogen, and sulfur, with molecular Resins weight range of 800-2000. Considered lower molecular weight asphaltenes.

Asphaltenes consist mainly of highly condensed aromatic compounds with one or two chromophores containing 4 to 10 fused rings each, with a
significant number of alkyl constituents. They have a molecular weight range of 500-1000.

The bitumen/ asphalt group is defined by the following six CAS Numbers: asphalt (penetration or hard) (CAS No. 8052-42-4); vacuum residues (CAS No. 64741-56-6); raffinates, residual oil decarbonization (CAS No. 64742-07-0); petroleum resins (CAS No. 64742-16-1); residues, hydrodesulfurised vacuum (CAS No. 64742-85-4); and asphalt, oxidized (CAS No. 64742-93-4). Small amounts of metals such as nickel, iron or vanadium may be present. Bitumen/ asphalt fumes must also be considered in an occupational setting and as fugitive emissions.

PHYSICAL STATE	Liquid
ODOR	No information available.
ODOR THRESHOLD	No information available.
PH	No information available.
MELTING POINT/FREEZING POINT	No information available.
INITIAL BOILING POINT AND BOILING RANGE	149ºC
FLASH POINT	47ºC
EVAPORATION RATE	No information available.
FLAMMABILITY	Flammable
UPPER/LOWER FLAMMABILITY/EXPLOSIVE LIMITS	Lower flammability limit (% vol): No information available. Upper flammability limit (% vol): No information available.
VAPOR PRESSURE	No information available.
VAPOR DENSITY (AIR =1)	No information available.
RELATIVE DENSITY (WATER = 1)	0.87-0.91
MOLECULAR WEIGHT	No information available.

SOLUBILITY(IES)	Immiscible
PARTITION COEFFICIENT: N-OCTANOL/WATER	No information available.
AUTO-IGNITION TEMPERATURE	> 200°C
SPECIFIC GRAVITY	No information available.
VISCOSITY	No information available.
PERCENT VOLATILITY	40-80 %

SECTION 10 – STABILITY AND REACTIVITY

REACTIVITY:	Refer to Section 7 – Handling and Storage
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	Refer to Section 7 – Handling and Storage
CONDITIONS TO AVOID	Refer to Section 7 – Handling and Storage
INCOMPATIBLE MATERIALS	Refer to Section 7 – Handling and Storage
HAZARDOUS DECOMPOSITION PRODUCTS	Refer to Section 5 – Fire Fighting Measures

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.

High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.

Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

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.

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 reliableindicator 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
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 The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.
Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.
Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.
Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed 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.

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). The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. 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 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. Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis
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 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. 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. 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. Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.
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. Exposure to H2S may produce pain, blurred vision, and irritation. These symptoms are temporary in all but severe cases. Eye irritation may produce conjunctivitis, photophobia, pain, and at higher concentrations blurred vision and corneal blistering Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.
CHRONIC	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly

due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses.

Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:
No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human. Long term exposure to coal tar dusts may produce chronic bronchitis or lung cancer. Dust, liquid or fume contact with skin may result in photosensitisation of skin areas and sunburn on frequent exposure to sunlight or ultra-violet radiation.
Workers exposed to hot tar and pitch showed abnormal serum protein levels due to liver dysfunction. Chronic exposure of mice to 0.3 mg/l of tar aerosols, for three 2 hour periods, produced necrotising tracheobronchitis and hyperplasia of the epithelium; these were occasionally accompanied by papillary infolding.

Exposed body surfaces and the scrotum of long-term coal-tar pitch workers may show kerato-acanthoma ("tar mollusca"), pitch warts or tar warts, even after exposure has ceased; the head, neck and other extremities are particularly prone. Pitch keratosis and acanthomas (cancerous or precancerous skin lesions) may also develop. Hyperpigmentation of the body surfaces and scrotum may be localised or diffuse.
Corneal ulcers, conjunctivitis and papillomata of the lids have also been described in workers chronically exposed to coal tar pitches. Workers exposed to petroleum, tar or pitch appear to show an elevated risk of cancer of the renal pelvis. Millwrights and welders in a stamping plant, occupationally exposed to coal-tars and coal-tar pitch showed a greater incidence of leukaemia and cancers of the lung and digestive organs.
Coal tar fumes or dusts have been implicated in the development of occupational cancers. A minimal time of exposure (1-5 years) has been cited. Similarly occupational cancers may develop many years after exposure ceases. Deaths from cancer of the lungs and pleura of retired gas workers was approximately twice the expected rate. Pot-room workers in the aluminium smelting industries showed an increased rate of lung-cancer mortality. One report from the former Soviet

Union associated such an increase with concentrations of tarry substances between 27 and 210 mg/m3 (B[a]P levels of 0.6 to 56 ug/m3). High respiratory mortality has been reported among coke oven workers in Great Britain whilst kidney and lung cancers were prevalent among American coke-oven workers predominantly exposed for more than 5 years.

A UK mortality analysis (in 1946) showed an increase in scrotal cancers in patent-fuel workers. Reports of skin and scrotal cancers are frequent amongst workers exposed to coal-tar fumes in coal gasification and coke production. A small excess of bladder cancer is described in tar distillers and patent-fuel workers.
Benzene extracts of atmospheric samples from a coal tar plant, painted on the intrascapular area of black mice, three times weekly, caused tumours to appear (some occurred within 465 days). Animal studies indicate that lung and kidney tumours were induced following exposure to coal tar aerosols. The degree of lung change of rats breathing air-contaminated with polycyclic aromatic hydrocarbons (PAHs) is dose-related.
Coal-tar containing ointments have been implicated in a number of human skin cancers. Evidence exists for mutagenic action (as seen in urine samples) after application of these ointments

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 unrecognized olfactory deficits. [Hirsch, A.R. - Occ. Env. Med.,1999, Vol 5, Iss 4, pp 284-287] 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 mixed 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. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

	TOXICITY	IRRITATION
Asphalt Based Primer	Not Available	Not Available
naphtha petroleum, light aromatic solvent	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >4.42 mg/L4h[1]	Skin: adverse effect observed (irritating)[1]
	Oral(Rat) LD50; >4500 mg/kg[1]	
1,2,4-trimethyl benzene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3160 mg/kg[2]	Not Available
	Inhalation(Rat) LC50; 18 mg/L4h[2]	
	Oral(Rat) LD50; 6000 mg/kg[1]	
Xylene (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]
cumene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 2000 mg/kg[2]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; 39 mg/L4h[2]	Eye (rabbit): 86 mg mild
	Oral(Rat) LD50; 1400 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 10 mg/24h mild
		Skin (rabbit):100 mg/24h moderate
		Skin: no adverse effect observed (not irritating)[1]

trimethylbenzene (mixed isomers)	**TOXICITY**	**IRRITATION**
	Oral(Rat) LD50; 8970 mg/kg[2]	Eye (rabbit): 500 mg/24h - mild
		Skin (rabbit): 500 mg/24h-moderate
1,3,5-trimethyl benzene	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >3460 mg/kg[1]	Eye (rabbit): 500 mg/24h mild
	Inhalation(Rat) LC50; 24 mg/L4h[2]	Eye: adverse effect observed (irritating)[1]
	Oral(Rat) LD50; 6000 mg/kg[1]	Skin (rabbit): 20 mg/24h moderate
		Skin: adverse effect observed (irritating)[1]
1,2,3-trimethyl benzene	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Not Available
	Oral(Rat) LD50; 3163 mg/kg[1]	
bitumen (petroleum)	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral(Rat) LD50; >5000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
naphtha petroleum, heavy, hydrotreated	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >1900 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >4.42 mg/L4h[1]	Skin: adverse effect observed (irritating)[1]
	Oral(Rat) LD50; >4500 mg/kg[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*	

Asphalt Based Primer	Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies. Data demonstrate that during inhalation exposure, aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following cessation of exposure, the level of aromatic hydrocarbons in body fats rapidly declines. Thus, the aromatic hydrocarbons are unlikely to bioaccumulate in the body. Selective partitioning of the aromatic hydrocarbons into the non-adipose tissues is unlikely. No data is available regarding distribution following dermal absorption. However, distribution following this route of exposure is likely to resemble the pattern occurring with inhalation exposure. Aromatics hydrocarbons may undergo several different Phase I dealkylation, hydroxylation and oxidation reactions which may or may not be followed by Phase II conjugation to glycine, sulfation or glucuronidation. However, the major predominant biotransformation pathway is typical of that of the alkylbenzenes and consists of: (1) oxidation of one of the alkyl groups to an alcohol moiety; (2) oxidation of the hydroxyl group to a carboxylic acid; (3) the carboxylic acid is then conjugated with glycine to form a hippuric acid. The minor metabolites can be expected to consist of a complex mixture of isomeric triphenols, the sulfate and glucuronide conjugates of dimethylbenzyl alcohols, dimethylbenzoic acids and dimethylhippuric acids. Consistent with the low propensity for bioaccumulation of aromatic hydrocarbons, these substances are likely to be significant inducers of their own metabolism. The predominant route of excretion of aromatic hydrocarbons following inhalation exposure involves either exhalation of the unmetabolized parent compound, or urinary excretion of its metabolites. When oral administration occurs, there is little exhalation of unmetabolized these hydrocarbons, presumably due to the first pass effect in the liver. Under these circumstances, urinary excretion of metabolites is the dominant route of excretion.

	The production of wood creosote, coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles, stems from the incomplete combustion or pyrolysis of carbon-containing materials. Creosotes, coal tar, coal tar pitch, and coal tar pitch volatiles are composed of many individual compounds of varying physical and chemical characteristics. In addition, the composition of each, although referred to by specific name (e.g., wood creosote or coal tar creosote) is not consistent. Coal tars are by-products of the carbonization of coal to produce coke or natural gas. Physically, they are usually viscous liquids or semisolids that are black or dark brown with a naphthalene-like odor. The coal tars are complex combinations of polycyclic aromatic hydrocarbons (PAHs), phenols, heterocyclic oxygen, sulfur, and nitrogen compounds. By comparison, coal tar creosotes are distillation products of coal tar. They have an oily liquid consistency and range in color from yellowish-dark green to brown. At least 75% of the coal tar creosote mixture is PAHs. Unlike the coal tars and coal tar creosotes, coal tar pitch is a residue produced during the distillation of coal tar. (Beech)wood creosote consists mainly of phenol, cresols, guaiacol, xylenol, and creosol. Creosote bush resin consists of phenolic (e.g., flavonoids and nordihydroguaiaretic acid), neutral (e.g., waxes), basic (e.g., alkaloids), and acidic (e.g., phenolic acids) compounds. The phenolic portion comprises 83-91% of the total resin. Nordihydroguaiaretic acid accounts for 5-10% of the dry weight of the leaves. It is likely that the toxicity of wood creosote, coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles is due largely to the major individual components, phenols, PAHs and others. For "distillates of coal tar" or 'creosotes. Critical Health Effects The critical health effects for risk characterisation are systemic long-term effects including carcinogenicity, mutagenicity, reproductive toxicity and developmental toxicity. The chemicals are also considered to be phototoxic and have the potential to cause skin irritation and sensitisation and mild respiratory irritation. Toxicokinetics Limited data are available. Toxicological data indicate that the chemicals are absorbed via all routes of exposure (WHO, 2004). The PAHs can be absorbed through the respiratory tract, the gastrointestinal tract and the skin. Following absorption, PAHs are widely distributed throughout the body to all internal organs. During metabolism, the parent compounds are converted via intermediate epoxides to phenols, diols, and tetrols, which then conjugate with sulfate or glucuronic acids or with glutathione (IPCS, 1998). Observation in humans Evidence of skin, eye and respiratory irritation in humans following exposure to creosote have been reported (ATSDR, 2002). Skin irritation, eczema and folliculitis were noted when an industrial health survey was conducted of workers exposed to coal tar creosote (ATSDR, 2002). In these workers, the effects of dermal irritation were reported as being exacerbated by exposure to ultraviolet (UV) light. The phototoxic effects of several PAHs were compared by treating human fibroblasts with these PAHs and then irradiating them with ultraviolet light (<400 nm). A good correlation was found between the phototoxic effects and known carcinogenic potential (IPCS, 1998). Studies involving workers included reported instances of irritation to superficial ocular tissues after being exposed to coal tar creosote; this was exacerbated after exposure to the sun (ATSDR, 2002). Skin Sensitisation Limited data are available. Distillates, coal tar, naphthalene oils (CAS No. 84650-04-4), gave positive results in a single local lymph node assay (LLNA). Creosote (CAS No. 8001-58-9) was found to induce dermal sensitisation when tested according to OECD TG 406 in a guinea pig maximisation test (GPMT) using Dunkin-Hartley guinea pigs (REACH). Overall, the available data support classification for all the chemicals in this

group.

An LLNA study (OECD TG 429) was conducted in female BALB/c mice (n = 5/concentration) with coal tar distillates, naphthalene oils (CAS No. 84650-04-4), using a 40 % dimethylacetamide, 30 % acetone and 30 % ethanol (DAE 433) mixture as a vehicle. The test concentrations of 0.3, 3 and 30 % had a simulation index (SI) of 1.36, 1.41 and 5.88 respectively. The positive control, dinitrochlorobenzene at a 0.5 % concentration, gave an SI of 11.55. The three-fold increase in lymphocyte proliferation (EC3 value) could not be calculated (REACHc).

In a GPMT (OECD TG 406) with creosote (CAS No. 8001-58-9), positive skin reactions were reported in 17/19 animals after 24 hours (average Draize score = 1.2) and 6/19 animals after 48 hours (average Draize score = 0.4) (REACHb).
Repeated Dose Toxicity

Oral
Limited data are available regarding the non-cancer effects of the chemicals.
The chemicals in this group are not considered to cause serious damage to health through repeated oral exposure based on the no observed adverse effect levels (NOAELs) (generally >100 mg/kg bw/day) reported for the following 2–4-ring PAHs:
-naphthalene;
-acenaphthene;
-fluorene;
-fluoranthene; and
- pyrene.

Effects on the liver, kidney and blood were observed at higher doses (IPCS, 1998).

Dermal
Limited data are available regarding the non-cancer effects of the chemicals.

Inhalation
Limited data are available regarding the non-cancer effect of the chemicals.
Male Fischer 344 rats were exposed to high-boiling coal liquid (heavy distillate) via inhalation (700 mg/m3) for six hours/day, five days/week for six weeks. A 20 % increase in arterial blood pressure and heart rate was reported, although it was not determined if the response was exposurerelated. The growth rate of the rats was reported as suppressed during the time of the study (ATSDR, 2002).
Repeated dose toxicity (inhalation) was determined by exposing 20 (sex/dose) Charles River (CD) rats to CAS No. 90640-86-1 (as distilled coal tar) (5.4, 49 and 106 mg/m3) for six hours/day, five days/week for 13 weeks. A decrease in body weight was recorded as significant in both sexes in the mid- and high-range dose groups during the sixth week of exposure. A treatment related increase in weight was reported in the lung/trachea/body weight ratio and was consistent with macroscopic observation of grey discolouration of the lungs and microscopic observation of macrophages in the lungs. Increases in liver weight (mid-dose group) and liver/body weight ratio (mid- and high-dose group) were recorded in
male animals. Increases in the liver weight (high-dose group), liver/body weight ratio and liver/brain weight ratio (mid- and high-dose group) were recorded in the female animals. Reversible hypertrophy of the thyroid follicular cells reported as related to a reduction of colloid was reported at all dose levels. A NOAEL of 5.4 mg/m3 was reported for this study (REACHb).

Observation in humans
Mild respiratory effects, including reduced lung function, have been reported in workers using coal tar creosote in wood preservative plants.

Genotoxicity
Several of the chemicals (CAS No. 73665-18-6, CAS No. 84650-03-3 and CAS No. 84650-04-4) are classified as hazardous—Category 2 mutagenic substance—with the

	risk phrase 'May cause heritable genetic damage' (T; R46) in the HSIS (Safe Work Australia). The available data support this classification for all the chemicals in this group, although the associated annotations will differ for each chemical (refer Recommendation section). For the chemicals CAS No. 84650-03-3 and CAS No. 84650-04-4, in vitro data using the reverse mutation assays with various strains of Salmonella typhimurium were negative for genotoxicity (REACH). No compositional information was available but these chemicals are lower boiling point distillate fractions that are likely to contain aromatics, tar bases and acids (see Grouping rationale). The classification of these chemicals is dependent on benzene concentration (refer to Existing Worker Health and Safety Controls: Hazard Classification section). Benzene is classified as hazardous—Category 2 mutagenic substance—with the risk phrase 'May cause heritable genetic damage' (T; R46) in the HSIS (Safe Work Australia). The chemical, CAS No. 90640-86-1 was positive in a reverse mutation assay in Salmonella typhimurium strains TA98 and TA1537 in the presence of metabolic activation. Weakly positive responses were also observed in strains TA100 and TA102. The sample was reported to contain >50 ppm B[a]P. Various creosotes have been reported to produce a positive response in vitro. Almost all creosotes tested showed mutagenic activity after metabolic activation (S9 mix) in the conventional Ames assay with S. typhimurium TA98. Positive results were also obtained with several other S. typhimurium TA or YG strains, or with the mouse lymphoma cell assay and the sister chromatid exchange test with Chinese hamster ovary cells. A common feature in the tests with Salmonella strains TA98 and TA100 (plus S9 mix) was that the mutagenicity appeared in the distillation fractions having the highest boiling point ranges (>290 °C) and high concentrations of known mutagenic PAHs (WHO, 2004). A creosote reported to contain <50 ppm B[a]P was tested according to OECD 476 (in vitro mouse lymphoma gene mutation assay). The chemical showed a weak positive mutagenic activity in the presence of metabolic activation. A creosote containing <50 ppm B[a]P did not induce chromosome aberrations in human lymphocytes cultures in the presence and absence of metabolic activation (REACHb). DNA adduct formation in mammalian systems has been observed following exposure to creosote, with adducts in rats (liver) and mice (lungs, forestomach and spleen) (ATSDR, 2002). A commercially available coal tar creosote was positive in an in vivo mouse micronucleus assay. The CD-1 male mice received two intraperitoneal (i.p.)injections (with an interval of 24 hours) of creosote (in olive oil) at concentrations of 92.5, 185, or 370 mg/kg bw. Dose-dependent increases in the frequency of micronucleated polychromatic erythrocytes in bone marrow were observed. A single intraperitoneal treatment of 370 mg/kg body weight also induced micronuclei (WHO, 2004). A creosote reported to contain <50 ppm B[a]P was reported to be negative in an in vivo mouse micronucleus test (REACHb). Genotoxicity of PAHs The chemicals have the potential to contain fluoranthene and chrysene as well as higher molecular weight PAHs that are genotoxic, including benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene (IARC, 2010; IARC, 2012; NICNAS). Positive effects were seen in most assays for the mutagenicity of B[a]P, including induced sperm abnormalities in mice (IPCS, 1998). Data for B[a]P are considered sufficient to indicate that the chemicals could induce mutations in germ cells. Carcinogenicity The chemicals are classified as hazardous—Category 2 carcinogenic substances—with the risk phrase 'May cause cancer' (T; R45) in the HSIS (Safe Work Australia). The available data support this classification for all the chemicals in this group, although the associated notes will differ for each chemical (refer Recommendation section). Several creosote or cresosote oils produced skin tumours in mice following dermal

application. Lung tumours were also reported in one study.
Worker exposure to creosotes has been associated with an increased risk of testicular cancer. The only available cohort study was considered limited by its small size (IARC, 1985; IARC, 2010).

The International Agency for Research on Cancer (IARC) concluded that creosotes are probably carcinogenic to humans (Group 2A). This was based on limited evidence of carcinogenicity in humans and sufficient evidence in experimental animals (IARC, 2010). There are a number of potential carcinogenic components of the chemicals. There is sufficient evidence in experimental animals for the carcinogenicity of four membered PAHs such as chrysene and pyrene and also several higher molecular weight PAHs (IARC, 2010; IARC 2012). The classification of a number of chemicals in this group is subject to note M (refer to Existing Worker Health and Safety Controls: Hazard Classification section), which exempts classification if it can be shown that the substance contains <0.005 % w/w B[a]P (50 ppm). No data have been identified regarding the rationale for note M. However, in the absence of detailed composition details, this is considered reasonable as, whilst several carcinogenic PAHs might be present as constituents in these chemicals at levels similar or higher than B[a]P, the cut-off concentration for mixtures containing category 1 carcinogens is 0.1 % (several orders of magnitude higher than 0.005 %).

The classification of some of the lower boiling point distillate fractions are subject to note J (refer to Existing Worker Health and Safety Controls: Hazard Classification section), which exempts classification if it can be shown that the substance contains <0.1% w/w benzene. Benzene is classified as hazardous, a Category 1 carcinogenic substance, with the risk phrase 'May cause cancer' (T; R45) in the HSIS (Safe Work Australia).

Reproductive and Developmental Toxicity
Overall, the reproductive and developmental data are limited for chemicals in the group, although the data for higher molecular weight PAHs are considered sufficient for classification for all chemicals except the lower boiling point distillate fractions (CAS Nos. 84650-03-3 and 84650-04-4).

The associated notes will differ for each chemical.
In a two-generation study, the chemical, distillates, coal tar, heavy oils (CAS No. 90640-86-1), was administered via oral gavage (25, 75 and 150 mg/kg bw/day) to male and female CD rats (26/sex/dose). At all dose levels, decrease in body weight during the pre-mating period was observed and recorded as dose-related. Decreased fertility and pregnancy indices in the F1 female parental rats were recorded at all dose levels (25, 75, 150 mg/kg bw/d). There was a significant dose-related reduction in the number of live F1 offspring at doses ³75 mg/kg bw/d. A dose-related decrease in growth of the F1 offspring was reported, starting at 25 mg/kg bw/d. Although the NOAEL is reported as 25 mg/kg bw/d (REACHb), reproductive effects were indicated at all doses.

In a developmental toxicity study, the chemical, distillates, coal tar, heavy oils (CAS No. 90640-86-1), was administered via oral gavage (25, 50 and 175 mg/kg bw/day) to 30 (per dose) mated female CD rats, during gestation day(GD) 6–15. Increases in post implantation loss, resorptions and a reduction in live foetuses were observed in 175 mg/kg bw/day group. Developmental toxicity was not observed at doses of 50 mg/kg bw/day or lower. Malformations were observed in all dose groups, although the incidences were significantly higher in the mid- and high-dose groups. These were historically common malformations and not considered by the study authors to be treatment related. There were no adverse effects observed for late intrauterine development of live foetuses in any dose group. The NOAEL for maternal toxicity was reported as 50 mg/kg bw/d and for teratogenicity 175 mg/kg bw/d (REACHb).

Coal tar creosote was tested for oestrogenic activity using an assay in ovariectomised (OVX) ICR and DBA/2 mice. The animals received oral doses (by gavage) once every 24 hours for four days and were euthanised on day five. No increase in absolute or relative uterine wet weight or vaginal cornification was observed.

A decrease in mean foetal body weight was observed in the offspring of female ICR mice dosed by gavage with 400 mg/kg petroleum creosote in DMSO on GD 5–9. Moderate maternal toxicity in the form of reduced body weight gain was observed for both creosote-treated and vehiclecontrol mice compared with untreated controls. (ATSDR, 2002; WHO, 2004).

Embryotoxicity of petroleum creosote has been studied in a mouse preimplantation embryo culture system. The ICR mice embryos (n = 15) collected on day 3.5 of gestation (blastocyst stage) were exposed for 1 hour to different concentrations of creosote in a serum-supplemented culture medium with and without rodent hepatic S9 microsomal fractions, and subsequently cultured in a control medium for 24–72 hours.

Embryonic viability was inversely related to petroleum creosote concentration (WHO, 2004).

An experiment with pregnant pigs, held on wooden platforms treated with coal tar creosote, resulted in adverse developmental effects. A significant number (24/41) of piglets died at birth and 11 piglets died by day three post farrowing.

The chemicals may contain several higher molecular weight PAHs that are embryotoxic. B[a]P also had adverse effects on female fertility, reproduction and postnatal development (IPCS, 1998).

The chemicals are recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

The classification criteria for mixtures should be applied to known components based on their concentrations in these UVCB substances. In the absence of detailed composition data the following notes should be applied.

Information on notes
A note should be added for the acute toxicity classification. The acute toxicity R23 classification need not apply if it can be shown that the chemical contains <8 % pyrene; however, R20 classification applies if the chemicals contains >1 % pyrene.

The current HSIS classification for carcinogenicity of the chemicals indicated Note H. Note H is no longer considered relevant for these chemicals as the acute, systemic and local effects of the chemicals have been evaluated.

The classification for CAS Nos. 61789-28-4, 65996-91-0, 65996-92-1, 68188-48-7, 73665-18-6, 84650-04-4 and 91995-51-6 are subject to Note M (refer to Existing Worker Health and Safety Controls: Hazard Classification section), which exempts classification if it can be shown that the substance contains <0.005 % w/w B[a]P (50 ppm). Given that Note M for carcinogenicity is considered appropriate for these chemicals and the cut-off concentration for mixtures is similar for the mutagenicity, reproductive/developmental and carcinogenicity classifications, a similar note for the proposed genotoxicity and reproductive/developmental classification is considered appropriate. Therefore, Note M should be slightly modified
as follows:
'Note M: The classification (with the exception of classification for acute toxicity and sensitisation) need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS no. 200-028-5). This note only applies to certain complex coal-derived substances in Annex I.'

The classification for CAS Nos. 84650-03-3, 84650-04-4 and 73665-18-6 are subject to Note J (refer to Existing Worker Health and Safety Controls: Hazard Classification section), which exempts classification if it can be shown that the substance contains <0.1% w/w benzene. These chemicals are described as including lower boiling point

	distillation fractions and therefore Note J is considered appropriate. Based on the description of CAS No. 65996-92-1 ('The distillate from coal tar having an approximate distillation range of 100 deg C to 450 deg C (212 deg F to 842 deg F). Composed primarily of two to four membered condensed ring aromatic hydrocarbons, phenolic compounds, and aromatic nitrogen bases.' (NCI)). Note J is also considered applicable to this chemical. The classification for CAS Nos. 8001-58-9 and 90640-86-1 are not subject to any notes. The lack of a note may be because the chemicals under these CAS Nos. might not be available in sufficiently purified forms. In the absence of further information, the addition of note M is not recommended. NICNAS HUMAN HEALTH TIER II ASSESSMENT FOR Coal Tar Distillates http://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1442
NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	For C9 aromatics (typically trimethylbenzenes - TMBs) Acute Toxicity Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50's range from 6,000 to 10,000 mg/m 3 for C9 aromatic naphtha and 18,000 to 24,000 mg/m3 for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines. Irritation and Sensitization Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified. Repeated Dose Toxicity Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m3). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m3, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m3) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs. The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m3. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m3). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m3 (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m3 for respiratory irritation and 250 ppm or 1230 mg/m3 for systemic effects. Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure.

Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

Mutagenicity
In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with Salmonella typhimurium and Escherichia coli bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In in vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m3) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category

Reproductive and Developmental Toxicity
Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m3 , respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex /group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex /group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.

Systemic Effects on Parental Generations:
The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body

	weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m3). Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m3). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation,, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m3), which excludes analysis of the highest concentration due to excessive mortality. Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m3) based on the body weights reductions observed in the F3 offspring. Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.* [Devoe] .
1,2,4-TRIMETHYL BENZENE	CHEMWATCH 2325 1,3,5-trimethylbenzene
XYLENE (XYLENE)	Reproductive effector in rats The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. 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.
CUMENE	Cumene is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals. Cumene caused tumours at several tissue sites, including lung and liver in mice and kidney in male rats. Several proposed mechanisms of carcinogenesis support the relevance to humans of lung and liver tumours in experimental animals. Specifically, there is evidence that humans and experimental animals metabolise cumene through similar metabolic pathways. There is also evidence that cumene is genotoxic in some tissues, based on findings of DNA damage in rodent lung and liver. Furthermore, mutations of the K-ras oncogene and p53 tumor-suppressor gene observed in cumene-induced lung tumours in mice, along with altered expression of many other genes, resemble molecular alterations found in human lung and other cancers. The relevance of the kidney tumors to cancer in

humans is uncertain; there is evidence that a species specific mechanism not relevant to humans contributes to their induction, but it is possible that other mechanisms relevant to humans, such as genotoxicity, may also contribute to kidney-tumour formation in male rats.

For aromatic terpenes:
Acute toxicity: Mammalian LD50 for p-cymene have shown it to have low toxic potential. Similar studies with cumene have concurred with these results

In general, the studies indicate that p-cymene (p-methylisopropylbenzene) or cumene (isopropylbenzene) is rapidly absorbed by oral or inhalation routes. They undergo oxidation (hydroxylation) of the side chain isopropyl substituent and, in the case of p-cymene, the methyl substituent to yield polar oxygenated metabolites. These metabolites are either excreted unchanged in the urine or undergo Phase II conjugation with glucuronic acid and/or glycine followed by excretion in the urine. Unchanged p-cymene or cumene were not detected in the urine or faeces.

Humans (5 males and 5 females/group) exposed to an atmosphere containing 49, 98, or 147 ppm cumene for 7 hours showed 64% absorption at 0.5 hours and 45% at 7 hours. Maximum excretion is observed at 6 to 8 hours and is essentially complete at 48 hours. Approximately 35% of the dose inhaled was excreted as 2-phenyl-2-propanol

Repeat Dose Toxicity: Subacute Studies: Groups of 7 to 12 male rats were exposed to 0, 50, or 250 ppm of p-cymene for 6 hours/day, 5 days/week for 4 weeks with an 8-week recovery period. there was no overt toxicity in the treated rats and no effect on body weight or terminal weight of the brain, cerebellum or whole brain. There was also no effect on regional enzyme activities, regional protein synthesis or regional neurotransmitter concentrations.
Cumene has been tested by the National Toxicology Program (NTP) in both rats and mice. Animals were exposed to up to 4,000 ppm cumene by whole-body inhalation for 12-13 days over a period of 16-17 days. In rats, all animals died at 4,000 ppm, and about half the animals died at the next exposure concentration (2,000 ppm). Varying degrees of ataxia were reported in surviving rats exposed to 500 to 2,000 ppm cumene. Increased relative liver and kidney weights were reported in rats exposed to cumene. In exposed male rats, hyaline droplets in the renal cortical tubules were reported. At 2,000 ppm, superlative inflammation of the lung was reported in 40% of the rats. In mice, all animals died at the 2 highest exposures (2,000 and 4,000 ppm). At 1,000 ppm, 80% of the female mice died and male mice showed varying degrees of ataxia. Increased relative liver and kidney weights were reported in mice exposed to cumene. Decreased thymus weight was reported in male mice exposed to 1,000 ppm of cumene. No histopathological findings accompanied the organ weight changes. A NOAEL of 1,000 ppm was determined for female rats and male mice and a NOAEL of 500 ppm was determined for female mice based on mortality and histopathological findings.

Chronic toxicity: The US EPA concluded that there is some evidence that suggests that cumene is not likely to produce a carcinogenic response (i.e., numerous genotoxic tests, including gene mutation, chromosomal aberration, and primary DNA damage tests, all but one of which were negative or not reproducible) In addition, EPA noted that cumene does not appear to metabolise to highly reactive chemical species and in terms of metabolism, cumene is analogous to methyl benzene for which a 2-year inhalation study was conducted by NTP and no evidence of carcinogenic activity was reported in either rats or mice.

Given that the only structural difference between p-cymene and cumene is the presence of a second alkyl substituent (isopropylbenzene versus p-methylisopropylbenzene), similar conclusions can be drawn for p-cymene, particularly since the pharmacokinetic, metabolic and toxicologic data that are available support this conclusion.

Reproductive toxicity: Taking into consideration the rapid metabolism and excretion of cumene, the US EPA concluded, “cumene has low potential for reproductive toxicity.”

	Developmental toxicity: Even at maternally toxic concentrations exposure to cumene vapor did not produce developmental toxicity in rats. However the US EPA determined that the changes in gestational parameters of the rabbits, though not significant, were consistent in indicating possible developmental effects and therefore set the NOAEL in rabbits for both developmental and maternal effects at 1,206 ppm and the LOAEL at 2,297 ppm, respectively (as reported in EPA, 1997). Since both cumene and p-cymene exhibit such similar pharmacokinetic and metabolic profiles, and show no evidence of toxicity at levels of exposure similar to those experienced by humans, further teratogenic or developmental testing is not recommended **Genotoxicity**: The genotoxicity database on p-cymene and cumene shows no mutagenic potential in the Ames assay. In cytogenetic assays, there is no evidence of a genotoxic potential in vitro. In whole animals, the genotoxicity results for cumene are mixed showing weakly positive results in micronuclei induction in rats, but no evidence of genotoxicity in mice. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [National Toxicology Program: U.S. Dep. of Health & Human Services 2002]
TRIMETHYLBENZENE (MIXED ISOMERS)	NOTE: This data is for mixed isomers of unstated proportions
1,3,5-TRIMETHYL BENZENE	CHEMWATCH 12171 1,2,4-trimethylbenzene
BITUMEN (PETROLEUM)	No significant acute toxicological data identified in literature search
NAPHTHA PETROLEUM, HEAVY, HYDROTREATED	for petroleum: Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic. This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss. This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents **Carcinogenicity**: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans. **Mutagenicity**: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays. **Reproductive Toxicity**: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed. **Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials. Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents

	lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.
Asphalt Based Primer & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & 1,2,4-TRIMETHYL BENZENE & CUMENE & TRIMETHYLBENZENE (MIXED ISOMERS) & 1,3,5-TRIMETHYL BENZENE & 1,2,3-TRIMETHYL BENZENE & BITUMEN (PETROLEUM)	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. 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. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
Asphalt Based Primer & NAPHTHA PETROLEUM, HEAVY, HYDROTREATED	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.
Asphalt Based Primer & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & 1,2,4-TRIMETHYL BENZENE & TRIMETHYLBENZENE (MIXED ISOMERS) & 1,3,5-TRIMETHYL BENZENE &	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

1,2,3-TRIMETHYL BENZENE	consisted of 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
1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE	Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene
XYLENE (XYLENE) & CUMENE & TRIMETHYLBENZENE (MIXED ISOMERS) & 1,3,5-TRIMETHYL BENZENE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
CUMENE & BITUMEN (PETROLEUM)	WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
TRIMETHYL BENZENE (MIXED ISOMERS) & 1,3,5-TRIMETHYL BENZENE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

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

SECTION 12 – ECOLOGICAL INFORMATION

TOXICITY:

Asphalt Based Primer

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

naphtha petroleum, light aromatic solvent

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
EC50	72h	Algae or other aquatic plants	19mg/l	1

	EC50	48h	Crustacea	6.14mg/l	1
	EC50	96h	Algae or other aquatic plants	64mg/l	2

1,2,4-trimethyl benzene

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	31-207	7
EC50(ECx)	96h	Algae or other aquatic plants	2.356mg/l	2
LC50	96h	Fish	3.41mg/l	2
EC50	96h	Algae or other aquatic plants	2.356mg/l	2
EC50	48h	Crustacea	ca.6.14mg/l	1

Xylene (xylene)

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

cumene

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	96h	Crustacea	0.4mg/l	1
EC50	72h	Algae or other aquatic plants	1.29mg/l	2
LC50	96h	Fish	2.7mg/l	2
EC50	48h	Crustacea	4mg/l	1

trimethylbenzene (mixed isomers)

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

1,3,5-trimethyl benzene

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	5.216mg/l	2
EC50	48h	Crustacea	13mg/L	5
BCF	1680h	Fish	23-342	7
NOEC(ECx)	384h	Crustacea	0.257mg/l	2
EC50	96h	Algae or other aquatic plants	3.084mg/l	2

1,2,3-trimethyl benzene

Endpoint	Test Duration (hr)	Species	Value	Source
BCF	1344h	Fish	133-217	7

bitumen (petroleum)

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

naphtha petroleum, heavy, hydrotreated

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	96h	Algae or other aquatic plants	64mg/l	2
EC50	96h	Algae or other aquatic plants	64mg/l	2

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

Harmful to aquatic organisms.
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. As a VOC, 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs.

Environmental fate:
Transport: ,1,2,4-Trimethylbenzene volatilises rapidly from surface waters as predicted by a Henry's law constant of 5.18 x 10-3 (vapor pressure, 2.03 mm Hg). The volatilization half-life from a model river is calculated to be 3.4 hours. The chemical also volatilises from soils, however, based on an estimated Koc of 472, moderate adsorption to soils and sediments may occur.
Transformation/Persistence
Air - 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) In the atmosphere, two estimates of the half-life are approximately 6 hours and, in the presence of hydroxyl radicals, 0.5 days
Soil - Volatilisation 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
Water - Because of 1,2,4-trimethylbenzene's water solubility and its vapor pressure of 2.03 mm Hg, the chemical will rapidly volatilise from surface waters Biodegradation of 1,2,4-trimethylbenzene occurred with inoculums from both seawater and ground water Various strains of Pseudomonas can biodegrade 1,2,4-trimethylbenzene.
Biota - The estimated bioconcentration factor (439) and high volatility of 1,2,4-trimethylbenzene indicates that bioaccumulation of the chemical will not be significant
Ecotoxicity:
Fish LC50 (96 h): fathead minnow 7.72 mg/l
No stress was observed in Oncorhynchus mykiss (rainbow trout, fingerling) or Petromyzon marinus (sea lamprey, larvae) at 5 mg/L for 24 hours
Daphnia magna EC50 (48 h): 3.61 mg/l
Cancer magister (dungeness crab) LC50 996 h): 5.1 mg/l
1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms; acute toxicity values fall within the range of greater than 1 mg/L and 100 mg/L. LC50 values for specific aquatic organisms range from approximately 5 to 8 mg/L which is orders of magnitude greater than any measured concentration in seawater (0.002 - 0.54 microgram/L) The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.

For aromatic hydrocarbons:
Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (Palaemonetes pugio) and brown shrimp (Penaeus aztecus) was dimethylnaphthalenes > methylnaphthalenes >naphthalenes.

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.
The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.
Anthracene is a phototoxic PAH . UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

Volatile furandiones and aldehydes are significant atmospheric oxidation products of aromatic compounds. Highly acidic dicarboxylic acids produced by the reactions between furandiones and water were shown to rapidly acidify an aqueous phase.

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 oxidized to an aldehyde, and finally to a carboxylic acid. The resulting fatty acid could be metabolised through the fatty acid degradation pathway.

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 laboraory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish

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

The tropical mysid Metamysidopsis insularis was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L this species has been shown to be as sensitive as temperate mysids to toxicants. However,However this study used nominal concentrations,and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (Crangon crangon) a 96-hour LC50 of 22 mg/L was determined. A “gas oil”was also tested and a 96-hour LC50 of 12 mg/L.was determined The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species . The diatom Phaeodactylum tricornutum showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga Isochrysis galbana was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L.
Finally, the green algae Chlorella salina was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L . All populations of phytoplankton returned to a steady state within 5 days of exposure In sandy soils, earthworm (Eisenia fetida) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight loss was recorded

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

For 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 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 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
Terrestrial fate: Measured Koc values of 166 and 182, indicate that 3-xylene is expected to have moderate mobility in soil. Volatilisation of p-xylene is expected to be important from moist soil surfaces given a measured Henry's Law constant of 7.18x10-3 atm-cu m/mole. The potential for volatilisation of 3-xylene from dry soil surfaces may exist based on a measured vapor pressure of 8.29 mm Hg. p-Xylene may be degraded during its passage through soil).

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. p-Xylene, present in soil samples contaminated with jet fuel, was completely degraded aerobically within 5 days. In aquifer studies under anaerobic conditions, p-xylene was degraded, usually within several weeks, with the production of 3-methylbenzylfumaric acid, 3-methylbenzylsuccinic acid, 3-methylbenzoate, and 3-methylbenzaldehyde as metabolites.

Aquatic fate: Koc values indicate that p-xylene may adsorb to suspended solids and sediment in water. p-Xylene is expected to volatilise from water surfaces based on the measured Henry's Law constant. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. BCF values of 14.8, 23.4, and 6, measured in goldfish, eels, and clams, respectively, indicate that bioconcentration in aquatic organisms is low. p-Xylene in water with added humic substances was 50% degraded following 3 hours irradiation suggesting that indirect photooxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. Although p-xylene is biodegradable and has been observed to degrade in pond water, there are insufficient data to assess the rate of this process in surface waters. p-Xylene has been observed to degrade in anaerobic and aerobic groundwater in several studies; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high.

Atmospheric fate:
Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere primarily by reaction with photochemically-produced hydroxyl radicals, with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylenes' susceptibility to photochemical oxidation in the troposphere is to the extent that they may contribute to photochemical smog formation. According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and from its vapour pressure, p-xylene, is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase p-xylene is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 16 hours. A half-life of 1.0 hr in summer and 10 hr in winter was measured for the reaction of p-xylene with photochemically-produced hydroxyl radicals.
p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers, with loss rates varying from 9-42% per hr. 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-dimethylp-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

Ecotoxicity:
for xylenes
Fish LC50 (96 h) Pimephales promelas 13.4 mg/l; Oncorhyncus mykiss 8.05 mg/l; Lepomis macrochirus 16.1 mg/l (all flow through values); Pimephales promelas 26.7 (static)
Daphnia EC50 948 h): 3.83 mg/l
Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/l
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 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 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.
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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.
DO NOT discharge into sewer or waterways.

PERSISTENCE & DEGRADABILITY

Ingredient	Persistence: Water/Soil	Persistence: Air
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
Xylene (xylene)	HIGH (Half-life = 360 days)	LOW (Half-life = 1.83 days)
Cumene	HIGH	HIGH
1,3,5-trimethyl benzene	HIGH	HIGH
1,2,3-trimethyl benzene	HIGH	HIGH

BIOACCUMULATION POTENTIAL

Ingredient	Bioaccumulation
1,2,4-trimethyl benzene	LOW (BCF = 275)
Xylene (xylene)	MEDIUM (BCF = 740)
Cumene	LOW (BCF = 35.5)
1,3,5-trimethyl benzene	LOW (BCF = 342)
1,2,3-trimethyl benzene	LOW (BCF = 259)

BIODEGRADATION MOBILITY (Mobility in soil)

Ingredient	Mobility
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
Cumene	LOW (KOC = 817.2)
1,3,5-trimethyl benzene	LOW (KOC = 703)
1,2,3-trimethyl benzene	LOW (KOC = 732.5)

SECTION 13 – DISPOSAL CONSIDERATIONS

DISPOSAL RECOMMENDATIONS

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.
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 NO

Land transport (TDG)

UN number 1999

UN proper shipping name TARS, LIQUID, including road oils, and cutback bitumens

Transport hazard class(es)

Class	3
Subrisk	Not Applicable

Packing group III

Environmental hazard Not Applicable

Special precautions for user

Special provisions	Not Applicable
Explosive Limit and Limited Quantity Index	5 L
ERAP Index	Not Applicable

Air transport (ICAO-IATA / DGR)

UN number 1999

UN proper shipping name Tars, liquid including road asphalt and oils, bitumen and cut backs

Transport hazard class(es)

ICAO/IATA Class	3
ICAO/ IATA Subrisk	Not Applicable
ERG Code	3L

Packing group: III

Environmental hazard: Not Applicable

Special precautions for user

Special provisions	A3
Cargo Only Packing Instructions	366
Cargo Only Maximum Qty / Pack	220 L
Passenger and Cargo Packing Instructions	355
Passenger and Cargo Maximum Qty / Pack	60 L
Passenger and Cargo Limited Quantity Packing Instructions	Y344
Passenger and Cargo Limited Maximum Qty / Pack	10 L

Sea transport (IMDG-Code / GGVSee)

UN number: 1999

UN proper shipping name: TARS, LIQUID including road oils, and cutback bitumens

Transport hazard class(es)

IMDG Class	3
IMDG Subrisk	Not Applicable

Packing group: III

Environmental hazard: Not Applicable

Special precautions for user

EMS Number	F-E, S-E
Special provisions	955
Limited Quantities	5 L

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
naphtha petroleum, light aromatic solvent	Not Available
1,2,4-trimethyl benzene	Not Available
Xylene (xylene)	Not Available
cumene	Not Available
trimethylbenzene (mixed isomers)	Not Available
1,3,5-trimethyl benzene	Not Available
1,2,3-trimethyl benzene	Not Available
bitumen (petroleum)	Not Available
naphtha petroleum, heavy, hydrotreated	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Group
naphtha petroleum, light aromatic solvent	Not Available
1,2,4-trimethyl benzene	Not Available
Xylene (xylene)	Not Available
cumene	Not Available

trimethylbenzene (mixed isomers)	Not Available
1,3,5-trimethyl benzene	Not Available
1,2,3-trimethyl benzene	Not Available
bitumen (petroleum)	Not Available
naphtha petroleum, heavy, hydrotreated	Not Available

SECTION 15 - REGULATIONS

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

WHMIS REGULATORY STATUS : This product has been classified in accordance with the hazard criteria of the Canadian Hazardous Products Regulations and the Safety Data Sheet contains all the information required by the Hazardous Products Regulations (WHMIS 2015). This product is WHMIS 2015 controlled.

naphtha petroleum, light aromatic solvent is found on the following regulatory lists

Canada Categorization decisions for all DSL substances
Canada Domestic Substances List (DSL)
Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS GHS
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

1,2,4-trimethyl benzene is found on the following regulatory lists

Canada Categorization decisions for all DSL substances
Canada Domestic Substances List (DSL)
Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS GHS

Xylene (xylene) is found on the following regulatory lists

Canada Categorization decisions for all DSL substances
Canada Domestic Substances List (DSL)
Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS GHS
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

cumene is found on the following regulatory lists

Canada Categorization decisions for all DSL substances
Canada Domestic Substances List (DSL)
Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS GHS
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

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

Canada Categorization decisions for all DSL substances
Canada Domestic Substances List (DSL)
Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS GHS

1,3,5-trimethyl benzene is found on the following regulatory lists

Canada Categorization decisions for all DSL substances
Canada Domestic Substances List (DSL)
Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS GHS

1,2,3-trimethyl benzene is found on the following regulatory lists

Canada Categorization decisions for all DSL substances
Canada Domestic Substances List (DSL)
Canada Toxicological Index Service - Workplace Hazardous Materials Information System - WHMIS GHS

bitumen (petroleum) is found on the following regulatory lists

Canada Categorization decisions for all DSL substances
Canada Domestic Substances List (DSL)
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

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

Canada Categorization decisions for all DSL substances
Canada CEPA Environmental Registry Substance Lists - List of substances on the DSL that are Persistent, Bioaccumulative, and Inherently Toxic to the Environment
Canada Domestic Substances List (DSL)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (naphtha petroleum, light aromatic solvent; 1,2,4-trimethyl benzene; Xylene (xylene); cumene; trimethylbenzene (mixed isomers); 1,3,5-trimethyl benzene; 1,2,3-trimethyl benzene; bitumen (petroleum); naphtha petroleum, heavy, hydrotreated)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (bitumen (petroleum); naphtha petroleum, heavy, hydrotreated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (1,2,3-trimethyl benzene)
Vietnam - NCI	Yes
Russia - FBEPH	No (1,2,3-trimethyl benzene)
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 OF SDS	March 18, 2022
REPLACES THE MSDS/SDS FROM	January 23, 2018
PREPARED BY	Research department
GENERAL INFORMATION	1-888-766-2468
WEBSITE	www.iko.com
OTHER INFO/DISCLAMERS	Read this Safety Data Sheet before handling or disposing of this product. This product safety information is provided to help our customers with health, safety and/or environmental matters. We have taken reasonable effort to ensure that the test methods and sources for this data are correct and reliable, however, we give no warranty, expressed or implied, regarding its correctness. Since conditions or methods of handling and using this product are beyond our control, we do not assume responsibility and expressly disclaim liability for damages resulting from or connected with the handling, storage, use or disposal of the product.