StaBil ® Fast Fix

Trico Products	Chemwatch Hazard Alert Code: 3
Chemwatch: 5530-86 Version No: 5.1 Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG reguirements	Issue Date: 30/06/2023 Print Date: 12/07/2023 L.GHS.AUS.EN
Salety Bala choice according to which regulations (nazaradas chemicals) / inchament 2520 and 7250 requirements	E.ONOEI

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

Product Identifier

Product name	StaBil ® Fast Fix
Chemical Name	Not Applicable
Synonyms	700012; Part No - 22303; 22304
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Fuel additives
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Trico Products		
Address	Unit 1, 80 Fairbank Road Clayton VIC 3169 Australia		
Telephone	61 3 9271 3288		
Fax	+61 3 9271 3290		
Website	http://www.tricoproducts.com		
Email	sales@tricoproducts.com.au		

Emergency telephone number

Association / Organisation	Trico Products CHEMWATCH EMERGENCY RESPONSE (24/7)	
Emergency telephone numbers	+61 3 9271 3288	+61 1800 951 288
Other emergency telephone numbers	13 11 26	+61 3 9573 3188

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

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

COMBUSTIBLE LIQUID, regulated for storage purposes only

Chemwatch Hazard Ratings

	Min	Max	
Flammability	1		
Toxicity	2	1	0 = Minimum
Body Contact	3		1 = Low
Reactivity	1		2 = Moderate
Chronic	2		3 = High 4 = Extreme

Poisons Schedule	S5
Classification ^[1]	Flammable Liquids Category 4, Acute Toxicity (Oral) Category 4, Aspiration Hazard Category 1, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Reproductive Toxicity Category 2, Hazardous to the Aquatic Environment Acute Hazard Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chernwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)



StaBil ® Fast Fix

Signal word Danger

Hazard statement(s)		
H227	Combustible liquid.	
H302	Harmful if swallowed.	
H304	May be fatal if swallowed and enters airways.	
H315	Causes skin irritation.	
H319	Causes serious eye irritation.	
H336	May cause drowsiness or dizziness.	
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.	
H401	Toxic to aquatic life.	
H412	Harmful to aquatic life with long lasting effects.	

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.	
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.	
P271	P271 Use only a well-ventilated area.	
P280	Wear protective gloves, protective clothing, eye protection and face protection.	
P261	Avoid breathing mist/vapours/spray.	
P264	Wash all exposed external body areas thoroughly after handling.	
P270	Do not eat, drink or smoke when using this product.	
P273	Avoid release to the environment.	

Precautionary statement(s) Response

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

Precautionary statement(s) Storage

P405	Store locked up.	
P403+P233	Store in a well-ventilated place. Keep container tightly closed.	

Precautionary statement(s) Disposal

P501

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight] Name		
64742-47-8	40-60	distillates. petroleum, light, hydrotreated	
111-76-2	40-60 ethylene glycol monobutyl ether		
64742-95-6.	1-10	naphtha petroleum. light aromatic solvent	
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L * EU IOELVs available		

SECTION 4 First aid measures

Description of first aid measures				
Eye Contact	If this product comes in contact with the eyes: Immediately hold eyelids apart and flush the eye continuously with running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper 			

StaBil ® Fast Fix

	 and lower lids. Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. Transport to hospital or doctor without delay. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	 If skin or hair contact occurs: Immediately flush body and clothes with large amounts of water, using safety shower if available. Quickly remove all contaminated clothing, including footwear. Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. Transport to hospital, or doctor.
Inhalation	 If fumes or combustion products are inhaled remove from contaminated area. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor, without delay.
Ingestion	 If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice. Avoid giving milk or oils. Avoid giving alcohol. If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

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

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
Advice for firefighters	
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear full body protective clothing with breathing apparatus. Prevent, by any means available, spillage from entering drains or water course. Use water delivered as a fine spray to control fire and cool adjacent area. Avoid spraying water onto liquid pools. DO NOT approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	 WARNING: In use may form flammable/ explosive vapour-air mixtures. Combustible. Slight fire hazard when exposed to heat or flame. Heating may cause expansion or decomposition leading to violent rupture of containers. On combustion, may emit toxic fumes of carbon monoxide (CO). May emit acrid smoke. Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO2) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills

Remove all ignition sources.Clean up all spills immediately.

	 Avoid breathing vapours and cor Control personal contact with the Contain and absorb spill with sai Wipe up. Place in a suitable, labelled cont 	e substand, ear	ance, by u h, inert ma	sing protective aterial or verm		
	Chemical Class: ester and ethers For release onto land: recommended	l sorbe	nts listed ir	n order of prio	prity.	
	SORBENT TYPE RANK APPLIC	ATION	COLLE	ECTION L	LIMITATIONS	
	LAND SPILL - SMALL					
	cross-linked polymer - particulate	1	shovel	shovel	R, W, SS	
	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	
	sorbent clay - particulate	2	shovel	shovel	R,I, P	
	wood fiber - particulate	3	shovel	shovel	R, W, P, DGC	
	wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT	
	treated wood fiber - pillow	3	throw	pitchfork	DGC, RT	
	LAND SPILL - MEDIUM					
	cross-linked polymer - particulate	1	blower	skiploader	R,W, SS	
	cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT	
	sorbent clay - particulate	3	blower	skiploader	R, I, P	
	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	
	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC	
Major Spills	wood fiber - particulate	4	blower	skiploader	R, W, P, DGC	
	Legend DGC: Not effective where ground cor R; Not reusable I: Not incinerable P: Effectiveness reduced when rainy RT:Not effective where terrain is rugg SS: Not for use within environmental W: Effectiveness reduced when winc Reference: Sorbents for Liquid Haza R.W Melvold et al: Pollution Technolo Moderate hazard. Clear area of personnel and mov Alert Fire Brigade and tell them I Wear breathing apparatus plus p Prevent, by any means available No smoking, naked lights or igni Increase ventilation. Stop leak if safe to do so. Contain spill with sand, earth or Collect recoverable product into Absorb remaining product with s Collect solid residues and seal ir Wash area and prevent runoff in I f contamination of drains or wat	y y rdous \$ ogy Re' ve upwi ocatior rotectir , spilla ion sou vermicu labelle and, ea h labelle to drair	itive sites Substance view No. 1 nd. and natur ve gloves. ge from en urces. ulite. d containee arch or verr ad drums fi is.	50: Noyes Da e of hazard. tering drains rs for recyclin niculite. or disposal.	ata Corporation 1988 or water course.	

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

SECTION 7 Handling and storage

Precautions for safe handling Safe handling	 DO NOT allow clothing wet with material to stay in contact with skin Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. Prevent concentration in hollows and sumps. DO NOT enter confined spaces until atmosphere has been checked. Avoid smoking, naked lights or ignition sources. 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.
Other information	 Store in original containers. Keep containers securely sealed. No smoking, naked lights or ignition sources. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	 DO NOT use aluminium or galvanised containers Metal can or drum Packaging as recommended by manufacturer. Check all containers are clearly labelled and free from leaks.
Storage incompatibility	 For alkyl aromatics: The alkyl side chain of aromatic rings can undergo axidation 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 primar 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 Monealkylenzenes 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-rearangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxide undergo Criegee rearrangement easily. Mkrowave 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 smog Oxidation of Alkylaromatics: T.S. S. Raa and Shubhra Awasth: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007 Ethylene glycol monobutyl ether (2-butoxyethanol) and its acetate: May form unstable peroxides in storage is incompatible with oxidisers, permanganates, peroxides, ammonium persulfate, bromine dioxide, nitrates, strong acids, sulfuric acid, nitric acid, perchloric acid Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and stron

X — Must not be stored together

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Х

0 — May be stored together with specific preventions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

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Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	distillates, petroleum, light, hydrotreated	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m3	242 mg/m3 / 50 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
distillates, petroleum, light, hydrotreated	140 mg/m3	1,500 mg/m3	8,900 mg/m3
ethylene glycol monobutyl ether	60 ppm	120 ppm	700 ppm
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
distillates, petroleum, light, hydrotreated	2,500 mg/m3	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available

MATERIAL DATA

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

Exposure controls

	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. Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate				
	protection. Supplied-air type respirator may be required in sp An approved self contained breathing apparatus (SCBA) may Provide adequate ventilation in warehouse or closed storage velocities which, in turn, determine the "capture velocities" of	ecial circumstances. Correct fit is essential to ensure adequ y be required in some situations. area. Air contaminants generated in the workplace possess	uate protection. s varying "escape"		
	Type of Contaminant:	······································	Air Speed:		
	solvent, vapours, degreasing etc., evaporating from tank (in	n still air).	0.25-0.5 m/s (50-100 f/min.)		
Appropriate engineering	aerosols, fumes from pouring operations, intermittent conta drift, plating acid fumes, pickling (released at low velocity ir		0.5-1 m/s (100-200 f/min.)		
controls	direct spray, spray painting in shallow booths, drum filling, generation into zone of rapid air motion)	conveyer loading, crusher dusts, gas discharge (active	1-2.5 m/s (200-500 f/min.)		
	grinding, abrasive blasting, tumbling, high speed wheel ger very high rapid air motion).	nerated dusts (released at high initial velocity into zone of	2.5-10 m/s (500-2000 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			
	Contaminants of low toxicity or of nuisance value only.	Contaminants of high toxicity			
	3: Intermittent, low production.	3: High production, heavy use			
	3: Intermittent, low production. 4: Large hood or large air mass in motion Simple theory shows that air velocity falls rapidly with distance with the square of distance from the extraction point (in simpl accordingly, after reference to distance from the contaminatir	3: High production, heavy use 4: Small hood-local control only e away from the opening of a simple extraction pipe. Veloci e cases). Therefore the air speed at the extraction point sho	ould be adjusted,		
Individual protection	4: Large hood or large air mass in motion Simple theory shows that air velocity falls rapidly with distance with the square of distance from the extraction point (in simpl	3: High production, heavy use 4: Small hood-local control only the away from the opening of a simple extraction pipe. Veloci the cases). Therefore the air speed at the extraction point shing source. The air velocity at the extraction fan, for example in a tank 2 meters distant from the extraction point. Other m	ould be adjusted, , should be a minimum of echanical considerations,		
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	 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 > 20 min Good when breakthrough time > 20 min Fair when breakthrough time > 20 min Por 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 sol 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.
Body protection	See Other protection below
Other protection	 Overalls. P.V.C apron. Barrier cream. Skin cleansing cream. Eye wash unit.

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 $\ensuremath{\textit{computer-generated}}$ selection:

Material	CPI
BUTYL	А
PE/EVAL/PE	А
SARANEX-23	А
NEOPRENE	В
NITRILE	В
PVC	В
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	С
PVA	С

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Type A 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	-	A-PAPR-AUS / Class 1
up to 50 x ES	-	A-AUS / Class 1	-
up to 100 x ES	-	A-2	A-PAPR-2 ^

^ - Full-face

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

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

SECTION 9 Physical and chemical properties

Information on ba	asic physical a	nd chemical p	properties

Appearance	Clear liquid with characteristic odour; does not mix with water		
Physical stateLiquidRelative density (Water = 1)0.8			0.8
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available

pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	3
Initial boiling point and boiling range (°C)	82	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	77	Taste	Not Available
Evaporation rate	>1 Ether = 1	Explosive properties	Not Available
Flammability	Combustible.	Oxidising properties	Not Available
Upper Explosive Limit (%)	7	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	0.8	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	2.3	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	>1	VOC g/L	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation of ten results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation hazard is increased at higher temperatures. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severely toxic effects; these may be fatal.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.
Skin Contact	 Skin contact with the material may produce toxic effects; systemic effects may result following absorption. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant, but mild, 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. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Open cuts, abraded or irritated skin should not be exposed to this material The material may accentuate any pre-existing dermatitis condition 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. Ethylene glycol monobutyl ether (2-butoxyethanol) penetrates the skin easily and toxic effects via this route may be more likely than by inhalation.
Eye	Evidence exists, or practical experience predicts, that the material may cause severe 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. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Chronic	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 servere 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. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to eause a strong suspicion of impaired fertility with vith are not a secondary non-specific consequence of other toxic effects. Univ appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible developmental toxicits (not the save dose such as a strong suspicion of developmental toxicity in the absence of signs of market material toxics and anaemican degenerative changes in the liver and kinkery. Chronic exposure by periodenem vorkers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthresis), psychological and neurophysiological deficits, since cause and with a dest-response relationship inicitating an association betwen routine workplace exposure to petroleum ny one of its constituents and skin cancer, particularly material of produces localised dermatoses. Surface cracking and ferosion may also increase susceptibility to infection by microorganisms. One epidemiclogical study of petroleum refinery w

StaBil ® Fast Fix	TOXICITY	IRRITATION	
	Not Available	Not Available	
	ΤΟΧΙΟΙΤΥ	IRRITATION	
distillates, petroleum, light,	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]	
hydrotreated	Inhalation(Rat) LC50: >4.3 mg/l4h ^[1]	Skin: adverse effect observed (irritating) ^[1]	
	Oral (Rat) LD50: >5000 mg/kg ^[2]		
	тохісіту	IRRITATION	
	dermal (guinea pig) LD50: 210 mg/kg ^[2]	Eye (rabbit): 100 mg SEVERE * [Union Carbide]	
	Inhalation(Rat) LC50: 450 ppm4h ^[2]	Eye (rabbit): 100 mg/24h-moderate	
ethylene glycol monobutyl ether	Oral (Rat) LD50: 250 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]	
chief		Skin (rabbit): 500 mg, open; mild	
		Skin: adverse effect observed (irritating) ^[1]	
		Skin: no adverse effect observed (not irritating) $\left[1 \right]$	
	тохісіту	IRRITATION	
naphtha petroleum, light	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]	
aromatic solvent	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 Substan specified data extracted from RTECS - Register of Toxic E	ces - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwis iffect of chemical Substances	

No significant acute toxicological data identified in literature search. For "kerosenes"

DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED

Acute toxicity: Oral LD50s for three kerosenes (Jet A, CAS No. 8008-20-6 and CAS No. 64742-81-0) ranged from > 2 to >20 g/kg The dermal LD50s of the same three kerosenes were all >2.0 g/kg. Inhalation LC50 values in Sprague-Dawley rats for straight run kerosene (CAS No.

8008-20-6) and hydrodesulfurised kerosene (CAS No. 64742-81-0) were reported to be > 5 and > 5.2 mg/l, respectively. No mortalities in rats were reported in rats when exposed for eight hours to saturated vapor of deodorised kerosene (probably a desulfurised kerosene). Six hour exposures of cats to the same material produced an LC50 of >6.4 mg/l When tested in rabbits for skin irritation, straight run kerosene (CAS No. 8008-20-6) produced "moderate" to "severe" irritation. Six additional skin irritation studies on a range of kerosenes produced "mild" to "severe" irritation An eve irritation in rabbits of straight run kerosene (CAS No. 8008-20-6) produced Draize scores of 0.7 and 2.0 (unwashed and washed eves) at 1 hour. By 24 hours, the Draize scores had returned to zero. Eye irritation studies have also been reported for hydrodesulfurized kerosene and jet fuel. These materials produced more irritation in the unwashed eves at 1 hour than had the straight run kerosene. The eve irritation persisted longer than that seen with straight run kerosene, but by day 7 had resolved. Straight run kerosene (CAS No. 8008-20-6), Jet A, and hydrodesulfurized kerosene (CAS No. 64742-81-0) have not produced sensitisation when tested in guinea pigs Repeat-Dose toxicity: Multiple repeat-dose toxicity studies have been reported on a variety of kerosenes or jet fuels. When applied dermally, kerosenes and jet fuels have been shown to produce dermal and systemic effects Dose levels of 200, 1000 and 2000 mg/kg of a straight run kerosene (CAS No. 8008-20-6) were applied undiluted to the skin of male and female New Zealand white rabbits The test material was applied 3x/week for 28 days. One male and one female in the 2000 mg/kg dose group found dead on days 10 and 24 respectively were thought to be treatment-related. Clinical signs that were considered to be treatment-related included: thinness, nasal discharge, lethargy, soiled anal area, anal discharge, wheezing. The high dose group appeared to have a treatment related mean body weight loss when compared to controls. Dose-related skin irritation was observed, ranging from "slight" to "moderate" in the low and high dose groups, respectively. Other treatment-related dermal findings included cracked, flaky and/or leathery skin, crusts and/or hair loss. Reductions in RBC, haemoglobin and haematocrit were seen in the male dose groups. There were no treatment related effects on a variety of clinical chemistry values. Absolute and relative weights for a number of organs were normal, with the following exceptions that were judged to be treatment-related: increased relative heart weights for the mid- and high- dose males and females. • increased absolute and relative spleen weights in treated females, and • differences in absolute and relative adrenal weights in both male and female treated animals (considered to be stress-related and therefore, indirectly related to treatment). Gross necropsy findings were confined largely to the skin. Enlarged spleens were seen in the female groups. Microscopic examination of tissues taken at necropsy found proliferative inflammatory changes in the treated skin of all male and female animals in the high dose group. These changes were, in the majority of animals, accompanied by an increase in granulopoiesis of the bone marrow. Four of six high dose males had testicular changes (multifocal or diffuse tubular hypoplasia) that were considered by the study authors to be secondary to the skin and/or weight changes In a different study, hydrodesulfurised kerosene was tested in a thirteen-week dermal study using Sprague-Dawley rats. Test material was applied 5x/week to the skin of male and female rats at dose levels of 165, 330 and 495 mg/kg. Aside from skin irritation at the site of application, there were no treatment-related clinical signs during the study. Screening of all animals using a functional observation battery (FOB) did not find any substance-related effects. Opthalomological examination of all animals also found no treatment-related effects. There were no treatmentrelated effects on growth rates, hematological or clinical chemical values, or absolute or relative organ weights. Microscopic examination of tissues from animals surviving to termination found no treatment-related changes, with the exception of a minimal degree of a proliferative and inflammatory changes in the skin. A hydrodesulfurised middle distillate (CAS no. 64742-80-9) has also been tested in a four week inhalation study. In the study, Sprague-Dawley rats were exposed to a nominal concentration of 25mg/m3 kerosene. Exposures were for approximately 6 hr/day, five days each week for four consecutive weeks. There were no treatment-related effects on clinical condition, growth rate, absolute or relative organ weights, or any of the hematological or clinical chemistry determinations. Microscopic examination found no treatment-related changes observed in any tissues. Carcinogenicity: In addition to the repeat-dose studies discussed above, a number of dermal carcinogenicity studies have been performed on kerosenes or jet fuels. .Following the discovery that hydrodesulfurised (HDS) kerosene caused skin tumors in lifetime mouse skin painting studies, the role of dermal irritation in tumor formation was extensively studied. HDS kerosene proved to be a mouse skin tumor promoter rather than initiator, and this promotion required prolonged dermal irritation . If the equivalent dose of kerosene was applied to the skin in manner that did not cause significant skin irritation (eg, dilution with a mineral oil) no skin tumors occurred . Dermal bioavailability studies in mice confirmed that the reduced irritation seen with samples in mineral oil was not due to decreased skin penetration. The effect of chronic acanthosis on the dermal tumorigenicity of a hydrodesulfurised kerosene was studied and the author concluded that hyperplasia was essential for tumor promotion. However, the author also concluded that subacute inflammation did not appear to be a significant factor A sample of a hydrodesulfurised kerosene has been tested in an initiation-promotion assay in male CD-1 mice . Animal survivals were not effected by exposure to the kerosene. The study's authors concluded that the kerosene was not an initiator but it did show tumor promoting activity. In-Vitro (Genotoxicity): The potential in vitro genotoxicities of kerosene and jet fuel have been evaluated in a variety of studies. Standard Ames assays on two kerosene samples and a sample of Jet A produced negative results with/without activation . Modified Ames assays on four kerosenes also produced negative results (with/without activation) except for one positive assay that occurred with activation . The testing of five kerosene and jet fuel samples in mouse lymphoma assays produced a mixture of negative and positive results . Hydrodesulfurized kerosene tested in a sister chromatid exchange assay produced negative results (with/without activation) In-Vivo Genotoxicity: Multiple in vivo genotoxicity studies have been done on a variety of kerosene-based materials. Four samples of kerosene were negative and a sample of Jet A was positive in in vivo bone marrow cytogenetic tests in Sprague-Dawley rats . One of the kerosen samples produced a positive response in male mice and negative results in females when tested in a sister chromatid exchange assay . Both deodorised kerosene and Jet A samples produced negative results in dominant lethal assays. The kerosene was administered to both mice and rats intraperitoneally, while the jet fuel was administered only to mice via inhalation. Reproductive/Developmental Toxicity Either 0, 20, 40 or 60% (v/v) kerosene in mineral oil was applied to the skin of the rats. The dose per body weight equivalents were 0, 165, 330 and 494 mg/kg. Test material was applied daily, 7 days/week from 14 days premating through 20 days of gestation. There were no treatment-related effects on mortality and no clinical signs of toxicity were observed. There were no compoundrelated effects on any of the reproductive/developmental parameters. The authors concluded that the no observable effect level (NOEL) for reproductive/developmental toxicity of HDS kerosene under the treatment conditions of the study was 494 mg/kg/day. Developmental toxicity screening studies on a kerosene and a sample of Jet A have been reported . There were no compound-related deaths in either study. While kerosene produced no clinical signs, the jet fuel produced a dose-related eye irritation (or infection). The signs of irritation lasted from 2 to 8 days with most animals showing signs for 3 days. Neither of the test materials had an effect on body weights or food consumption. Examination of offspring at delivery did not reveal any treatment-related abnormalities, soft tissue changes or skeletal abnormalities. The sex ratio of the fetuses was also unaffected by treatment with either of the compounds. NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the ETHYLENE GLYCOL spongy layer (spongiosis) and intracellular oedema of the epidermis. MONOBUTYL ETHER For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing

with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE in vitro than those of rats. Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA in vitro and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA in vitro. Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in S. typhimurium strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. In vitro cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic. Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE). Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species. At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol. Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility. Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma. 1: NTP Toxicology Program Technical report Series 484, March 2000. Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe] For Low Boiling Point Naphthas (LBPNs): Acute toxicity: LBPNs generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure Most LBPNs are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices. Sensitisation: LBPNs do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies Repeat dose toxicity: The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPN substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNs in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNs, were considered species- and sex-specific These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values. NAPHTHA PETROLEUM, Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNs. The lowest LOAEC identified in LIGHT AROMATIC SOLVENT these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 ma/m3 No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNs and very few non-cancer chronic toxicity studies were identified for other LBPNs. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3 A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported. Genotoxicity: Although few genotoxicity studies were identified for the site-restricted LBPNs, the genotoxicity of several other LBPN substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results

For in vivo genotoxicity tests, LBPNs exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material

was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells. For in vitro genotoxicity studies, LBPNs were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNs exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative for UDS and the mouse lymphoma assay.

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

Carcinogenicity:

Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effects of human exposure to LBPN substances. No inhalation studies assessing the carcinogenicity of the site-restricted LBPNs were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m3 of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously.Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPN group. Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPN substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPN, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans).

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

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

NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m3 (CAS RN 8052-41-3) to 27 687 mg/m3 (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m3 to 27 059 mg/m3 for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m3, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted. For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13.

For most LBPNs, no treatment-related developmental effects were observed by the different routes of exposure However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m3 delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

For trimethylbenzenes:

Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption . 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion . After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of 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-dimethylbippuric 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. 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 nervou

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 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

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/m 3 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.

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 tests 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 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 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 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 F3 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. For petroleum: This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-c		
DISTILLATES, PETROLEUM, LIGHT, HYDROTREATED & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	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.		
Acute Toxicity	✓	Carcinogenicity	×
Skin Irritation/Corrosion	×	Reproductivity	×
Serious Eye Damage/Irritation	 ✓ 	STOT - Single Exposure	×
Respiratory or Skin sensitisation	×	STOT - Repeated Exposure	×

SECTION 12 Ecological information

Mutagenicity X

	Endpoint	Test Duration (hr)	Species	Value	Source
StaBil ® Fast Fix	Not Available	Not Available	Not Available	Not Available	Not Availabl
	Endpoint	Test Duration (hr)	Species	Value	Source
distillates, petroleum, light, hydrotreated	LC50	96h	Fish	2.2mg/l	4
nyuroneateu	NOEC(ECx)	3072h	Fish	1mg/l	1
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	623mg/l	2
	EC50	48h	Crustacea	164mg/l	2
ethylene glycol monobutyl ether	EC50	96h	Algae or other aquatic plants	720mg/l	2
L	LC50	96h	Fish	1700mg/l	Not Availabl
	EC10(ECx)	48h	Crustacea	7.2mg/l	2
	Endpoint	Test Duration (hr)	Species	Value	Sourc
naphtha petroleum, light aromatic solvent	EC50	72h	Algae or other aquatic plants	19mg/l	1
	EC50	48h	Crustacea	6.14mg/l	1
aromatic solvent	EC50	96h	Algae or other aquatic plants	64mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1

E Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA,

Aspiration Hazard 🗸

Legend:

➤ - Data either not available or does not fill the criteria for classification ➤ - Data available to make classification Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment. **DO NOT** discharge into sewer or waterways.

Persistence and degradability

Persistence: Water/Soil	Persistence: Air
LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
Bioaccumulation	
LOW (BCF = 159)	
LOW (BCF = 2.51)	
Mobility	
HIGH (KOC = 1)	
	LOW (Half-life = 56 days) Bioaccumulation LOW (BCF = 159) LOW (BCF = 2.51) Mobility

SECTION 13 Disposal considerations

Waste treatment methods	
Product / Packaging disposal	 Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. Otherwise: If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product. DO NOT allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. Recycle wherever possible or consult manufacturer for recycling options. Consult State Land Waste Authority for disposal. Bury or incinerate residue at an approved site. Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

COMBUSTIBLE LIQUID	COMBUSTIBLE LIQUID, regulated for storage purposes only	
Marine Pollutant	NO	
HAZCHEM	Not Applicable	

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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
distillates, petroleum, light, hydrotreated	Not Available
ethylene glycol monobutyl ether	Not Available
naphtha petroleum, light aromatic solvent	Not Available

Transport in bulk in accordance with the IGC Code

Product name	Ship Type		
distillates, petroleum, light, hydrotreated	Not Available		
ethylene glycol monobutyl ether	Not Available		
naphtha petroleum, light aromatic solvent	Not Available		

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

distillates, petroleum, light, hydrotreated is found on the following regulatory lists	
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC
Australian Inventory of Industrial Chemicals (AIIC)	Monographs
Chemical Footprint Project - Chemicals of High Concern List	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
ethylene glycol monobutyl ether is found on the following regulatory lists	
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
naphtha petroleum, light aromatic solvent is found on the following regulatory lists	
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	Chemical Footprint Project - Chemicals of High Concern List

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

Chemical Footprint Project - Chemicals of High Concern List International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

National Inventory Status

National Inventory	Status			
Australia - AIIC / Australia Non-Industrial Use	Yes			
Canada - DSL	Yes			
Canada - NDSL	No (distillates, petroleum, light, hydrotreated; ethylene glycol monobutyl ether; naphtha petroleum, light aromatic solvent)			
China - IECSC	Yes			
Europe - EINEC / ELINCS / NLP	Yes			
Japan - ENCS	/es			
Korea - KECI	Yes			
New Zealand - NZIoC	Yes			
Philippines - PICCS	Yes			
USA - TSCA	Yes			
Taiwan - TCSI	Yes			
Mexico - INSQ	Yes			
Vietnam - NCI	Yes			
Russia - FBEPH	Yes			
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	30/06/2023
Initial Date	01/05/2022

SDS Version Summary

Version	Date of Update	Sections Updated
4.1	10/03/2023	Classification change due to full database hazard calculation/update.
5.1	30/06/2023	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), First Aid measures - Advice to Doctor, Toxicological information - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Exposure controls / personal Protection (other), Exposure controls / personal Protection (eye), Exposure controls / personal Protection (other), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major)

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chernwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

- PC TWA: Permissible Concentration-Time Weighted Average
- PC STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations ES: Exposure Standard

end of SDS

OSF: Odour Safety Factor NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index AIIC: Australian Inventory of Industrial Chemicals DSL: Domestic Substances List NDSL: Non-Domestic Substances List IECSC: Inventory of Existing Chemical Substance in China EINECS: European INventory of Existing Commercial chemical Substances ELINCS: European List of Notified Chemical Substances NLP: No-Longer Polymers ENCS: Existing and New Chemical Substances Inventory KECI: Korea Existing Chemicals Inventory NZIOC: New Zealand Inventory of Chemicals PICCS: Philippine Inventory of Chemicals and Chemical Substances TSCA: Toxic Substances Control Act TCSI: Taiwan Chemical Substance Inventory INSQ: Inventario Nacional de Sustancias Químicas NCI: National Chemical Inventory FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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