ANDREW MINERAL TURPENTINE

Damar Industries Limited

Version No: 1.3

Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Chemwatch Hazard Alert Code: 2

Issue Date: **15/11/2021** Print Date: **02/06/2022** L.GHS.NZL.EN

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

Product Identifier					
Product name	ANDREW MINERAL TURPENTINE				
Chemical Name	Not Applicable				
Synonyms	ALE0103; ALF0103; ALG0103; ALK0103; ALP0103; ALQ0103				
Proper shipping name	TURPENTINE SUBSTITUTE				
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 Industrial solvent: Cleaning and degreasing

Details of the supplier of the safety data sheet

Registered company name	Damar Industries Limited			
Address	800 Te Ngae Road, Eastgate Park, Rotorua 3042 New Zealand			
Telephone	7 345 6007			
Fax	+64 7 345 6019			
Website	www.damarindustries.com			
Email	info@damarindustries.co.nz			

Emergency telephone number

Association / Organisation	CHEMCALL		
Emergency telephone numbers	0800 243 622		
Other emergency telephone numbers	1800 127 406 (outside New Zealand)		

SECTION 2 Hazards identification

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Classified as Dangerous Goods for transport purposes.

Classification [1]	Flammable Liquids Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Reproductive Toxicity Category 2, Aspiration Hazard Category 1, Carcinogenicity Category 2			
Legend:	. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI			
Determined by Chemwatch using GHS/HSNO criteria	3.1C, 6.1E (aspiration), 6.7B, 6.8B, 6.9B, 9.1B, 6.1E (respiratory tract irritant)			

Label elements

Hazard pictogram(s)









Signal word Dange

Hazard statement(s)

H226	Flammable liquid and vapour.		
H411	Toxic to aquatic life with long lasting effects.		
H373	lay cause damage to organs through prolonged or repeated exposure.		
H335	May cause respiratory irritation.		
H361	Suspected of damaging fertility or the unborn child.		

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H304	May be fatal if swallowed and enters airways.		
H351	Suspected of causing cancer.		

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.			
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.			
P260	Do not breathe mist/vapours/spray.			
P271	se only a well-ventilated area.			
P280	ear protective gloves and protective clothing.			
P240	Ground and bond container and receiving equipment.			
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.			
P242	Use non-sparking tools.			
P243	Take action to prevent static discharges.			
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	case of fire: Use alcohol resistant foam or normal protein foam to extinguish.			
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.			
P391	Collect spillage.			
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].			
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.			

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name		
64742-82-1.	48-58	naphtha petroleum, heavy, hydrodesulfurised		
Not Available	:	Contains		
95-63-6	2-9	1.2.4-trimethyl benzene		
108-67-8	0.6-3	1.3.5-trimethyl benzene		
100-41-4	<=0.3	ethylbenzene		
64742-95-6	47-52	naphtha petroleum, light aromatic solvent		
Not Available	:	contains		
98-82-8	1-<5	cumene		
108-67-8*	5-<10	Mesitylene (1,3,5-trimethyl benzene)		
91-20-3	<1	naphthalene		
95-63-6*	30-35	Pseudocumene (1,2,4-trimethylbenzene)		
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L * EU IOELVs available			

SECTION 4 First aid measures

Description of first aid measures

Eye Contact

If this product comes in contact with the eyes:

- Wash out immediately with fresh running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.
- Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

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Skin Contact	If skin contact occurs: Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available). Seek medical attention in event of irritation.					
Inhalation	 If fumes, aerosols or combustion products are inhaled remove from contaminated area. Other measures are usually unnecessary. 					
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. 					

Indication of any immediate medical attention and special treatment needed

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

for naphthalene intoxication; Naphthalene requires hepatic and microsomal activation prior to the production of toxic effects. Liver microsomes catalyse the initial synthesis of the reactive 1.2-epoxide intermediate which is subsequently oxidised to naphthalene dihydrodiol and alpha-naphthol. The 2-naphthoguinones are thought to produce haemolysis, the 1,2-naphthoquinones are thought to be responsible for producing cataracts in rabbits, and the glutathione-adducts of naphthalene-1,2-oxide are probably responsible for pulmonary toxicity. Suggested treatment regime:

- Induce emesis and/or perform gastric lavage with large amounts of warm water where oral poisoning is suspected.
- Instill a saline cathartic such as magnesium or sodium sulfate in water (15 to 30g).
- Demulcents such as milk, egg white, gelatin, or other protein solutions may be useful after the stomach is emptied but oils should be avoided because they promote absorption.
- If eyes/skin contaminated, flush with warm water followed by the application of a bland ointment.
- Severe anaemia, due to haemolysis, may require small repeated blood transfusions, preferably with red cells from a non-sensitive individual.
- Where intravascular haemolysis, with haemoglobinuria occurs, protect the kidneys by promoting a brisk flow of dilute urine with, for example, an osmotic diuretic such as mannitol. It may be useful to alkalinise the urine with small amounts of sodium bicarbonate but many researchers doubt whether this prevents blockage of the renal tubules
- Use supportive measures in the case of acute renal failure. GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, 5th Ed.

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 Alert Fire Brigade and tell them location and nature of hazard. May be violently or explosively reactive Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. If safe, switch off electrical equipment until vapour fire hazard removed. Fire Fighting 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.
- 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).
- Fire/Explosion Hazard Combustion products include:

carbon monoxide (CO)

carbon dioxide (CO2)

other pyrolysis products typical of burning organic material.

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May emit clouds of acrid smoke

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

- Remove all ignition sources.
- Clean up all spills immediately.
- Minor Spills
- 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.

Chemical Class: aromatic hydrocarbons

For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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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

Major Spills

Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT:Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

- ► Clear area of personnel and move upwind.
- Alert Fire Brigade and tell them location and nature of hazard.
- May be violently or explosively reactive.
- Wear breathing apparatus plus protective gloves.
- Prevent, by any means available, spillage from entering drains or water course.
- Consider evacuation (or protect in place).
- ▶ No smoking, naked lights or ignition sources.
- Increase ventilation.
- Stop leak if safe to do so.
- ► Water spray or fog may be used to disperse /absorb vapour.
- Contain spill with sand, earth or vermiculite.
- ▶ Use only spark-free shovels and explosion proof equipment.
- ▶ Collect recoverable product into labelled containers for recycling.
- Absorb remaining product with sand, earth or vermiculite.
 Collect solid residues and seal in labelled drums for disposal
- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling

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

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

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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.
- 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.
- Find the 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

Other information

Storage incompatibility

- 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) Suitable container
 - 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.

- ready 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
- Low molecular weight alkanes:

May react violently with strong oxidisers, chlorine, chlorine dioxide, dioxygenyl tetrafluoroborate.

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- ▶ 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.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	naphtha petroleum, heavy, hydrodesulfurised	White spirits (Stoddard solvent)	100 ppm / 525 mg/m3	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	ethylbenzene	Ethyl benzene	100 ppm / 434 mg/m3	543 mg/m3 / 125 ppm	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	cumene	Cumene	25 ppm / 125 mg/m3	375 mg/m3 / 75 ppm	Not Available	(skin)-Skin absorption
New Zealand Workplace Exposure Standards (WES)	naphthalene	Naphthalene	0.5 ppm / 2.6 mg/m3	10 mg/m3 / 2 ppm	Not Available	(skin)-Skin absorption 6.7B-Suspected carcinogen

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
naphtha petroleum, heavy, hydrodesulfurised	300 mg/m3	1,800 mg/m3	29500** 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
1,3,5-trimethyl benzene	Not Available	Not Available	480 ppm
ethylbenzene	Not Available	Not Available	Not Available
naphtha petroleum, light aromatic solvent	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
cumene	Not Available	Not Available	Not Available
Mesitylene (1,3,5-trimethyl benzene)	Not Available	Not Available	480 ppm
naphthalene	15 ppm	83 ppm	500 ppm
Pseudocumene (1,2,4-trimethylbenzene)	140 mg/m3	360 mg/m3	2,200 mg/m3
Pseudocumene (1,2,4-trimethylbenzene)	Not Available	Not Available	480 ppm

Ingredient	Original IDLH	Revised IDLH
naphtha petroleum, heavy, hydrodesulfurised	20,000 mg/m3	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
ethylbenzene	800 ppm	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
cumene	900 ppm	Not Available
Mesitylene (1,3,5-trimethyl benzene)	Not Available	Not Available
naphthalene	250 ppm	Not Available
Pseudocumene (1,2,4-trimethylbenzene)	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
1,2,4-trimethyl benzene	E	≤ 0.1 ppm
1,3,5-trimethyl benzene	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.

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Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
naphtha petroleum, light aromatic solvent	Е	≤ 0.1 ppm
Mesitylene (1,3,5-trimethyl benzene)	E	≤ 0.1 ppm
Pseudocumene (1,2,4-trimethylbenzene)	E	≤ 0.1 ppm
Notes:	Occupational exposure banding is a process of assigning chemicals into sadverse health outcomes associated with exposure. The output of this processing of exposure concentrations that are expected to protect worker hea	ocess is an occupational exposure band (OEB), which corresponds to a

MATERIAL DATA

for naphthalene:

Odour Threshold Value: 0.038 ppm

The TLV-TWA is thought to be low enough to prevent ocular toxicity but the margin of safety associated with the TLV for hypersusceptible individuals (with glucose-6-phosphate dehydrogenase defective erythrocytes) to naphthalene-induced blood dyscrasias is unknown. Individual sensitivity to inhaled naphthalene-induced haemotoxicity varies greatly with even small doses producing acute haemolysis in some.

Odour Safety Factor(OSF)

OSF=1.2E2 (NAPHTHALENE)

For white spirit:

Low and high odour thresholds of 5.25 and 157.5 mg/m3, respectively, were considered to provide a rather useful index of odour as a warning property.

The TLV-TWA is calculated from data on the toxicities of the major ingredients and is intended to minimise the potential for irritative and narcotic effects, polyneuropathy and kidney damage produced by vapours.

The NIOSH (USA) REL-TWA of 60 ppm is the same for all refined petroleum solvents. NIOSH published an occupational "action level" of 350 mg/m3 for exposure to Stoddard solvent, assuming a 10-hour work shift and a 40-hour work-week. The NIOSH-REL ceiling of 1800 mg/m3 was established to protect workers from short-term effects that might produce vertigo or other adverse effects which might increase the risk of occupational accidents. Combined (gross) percutaneous absorption and inhalation exposure (at concentrations associated with nausea) are thought, by some, to be responsible for the development of frank hepatic toxicity and jaundice.

Odour Safety Factor (OSF)

OSF=0.042 (white spirit)

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:

ClassOSF Description

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

for ethyl benzene:

Odour Threshold Value: 0.46-0.60 ppm

NOTE: Detector tubes for ethylbenzene, measuring in excess of 30 ppm, are commercially available.

Ethyl benzene produces irritation of the skin and mucous membranes and appears to produce acute and chronic effects on the central nervous system. Animal experiments also suggest the effects of chronic exposure include damage to the liver, kidneys and testes. In spite of structural similarities to benzene, the material does not appear to cause damage to the haemopoietic system. The TLV-TWA is thought to be protective against skin and eye irritation. Exposure at this concentration probably will not result in systemic effects. Subjects exposed at 200 ppm experienced transient irritation of the eyes; at 1000 ppm there was eye irritation with profuse lachrymation; at 2000 ppm eye irritation and lachrymation were immediate and severe accompanied by moderate nasal irritation, constriction in the chest and vertigo; at 5000 ppm exposure produced intolerable irritation of the eyes and throat.

Odour Safety Factor(OSF)

OSF=43 (ETHYL BENZENE)

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.

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.

Appropriate engineering controls

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

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direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active

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circulating air required to effectively remove the contaminant. Air Speed: Type of Contaminant: 0.25-0.5 m/s solvent, vapours, degreasing etc., evaporating from tank (in still air). (50-100 f/min.) 0.5-1 m/s aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, (100-200 plating acid fumes, pickling (released at low velocity into zone of active generation) f/min.)

Within each range the appropriate value depends on:

generation into zone of rapid air motion)

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.

- Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance.
- Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures.
- Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered.. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)

Personal protection









Eye and face protection

Safety glasses with side shields.

Chemical goggles

Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

Body protection

See Other protection below

- Overalls.
- PVC Apron.
- PVC protective suit may be required if exposure severe.
- Evewash unit.
- Ensure there is ready access to a safety shower.

Other protection

- 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:

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Material	СРІ
BUTYL	С
NATURAL RUBBER	С

1-2.5 m/s

(200-500

f/min.)

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NEOPRENE	C
NITRILE	С
PVA	С
TEFLON	C
VITON	С

^{*} CPI - Chemwatch Performance Index

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Clear, colourless liquid		
Physical state	Liquid	Relative density (Water = 1)	0.81-0.82
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>200
pH (as supplied)	Not Applicable	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	154-192	Molecular weight (g/mol)	Not Available
Flash point (°C)	41	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Flammable.	Oxidising properties	Not Available
Upper Explosive Limit (%)	0.7	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	0.6	Volatile Component (%vol)	100
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (Not Available%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
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

The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.

Inhalation hazard is increased at higher temperatures.

Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo

Inhaled

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

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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.

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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.

Inhalation of naphthalene vapour has been associated with headache, loss of appetite and nausea. Other conditions associated with exposure to the vapour include optic neuritis, corneal injury and kidney damage. Animals exposed to aerosols of a refined commercial solvent mixture consisting primarily of mono-methylated naphthalenes, exhibited dyspnoea. When animals were exposed to this mixture for 27 daily one-hour exposures over a 35-day period, they showed dyspnoea, listlessness, prostration and marked salivation. Weight loss was evident in mice but not in other species. Pathological changes occurred in the lungs, liver and skin. Pulmonary changes consisted mainly of oedema, bronchopneumonia, emphysema, and thickening of the parabronchiolar alveolar septa. Haematology did not identify significant changes. 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 Exposure to white spirit, in a controlled inhalation study using volunteers either at rest or during exercise, (1000 or 2500 mg/m3 for 30 minutes) produced a linear relationship between alveolar and arterial concentrations of the individual solvent components. Pulmonary absorption of the aliphatics ranged from 46-59%, whilst that of aromatic ranged from 58-70%. Although systemic absorption was greater during exercise, the proportion of circulating aliphatic to aromatic components decreased with increased activity. Exposure to 2500 - 5000 mg/m3 produces nausea

The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.

Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.

The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.

Ingestion

Ingestion of naphthalene and its congeners may produce abdominal cramps with nausea, vomiting, diarrhoea, headache, profuse perspiration, listlessness, confusion, and in severe poisonings, coma with or without convulsions. Irritation of the urinary bladder may also occur (presumably due to the excretory products of naphthalene metabolism) and produce urgency, dysuria, and the passage of brown or black urine with or without albumin or casts. These effects may disappear within a few days and have not been associated with haemolysis which is a prominent finding in naphthalene poisoning. Severe naphthalene poisoning in humans produces haemoglobinuria, methaemoglobinaemia, the production of Heinz bodies and death. Methaemoglobinemia produces a form of oxygen starvation (anoxia). Symptoms include cyanosis (a bluish discolouration skin and mucous membranes) and breathing difficulties. Symptoms may not be evident until several hours after exposure. At about 15% concentration of blood methaemoglobin there is observable cyanosis of the lips, nose and earlobes. Symptoms may be absent although euphoria, flushed face and headache are commonly experienced. At 25-40%, cyanosis is marked but little disability occurs other than that produced on physical exertion. At 40-60%, symptoms include weakness, dizziness, lightheadedness, increasingly severe headache, ataxia, rapid shallow respiration, drowsiness, nausea, vomiting, confusion, lethargy and stupor. Above 60% symptoms include dyspnea, respiratory depression, tachycardia or bradycardia, and convulsions. Levels exceeding 70% may be fatal. In those who survive haemotoxic effects, life-threatening acute renal failure, secondary to renal blockade, occurs. The acute lethal dose of naphthalene is estimated to be between 5 and 15 grams, although certain susceptible individuals have died after ingestion of a total dose of 2 grams. Hypersusceptibility, based on congenital deficiency of glucose-6-phosphate dehydrogenase activity, has been identified and is more common amongst Asians, Arabs, Caucasians of Latin ancestry and American and African blacks; males in particular are sensitive.

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.

Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.

Skin Contact

Workers sensitised to naphthalene and its congeners show exfoliative dermatitis. Hypersensitivity, with positive patch tests, has been demonstrated in certain individuals. Percutaneous absorption is apparently inadequate to produce acute systemic reactions, except in new-born babies. Tests with a refined commercial liquid grade of methylnaphthalene (MN), placed under a patch for 48 hours on human skin produced slight to moderate reactions. In rabbits, a single dermal exposure to MN produced loss of appetite (anorexia). Repeated application of the refined commercial grade of MN to rabbit skin at 1-4 mg/kg/day for up to 21 days produced severe skin irritation and necrosis. Anorexia, moderate weight loss and fatalities were also recorded. Pathological changes in one animal were identified in the liver, stomach, heart, bone marrow, spleen, thyroid and thigh muscle. In another animal there was moderate hyperplasia of the bone marrow and thyroid. Photosensitisation has been recorded amongst workers exposed to naphthalene. An abnormal adverse reaction to ultraviolet (UV) and/or visible radiation results in sun-burn like responses or oedematous, vesiculated lesions or bullae.

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 material may accentuate any pre-existing dermatitis condition

Skin contact with the material may be harmful; systemic effects may result following absorption.

Eye

Exposure to naphthalene and its congeners has produced cataracts in animals and workers. In one study, eight of twenty-one workers, exposed to naphthalene for 5-years, showed opacities of the lens.

Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.

The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with

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possible permanent impairment of vision, if not promptly and adequately treated.

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 conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.

Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects.

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

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:

myelotoxic compounds, the most notable being benzene.

Chronic

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. Follicular dermatitis may develop rapidly on repeated immersion of the hands and forearms in white spirits. A Belgian report, produced in 1958, described sub-chronic toxicity amongst workers exposed to white spirit (83% paraffins, 17% aromatics) over a 4 month period. These workers complained of nausea and vomiting and one developed aplastic anaemia; bone marrow depression was confirmed. This employee died several

In a two-year inhalation study, groups of mice were exposed at 0, 10 or 30 ppm naphthalene, 6 hours/day, 5 days/week for 103 weeks. Female mice showed an increase of pulmonary alveolar/bronchiolar adenomas at 30 ppm. There was no increase in the incidence of tumours in male mice. Naphthalene inhalation was associated with an increase in the incidence and severity of chronic inflammation, metaplasia of the olfactory epithelium, and hyperplasia of the respiratory epithelium in the nose, and chronic inflammation of the lungs of both sexes.

months later as a result of septicaemia. Bone marrow depression, associated with human exposure, might be explained by the presence of

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.

Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]

Prolonged or repeated contact with xylenes may cause defatting dermatitis with drying and cracking. Chronic inhalation of xylenes has been associated with central nervous system effects, loss of appetite, nausea, ringing in the ears, irritability, thirst anaemia, mucosal bleeding, enlarged liver and hyperplasia. Exposure may produce kidney and liver damage. In chronic occupational exposure, xylene (usually mix ed with other solvents) has produced irreversible damage to the central nervous system and ototoxicity (damages hearing and increases sensitivity to noise), probably due to neurotoxic mechanisms.

Industrial workers exposed to xylene with a maximum level of ethyl benzene of 0.06 mg/l (14 ppm) reported headaches and irritability and tired quickly. Functional nervous system disturbances were found in some workers employed for over 7 years whilst other workers had enlarged livers. Xylene has been classed as a developmental toxin in some jurisdictions.

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

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	Not Available	Not Available
	TOXICITY	IRRITATION
naphtha petroleum, heavy,	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
hydrodesulfurised	Inhalation(Rat) LC50; >1.58 mg/l4h ^[1]	Skin: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50; >4500 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
1,2,4-trimethyl benzene	Dermal (rabbit) LD50: >3160 mg/kg ^[2]	Not Available

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Inhalation(Rat) LC50; 18 mg/L4h^[2] Oral (Rat) LD50; 6000 mg/kg^[1] TOXICITY IRRITATION Eye (rabbit): 500 mg/24h mild dermal (rat) LD50: >3460 mg/kg[1] Inhalation(Rat) LC50; 24 mg/L4h^[2] Eye: adverse effect observed (irritating)^[1] 1,3,5-trimethyl benzene Skin (rabbit): 20 mg/24h moderate Oral (Rat) LD50; 6000 mg/kg^[1] Skin: adverse effect observed (irritating)^[1] TOXICITY IRRITATION Dermal (rabbit) LD50: 17800 $mg/kg^{[2]}$ Eye (rabbit): 500 mg - SEVERE Inhalation(Rat) LC50; 17.2 mg/l4h $^{[2]}$ Eye: no adverse effect observed (not irritating) $^{[1]}$ ethylbenzene Oral (Rat) LD50; 3500 mg/kg^[2] Skin (rabbit): 15 mg/24h mild Skin: no adverse effect observed (not irritating)^[1] TOXICITY Dermal (rabbit) LD50: >1900 mg/kg^[1] Eye: no adverse effect observed (not irritating)^[1] naphtha petroleum, light aromatic solvent Skin: adverse effect observed (irritating)^[1] Inhalation(Rat) LC50; >4.42 mg/L4h^[1] Oral (Rat) LD50; >4500 mg/kg^[1] 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] cumene Skin (rabbit): 10 mg/24h mild Skin (rabbit):100 mg/24h moderate Skin: no adverse effect observed (not irritating)^[1] TOXICITY IRRITATION dermal (rat) LD50: >3460 mg/kg^[1] Eye: adverse effect observed (irritating)^[1] Mesitylene (1,3,5-trimethyl benzene) Inhalation(Rat) LC50; 24 mg/L4h^[2] Skin: adverse effect observed (irritating)[1] Oral (Rat) LD50; 6000 mg/kg^[1] TOXICITY IRRITATION dermal (rat) LD50: >2500 mg/kg[2] Eye (rabbit): 100 mg - mild naphthalene Inhalation(Rat) LC50; >0.4 mg/l4h[1] Skin (rabbit):495 mg (open) - mild Oral (Rat) LD50; 490 mg/kg^[2] IRRITATION TOXICITY Not Available Dermal (rabbit) LD50: >3160 mg/kg^[2] Pseudocumene (1.2.4trimethylbenzene) Inhalation(Rat) LC50; 18 mg/L4h^[2] Oral (Rat) LD50; 6000 mg/kg^[1] 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise Leaend: specified data extracted from RTECS - Register of Toxic Effect of chemical Substances Data demonstrate that during inhalation exposure, aromatic hydrocarbons undergo substantial partitioning into adipose tissues. Following

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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

NAPHTHA PETROLEUM. HEAVY. **HYDRODESULFURISED**

No significant acute toxicological data identified in literature search.

For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead Version No: **1.3** Page **13** of **24** Issue Date: **15/11/2021**

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to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation.

Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans.

Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants).

Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus.

Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials.

Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.

1,2,4-TRIMETHYL BENZENE

CHEMWATCH 2325 1,3,5-trimethylbenzene

1,3,5-TRIMETHYL BENZENE

CHEMWATCH 12171 1,2,4-trimethylbenzene

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

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

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

ETHYLBENZENE

Hearing loss has been reported in rats (but not guinea pigs) exposed to relatively high exposures (400 ppm and greater) of ethylbenzene In chronic toxicity/carcinogenicity studies, both rats and mice were exposed via inhalation to 0, 75, 250 or 750 ppm for 104 weeks. In rats, the kidney was the target organ of toxicity, with renal tubular hyperplasia noted in both males and females at the 750 ppm level only. In mice, the liver and lung were the principal target organs of toxicity. In male mice at 750 ppm, lung toxicity was described as alveolar epithelial metaplasia, and liver toxicity was described as hepatocellular syncitial alteration, hypertrophy and mild necrosis; this was accompanied by increased follicular cell hyperplasia in the thyroid. As a result the NOAEL in male mice was determined to be 250 ppm. In female mice, the 750 ppm dose group had an increased incidence of eosinophilic foci in the liver (44% vs 10% in the controls) and an increased incidence in follicular cell hyperplasia in the thyroid pland.

In studies conducted by the U.S. National Toxicology Program, inhalation of ethylbenzene at 750 ppm resulted in increased lung tumors in male mice, liver tumors in female mice, and increased kidney tumors in male and female rats. No increase in tumors was reported at 75 or 250 ppm. Ethylbenzene is considered to be an animal carcinogen, however, the relevance of these findings to humans is currently unknown. Although no reproductive toxicity studies have been conducted on ethylbenzene, repeated-dose studies indicate that the reproductive organs are not a target for ethylbenzene toxicity

Ethylbenzene was negative in bacterial gene mutation tests and in a yeast assay on mitotic recombination.

NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT

* [Devoe] .

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.

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

CUMENE

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 payments.

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 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."

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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]

ANDREW MINERAL TURPENTINE & NAPHTHA PETROLEUM, HEAVY, HYDRODESULFURISED 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.

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

For trimethylbenzenes: Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the

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-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

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) tosted.

headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had

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 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/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.

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

ANDREW MINERAL
TURPENTINE & NAPHTHA
PETROLEUM, HEAVY,
HYDRODESULFURISED &
1,2,4-TRIMETHYL BENZENE &
1,3,5-TRIMETHYL BENZENE &
NAPHTHA PETROLEUM,
LIGHT AROMATIC SOLVENT &
Mesitylene (1,3,5-trimethyl
benzene) & Pseudocumene
(1,2,4-trimethylbenzene)

NAPHTHA PETROLEUM, HEAVY, HYDRODESULFURISED & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT Version No: **1.3** Page **15** of **24** Issue Date: **15/11/2021**

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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 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.

1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE

Other Toxicity data is available for CHEMWATCH 12172 1,2,3-trimethylbenzene

1,2,4-TRIMETHYL BENZENE &
1,3,5-TRIMETHYL BENZENE &
NAPHTHA PETROLEUM,
LIGHT AROMATIC SOLVENT &
CUMENE & Mesitylene (1,3,5trimethyl benzene) &
Pseudocumene (1,2,4trimethylbenzene)

Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

1,3,5-TRIMETHYL BENZENE & NAPHTHALENE

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

1,3,5-TRIMETHYL BENZENE & CUMENE

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.

ETHYLBENZENE & NAPHTHALENE

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

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ANDREW MINERAL TURPENTINE

ETHYLBENZENE & CUMENE & NAPHTHALENE WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. **Acute Toxicity** Carcinogenicity × ~ Skin Irritation/Corrosion Reproductivity Serious Eye Damage/Irritation STOT - Single Exposure Respiratory or Skin × STOT - Repeated Exposure sensitisation × Mutagenicity **Aspiration Hazard**

Legend:

X − Data either not available or does not fill the criteria for classification
 y − Data available to make classification

SECTION 12 Ecological information

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Тο		

ANDREW MINERAL	Endpoint	Test Duration (hr)		Species		Value	Source
TURPENTINE Not Avail		Not Available		Not Available		Not Available	Not Availab
	Endpoint	Test Duration (hr)		Species		Value	Source
	EC50(ECx)	72h		Algae or other aquatic plants		391mg/l	2
	EC50	72h		Algae or other aquatic plants		391mg/l	2
naphtha petroleum, heavy,	NOEC(ECx)	504h		Crustacea		0.097mg/l	2
	EC50	72h		Algae or other aquatic plants		0.53mg/l	2
hydrodesulfurised	EC50	96h		Algae or other aquatic plants		0.58mg/l	2
	NOEC(ECx)	720h	Crustacea		0.024mg/l	2	
	LC50	96h		Fish		0.14mg/l	2
	EC50	96h		Algae or other aquatic plants		0.277mg/l	2
	Endpoint	Test Duration (hr)	:	Species		Value	Source
	BCF	1344h		Fish		31-207	7
1,2,4-trimethyl benzene	EC50(ECx)	96h		Algae or other aquatic plants		2.356mg/l	2
	LC50	96h		Fish		3.41mg/l	2
	EC50	48h		Crustacea		ca.6.14mg/l	1
	EC50	96h		Algae or other aquatic plants		2.356mg/l	2
	Endpoint	Test Duration (hr)		Species		Value	Sour
	BCF	1680h	Fish			23-342	7
	EC50	48h	Crustacea			13mg/L	5
1,3,5-trimethyl benzene	EC50	96h Algae or other aquatic plants			3.084mg/l	2	
	NOEC(ECx)	384h Crustacea			0.257mg/l	2	
	LC50	96h		Fish		5.216mg/l	2
	Endpoint	Test Duration (hr)	Sp	pecies	Valu	ıe	Sour
	NOEC(ECx)	720h	Fis	sh	0.38	31mg/L	4
-4h.db	LC50	96h	Fis	sh	3.38	31-4.075mg/L	4
ethylbenzene	EC50	72h	Alg	gae or other aquatic plants	4.6r	ng/l	1
	EC50	48h	Cr	ustacea	1.37	7-4.4mg/l	4
	EC50	96h	Alg	gae or other aquatic plants	3.6r	ng/l	2
	Endpoint	Test Duration (hr)		Species		Value	Sour
manhtha naturburu Pata	NOEC(ECx)	72h		Algae or other aquatic plants		1mg/l	1
naphtha petroleum, light aromatic solvent	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
	Endpoint	Test Duration (hr)		Species		Value	Source
	NOEC(ECx)	96h		Crustacea		0.4mg/l	1
cumene	LC50	96h		Fish		2.7mg/l	2
cumene	2000	72h		Algae or other aquatic plants 1.29			
cumene	EC50	72h		Algae or other aquatic plants		1.29mg/l	2

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	Endpoint	Test Duration (hr)	Species	Value	Sourc
	BCF	1680h	Fish	23-342	7
Mesitylene (1,3,5-trimethyl	EC50	48h	Crustacea	13mg/L	5
benzene)	EC50	96h	Algae or other aquatic plants	Algae or other aquatic plants 3.084mg/l	
	NOEC(ECx)	384h	Crustacea	Crustacea 0.257mg/l	
	LC50	96h	Fish	5.216mg/l	2
	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	23-146	7
	NOEC(ECx)	48h	Fish	0.013mg/L	4
naphthalene	LC50	96h	Fish	0.51mg/l	4
	EC50	72h	Algae or other aquatic plants	~0.4~0.5mg/l	2
	EC50	48h	Crustacea	1.09-3.4mg/l	4
	Endpoint	Test Duration (hr)	Species	Value	Sourc
	BCF	1344h	Fish	31-207	7
Pseudocumene (1,2,4-	EC50(ECx)	96h	Algae or other aquatic plants	2.356mg/l	2
trimethylbenzene)	LC50	96h	Fish	3.41mg/l	2
	EC50	48h	Crustacea	ca.6.14mg/l	1
	EC50	96h	Algae or other aquatic plants	2.356mg/l	2
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

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

oxygen transfer between the air and the water

Oils of any kind can cause:

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

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

For 1,2,4 - Trimethylbenzene:

Half-life (hr) air: 0.48-16;

Half-life (hr) H2O surface water: 0.24 -672;

Half-life (hr) H2O ground: 336-1344;

Half-life (hr) soil: 168-672;

Henry's Pa m3 /mol: 385 -627;

Bioaccumulation: not significant. 1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance.

Atmospheric Fate: 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs. Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals. Reaction also occurs with ozone but very slowly (half life 8820 days).

Aquatic Fate: 1,2,4-Trimethylbenzene volatilizes rapidly from surface waters with volatilization half-life from a model river calculated to be 3.4 hours. Biodegradation of 1,2,4-trimethylbenzene has been noted in both seawater and ground water. Various strains of Pseudomonas can biodegrade 1,2,4-trimethylbenzene.

Terrestrial Fate: 1,2,4-Trimethylbenzene also volatilizes from soils however; moderate adsorption to soils and sediments may occur. Volatilization is the major route of removal of 1,2,4-trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.

Ecotoxicity: No significant bioaccumulation has been noted. 1,2,4-Trimethylbenzene is moderately toxic to fathead minnow and slightly toxic to dungeness crab. 1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms. No stress was observed in rainbow trout, sea lamprey and Daphnia magna water fleas. The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.

For Aromatic Substances Series:

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

Atmospheric Fate: PAHs are 'semi-volatile substances' which can move between the atmosphere and the Earth's surface in repeated, temperature-driven cycles of deposition and volatilization. Terrestrial Fate: BTEX compounds have the potential to move through soil and contaminate ground water, and their vapors are highly flammable and explosive. Ecotoxicity - Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. The order of most toxic to least in a study using grass shrimp and brown shrimp was dimethylnaphthalenes > methylnaphthalenes > naphthalenes. Anthrcene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Biological resources in strong sunlight are at more risk than those that are not. PAHs in general are more frequently associated with chronic risks.

For 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);

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- (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 potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances. Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5

In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.

Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs

These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however,

one study suggests that some alkyl-PAHs may biomagnify. While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.

In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.

Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic components as compared to fish.

This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal

Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish Ecotoxicity:

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

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.

 $log\ Koc: 2.05-3.08;\ Koc: 25.4-204;\ Half-life\ (hr)\ air: 0.24-42;\ Half-life\ (hr)\ H2O\ surface\ water: 24-672;\ Half-life\ (hr)\ H2O\ ground: 336-8640;\ Half-life\ (hr)\ soil: 52-672;\ Henry's\ Pa\ m3\ /mol: 637-879;\ Henry's\ atm\ m3\ /mol - 7.68E-03;\ BOD\ 5\ if\ unstated: -1.4,1%;\ COD\ - 2.56,13%\ ThOD\ - 3.125:\ BCF: 23;\ log\ BCF: 1.17-2.41.$

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

Atmospheric Fate: Xylene evaporates quickly into the air from surface soil and water and can remain in the air for several days until it is broken down by sunlight into other less harmful chemicals. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere with an estimated atmospheric

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4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

lifetime of about 0.5 to 2 days. Xylene may contribute to photochemical smog formation. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzylnitrate, m-tolualdehyde,

Aquatic Fate: p-xylene may adsorb to suspended solids and sediment in water and is expected to volatilise from water surfaces. Estimated volatilisation half-lives for a model river and model lake are 3 hours and 4 days, respectively. Measurements taken from goldfish, eels and clams indicate that bioconcentration in aquatic organisms is low. Photo-oxidation in the presence of humic acids may play an important role in the abiotic degradation of p-xylene. p-Xylene is biodegradable and has been observed to degrade in pond water however; it is unclear if it degrades in surface waters. p-Xylene has been observed to degrade in anaerobic groundwater; however, it is known to persist for many years in groundwater, at least at sites where the concentration might have been quite high. Ecotoxicity: Xylenes are slightly toxic to fathead minnow, rainbow trout and bluegill and not acutely toxic to water fleas. For Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/L. and Gammarus lacustris LC50 (48 h): 0.6 mg/L. for naphthalene:

Environmental fate:

Naphthalene released to the atmosphere may be transported to surface water and/or soil by wet or dry deposition. Since most airborne naphthalene is in the vapor phase, deposition is expected to be very slow (about 0.04-0.06 cm/sec). It has been estimated that about 2-3% of naphthalene emitted to air is transported to other environmental media, mostly by dry deposition.

Naphthalene in surface water may volatilise to the atmosphere. The rate of volatilization also depends upon several environmental conditions, including temperature, wind velocity, and mixing rates of the air and water columns

Log octanol/water partition coefficients (Kow) for naphthalene range from 3.29 to 3.37 and log organic carbon coefficients (Koc) range from 2.97 to 3.27. The reported experimentally determined log Koc is 3.11. Based on the magnitude of these values, it is expected that only a small fraction (<10%) of naphthalene in typical surface water would be associated with particulate matter. Thus, naphthalene discharged to surface waters would remain largely in solution, with smaller quantities being associated with suspended solids and benthic sediments.

Naphthalene is easily volatilized from aerated soils and is adsorbed to a moderate extent (10%). The extent of sorption depends on the organic carbon content of the soil, with rapid movement expected through sandy soils. The estimated soil adsorption coefficient for naphthalene in a soil with <0.6% organic carbon is 1.8. Because it adsorbs to aquifer material, naphthalene's passage through groundwater will be somewhat retarded. However, sorption of naphthalene to aquifer materials with low organic carbon content (<0.03%) may be enhanced by the presence of nonionic low-polarity organics, such as tetrachloroethene, commonly found at hazardous waste sites. Bioconcentration factors (BCFs) for naphthalene have been measured and calculated from the Kow, Koc, or water solubility. The values reported for log BCF range from 1.6 to 3, indicating moderate bioconcentration in aquatic organisms. Naphthalene is reported to be rapidly eliminated from invertebrates when the organisms are placed in pollutant-free water, and naphthalene is readily metabolized in fish. Based on the magnitude of the Kow, bioaccumulation in the food chain is not expected to occur. However, naphthalene exposure of cows and chickens could lead to the presence of naphthalene in milk and eggs

Limited data were located on transport and partitioning of methylnaphthalenes in the environment. The respective vapor pressures (0.054 and 0.068 mmHg), water solubilities (25.8 and 24.6 mg/L), and Henry's law constants (3.60x10-4 and 4.99x10-4 atm-m3/mol) for 1-methylnaphthalene and 2-methylnaphthalene are of similar magnitude to these properties for naphthalene. Thus, it is likely that loss of methylnaphthalenes from ambient water occurs by volatilization. Based on the magnitude of log Kow for 1-methylnaphthalene and 2-methylnaphthalene (3.87 and 3.86, respectively) and the experimental log Koc for 2-methylnaphthalene (3.93), these chemicals may partition similarly to naphthalene in environmental media and are expected to be slightly mobile to immobile in soils. Log BCFs calculated for 2-methylnaphthalene range from 2 to 2.8 and measured log BCFs for 1-methylnaphthalene and 2-methylnaphthalene in oysters ranged from 2.7 to 4.1. Methylnaphthalenes are also metabolised and excreted rapidly by fish and shellfish when they are removed from polluted waters.

The most important atmospheric removal process for naphthalene is reaction with photochemically

produced hydroxyl radicals. The major products of this reaction are 1- and 2-naphthol and 1- and 2-nitronaphthalene. Naphthalene also reacts with N2O5, nitrate radicals, and ozone in the atmosphere and photolysis is expected to occur. Methylnaphthalenes also react with hydroxyl radicals. The reported rate constants are 5.30x10-11 and 5.23x10-11 cm3/molecule-sec for 1-methylnaphthalene and 2-methylnaphthalene, respectively. Based on an atmospheric hydroxyl radical concentration of 1x10 6/cm3, the corresponding atmospheric half-lives are 3.6 and 3.7 hours. Reactions of 1-methylnaphthalene and 2-methylnaphthalene with N2O5 radicals have half-lives of 24 and 19 days, respectively. These chemicals also react with atmospheric ozone.

Naphthalene and methylnaphthalenes are degraded in water by photolysis and biological processes. The half-life for photolysis of naphthalene in surface water is estimated to be about 71 hours, but the half-life in deeper water (5 m) is estimated at 550 days. The half-lives for photolysis of 1-methylnaphthalene and 2-methylnaphthalene were estimated at 22 and 54 hours, respectively.

Biodegradation of naphthalene is sufficiently rapid for it to be a dominant fate process in aquatic systems. Data on biodegradation of naphthalene in biodegradability tests and natural systems suggest that biodegradation occurs after a relatively short period of acclimation. Methylnaphthalenes are biodegraded under aerobic conditions after adaptation. The highest degradation rates were reported in water constantly polluted with petroleum.

Naphthalene biodegradation rates are about 8-20 times higher in sediment than in the water column above the sediment. Methylnaphthalenes biodegrade more slowly. Reported half-lives in sediments were 46 weeks for 1-methylnaphthalene and ranged from 14 to 50 weeks for 2-methylnaphthalene.

In soils, biodegradation potential is important to biological remediation of soil. Studies on biodegradation of PAHs suggest that adsorption to the organic matter significantly reduces the bioavailability for microorganisms, and thus the biodegradability, of PAHs, including naphthalene. Biodegradation is accomplished through the action of aerobic microorganisms and declines precipitously when soil conditions become anaerobic. Studies indicate that naphthalene biodegrades to carbon dioxide in aerobic soils, with salicylate as an intermediate product. Abiotic degradation of naphthalene seldom occurs in soils. The behavior of 1-methylnaphthalene in sandy loam was very similar to that of naphthalene. 1-Methylnaphthalene was easily volatilised from aerated soil, and the biodegradation half-life averaged between 1.7 and 2.2 days.

Acute toxicity data on naphthalene are available for several fish species (freshwater and marine). If low reliable data (too old, static test, nominal concentrations) are excluded, 96h LC50 values range from 1.8 to 7.8 mg/L. Comparable results were obtained with other vertebrates (amphibians).

From chronic toxicity tests, a precise NOEL is not clearly determined. A NOEC of 0.12 mg/L was observed in a 40 days test on juvenile pink salmon, but 50% mortality at 0.11 mg/L was calculated for trout fry exposed during hatching.

Several data are also available for invertebrates, showing 48h EC50 values ranging from 2.1 to 24 mg/L. Also in this case, higher figures must be taken with care due to nominal

Chronic data on freshwater invertebrates are methodologically unclear or questionable.

On algae too, data available are obtained with hardly comparable methodological approaches. 50% photosynthesis reduction was observed at 2.8 mg/L in 4 hours experiments. QSAR predictions using equations for narcosis give results consistent with experimental short-term data on fish daphnia and algae.

DO NOT discharge into sewer or waterways

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
1,2,4-trimethyl benzene	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)
1,3,5-trimethyl benzene	HIGH	HIGH
ethylbenzene	HIGH (Half-life = 228 days)	LOW (Half-life = 3.57 days)
cumene	HIGH	HIGH
Mesitylene (1,3,5-trimethyl benzene)	HIGH	HIGH
naphthalene	HIGH (Half-life = 258 days)	LOW (Half-life = 1.23 days)
Pseudocumene (1,2,4-trimethylbenzene)	LOW (Half-life = 56 days)	LOW (Half-life = 0.67 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
1,2,4-trimethyl benzene	LOW (BCF = 275)
1,3,5-trimethyl benzene	LOW (BCF = 342)

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Ingredient	Bioaccumulation
ethylbenzene	LOW (BCF = 79.43)
cumene	LOW (BCF = 35.5)
Mesitylene (1,3,5-trimethyl benzene)	LOW (BCF = 342)
naphthalene	HIGH (BCF = 18000)
Pseudocumene (1,2,4- trimethylbenzene)	LOW (BCF = 275)

Mobility in soil

Ingredient	Mobility
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
1,3,5-trimethyl benzene	LOW (KOC = 703)
ethylbenzene	LOW (KOC = 517.8)
cumene	LOW (KOC = 817.2)
Mesitylene (1,3,5-trimethyl benzene)	LOW (KOC = 703)
naphthalene	LOW (KOC = 1837)
Pseudocumene (1,2,4-trimethylbenzene)	LOW (KOC = 717.6)

SECTION 13 Disposal considerations

Waste treatment methods

- ▶ 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. Product / Packaging disposal
 - 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.

Ensure that the hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package. The package must be disposed according to the manufacturer's directions taking into account the material it is made of. Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous. DO NOT deposit the hazardous substance into or onto a landfill or a sewage facility.

Burning the hazardous substance must happen under controlled conditions with no person or place exposed to

(1) a blast overpressure of more than 9 kPa; or

(2) an unsafe level of heat radiation.

The disposed hazardous substance must not come into contact with class 1 or 5 substances.

SECTION 14 Transport information

Labels Required



Marine Pollutant



HAZCHEM

3Y

Land transport (UN)

zana tranoport (Ott)					
UN number	1300	1300			
UN proper shipping name	TURPENTII	TURPENTINE SUBSTITUTE			
Transport hazard class(es)	Class Subrisk	3 Not Applicable			
Packing group	III				

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Environmental hazard	Environmentally hazar	Environmentally hazardous			
Special precautions for user	Special provisions Limited quantity	223 5 L			

Air transport (ICAO-IATA / DGR	2)				
UN number	1300				
UN proper shipping name	Turpentine substitute				
	ICAO/IATA Class	3			
Transport hazard class(es)	ICAO / IATA Subrisk	Not Applicable			
	ERG Code	3L			
Packing group	III	III			
Environmental hazard	Environmentally hazardo	Environmentally hazardous			
	Special provisions		А3		
	Cargo Only Packing In	structions	366		
	Cargo Only Maximum	Qty / Pack	220 L		
Special precautions for user	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	1300	
UN proper shipping name	TURPENTINE SUBSTITUTE	
Transport hazard class(es)	IMDG Class 3 IMDG Subrisk Not Applicable	
Packing group	III	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number F-E, S-E Special provisions 223 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, heavy, hydrodesulfurised	Not Available
Contains	Not Available
1,2,4-trimethyl benzene	Not Available
1,3,5-trimethyl benzene	Not Available
ethylbenzene	Not Available
naphtha petroleum, light aromatic solvent	Not Available
contains	Not Available
cumene	Not Available
Mesitylene (1,3,5-trimethyl benzene)	Not Available
naphthalene	Not Available
Pseudocumene (1,2,4-trimethylbenzene)	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
naphtha petroleum, heavy, hydrodesulfurised	Not Available
Contains	Not Available
1,2,4-trimethyl benzene	Not Available
1,3,5-trimethyl benzene	Not Available

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ANDREW MINERAL TURPENTINE

Ship Type Product name ethylbenzene Not Available naphtha petroleum, light Not Available aromatic solvent contains Not Available cumene Not Available Mesitylene (1,3,5-trimethyl Not Available benzene) naphthalene Not Available Pseudocumene (1.2.4-

SECTION 15 Regulatory information

trimethylbenzene)

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

This substance is to be managed using the conditions specified in an applicable Group Standard

Not Available

HSR Number	Group Standard	
HSR002652	Solvents Flammable Carcinogenic Group Standard 2020	

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

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

Chemical Footprint Project - Chemicals of High Concern List

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

New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

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

New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

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

New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

ethylbenzene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List International Agency for Research on Cancer (IARC) - Agents Classified by the IARC

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

New Zealand Approved Hazardous Substances with controls

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

Chemical Footprint Project - Chemicals of High Concern List

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

New Zealand Approved Hazardous Substances with controls

cumene is found on the following regulatory lists

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

New Zealand Approved Hazardous Substances with controls

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

New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

naphthalene is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

New Zealand Approved Hazardous Substances with controls

Pseudocumene (1,2,4-trimethylbenzene) is found on the following regulatory lists

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

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New Zealand Approved Hazardous Substances with controls

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data

New Zealand Inventory of Chemicals (NZIoC)

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Quantity (Closed Containers)	Quantity (Open Containers)
3.1C	500 L in containers more than 5 L	250 L
3.1C	1 500 L in containers up to and including 5 L	250 L

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
3.1C or 3.1D				10 L

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status	
Australia - AIIC / Australia Non-Industrial Use	No (contains)	
Canada - DSL	No (contains)	
Canada - NDSL	No (naphtha petroleum, heavy, hydrodesulfurised; Contains; 1,2,4-trimethyl benzene; 1,3,5-trimethyl benzene; ethylbenzene; naphtha petroleum, light aromatic solvent; contains; cumene; Mesitylene (1,3,5-trimethyl benzene); naphthalene; Pseudocumene (1,2,4-trimethylbenzene))	
China - IECSC	No (contains)	
Europe - EINEC / ELINCS / NLP	No (contains)	
Japan - ENCS	No (contains)	
Korea - KECI	No (contains)	
New Zealand - NZIoC	No (contains)	
Philippines - PICCS	No (contains)	
USA - TSCA	No (contains)	
Taiwan - TCSI	Yes	
Mexico - INSQ	Yes	
Vietnam - NCI	No (contains)	
Russia - FBEPH	No (contains)	
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	15/11/2021
Initial Date	11/05/2020

SDS Version Summary

Version	Date of Update	Sections Updated
0.3	15/11/2021	Ingredients, Physical Properties, Transport Information

Other information

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

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

Definitions and abbreviations

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

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STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit。

IDLH: Immediately Dangerous to Life or Health Concentrations

ES: Exposure Standard OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value

BCF: BioConcentration Factors BEI: Biological Exposure Index

AIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European Inventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act TCSI: Taiwan Chemical Substance Inventory INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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