

Liqui Moly Diesel Clean & Boost

Motor Active

Chemwatch Hazard Alert Code: 2

Chemwatch: 4762-81

Version No: 2.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Issue Date: 27/06/2017

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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Liqui Moly Diesel Clean & Boost
Synonyms	Product Code: 2769
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains 2-ethylhexyl nitrate and solvent naphtha petroleum, heavy aromatic)
Other means of identification	Not Available

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

Relevant identified uses	Use according to manufacturer's directions. Cleaner; consumer applications; professional applications.
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Details of the supplier of the safety data sheet

Registered company name	Motor Active
Address	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia
Telephone	+61 2 9737 9422 1800 350 622
Fax	+61 2 9737 9414
Website	www.motoractive.com.au
Email	andrew.spira@motoractive.com.au

Emergency telephone number

Association / Organisation	MotorActive
Emergency telephone numbers	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
Other emergency telephone numbers	13 11 26 (In Case of Emergency contact: Poison Information Hotline)

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

COMBUSTIBLE LIQUID, regulated for storage purposes only

CHEMWATCH HAZARD RATINGS

	Min	Max	
Flammability	1	1	
Toxicity	1	1	
Body Contact	2	2	
Reactivity	1	1	
Chronic	2	2	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	S6
Classification [1]	Flammable Liquid Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Carcinogenicity Category 2, Reproductive Toxicity Category 2, Specific target organ toxicity - single exposure Category 3 (respiratory tract irritation), Specific target organ toxicity - single exposure Category 3 (narcotic effects), Aspiration Hazard Category 1, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
SIGNAL WORD	DANGER

Hazard statement(s)

H227	Combustible liquid.
H315	Causes skin irritation.

Continued...

Liqui Moly Diesel Clean & Boost

H319	Causes serious eye irritation.
H351	Suspected of causing cancer.
H361	Suspected of damaging fertility or the unborn child.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H304	May be fatal if swallowed and enters airways.
H411	Toxic to aquatic life with long lasting effects.
AUH044	Risk of explosion if heated under confinement.
AUH066	Repeated exposure may cause skin dryness and cracking.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P271	Use only outdoors or in a well-ventilated area.
P281	Use personal protective equipment as required.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

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

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 in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64742-82-1.	30-60	<u>naphtha, petroleum, hydrodesulfurised heavy</u>
64742-94-5	10-30	<u>solvent naphtha petroleum, heavy aromatic</u>
27247-96-7	<10	<u>2-ethylhexyl nitrate</u>
64742-95-6.	<10	<u>naphtha petroleum, light aromatic solvent</u>
95-63-6	<10	<u>1,2,4-trimethyl benzene</u>
91-20-3	<10	<u>naphthalene</u>
108-67-8	<10	<u>1,3,5-trimethyl benzene</u>
104-76-7	<10	<u>2-ethylhexanol</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- ▶ Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO₂ 50 mm Hg) should be intubated.
- ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
- ▶ Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

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

for 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-naphthoquinones 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

- ▶ Water spray or fog.
- ▶ Alcohol stable foam.
- ▶ Dry chemical powder.
- ▶ Carbon dioxide.

Do not use a water jet to fight fire.

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
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers.

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- ▶ On combustion, may emit toxic fumes of carbon monoxide (CO).
- ▶ May emit acrid smoke.
- ▶ Mists containing combustible materials may be explosive.

Combustion products include:

- , carbon dioxide (CO₂)
- , other pyrolysis products typical of burning organic material.

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SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal. 																																																																											
Major Spills	<p>Environmental hazard - contain spillage. Chemical Class: aliphatic hydrocarbons For release onto land: recommended sorbents listed in order of priority.</p> <table border="1"> <thead> <tr> <th>SORBENT TYPE</th> <th>RANK</th> <th>APPLICATION</th> <th>COLLECTION</th> <th>LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>shovel</td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>wood fiber - pillow</td> <td>2</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td>treated wood fibre- pillow</td> <td>2</td> <td>throw</td> <td>pitchfork</td> <td>DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, I, P</td> </tr> <tr> <td>foamed glass - pillow</td> <td>3</td> <td>throw</td> <td>pitchfork</td> <td>R, P, DGC, RT</td> </tr> <tr> <td colspan="5">LAND SPILL - MEDIUM</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>blower</td> <td>skiploader</td> <td>R,W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>2</td> <td>throw</td> <td>skiploader</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>R, I, P</td> </tr> <tr> <td>polypropylene - particulate</td> <td>3</td> <td>blower</td> <td>skiploader</td> <td>W, SS, DGC</td> </tr> <tr> <td>expanded mineral - particulate</td> <td>4</td> <td>blower</td> <td>skiploader</td> <td>R, I, W, P, DGC</td> </tr> <tr> <td>polypropylene - mat</td> <td>4</td> <td>throw</td> <td>skiploader</td> <td>DGC, RT</td> </tr> </tbody> </table> <p>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 Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ 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. 	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	LAND SPILL - SMALL					cross-linked polymer - particulate	1	shovel	shovel	R, W, SS	cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT	wood fiber - pillow	2	throw	pitchfork	R, P, DGC, RT	treated wood fibre- pillow	2	throw	pitchfork	DGC, RT	sorbent clay - particulate	3	shovel	shovel	R, I, P	foamed glass - pillow	3	throw	pitchfork	R, P, DGC, RT	LAND SPILL - MEDIUM					cross-linked polymer - particulate	1	blower	skiploader	R,W, SS	cross-linked polymer - pillow	2	throw	skiploader	R, DGC, RT	sorbent clay - particulate	3	blower	skiploader	R, I, P	polypropylene - particulate	3	blower	skiploader	W, SS, DGC	expanded mineral - particulate	4	blower	skiploader	R, I, W, P, DGC	polypropylene - mat	4	throw	skiploader	DGC, RT
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Personal Protective Equipment advice is contained in Section 8 of the SDS.

Continued...

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ 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. ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Electrostatic discharge may be generated during pumping - this may result in fire. ▶ Ensure electrical continuity by bonding and grounding (earthing) all equipment. ▶ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<=1 m/sec until fill pipe submerged to twice its diameter, then <= 7 m/sec). ▶ Avoid splash filling. ▶ Do NOT use compressed air for filling discharging or handling operations. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents <p>acids alkalies</p>

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	naphtha, petroleum, hydrodesulfurised heavy	White spirits	790 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	naphthalene	Naphthalene	10 ppm / 52 mg/m3	79 mg/m3 / 15 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
naphtha, petroleum, hydrodesulfurised heavy	Naphtha, hydrotreated heavy; (Isopar L-rev 2)	350 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	Petroleum distillates; petroleum ether; includes clay-treated light naphthenic [64742-45-6]; low boiling [68477-31-6]; petroleum extracts [64742-06-9]; petroleum base oil [64742-46-7]; petroleum 50 thinner, petroleum spirits [64475-85-0], Soltrol, VM&P naphtha [8032-32-4]; Ligroine, and paint solvent; petroleum paraffins C5-C20 [64771-72-8]; hydrotreated light naphthenic [64742-53-6]; solvent refined light naphthenic [64741-97-5]; and machine coolant 1	1,100 mg/m3	1,800 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	Naphtha (coal tar); includes solvent naphtha, petroleum (64742-88-7), naphtha (petroleum) light aliphatic, rubber solvent (64742-89-8), heavy catalytic cracked (64741-54-4), light straight run (64741-46-4), heavy aliphatic solvent (64742-96-7), high flash aromatic and aromatic solvent naphtha (64742-95-6)	1,200 mg/m3	6,700 mg/m3	40,000 mg/m3
naphtha, petroleum, hydrodesulfurised heavy	Stoddard solvent; (Mineral spirits, 85% nonane and 15% trimethyl benzene)	300 mg/m3	1,800 mg/m3	29500 mg/m3
1,2,4-trimethyl benzene	Permafluor E+	140 mg/m3	360 mg/m3	2,200 mg/m3
1,2,4-trimethyl benzene	Trimethylbenzene, 1,2,4-; (Pseudocumene)	Not Available	Not Available	480 ppm
naphthalene	Naphthalene	15 ppm	83 ppm	500 ppm
1,3,5-trimethyl benzene	Mesitylene; (1,3,5-Trimethylbenzene)	Not Available	Not Available	480 ppm
2-ethylhexanol	Ethyl-1-hexanol, 2-	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
naphtha, petroleum, hydrodesulfurised heavy	20000 mg/m ³ / 1,100 [LEL] ppm / 1,000 [LEL] ppm	Not Available
solvent naphtha petroleum, heavy aromatic	Not Available	Not Available
2-ethylhexyl nitrate	Not Available	Not Available
naphtha petroleum, light aromatic solvent	Not Available	Not Available
1,2,4-trimethyl benzene	Not Available	Not Available
naphthalene	250 ppm	Not Available
1,3,5-trimethyl benzene	Not Available	Not Available
2-ethylhexanol	Not Available	Not Available

MATERIAL DATA

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)

Odour threshold: 0.25 ppm.

The TLV-TWA is protective against ocular and upper respiratory tract irritation and is recommended for bulk handling of gasoline based on calculations of hydrocarbon content of gasoline vapour.

A STEL is recommended to prevent mucous membrane and ocular irritation and prevention of acute depression of the central nervous system. Because of the wide variation in molecular weights of its components, the conversion of ppm to mg/m³ is approximate. Sweden recommends hexane type limits of 100 ppm and heptane and octane type limits of 300 ppm. Germany does not assign a value because of the widely differing compositions and resultant differences in toxic properties.

Odour Safety Factor (OSF)

OSF=0.042 (gasoline)

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

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

The Odour Safety Factor (OSF) is defined as:

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

Classification into classes follows:

Class OSF Description

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

B 26-550 As "A" for 50-90% of persons being distracted

C 1-26 As "A" for less than 50% of persons being distracted

D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached

E <0.18 As "D" for less than 10% of persons aware of being tested

NOTE M: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.005% w/w benzo[a]pyrene (EINECS No 200-028-5). This note applies only to certain complex oil-derived substances in Annex IV.

European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

NOTE 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

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.


Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection.

Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.

An approved self contained breathing apparatus (SCBA) may be required in some situations.

Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

	<p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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.</p>	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
Lower end of the range	Upper end of the range										
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3: Intermittent, low production.	3: High production, heavy use										
4: Large hood or large air mass in motion	4: Small hood-local control only										
Personal protection											
Eye and face protection	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturizer is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> • frequency and duration of contact, • chemical resistance of glove material, • glove thickness and • dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> • When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. • When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. • Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. • Contaminated gloves should be replaced. <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> • Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. • Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>										
Body protection	See Other protection below										
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. 										

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-**

generated selection:

Liqui Moly Diesel Clean & Boost

Material	CPI
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Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

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BUTYL	C
NEOPRENE	C
PVA	C
TEFLON	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

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

^ - Full-face

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

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Clear light brown liquid with a characteristic odour; not miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.869
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	<0.07
Initial boiling point and boiling range (°C)	180	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	63 (CC)	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Combustible.	Oxidising properties	Not Available
Upper Explosive Limit (%)	7	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	0.6	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	>1	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>The acute toxicity of inhaled alkylbenzenes is best described by central nervous system depression. As a rule, these compounds may also act as general anaesthetics.</p> <p>Systemic poisoning produced by general anaesthesia is characterised by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness and</p>
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	<p>respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced. Inhaled alkylbenzene vapours cause death in animals at air levels that are relatively similar (typically LC50s are in the range 5000 -8000 ppm for 4 to 8 hour exposures). It is likely that acute inhalation exposure to alkylbenzenes resembles that to general anaesthetics.</p> <p>Alkylbenzenes are not generally toxic other than at high levels of exposure. This may be because their metabolites have a low order of toxicity and are easily excreted. There is little or no evidence to suggest that metabolic pathways can become saturated leading to spillover to alternate pathways. Nor is there evidence that toxic reactive intermediates, which may produce subsequent toxic or mutagenic effects, are formed</p> <p>Excessive exposure to 2-ethyl hexanol may result in headache, muscle weakness, nausea, giddiness, ataxia and central nervous system depression. Inhalation hazard is increased at higher temperatures.</p> <p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitisers and may cause ventricular fibrillations.</p> <p>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.</p> <p>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.</p> <p>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 parabrachial alveolar septa. Haematology did not identify significant changes.</p> <p>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</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Considered an unlikely route of entry in commercial/industrial environments. The liquid may produce gastrointestinal discomfort and may be harmful if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Rabbits exposed dermally to 2 ml/kg 2-ethyl hexanol, for 12 days, showed evidence of liver, lung, kidney and heart effects.</p> <p>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 methyl naphthalene (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.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p> <p>Aromatic hydrocarbons may produce skin irritation, vasodilation with erythema and changes in endothelial cell permeability. Systemic intoxication, resulting from contact with the light aromatics, is unlikely due to the slow rate of permeation. Branching of the side chain appears to increase percutaneous absorption.</p>
Eye	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>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.</p> <p>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.</p>
Chronic	<p>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.</p> <p>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.</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p>

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals.

Prolonged or repeated skin contact to 2-ethyl hexanol may result in irritation, reddening and sensitisation. Repeated oral and dermal exposures to animals has resulted in adverse liver and kidney effects.

Male and female rats receiving 1.25% 2-ethyl hexanol in the diet for 90 days showed evidence of kidney and liver effects.

The material produces teratogenic effects in laboratory rats when administered orally to pregnant Wistar rats on day 12 of gestation (1 and 2 ml/kg). These rats were sacrificed on day 20; the percent surviving foetuses with malformations in had increased to 2% and 22.2% in the low and high doses groups respectively. Maternal toxicity may have been a factor. Two subsequent studies indicate that birth defects in rats do not occur following inhalation of the vapour at maximum saturation concentration (850 mg/m³), or dermally at 3 ml/kg doses. Preliminary evidence suggests that absorption through human skin occurs more slowly than in rat.

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.

Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human

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.

Repeated application of mildly hydrotreated oils (principally paraffinic), to mouse skin, induced skin tumours; no tumours were induced with severely hydrotreated oils.

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

Liqui Moly Diesel Clean & Boost	TOXICITY	IRRITATION
	Not Available	Not Available
naphtha, petroleum, hydrosulfurised heavy	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Not Available
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	
	dermal (rat) LD50: 28000 mg/kg ^[2]	
	Inhalation (rat) LC50: >2796.8052 mg/l/8H ^[2]	
	Inhalation (rat) LC50: 3396.1206 mg/l/4H ^[2]	
	Inhalation (rat) LC50: 61 mg/l/4H ^[2]	
	Oral (rat) LD50: >4300 mg/kg ^[2]	
	Oral (rat) LD50: >4500 mg/kg ^[1]	
	Oral (rat) LD50: >4500 mg/kg ^[1]	
	Oral (rat) LD50: >4500 mg/kg ^[1]	
Oral (rat) LD50: >5000 mg/kg ^[1]		
solvent naphtha petroleum, heavy aromatic	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): Irritating
	Inhalation (rat) LC50: >0.59 mg/l/4H ^[2]	
	Oral (rat) LD50: >2000 mg/kg ^[1]	
2-ethylhexyl nitrate	TOXICITY	IRRITATION
	dermal (rat) LD50: >4820 mg/kg ^[2]	Not Available
	Inhalation (rat) LC50: >1.15 mg/l/1h. ^[2]	
	Oral (rat) LD50: 7500 mg/kg ^[2]	

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naphtha petroleum, light aromatic solvent	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg ^[1]	Not Available
	Inhalation (rat) LC50: >7331.62506 mg/l/8h ^[2]	
	Oral (rat) LD50: >4500 mg/kg ^[1]	
1,2,4-trimethyl benzene	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 18 mg/l/4hd ^[2]	Not Available
	Oral (rat) LD50: 3280 mg/kg ^[1]	
naphthalene	TOXICITY	IRRITATION
	dermal (rat) LD50: >2500 mg/kg ^[2]	Eye (rabbit): 100 mg - mild
	Oral (rat) LD50: 490 mg/kg ^[2]	Skin (rabbit):495 mg (open) - mild
1,3,5-trimethyl benzene	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 24 mg/l/4hd ^[2]	Eye (rabbit): 500 mg/24h mild
	Oral (rat) LD50: 3280 mg/kg ^[1]	Skin (rabbit): 20 mg/24h moderate
2-ethylhexanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1970 mg/kg ^[2]	Eye (rabbit): 20 mg/24h - moderate
	Oral (rat) LD50: 2049 mg/kg ^[2]	Eye (rabbit): 4.17 mg - SEVERE
		Skin (rabbit): 415 mg (open)-mild
		Skin (rabbit): 500 mg/24h-moderate

Legend:

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

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	<p>For Low Boiling Point Naphthas (LBPNS):</p> <p>Acute toxicity: LBPNS generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure Most LBPNS are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices.</p> <p>Sensitisation: LBPNS do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies</p> <p>Repeat dose toxicity: The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPNS substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNS in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNS, were considered species- and sex-specific. These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.</p> <p>Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNS. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3 No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNS and very few non-cancer chronic toxicity studies were identified for other LBPNS. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3 A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.</p> <p>Genotoxicity: Although few genotoxicity studies were identified for the site-restricted LBPNS, the genotoxicity of several other LBPNS substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results. For in vivo genotoxicity tests, LBPNS exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells. For in vitro genotoxicity studies, LBPNS were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNS exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay . Mixed results were observed for UDS and the mouse lymphoma assay. While the majority of in vivo genotoxicity results for LBPNS substances are negative, the potential for genotoxicity of LBPNS as a group cannot be discounted based on the mixed in vitro genotoxicity results.</p> <p>Carcinogenicity:</p>
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Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effects of human exposure to LBP substances.

No inhalation studies assessing the carcinogenicity of the site-restricted LBPNs were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m³ of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously. Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol. However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBP group.

Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBP substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBP, as a Group 2B carcinogen (possibly carcinogenic to humans) and "occupational exposures in petroleum refining" as Group 2A carcinogens (probably carcinogenic to humans).

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

or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol.

Reproductive/ Developmental toxicity:

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

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

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

Low Boiling Point Naphthas [Site-Restricted]

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/m³ for C9 aromatic naphtha and 18,000 to 24,000 mg/m³ for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.

Irritation and Sensitization

Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.

Repeated Dose Toxicity

Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m³). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neuro/behavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m³, the highest concentration tested. In an inhalation study of a commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m³) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.

The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m³. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4-and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m³). Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m³ (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m³ for respiratory irritation and 250 ppm or 1230 mg/m³ for systemic effects.

Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg/bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with *Salmonella typhimurium* and *Escherichia coli* bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly

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	<p>positive. 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</p> <p>Reproductive and Developmental Toxicity</p> <p>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.</p> <p>Systemic Effects on Parental Generations:</p> <p>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).</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Inhalation (rat) TCLo: 1320 ppm/6h/90D-I * [Devoe]</p>
1,2,4-TRIMETHYL BENZENE	CHEMWATCH 2325 1,3,5-trimethylbenzene
NAPHTHALENE	<p>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.</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
1,3,5-TRIMETHYL BENZENE	CHEMWATCH 12171 1,2,4-trimethylbenzene
2-ETHYLHEXANOL	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>For alkyl alcohols C6-13:</p> <p>This group of products are very similar in terms of physicochemical and toxicological properties. Interpolation of data can be used to assess the alkyl alcohols for which data is not available.</p> <p>Acute toxicity: All of these alcohols have a low order of toxicity in rats via the oral route. The LD50 for C6-branched and linear alcohols were >3700 mg/kg; LD50s for the C6-8, C7-9, C8-10, C9-11 and C11-14 branched alkyl alcohols were all >2000 mg/kg.</p> <p>These alcohols have a low order of toxicity via the dermal route. Dermal LD50s were greater than 2600 mg/kg.</p> <p>Subchronic toxicity: Repeat dose studies indicate these alcohols have a low order of subchronic toxicity by both the oral and dermal route. Further they demonstrate that these alcohols display a consistent degree of subchronic toxicity by these routes</p> <p>Developmental toxicity: Studies demonstrate that the alcohols are not selective developmental toxicants by either the oral or inhalation route of exposure. Inhalation of alkyl alcohols C6-13 is a primary concern during industrial use, particularly for lower molecular weight alcohols.</p> <p>Collectively the weight of evidence demonstrates that these alcohols have a low order of maternal toxicity and do not induce signs of developmental toxicity until maternal toxicity is observed. The NOAELs for inhalation reflect the maximum achievable vapour concentration.</p> <p>Reproductive toxicity: Developmental toxicity studies for several of these alcohols, conducted by the oral route, produce consistent results and demonstrate that these substances do not affect reproductive parameters. Although a slight increase in resorptions was observed in several studies, this occurred only in the highest dose group and in the presence of overt maternal toxicity.</p> <p>Genotoxicity: The weight of evidence from existing data supports the conclusion that these materials are not genotoxic.</p> <p>Further data to support this assessment comes from a series of alkyl acetates C6-13. Alkyl acetates are produced from alkyl alcohols and undergo metabolism by esterases to produce acetic acid and the corresponding alkyl alcohol. There is no evidence for genotoxicity with these compounds in a variety of strains of <i>S. typhimurium</i> in the presence or absence of metabolic activation. C6, C6-8, C7-9 and C11-14 alkyl acetates produced negative results in the Ames test.</p> <p>Based on data for structurally similar substances these alcohols are not expected to be clastogenic. Alkyl acetates can also be used to predict clastogenic potential of alkyl alcohols. Although there is evidence of cytotoxicity at extremely high doses, no clastogenic activity was seen in a homologous family of alkyl acetates.</p> <p>Metabolism: Alkyl alcohols are broken down, in the body, by mitochondrial beta-oxidation or by cytochrome P450 omega and omega-minus oxidation. The alcohol undergoes various oxidative steps to yield other alcohols, ketones, aldehydes, carboxylic acids and carbon dioxide. Data for monohydric, aliphatic alcohols show a systematic variation according to molecular weight in a manner similar to other homologous series. The body handles aliphatic hydrocarbons in a similar manner via oxidative conversion to alcohols, ketones, and eventual elimination as carbon dioxide and carboxylic acids. The undegraded alcohols can be conjugated either directly or as a metabolite with glucuronic acid, sulfuric acid or glycine and are rapidly excreted. Intermediate aldehydes may be reactive and bind with DNA and/or proteins.</p> <p>The Branched Chain Saturated Alcohol (BCSA) group of fragrance ingredients was evaluated for safety.</p> <p>The 15 materials tested have a low order of acute toxicity.</p> <p>Following repeated application, seven BCSA tested were of low systemic toxicity</p> <p>In humans, no evidence of skin irritation was found at concentrations of 2-10%. Undiluted, 11 materials evaluated caused moderate to severe eye irritation. As current end product use levels are between 0.001% and 1.7%, eye irritation is not a concern. The materials have no or low sensitizing potential.</p>

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	<p>For individuals who are already sensitized, an elicitation reaction is possible. Due to lack of UVA/UVB light-absorbing structures, and review of phototoxic/photoallergy data, the BCSA are not expected to elicit phototoxicity or photoallergy.</p> <p>Studies performed on eight BCSA and three metabolites show no in vivo or in vitro genotoxicity. A valid carcinogenicity study showed that 2-ethyl-1-hexanol is a weak inducer of liver tumours in female mice, however, the relevance of this effect and mode of action to humans is still a matter of debate. Current opinion holds that there are no safety concerns regarding BCSA under the present levels of use and exposure.</p> <p>The common characteristic structural elements of the alcohols with saturated branched chain are one hydroxyl group per molecule, a C4-C12 carbon chain with one or several methyl side chains. Two members of the group, 2-ethyl-1-butanol and 2-ethyl-1-hexanol, contain an ethyl side chain. One member contains a methoxy group. Metabolism studies are lacking for this compound, however, a methoxy group is enzymatically not readily cleaved and if it were so, another primary alcohol group would be formed.</p> <p>The Research Institute for Fragrance Materials (RIFM) Expert Panel</p>
<p>Liqui Moly Diesel Clean & Boost & NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>Liqui Moly Diesel Clean & Boost & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT & 1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE</p>	<p>For trimethylbenzenes:</p> <p>Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption. 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid. The major routes of excretion of 1,2,4-trimethylbenzene 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.</p> <p>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.</p> <p>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 trimethylbenzenes at 1700 ppm for 10 to 21 days</p> <p>Neurotoxicity 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes</p> <p>Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.</p> <p>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</p> <p>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.</p> <p>Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella typhimurium/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.</p> <p>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 developmental neurotoxicity, was evident in rats in a 3-generation reproductive study</p> <p>No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethylbenzenes, 4-6 hours/day, 5 days/week over one generation</p>
<p>Liqui Moly Diesel Clean & Boost & SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT</p>	<p>for petroleum:</p> <p>Altered mental state, drowsiness, peripheral motor neuropathy, irreversible brain damage (so-called Petrol Sniffer's Encephalopathy), delirium, seizures, and sudden death have been reported from repeated overexposure to some hydrocarbon solvents, naphthas, and gasoline</p> <p>This product may contain benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.</p> <p>This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.</p> <p>This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents</p> <p>Carcinogenicity: Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.</p> <p>Mutagenicity: There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.</p> <p>Reproductive Toxicity: Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.</p> <p>Human Effects: Prolonged/repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.</p> <p>Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.</p>

Liqui Moly Diesel Clean & Boost & 2-ETHYLHEXYL NITRATE	Chemical with the aliphatic nitro group (-C-NO2) have been added to a list of DNA-reactive subgroups recognised by the National Toxicological Program (NTP, U.S. Dept Health and Human Services) for possible carcinogenic activity.
NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY & NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than 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.
1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE & 2-ETHYLHEXANOL	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.
1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE	Other Toxicity data is available for
1,2,4-TRIMETHYL BENZENE & 1,3,5-TRIMETHYL BENZENE	CHEMWATCH 12172 1,2,3-trimethylbenzene
NAPHTHALENE & 1,3,5-TRIMETHYL BENZENE	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
1,3,5-TRIMETHYL BENZENE & 2-ETHYLHEXANOL	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.

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

Legend: ✘ – Data available but does not fill the criteria for classification
✔ – Data available to make classification
☹ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Liqui Moly Diesel Clean & Boost	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available

naphtha, petroleum, hydrodesulfurised heavy	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	EC50	72	Algae or other aquatic plants	=13mg/L	1
	NOEC	72	Algae or other aquatic plants	=0.1mg/L	1
	EC50	48	Crustacea	>100mg/L	1
	EC50	96	Algae or other aquatic plants	=450mg/L	1
	EC50	72	Algae or other aquatic plants	=6.5mg/L	1
	NOEC	72	Algae or other aquatic plants	<0.1mg/L	1
	LC50	96	Fish	0.00746mg/L	4
	EC50	48	Crustacea	0.058mg/L	4
	BCF	96	Fish	0.2mg/L	4
	NOEC	168	Crustacea	<=0.05mg/L	4
	LC50	96	Fish	8.8mg/L	4
	EC50	48	Crustacea	3.7mg/L	4
	EC50	72	Algae or other aquatic plants	=6.5mg/L	1
	NOEC	72	Algae or other aquatic plants	<0.1mg/L	1
	EC50	72	Algae or other aquatic plants	=6.5mg/L	1
	NOEC	72	Algae or other aquatic plants	<0.1mg/L	1

Liqui Moly Diesel Clean & Boost

	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	solvent naphtha petroleum, heavy aromatic	LC50	96	Fish	0.58mg/L
EC50		48	Crustacea	0.76mg/L	2
EC50		72	Algae or other aquatic plants	<1mg/L	1
NOEC		72	Algae or other aquatic plants	0.3mg/L	2
2-ethylhexyl nitrate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	2mg/L	2
	NOEC	96	Fish	1.52mg/L	2
	naphtha petroleum, light aromatic solvent	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE
EC50		48	Crustacea	=6.14mg/L	1
EC50		72	Algae or other aquatic plants	3.29mg/L	1
EC10		72	Algae or other aquatic plants	1.13mg/L	1
	NOEC	72	Algae or other aquatic plants	=1mg/L	1
	1,2,4-trimethyl benzene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE
LC50		96	Fish	7.72mg/L	2
	EC50	48	Crustacea	ca.6.14mg/L	1
naphthalene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.213mg/L	4
	EC50	48	Crustacea	1.6mg/L	4
	EC50	72	Algae or other aquatic plants	ca.0.4- ca.0.5mg/L	2
	BCF	12	Fish	10.2mg/L	4
	NOEC	48	Fish	0.012817mg/L	4
1,3,5-trimethyl benzene	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	12.52mg/L	4
	EC50	48	Crustacea	13mg/L	5
	EC0	24	Crustacea	=40mg/L	4
	NOEC	504	Crustacea	0.4mg/L	4
2-ethylhexanol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	<0.075mg/L	4
	EC50	48	Crustacea	39mg/L	1
	EC50	72	Algae or other aquatic plants	=11.5mg/L	1
	EC10	72	Algae or other aquatic plants	=1.3mg/L	1
	NOEC	48	Algae or other aquatic plants	=10mg/L	1

Legend:

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

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

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For 1,2,4-trimethylbenzene:

Half-life (hr) air : 0.48-16

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

Half-life (hr) H₂O ground : 336-1344

Half-life (hr) soil : 168-672

Henry's Pa m³ /mol: 385-627

Bioaccumulation : not significant

1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance. As a VOC, 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs.

Environmental fate:

Transport: 1,2,4-Trimethylbenzene volatilises rapidly from surface waters as predicted by a Henry's law constant of 5.18×10^{-3} (vapor pressure, 2.03 mm Hg). The volatilisation half-life from a model river is calculated to be 3.4 hours. The chemical also volatilises from soils, however, based on an estimated K_{oc} of 472, moderate adsorption to soils and sediments may occur

Transformation/Persistence

Air - Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals. Reaction also occurs with ozone but very slowly (half life, 8820 days). In the atmosphere, two estimates of the half-life are approximately 6 hours and, in the presence of hydroxyl radicals, 0.5 days.

Soil - Volatilisation is the major route of removal of 1,2,4-trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.

Water - Because of 1,2,4-trimethylbenzene's water solubility and its vapor pressure of 2.03 mm Hg, the chemical will rapidly volatilise from surface waters. Biodegradation of 1,2,4-trimethylbenzene occurred with inoculums from both seawater and ground water. Various strains of *Pseudomonas* can biodegrade 1,2,4-trimethylbenzene.

Biota - The estimated bioconcentration factor (439) and high volatility of 1,2,4-trimethylbenzene indicates that bioaccumulation of the chemical will not be significant.

Ecotoxicity:

Fish LC50 (96 h): fathead minnow 7.72 mg/l

No stress was observed in *Oncorhynchus mykiss* (rainbow trout, fingerling) or *Petromyzon marinus* (sea lamprey, larvae) at 5 mg/L for 24 hours

Daphnia magna EC50 (48 h): 3.61 mg/l

Cancer magister (dungeness crab) LC50 996 h): 5.1 mg/l

1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms; acute toxicity values fall within the range of greater than 1 mg/L and 100 mg/L. LC50 values for specific aquatic organisms range from approximately 5 to 8 mg/L which is orders of magnitude greater than any measured concentration in seawater (0.002 - 0.54 microgram/L). The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.

For nitroalkanes:

The stabilities of nitroalkanes in both water and aqueous mineral acids have been determined in a well conducted study. Nitromethane, nitroethane, 1-nitropropane and 2-nitropropane did not hydrolyse appreciably in neutral water, even at elevated temperatures. The first three (primary) nitroalkanes underwent hydrolysis in aqueous mineral acid solution, and the hydrolysis rate constants were determined. 2-Nitropropane itself (a secondary nitroalkane) was stable to hydrolysis even in hot mineral acids.

Nitroalkanes absorb UV light >290 nm and undergo a primary dissociative process forming free radicals

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

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

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 1x10⁶ 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.

Ecotoxicity:

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 concentrations. 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)
naphthalene	HIGH (Half-life = 258 days)	LOW (Half-life = 1.23 days)
1,3,5-trimethyl benzene	HIGH	HIGH
2-ethylhexanol	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
solvent naphtha petroleum, heavy aromatic	LOW (BCF = 159)
1,2,4-trimethyl benzene	LOW (BCF = 275)

naphthalene	HIGH (BCF = 18000)
1,3,5-trimethyl benzene	LOW (BCF = 342)
2-ethylhexanol	LOW (BCF = 27)



Mobility in soil

Ingredient	Mobility
1,2,4-trimethyl benzene	LOW (KOC = 717.6)
naphthalene	LOW (KOC = 1837)
1,3,5-trimethyl benzene	LOW (KOC = 703)
2-ethylhexanol	LOW (KOC = 26.01)

SECTION 13 DISPOSAL CONSIDERATIONS**Waste treatment methods**

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 TRANSPORT INFORMATION**Labels Required**

	
Marine Pollutant	NO 
HAZCHEM	*3Z

Land transport (DOT)

UN number	3082
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains 2-ethylhexyl nitrate and solvent naphtha petroleum, heavy aromatic)
Transport hazard class(es)	Class : 9 Subrisk : Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions : 274 331 335 375 AU01 Limited quantity : 5 L

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in;

- (a) packagings;
(b) IBCs; or

(c) any other receptacle not exceeding 500 kg(L).

- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

UN number	3082	
UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. * (contains 2-ethylhexyl nitrate and solvent naphtha petroleum, heavy aromatic)	
Transport hazard class(es)	ICAO/IATA Class	9
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	9L
Packing group	III	
Environmental hazard	Environmentally hazardous	
Special precautions for user	Special provisions	A97 A158 A197
	Cargo Only Packing Instructions	964
	Cargo Only Maximum Qty / Pack	450 L
	Passenger and Cargo Packing Instructions	964
	Passenger and Cargo Maximum Qty / Pack	450 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y964
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3082	
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains 2-ethylhexyl nitrate and solvent naphtha petroleum, heavy aromatic)	
Transport hazard class(es)	IMDG Class	9
	IMDG Subrisk	Not Applicable
Packing group	III	
Environmental hazard	Marine Pollutant	
Special precautions for user	EMS Number	F-A , S-F
	Special provisions	274 335 969
	Limited Quantities	5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture****NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY(64742-82-1.) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Inventory of Chemical Substances (AICS)	International Air Transport Association (IATA) Dangerous Goods Regulations - Prohibited List Passenger and Cargo Aircraft
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)	

SOLVENT NAPHTHA PETROLEUM, HEAVY AROMATIC(64742-94-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Inventory of Chemical Substances (AICS)	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

2-ETHYLHEXYL NITRATE(27247-96-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

NAPHTHA PETROLEUM, LIGHT AROMATIC SOLVENT(64742-95-6.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)
Australia Inventory of Chemical Substances (AICS)	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

1,2,4-TRIMETHYL BENZENE(95-63-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

NAPHTHALENE(91-20-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix G
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Inventory of Chemical Substances (AICS)	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix E (Part 2)	Australia Work Health and Safety Regulations 2016 - Hazardous chemicals (other than lead) requiring health monitoring
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix F (Part 3)	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

1,3,5-TRIMETHYL BENZENE(108-67-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

2-ETHYLHEXANOL(104-76-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Appendix B (Part 3)
Australia Inventory of Chemical Substances (AICS)	

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (naphthalene; naphtha petroleum, light aromatic solvent; 1,3,5-trimethyl benzene; 2-ethylhexanol; 2-ethylhexyl nitrate; 1,2,4-trimethyl benzene; naphtha, petroleum, hydrodesulfurised heavy; solvent naphtha petroleum, heavy aromatic)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Y
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	27/06/2017
Initial Date	Not Available

Other information**Ingredients with multiple cas numbers**

Name	CAS No
naphtha, petroleum, hydrodesulfurised heavy	64742-82-1., 64741-92-0., 8052-41-3., 1030262-12-4., 8032-32-4., 8030-30-6., 64742-88-7., 64742-89-8., 8002-05-9., 61789-95-5., 64742-48-9., 101795-02-2., 8031-06-9., 8030-31-7., 50813-73-5., 54847-97-1., 121448-83-7., 8031-38-7., 8031-39-8.
naphtha petroleum, light aromatic solvent	64742-95-6., 25550-14-5.
2-ethylhexanol	104-76-7, 111675-57-1, 68526-83-0, 68526-84-1

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

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

Definitions and abbreviations

PC— TWA: Permissible Concentration-Time Weighted Average
 PC— STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit,
 IDLH: Immediately Dangerous to Life or Health Concentrations
 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

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