

# Altex RIPO

Altex Coatings Ltd

Chemwatch Hazard Alert Code: 2

Chemwatch: 9-41586

Version No: 1.3

Safety Data Sheet according to HSNO Regulations

Issue Date: 28/11/2013

Print Date: 24/12/2013

S.GHS.NZL.EN

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

### Product Identifier

Product name	Altex RIPO
Chemical Name	Not Applicable
Proper shipping name	PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
Chemical formula	Not Applicable
Other means of identification	Not Available
CAS number	Not Applicable

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

Relevant identified uses	Use according to manufacturer's directions.
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### Details of the supplier of the safety data sheet

Registered company name	Altex Coatings Ltd
Address	New Zealand
Telephone	+64 7 5411221
Fax	+64 7 5411310
Website	www.altexcoatings.co.nz
Email	Not Available

### Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	0800 764766
Other emergency telephone numbers	0800 764766

### CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
+800 2436 2255	+612 9186 1132	Not Available

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

## SECTION 2 HAZARDS IDENTIFICATION

### Classification of the substance or mixture

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

GHS Classification <sup>[1]</sup>	Flammable Liquid Category 3, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, STOT - SE (Resp. Irr.) Category 3, STOT - SE (Narcosis) Category 3, STOT - RE Category 2, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	3.1C, 6.1D (dermal), 6.1D (inhalation), 6.3A, 6.4A, 6.9 (narcotic), 6.9 (respiratory), 6.9B (inhalation), 9.1B, 9.1D

## Label elements

GHS label elements	
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SIGNAL WORD	<b>WARNING</b>
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## Hazard statement(s)

H226	Flammable liquid and vapour
H312	Harmful in contact with skin
H332	Harmful if inhaled
H315	Causes skin irritation
H319	Causes serious eye irritation
H335	May cause respiratory irritation
H336	May cause drowsiness or dizziness
H373	May cause damage to organs through prolonged or repeated exposure
H401	Toxic to aquatic life
H411	Toxic to aquatic life with long lasting effects

## Supplementary statement(s)

Not Applicable

## Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P240	Ground/bond container and receiving equipment.
P241	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
P242	Use only non-sparking tools.
P243	Take precautionary measures against static discharge.

## Precautionary statement(s) Response

P321	Specific treatment (see advice on this label).
P370+P378	In case of fire: Use... to extinguish.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/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 water and soap
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

## Precautionary statement(s) Storage

P403+P235	Store in a well-ventilated place. Keep cool.
P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

**Precautionary statement(s) Disposal**

<b>P501</b>	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration
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**SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS****Substances**

See section below for composition of Mixtures

**Mixtures**

CAS No	%[weight]	Name
64742-95-6.	50-60	<a href="#">aromatic hydrocarbon solvent</a>
8001-26-1	20-30	<a href="#">linseed oil</a>
1330-20-7	10-20	<a href="#">xylene</a>
9006-03-5	1-10	<a href="#">chlorinated rubber - carbon tetrachloride free</a>
64742-82-1.	1-10	<a href="#">naphtha, petroleum, hydrodesulfurised heavy</a>
108-88-3	1-10	<a href="#">toluene</a>

**SECTION 4 FIRST AID MEASURES**

NZ Poisons Centre 0800 POISON (0800 764 766) | NZ Emergency Services: 111

**Description of first aid measures**

<b>Eye Contact</b>	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> <li>▶ Wash out immediately with fresh running water.</li> <li>▶ 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.</li> <li>▶ Seek medical attention without delay; if pain persists or recurs seek medical attention.</li> <li>▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.</li> </ul>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul>
<b>Inhalation</b>	<ul style="list-style-type: none"> <li>▶ If fumes or combustion products are inhaled remove from contaminated area.</li> <li>▶ Lay patient down. Keep warm and rested.</li> <li>▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ 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.</li> <li>▶ Transport to hospital, or doctor, without delay.</li> </ul>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ <b>If swallowed do NOT induce vomiting.</b></li> <li>▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.</li> <li>▶ Observe the patient carefully.</li> <li>▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.</li> <li>▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.</li> <li>▶ Seek medical advice.</li> <li>▶ Avoid giving milk or oils.</li> <li>▶ Avoid giving alcohol.</li> <li>▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.</li> </ul>

**Indication of any immediate medical attention and special treatment needed**

<b>Indication of any immediate medical attention and special treatment needed</b>	<p>Treat symptomatically.</p> <p>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.</p> <p>For acute or short term repeated exposures to xylene:</p> <ul style="list-style-type: none"> <li>▶ Gastro-intestinal absorption is significant with ingestions. For ingestions exceeding 1-2 ml (xylene)/kg,</li> </ul>
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- intubation and lavage with cuffed endotracheal tube is recommended. The use of charcoal and cathartics is equivocal.
- Pulmonary absorption is rapid with about 60-65% retained at rest.
- Primary threat to life from ingestion and/or inhalation, is respiratory failure.
- Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO<sub>2</sub> < 50 mm Hg or pCO<sub>2</sub> > 50 mm Hg) should be intubated.
- Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
- A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
- Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.

## BIOLOGICAL EXPOSURE INDEX - BEI

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

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

## SECTION 5 FIREFIGHTING MEASURES

## Extinguishing media

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

## Special hazards arising from the substrate or mixture

## Fire Incompatibility

- Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

## Advice for firefighters

## Fire Fighting

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

- Liquid and vapour are flammable.
- Moderate fire hazard when exposed to heat or flame.
- Vapour forms an explosive mixture with air.
- Moderate explosion hazard when exposed to heat or flame.
- Vapour may travel a considerable distance to source of ignition.
- Heating may cause expansion or decomposition leading to violent rupture of containers.
- On combustion, may emit toxic fumes of carbon monoxide (CO).

Combustion products include:

- , carbon monoxide (CO)
- , carbon dioxide (CO<sub>2</sub>)
- , acrolein
- , other pyrolysis products typical of burning organic material

May emit clouds of acrid smoke

## SECTION 6 ACCIDENTAL RELEASE MEASURES

### Personal precautions, protective equipment and emergency procedures

<b>Minor Spills</b>	<ul style="list-style-type: none"> <li>▸ Remove all ignition sources.</li> <li>▸ Clean up all spills immediately.</li> <li>▸ Avoid breathing vapours and contact with skin and eyes.</li> <li>▸ Control personal contact with the substance, by using protective equipment.</li> <li>▸ Contain and absorb small quantities with vermiculite or other absorbent material.</li> <li>▸ Wipe up.</li> <li>▸ Collect residues in a flammable waste container.</li> </ul>
<b>Major Spills</b>	<ul style="list-style-type: none"> <li>▸ Clear area of personnel and move upwind.</li> <li>▸ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▸ May be violently or explosively reactive.</li> <li>▸ Wear breathing apparatus plus protective gloves.</li> <li>▸ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▸ Consider evacuation (or protect in place).</li> <li>▸ No smoking, naked lights or ignition sources.</li> <li>▸ Increase ventilation.</li> <li>▸ Stop leak if safe to do so.</li> <li>▸ Water spray or fog may be used to disperse /absorb vapour.</li> <li>▸ Contain spill with sand, earth or vermiculite.</li> <li>▸ Use only spark-free shovels and explosion proof equipment.</li> <li>▸ Collect recoverable product into labelled containers for recycling.</li> <li>▸ Absorb remaining product with sand, earth or vermiculite.</li> <li>▸ Collect solid residues and seal in labelled drums for disposal.</li> <li>▸ Wash area and prevent runoff into drains.</li> <li>▸ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

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

## SECTION 7 HANDLING AND STORAGE

### Precautions for safe handling

<b>Safe handling</b>	<ul style="list-style-type: none"> <li>▸ Containers, even those that have been emptied, may contain explosive vapours.</li> <li>▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.</li> <li>▸ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> <li>▸ Electrostatic discharge may be generated during pumping - this may result in fire.</li> <li>▸ Ensure electrical continuity by bonding and grounding (earthing) all equipment.</li> <li>▸ Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (<math>\leq 1</math> m/sec until fill pipe submerged to twice its diameter, then <math>\leq 7</math> m/sec).</li> <li>▸ Avoid splash filling.</li> <li>▸ Do NOT use compressed air for filling discharging or handling operations.</li> <li>▸ Avoid all personal contact, including inhalation.</li> <li>▸ Wear protective clothing when risk of overexposure occurs.</li> <li>▸ Use in a well-ventilated area.</li> <li>▸ Prevent concentration in hollows and sumps.</li> <li>▸ <b>DO NOT enter confined spaces until atmosphere has been checked.</b></li> <li>▸ Avoid smoking, naked lights or ignition sources.</li> <li>▸ Avoid generation of static electricity.</li> <li>▸ <b>DO NOT use plastic buckets.</b></li> <li>▸ Earth all lines and equipment.</li> <li>▸ Use spark-free tools when handling.</li> <li>▸ Avoid contact with incompatible materials.</li> <li>▸ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▸ Keep containers securely sealed when not in use.</li> <li>▸ Avoid physical damage to containers.</li> <li>▸ Always wash hands with soap and water after handling.</li> <li>▸ Work clothes should be laundered separately.</li> <li>▸ Use good occupational work practice.</li> <li>▸ Observe manufacturer's storage and handling recommendations contained within this MSDS.</li> <li>▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.</li> </ul>
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## Other information

- Store in original containers in approved flammable liquid storage area.
  - Store away from incompatible materials in a cool, dry, well-ventilated area.
  - **DO NOT store in pits, depressions, basements or areas where vapours may be trapped.**
  - No smoking, naked lights, heat or ignition sources.
  - Storage areas should be clearly identified, well illuminated, clear of obstruction and accessible only to trained and authorised personnel - adequate security must be provided so that unauthorised personnel do not have access.
  - Store according to applicable regulations for flammable materials for storage tanks, containers, piping, buildings, rooms, cabinets, allowable quantities and minimum storage distances.
  - Use non-sparking ventilation systems, approved explosion proof equipment and intrinsically safe electrical systems.
  - Have appropriate extinguishing capability in storage area (e.g. portable fire extinguishers - dry chemical, foam or carbon dioxide) and flammable gas detectors.
  - Keep adsorbents for leaks and spills readily available.
  - Protect containers against physical damage and check regularly for leaks.
  - Observe manufacturer's storage and handling recommendations contained within this MSDS.
- In addition, for tank storages (where appropriate):
- Store in grounded, properly designed and approved vessels and away from incompatible materials.
  - For bulk storages, consider use of floating roof or nitrogen blanketed vessels; where venting to atmosphere is possible, equip storage tank vents with flame arrestors; inspect tank vents during winter conditions for vapour/ice build-up.
  - Storage tanks should be above ground and diked to hold entire contents.

## Conditions for safe storage, including any incompatibilities

## Suitable container

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

## Storage incompatibility

- Xylenes:
- may ignite or explode in contact with strong oxidisers, 1,3-dichloro-5,5-dimethylhydantoin, uranium fluoride
  - attack some plastics, rubber and coatings
  - may generate electrostatic charges on flow or agitation due to low conductivity.
- For cumene:
- reacts violently with strong acids, strong oxidisers, chlorosulfonic acid, nitric acid, oleum, oxygen
  - unless inhibited can form unstable peroxides
  - prolonged exposure to air forms the highly reactive oxidiser, cumyl hydroperoxide
  - attacks rubber
  - may generate electrostatic charges due to low conductivity
  - Vigorous reactions, sometimes amounting to explosions, can result from the contact between aromatic rings and strong oxidising agents.
  - Aromatics can react exothermically with bases and with diazo compounds.
- For alkyl aromatics:
- The alkyl side chain of aromatic rings can undergo oxidation by several mechanisms. The most common and dominant one is the attack by oxidation at benzylic carbon as the intermediate formed is stabilised by resonance structure of the ring.
- Following reaction with oxygen and under the influence of sunlight, a hydroperoxide at the alpha-position to the aromatic ring, is the primary oxidation product formed (provided a hydrogen atom is initially available at this position) - this product is often short-lived but may be stable dependent on the nature of the aromatic substitution; a secondary C-H bond is more easily attacked than a primary C-H bond whilst a tertiary C-H bond is even more susceptible to attack by oxygen
  - Monoalkylbenzenes may subsequently form monocarboxylic acids; alkyl naphthalenes mainly produce the corresponding naphthalene carboxylic acids.
  - Oxidation in the presence of transition metal salts not only accelerates but also selectively decomposes the hydroperoxides.
  - Hock-rearrangement by the influence of strong acids converts the hydroperoxides to hemiacetals. Peresters formed from the hydroperoxides undergo Criegee rearrangement easily.
  - Alkali metals accelerate the oxidation while CO<sub>2</sub> as co-oxidant enhances the selectivity.

- Microwave conditions give improved yields of the oxidation products.
- Photo-oxidation products may occur following reaction with hydroxyl radicals and NO<sub>x</sub> - these may be components of photochemical smogs.

Oxidation of Alkylaromatics: T.S.S Rao and Shubhra Awasthi: E-Journal of Chemistry Vol 4, No. 1, pp 1-13 January 2007

Contact with high pressure oxygen may cause ignition / combustion.

#### PACKAGE MATERIAL INCOMPATIBILITIES

### SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

#### Control parameters

#### OCCUPATIONAL EXPOSURE LIMITS (OEL)

#### INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
New Zealand Workplace Exposure Standards (WES)	xylene	Xylene (o-, m-, p-isomers)	217 (mg/m <sup>3</sup> ) / 50 (ppm)	Not Available	Not Available	Not Available
New Zealand Workplace Exposure Standards (WES)	toluene	Toluene	188 (mg/m <sup>3</sup> ) / 50 (ppm)	Not Available	Not Available	Skin absorption

#### EMERGENCY LIMITS

Ingredient	TEEL-0	TEEL-1	TEEL-2	TEEL-3
aromatic hydrocarbon solvent	500(ppm)	750(ppm)	750(ppm)	750(ppm)
linseed oil	0.5 / 7.5(ppm)	1.5 / 25(ppm)	10 / 150(ppm)	60 / 500(ppm)
xylene	100(ppm)	130(ppm)	920(ppm)	2500(ppm)
toluene	200(ppm)	200(ppm)	510(ppm)	2900(ppm)

Ingredient	Original IDLH	Revised IDLH
xylene	1,000(ppm)	900(ppm)
toluene	2,000(ppm)	500(ppm)

#### 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.

For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

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	<p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="443 237 1524 495"> <thead> <tr> <th data-bbox="443 237 981 277">Lower end of the range</th> <th data-bbox="981 237 1524 277">Upper end of the range</th> </tr> </thead> <tbody> <tr> <td data-bbox="443 277 981 344">1: Room air currents minimal or favourable to capture</td> <td data-bbox="981 277 1524 344">1: Disturbing room air currents</td> </tr> <tr> <td data-bbox="443 344 981 412">2: Contaminants of low toxicity or of nuisance value only.</td> <td data-bbox="981 344 1524 412">2: Contaminants of high toxicity</td> </tr> <tr> <td data-bbox="443 412 981 452">3: Intermittent, low production.</td> <td data-bbox="981 412 1524 452">3: High production, heavy use</td> </tr> <tr> <td data-bbox="443 452 981 495">4: Large hood or large air mass in motion</td> <td data-bbox="981 452 1524 495">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
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<p><b>Personal protection</b></p>											
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens 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]</li> </ul>										
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>										
<p><b>Hand protection</b></p>	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <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>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"> <li>▶ frequency and duration of contact,</li> <li>▶ chemical resistance of glove material,</li> <li>▶ glove thickness and</li> <li>▶ dexterity</li> </ul> <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"> <li>▶ 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.</li> <li>▶ 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.</li> <li>▶ Contaminated gloves should be replaced.</li> </ul> <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>										
<p><b>Body protection</b></p>	<p>See Other protection below</p>										
<p><b>Other protection</b></p>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ PVC Apron.</li> <li>▶ PVC protective suit may be required if exposure severe.</li> <li>▶ Eyewash unit.</li> <li>▶ Ensure there is ready access to a safety shower.</li> <li>▶ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.</li> <li>▶ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or</li> </ul>										

► pockets), non sparking safety footwear.

### Thermal hazards

## 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:

Altex RIPO Not Available

Material	CPI
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\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

## Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	Air-line*	-	-
up to 100 x ES	-	A-3 P2	-
100+ x ES	-	Air-line**	-

\* - Continuous-flow; \*\* - Continuous-flow or positive pressure demand

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(SO<sub>2</sub>), G = Agricultural chemicals, K = Ammonia(NH<sub>3</sub>), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

## SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

### Information on basic physical and chemical properties

<b>Appearance</b>	Coloured with Characteristic Odour		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	0.89
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	465
<b>pH (as supplied)</b>	Not Available	<b>Decomposition temperature</b>	Not Available
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	146	<b>Molecular weight (g/mol)</b>	Not Available
<b>Flash point (°C)</b>	Not Available	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	0.7	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Available	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	7.1	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	0.7	<b>Volatile Component (%vol)</b>	69
<b>Vapour pressure (kPa)</b>	1.35	<b>Gas group</b>	Not Available
<b>Solubility in water (g/L)</b>	Immiscible	<b>pH as a solution(1%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	3.9		

## SECTION 10 STABILITY AND REACTIVITY

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>► Presence of incompatible materials.</li> <li>► Product is considered stable.</li> <li>► Hazardous polymerisation will not occur.</li> </ul>

<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 TOXICOLOGICAL INFORMATION

### Information on toxicological effects

<b>Inhaled</b>	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <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>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 respiratory depression and arrest. Cardiac arrest may result from cardiovascular collapse. Bradycardia, and hypotension may also be produced.</p> <p>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>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.</p> <p>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>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>Headache, fatigue, lassitude, irritability and gastrointestinal disturbances (e.g., nausea, anorexia and flatulence) are the most common symptoms of xylene overexposure. Injury to the heart, liver, kidneys and nervous system</p>
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	<p>has also been noted amongst workers. Transient memory loss, renal impairment, temporary confusion and some evidence of disturbance of liver function was reported in three workers overcome by gross exposure to xylene (10000 ppm). One worker died and autopsy revealed pulmonary congestion, oedema and focal alveolar haemorrhage. Volunteers inhaling xylene at 100 ppm for 5 to 6 hours showed changes in manual coordination reaction time and slight ataxia. Tolerance developed during the workweek but was lost over the weekend. Physical exercise may antagonise this effect. Xylene body burden in humans exposed to 100 or 200 ppm xylene in air depends on the amount of body fat with 4% to 8% of total absorbed xylene accumulating in adipose tissue. Xylene is a central nervous system depressant. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual. Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result. Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis). Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption. The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either</p> <ul style="list-style-type: none"> <li>▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▸ produces significant, but moderate, 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.</li> </ul> <p>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>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>The liquid may be miscible with fats or oils and may degrease the skin, producing a skin reaction described as non-allergic contact dermatitis. The material is unlikely to produce an irritant dermatitis as described in EC Directives .</p> <p>The material may accentuate any pre-existing dermatitis condition</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>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. Evidence exists, or practical experience predicts, that the material may cause severe eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Eye contact may cause significant inflammation with pain. Corneal injury may occur; permanent impairment of vision may result unless treatment is prompt and adequate. Repeated or prolonged exposure to irritants may cause inflammation characterised by a temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>The liquid produces a high level of eye discomfort and is capable of causing pain and severe conjunctivitis. Corneal injury may develop, with possible permanent impairment of vision, if not promptly and adequately treated.</p>
Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance)</p>

is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

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

There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.

Human and animal exposures to the phytoestrogens (for example the isoflavones, some flavonoids, saponin, coumestans and lignans) can be high because these compounds are found in many foods. Interest in the dietary phytoestrogens derives from their apparent protective effects against cancer, cardiovascular disease and osteoporosis. High levels, over extended periods, may produce toxic effects.

However, toxicological studies revealed that when administered in isolated or enriched form or at high doses isoflavones impair the function of the thyroid gland. It cannot be ruled out that this oestrogen-like effect also encourages the onset of breast cancer. Since women are more at risk of developing cancer in any case after menopause, the intake of food supplements with a high isoflavone content may present unexpected risks for this group of consumers.

Although phytoestrogens exist as the inactive glycoside in food products, bacterial beta-glycosidases, in the colon, hydrolyse the glycosides to the active aglycones.

A common feature of the phytoestrogens is their striking similarity to 17 $\beta$ -oestradiol and the synthetic oestrogen, diethylstilboestrol. There is evidence that phytoestrogens may mediate oestrogen-like effects by direct interaction with the oestrogen receptor of cells. Although the hormonal activity of phytoestrogens is two to five orders of magnitude below that of oestradiol, their high concentration in certain plants and their slower metabolic disposition, can lead to tissue levels exceeding those of endogenous oestrogens by a factor of a thousand or more.

There is also evidence that phytoestrogens may influence animal and human health by acting as antioxidants and hydrogen peroxide scavengers or by interfering with eicosanoid and cytokine production and cell signalling. Anogenital distance, puberty onset, oestrus cycling, growth, sex-organ weight and hormonal profile are indicators of oestrogen- or anti-oestrogen like activity. Of interest is the finding that low doses of the dietary isoflavone, genistein, taken by pregnant rats produced shorter anogenital distances in the offspring; high doses did not produce this effect. Exposure to a 5% flaxseed diet (high in lignans) during pregnancy and lactation, resulted in the delayed puberty onset in rats (anti-oestrogenic effect). By contrast, a 10% flaxseed diet produced an earlier onset of puberty (an oestrogenic effect), but longer oestrus cycles due to prolonged dioestrus (an antioestrogenic effect).

There have been many reports of phytoestrogens disrupting reproductive activity in sheep. Infertility in sheep (so-called "clover disease") has been traced to isoflavone concentrations in clover (up to 5% dry weight).

Temporary infertility is attributed to increased embryo mortality and a reduction or cessation in ovulation. Permanent infertility, in sheep, is purported to occur after 3 years of exposure to dietary oestrogenic compounds; this infertility is due to permanent changes in the architecture of the cervix and also changes in the viscoelasticity of the cervical mucous which prevents the transport of sperm through the cervix. In addition to these effects, phytoestrogens exert effects on oestrogen-sensitive tissues such as the mammary gland and female reproductive organs of the ewe. Cattle have also been shown to be sensitive to the oestrogen-like effects of dietary phytoestrogens. Specific observations include swelling of the vulva, discharge of cervical mucous, uterus enlargements and cystic ovaries. Irregular oestrus cycles, including periods of anoestrus, and decreased rates of conception have also been reported. The impact on reproductive activity, by phytoestrogens on humans, is unknown.

The recent practice of feeding infants soy-based formula raises issues related to the long-term health effects of exposure during development. It has been recognised, for example, that the practice may be associated with goiter (thyroid enlargement associated with thyroid hormone deficiency) in humans and animals. Soy phytoestrogens inhibit thyroid hormone synthesis at concentrations which occur in infant formula.

If sufficient inhibition of iodide uptake by the goiter occurs, formation of thyroid hormones is depressed. These hormones are essential to the regulation of oxygen consumption and metabolism throughout the body. Clinical manifestations of this so-called "**hypothyroidism (or athyrea)**" include low metabolic rate, a tendency to gain weight, somnolence, and *myxoedema* (a relatively hard oedema of the subcutaneous tissue), dryness and loss of hair, low body temperature, hoarseness, muscle weakness, a slow return of the muscle after tendon jerk, and slow mentation. When hypothyroidism occurs in women, early in pregnancy, the foetus is at risk of impaired physical and mental development, the severity of the impairment depending on the degree of hypothyroidism.

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

## Altex RIPO

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.

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.

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

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

Xylene has been classed as a developmental toxin in some jurisdictions.

Small excess risks of spontaneous abortion and congenital malformation were reported amongst women exposed to xylene in the first trimester of pregnancy. In all cases, however, the women were also been exposed to other substances. Evaluation of workers chronically exposed to xylene has demonstrated lack of genotoxicity.

Exposure to xylene has been associated with increased risks of haemopoietic malignancies but, again, simultaneous exposure to other substances (including benzene) complicates the picture. A long-term gavage study to mixed xylenes (containing 17% ethyl benzene) found no evidence of carcinogenic activity in rats and mice of either sex.

Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

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

Altex RIPO	TOXICITY	IRRITATION
	Not Available	Not Available
aromatic hydrocarbon solvent	TOXICITY	IRRITATION
	Not Available	Not Available
linseed oil	TOXICITY	IRRITATION
	Oral (Rat) LD50: >2000 mg/kg *	Skin (human):300 mg/3days-moderate
	Not Available	Not Available
xylene	TOXICITY	IRRITATION
	Inhalation (rat) LC50: 5000 ppm/4h	Eye (human): 200 ppm irritant
	Intraperitoneal (Mouse) LD50: 1548 mg/kg	Eye (rabbit): 5 mg/24h SEVERE
	Intraperitoneal (Rat) LD50: 2459 mg/kg	Eye (rabbit): 87 mg mild
	Oral (Mouse) LD50: 2119 mg/kg	Skin (rabbit):500 mg/24h moderate
	Oral (rat) LD50: 4300 mg/kg	
	Subcutaneous (Rat) LD50: 1700 mg/kg	
	Not Available	Not Available
chlorinated rubber - carbon tetrachloride free	TOXICITY	IRRITATION
	Not Available	Not Available
naphtha, petroleum, hydrosulfurised heavy	TOXICITY	IRRITATION
	Not Available	Not Available
toluene	TOXICITY	IRRITATION

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Dermal (rabbit) LD50: 12124 mg/kg	Eye (rabbit): 2mg/24h - SEVERE
Inhalation (rat) LC50: >26700 ppm/1h	Eye (rabbit):0.87 mg - mild
Oral (rat) LD50: 636 mg/kg	Eye (rabbit):100 mg/30sec - mild
	Skin (rabbit):20 mg/24h-moderate
	Skin (rabbit):500 mg - moderate
Not Available	Not Available

## Altex RIPO

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

No significant acute toxicological data identified in literature search.

Group A aliphatic monoesters (fatty acid esters)

According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group A substances are simple monoesters derived from a monofunctional alcohol, such as 2-ethylhexyl alcohol (C8-alcohol) or tridecyl alcohol (C13 alcohol) and **fatty acids** such as palmitic, stearic, oleic or linoleic acid. Metabolism of the parent esters is expected to yield the corresponding fatty acids and alcohols. The fatty acids are naturally occurring and have a low order of toxicity.

Group A substances are rather lipophilic (log Kow 10-15) in character due to the large number of carbon numbers in the ester molecule (e.g., 24,26, 31 carbons) and have relatively high boiling points. Owing to the non-volatile nature of these esters, their vapour pressures are very low and difficult to determine experimentally. Water solubility is also very low.

**Mammalian Toxicity:**

**Acute Toxicity.** Many higher fatty acid esters, such as the stearates, oleates and palmitates, have been cleared for use in the food industry ; thus, their general physiological response and toxicity are very low. Many of the higher fatty acid esters are considered safe for use in cosmetics.

Available acute toxicity data indicate that the fatty acid esters in Group A, in general, have a low order of toxicity [e.g., palmitic acid, 2-ethylhexyl ester (LD50 > 5 g/kg) and tall oil fatty acid 2-ethylhexyl ester (LD50 > 64 g/kg)]. Consistent with that, available data spanning the carbon range of C22 to C34 indicate that the alkyl fatty acid esters are not toxic by oral administration [rat LD50 (oral) > 5g/kg, with range from 5 g/kg to 64 kg/kg]. Butyl stearate is tolerated by rats without lethal effects at oral doses of 32 g/kg while octyl oleate has a reported LD50 of >40 ml/kg.

In addition, many alkyl fatty acid esters, such as the stearates, oleates and palmitates, have been demonstrated to be not toxic by dermal administration

Because of the low volatility of these substances, inhalation exposure at toxicological significant levels is not expected.

**Repeated Dose Toxicity.** 28-Day oral gavage studies in rats with decyl oleate (CAS 3687-46-5) at doses of 100,500 and 1000 mg/kg showed no toxicity as noted with respect to clinical symptoms, biochemistry, hematology, gross lesions or tissue/organ histopathology. The NOAEL was estimated to be 1000 mg/kg. Similarly, octyl or (2-ethylhexyl) stearate showed a NOAEL of 1000 mg/kg in 28-day oral gavage studies in rats.

In chronic two-year feeding studies with butyl stearate at concentrations of 1.25% or 6.25% in the diet, exposed rats showed no significant difference from control animals with respect to growth, survival, blood counts or other haematological parameters.

Besides the two substances above, various other long-chain fatty acid esters have also been studied for their repeated dose toxicity and the findings support a low order of toxicity.

**Genotoxicity:**

**Genetic Toxicity (Salmonella).** Fatty acid, C 16- 18 saturated and C 18 unsaturated, 2-ethylhexanoate (CAS 85049-37-2); octyl stearate (CAS 109-36-4); and decyl oleate (CAS 3687-46-5) were shown to be negative in the Ames assay. Since the monoesters are similar in chemical structure and carbon-number range, it is unlikely that esters in Group A will induce point mutation. In addition, the chemistry of the long-chain fatty acids does not suggest the likelihood that these substances or their constituent substructures (i.e., fatty acids, alcohols) are reactive or electrophilic in nature.

**Genetic Toxicity (Chromosomal Aberrations).** The chemistry of the long-chain fatty acid esters does not suggest the likelihood that these substances or their constituent substructures (i.e., fatty acids, alcohols) are reactive or electrophilic in nature. Therefore, the likelihood that the fatty acid monoesters may cause chromosomal mutation is very low.

**Reproductive toxicity:** Assessment of reproductive effects of alkyl fatty acid esters in Group A is based primarily on studies with butyl stearate. Fertility, litter size and survival of offspring were normal in rats fed diets containing 6.25% butyl stearate for 10 weeks. However, growth was reduced in offspring during the pre-weaning and post-weaning periods. No gross lesions were noted among the offspring killed at the end of the 21-day post-weaning periods. These results indicate that long-chain fatty acid esters do not cause reproductive toxicity in rats. Given the relative low order of toxicity for long-chain fatty acid esters and their relative non-electrophilic and non-reactive nature, it seems unlikely that the long-chain fatty acid esters would present serious reproductive concerns.

**Developmental Toxicity/ Teratogenicity.** Assessment of developmental effects for the long-chain fatty acid esters in this group was based primarily on data reported for fatty acid, C16-18, 2-ethylhexyl ester (CAS 91031-48-0). In oral gavage studies in rats administered doses of 100,300 and 1000 mg/kg during gestation, the maternal NOAEL was > 1000 mg/kg and the NOAEL for teratogenicity was >1000 mg/kg. Based on these findings and the fact Group A substances, are very chemically similar to the structure of the tested material, read-across assessment is thought to be appropriate.

For Group E aliphatic esters (polyol esters):

According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group E substances are esters of monoacids, mainly common fatty acids, and trihydroxy or polyhydroxyalcohols or polyols, such as pentaerythritol (PE), 2-ethyl-2-(hydroxymethyl)- 1,3-propanediol or trimethylolpropane (TMP), and dipentaerythritol (diPE). The Group E substances often are referred to as "polyol esters" The polyol esters are unique in their chemical characteristics since they lack beta-tertiary hydrogen atoms, thus leading to stability against oxidation and elimination. The fatty acids often range from C5-C10 to as high as C18 (e.g., oleic, stearic, isostearic, tall oil fatty acids) in carbon number and generally are derived from naturally occurring sources. Group E esters may have multiple ester linkages and may include mixed esters derived from different carbon-length fatty acid mixtures. The lack of beta-tertiary hydrogen atoms in the structure of the polyol esters makes them characteristically and chemically stable against oxidation and elimination in comparison to other ester classes or groups. For these reasons, trimethylolpropane (TMP) and pentaerythritol (PE) esters with fatty acids of C5 to C10 carbon-chain length have applications as synthetic lubricants for passenger car motor oil and military and civilian jet engines. TMP and PE esters of C18 acids (e.g., isostearic and oleic acids) also have found use in synthetic lubricant applications, including refrigeration lubricants and hydraulic fluids. Because of their higher thermal stability characteristics, they also find use in a variety of high temperature applications such as industrial oven chain oils, high temperature greases, fire resistant transformer coolants and turbine engines

Polyol esters that are extensively esterified also have greater polarity, less volatility and enhanced lubricity characteristics. **Acute toxicity:** Depending on the degree of esterification, the polyol esters can be resistant or slow towards chemical or enzymatic hydrolysis (i.e., esterase or lipases) as a result of steric hindrance. PE and diPE esters that are capable of being enzymatically hydrolyzed will generate pentaerythritol or dipentaerythritol, and the corresponding fatty acids which, for most of the Group E esters, are comprised mainly of oleic, linoleic and stearic acids as well as the fatty acids in the C5-10 carbon-length. Similarly, TMP esters can undergo metabolism to yield trimethylolpropane (2-ethyl-2-hydroxymethyl-1,3-propanediol) and fatty acid constituents. Pentaerythritol and trimethylolpropane have been reported to have a low order of toxicity. The acute oral LD50 for these substances was greater than 2000 mg/kg indicating a relatively low order of toxicity. The similarity in the low order of toxicity for these substances is consistent with their similar chemical structure and physicochemical properties

**Repeat dose toxicity:** Polyol esters are generally well tolerated by rats in 28-day oral toxicity studies. NOAEL for these substances was 1000 mg/kg/day in Sprague-Dawley rats. The TMP ester of heptanoic and octanoic acid did not produce signs of overt systemic toxicity at any dose levels tested (i.e., 100, 300, and 1000 mg/kg/day). There were no treatment-related clinical in-life, functional observation battery, or gross postmortem findings. There were no treatment related mortality, and no adverse effects on body weight, food consumption, clinical laboratory parameters, or organ weights. However, there were increased numbers of hyaline droplets in the proximal cortical tubular epithelium of the 300 and 1000 mg/kg/day in male rats. Based on these findings (hyaline droplets), the NOAEL for this polyol ester was established at 100 mg/kg/day for male rats. Hyaline droplet formation observed in the male kidneys is believed to be a sex/species condition specific to only male rats, which has little relevance to humans. The results from these repeated dose dermal toxicity studies suggest that polyol esters exhibit a low order of toxicity following repeated application. This may be attributable to similarities in their chemical structures, physicochemical properties, and common metabolic pathways (i.e., esters can be enzymatically hydrolyzed to the corresponding polyalcohol and the corresponding fatty acids) The polyol, hexanedioic acid, mixed esters with decanoic acid, heptanoic acid, octanoic acid and PE, was applied to the skin of groups of 10 (male and female) rats for five days a week for four (4) weeks at dose levels of 0, 125, 500 and 2000 mg/kg/day. Treated animals exhibited no signs indicative of systemic toxicity. No visible signs of irritation were observed at treatment sites. Microscopically, treated skin (viz., greater than or equal to 500 mg/kg/day) exhibited a dose-related increased incidence and severity of hyperplasia and hyperkeratosis of the epidermis and sebaceous gland hyperplasia. These effects were reversible. None of the minor changes in haematology and serum chemistry parameters were considered biologically significant. High dose females (2000 mg/kg/day) exhibited a significant increase in relative adrenal and brain weights when compared to the controls. These

differences were attributed to the lower final body weight of the female animals. The NOAEL in this study for systemic toxicity was established as 500 mg /kg/day and 125 mg/kg/day for skin irritation.

**Reproductive and developmental toxicity:** Since metabolism of the polyol esters can occur, leading to the generation of the corresponding fatty acids and the polyol alcohol (such as pentaerythritol, trimethylolpropane, and dipentaerythritol), the issue of whether these metabolites may pose any potential reproductive/developmental toxicity concerns is important. However, the polyol alcohols such as pentaerythritol, trimethylolpropane, and dipentaerythritol, would be expected to undergo further metabolism, conjugation and excretion in the urine. Available evidence indicates that these ester hydrolysates (i.e., hydrolysis products), primarily fatty acids (e.g., heptanoic, octanoic, and decanoic acids) and secondarily the polyol alcohols should exhibit a low order of reproductive toxicity. It can be concluded that this group of high molecular weight polyol esters should not produce profound reproductive effects in rodents.

**Genotoxicity:** Polyols tested for genetic activity in the Salmonella assay, have been found to be inactive. Several polyol esters have been adequately tested for chromosomal mutation in the in vitro mammalian chromosome aberration assay, and all were inactive. Two TMP esters were also tested for in vivo chromosomal aberration in rats, and both demonstrated no activity. Thus, it is unlikely that these substances are chromosomal mutagens.

For trimethylbenzenes:

Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption. 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-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.

**Acute Toxicity** Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis. High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness. The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes. Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg). Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels. No effects were reported for rats exposed to a mixture of trimethylbenzenes at 1700 ppm for 10 to 21 days.

**Neurotoxicity** 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes. Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.

**Subchronic/Chronic Toxicity** Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; hematological effects may have been due to trace amounts of benzene.

Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia.

**Genotoxicity:** Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella 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.

**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.

No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethyl- benzenes, 4-6 hours/day, 5 days/week over one generation.

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

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

NOTE: C12, 60% chlorinated paraffin [CAS RN 108171-26-2] is classified by IARC as Group 2B. Possibly carcinogenic to humans.

Studies using the C12, 59% chlorinated variant (in combination with corn oil) caused tumors when force fed at very high doses over long periods of time. Pregnant rats fed C16, 52% chlorinated paraffin had offspring which died during weaning.

High molecular weight liquid chloroparaffins are considered to be practically non-harmful. Special consideration should be given to solid grades of the material (eg Cereclor 70) because of relatively high levels of carbon tetrachloride remaining as a residual reactant. Vapours are readily absorbed through intact skin, requiring additional precautions in handling.

Lifetime studies have been carried out with two grades of chlorinated paraffins. A short-chain grade with 58% chlorine caused tumours in rats and mice. Male mice exposed to long-chain grades with 40% chlorine showed an excess of tumours at one site. It has been shown that the mechanisms by which short-term paraffins cause tumours are specific to rodents and may not have relevance to human health. Furthermore, chlorinated paraffins have been shown to non-genotoxic.

The Regulatory regime in various countries differs with respect to chlorinated paraffins.

In the USA, the short-chain (C12), 58% chlorine product has been classified and labelled as a carcinogen.

In Germany the MAK Commission has classified most chlorinated paraffins as Category IIIB (suspect carcinogens). They are not however included in the list of substances (TRGS 905) required to be labelled.

All EU Member States are required to classify short chain chlorinated paraffins as Category 3 carcinogens.

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

For trimethylbenzenes:

Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption. 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells.

Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid. The major routes of excretion of 1,2,4-trimethyl- benzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.

**Acute Toxicity** Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis. High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness. The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm

#### AROMATIC HYDROCARBON SOLVENT

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.

**Neurotoxicity** 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes.

Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.

**Subchronic/Chronic Toxicity** Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene.

Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia.

**Genotoxicity:** Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella 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.

**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.

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.

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

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

**for petroleum:**

This product contains benzene which is known to cause acute myeloid leukaemia and n-hexane which has been shown to metabolize to compounds which are neuropathic.

This product contains toluene. There are indications from animal studies that prolonged exposure to high concentrations of toluene may lead to hearing loss.

This product contains ethyl benzene and naphthalene from which there is evidence of tumours in rodents.

**Carcinogenicity:** Inhalation exposure to mice causes liver tumours, which are not considered relevant to humans. Inhalation exposure to rats causes kidney tumours which are not considered relevant to humans.

**Mutagenicity:** There is a large database of mutagenicity studies on gasoline and gasoline blending streams, which use a wide variety of endpoints and give predominantly negative results. All in vivo studies in animals and recent studies in exposed humans (e.g. petrol service station attendants) have shown negative results in mutagenicity assays.

**Reproductive Toxicity:** Repeated exposure of pregnant rats to high concentrations of toluene (around or exceeding 1000 ppm) can cause developmental effects, such as lower birth weight and developmental neurotoxicity, on the foetus. However, in a two-generation reproductive study in rats exposed to gasoline vapour condensate, no adverse effects on the foetus were observed.

**Human Effects:** Prolonged/ repeated contact may cause defatting of the skin which can lead to dermatitis and may make the skin more susceptible to irritation and penetration by other materials.

Lifetime exposure of rodents to gasoline produces carcinogenicity although the relevance to humans has been questioned. Gasoline induces kidney cancer in male rats as a consequence of accumulation of the alpha2-microglobulin protein in hyaline droplets in the male (but not female) rat kidney. Such abnormal accumulation

represents lysosomal overload and leads to chronic renal tubular cell degeneration, accumulation of cell debris, mineralisation of renal medullary tubules and necrosis. A sustained regenerative proliferation occurs in epithelial cells with subsequent neoplastic transformation with continued exposure. The alpha2-microglobulin is produced under the influence of hormonal controls in male rats but not in females and, more importantly, not in humans.

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 of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

Group A aliphatic monoesters (fatty acid esters)

According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group A substances are simple monoesters derived from a monofunctional alcohol, such as 2-ethylhexyl alcohol (C8-alcohol) or tridecyl alcohol (C13 alcohol) and **fatty acids** such as palmitic, stearic, oleic or linoleic acid. Metabolism of the parent esters is expected to yield the corresponding fatty acids and alcohols. The fatty acids are naturally occurring and have a low order of toxicity.

Group A substances are rather lipophilic (log Kow 10-15) in character due to the large number of carbon numbers in the ester molecule (e.g., 24,26, 31 carbons) and have relatively high boiling points. Owing to the non-volatile nature of these esters, their vapour pressures are very low and difficult to determine experimentally. Water solubility is also very low.

#### **Mammalian Toxicity:**

**Acute Toxicity.** Many higher fatty acid esters, such as the stearates, oleates and palmitates, have been cleared for use in the food industry ; thus, their general physiological response and toxicity are very low.

Many of the higher fatty acid esters are considered safe for use in cosmetics.

Available acute toxicity data indicate that the fatty acid esters in Group A, in general, have a low order of toxicity [e.g., palmitic acid, 2-ethylhexyl ester (LD50 > 5 g/kg) and tall oil fatty acid 2-ethylhexyl ester (LD50 > 64 g/kg)]. Consistent with that, available data spanning the carbon range of C22 to C34 indicate that the alkyl fatty acid esters are not toxic by oral administration [rat LD50 (oral) > 5g/kg, with range from 5 g/kg to 64 kg/kg]. Butyl stearate is tolerated by rats without lethal effects at oral doses of 32 g/kg while octyl oleate has a reported LD50 of >40 ml/kg.

In addition, many alkyl fatty acid esters, such as the stearates, oleates and palmitates, have been demonstrated to be not toxic by dermal administration

Because of the low volatility of these substances, inhalation exposure at toxicological significant levels is not expected.

**Repeated Dose Toxicity.** 28-Day oral gavage studies in rats with decyl oleate (CAS 3687-46-5) at doses of 100,500 and 1000 mg/kg showed no toxicity as noted with respect to clinical symptoms, biochemistry, hematology, gross lesions or tissue/organ histopathology. The NOAEL was estimated to be 1000 mg/kg. Similarly, octyl or (2-ethylhexyl) stearate showed a NOAEL of 1000 mg/kg in 28-day oral gavage studies in rats.

In chronic two-year feeding studies with butyl stearate at concentrations of 1.25% or 6.25% in the diet, exposed rats showed no significant difference from control animals with respect to growth, survival, blood counts or other haematological parameters.

Besides the two substances above, various other long-chain fatty acid esters have also been studied for their repeated dose toxicity and the findings support a low order of toxicity.

#### **Genotoxicity:**

**Genetic Toxicity (Salmonella).** Fatty acid, C 16- 18 saturated and C 18 unsaturated, 2-ethylhexanoate (CAS 85049-37-2); octyl stearate (CAS 109-36-4); and decyl oleate (CAS 3687-46-5)] were shown to be negative in the Ames assay. Since the monoesters are similar in chemical structure and carbon-number range, it is unlikely that esters in Group A will induce point mutation. In addition, the chemistry of the long-chain fatty acids does not suggest the likelihood that these substances or their constituent substructures (i.e., fatty acids, alcohols) are reactive or electrophilic in nature.

**Genetic Toxicity (Chromosomal Aberrations).** The chemistry of the long-chain fatty acid esters does not suggest the likelihood that these substances or their constituent substructures (i.e., fatty acids, alcohols) are reactive or electrophilic in nature. Therefore, the likelihood that the fatty acid monoesters may cause chromosomal mutation is very low.

**Reproductive toxicity:** Assessment of reproductive effects of alkyl fatty acid esters in Group A is based primarily on studies with butyl stearate. Fertility, litter size and survival of offspring were normal in rats fed diets containing 6.25% butyl stearate for 10 weeks. However, growth was reduced in offspring during the pre-weaning and post-weaning periods. No gross lesions were noted among the offspring killed at the end of the 21-day post-weaning periods. These results indicate that long-chain fatty acid esters do not cause reproductive toxicity in rats..Given the relative low order of toxicity for long-chain fatty acid esters and their relative non-electrophilic and non-reactive nature, it seems unlikely that the long-chain fatty acid esters would present serious reproductive concerns.

**Developmental Toxicity/ Teratogenicity.** Assessment of developmental effects for the long-chain fatty acid esters in this group was based primarily on data reported for fatty acid, C16-18, 2-ethylhexyl ester (CAS 91031-48-0). In oral gavage studies in rats administered doses of 100,300 and 1000 mg/kg during gestation, the maternal NOAEL was > 1000 mg/kg and the NOAEL for teratogenicity was >1000 mg/kg. Based on these findings and the fact Group A substances, are very chemically similar to the structure of the tested material,

## LINSEED OIL

	<p>read-across assessment is thought to be appropriate. * Akzo Nobel SDS</p>
<p style="text-align: center;"><b>XYLENE</b></p>	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. The substance is classified by IARC as Group 3: <b>NOT</b> classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. Reproductive effector in rats</p>
<p style="text-align: center;"><b>CHLORINATED RUBBER - CARBON TETRACHLORIDE FREE</b></p>	<p>No significant acute toxicological data identified in literature search. NOTE: C12, 60% chlorinated paraffin [CAS RN 108171-26-2] is classified by IARC as Group 2B. Possibly carcinogenic to humans. Studies using the C12, 59% chlorinated variant (in combination with corn oil) caused tumors when force fed at very high doses over long periods of time. Pregnant rats fed C16, 52% chlorinated paraffin had offspring which died during weaning. High molecular weight liquid chloroparaffins are considered to be practically non-harmful. Special consideration should be given to solid grades of the material (eg Cereclor 70) because of relatively high levels of carbon tetrachloride remaining as a residual reactant. Vapours are readily absorbed through intact skin, requiring additional precautions in handling. Lifetime studies have been carried out with two grades of chlorinated paraffins. A short-chain grade with 58% chlorine caused tumours in rats and mice. Male mice exposed to long-chain grades with 40% chlorine showed an excess of tumours at one site. It has been shown that the mechanisms by which short-term paraffins cause tumours are specific to rodents and may not have relevance to human health. Furthermore, chlorinated paraffins have been shown to non-genotoxic. The Regulatory regime in various countries differs with respected to chlorinated paraffins. In the USA, the short-chain (C12), 58% chlorine product has been classified and labelled as a carcinogen. In Germany the MAK Commission has classified most chlorinated paraffins as Category IIIB (suspect carcinogens). They are not however included in the list of substances (TRGS 905) required to be labelled. All EU Member States are required to classify short chain chlorinated paraffins as Category 3 carcinogens.</p>
<p style="text-align: center;"><b>NAPHTHA, PETROLEUM, HYDRODESULFURISED HEAVY</b></p>	<p>No significant acute toxicological data identified in literature search.</p>
<p style="text-align: center;"><b>TOLUENE</b></p>	<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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. For toluene: <b>Acute Toxicity</b> Humans exposed to intermediate to high levels of toluene for short periods of time experience adverse central nervous system effects ranging from headaches to intoxication, convulsions, narcosis, and death. Similar effects are observed in short-term animal studies. <b>Humans</b> - Toluene ingestion or inhalation can result in severe central nervous system depression, and in large doses, can act as a narcotic. The ingestion of about 60 mL resulted in fatal nervous system depression within 30 minutes in one reported case. Constriction and necrosis of myocardial fibers, markedly swollen liver, congestion and haemorrhage of the lungs and acute tubular necrosis were found on autopsy. Central nervous system effects (headaches, dizziness, intoxication) and eye irritation occurred following inhalation exposure to 100 ppm toluene 6 hours/day for 4 days. Exposure to 600 ppm for 8 hours resulted in the same and more serious symptoms including euphoria, dilated pupils, convulsions, and nausea . Exposure to 10,000-30,000 ppm has been reported to cause narcosis and death Toluene can also strip the skin of lipids causing dermatitis <b>Animals</b> - The initial effects are instability and incoordination, lachrymation and sniffles (respiratory exposure), followed by narcosis. Animals die of respiratory failure from severe nervous system depression. Cloudy swelling of the kidneys was reported in rats following inhalation exposure to 1600 ppm, 18-20 hours/day for 3 days <b>Subchronic/Chronic Effects:</b> Repeat doses of toluene cause adverse central nervous system effects and can damage the upper respiratory system, the liver, and the kidney. Adverse effects occur as a result from both oral and the inhalation exposures. A reported lowest-observed-effect level in humans for adverse neurobehavioral effects is 88 ppm. <b>Humans</b> - Chronic occupational exposure and incidences of toluene abuse have resulted in hepatomegaly and</p>

liver function changes. It has also resulted in nephrotoxicity and, in one case, was a cardiac sensitiser and fatal cardiotoxin.

Neural and cerebellar dystrophy were reported in several cases of habitual "glue sniffing." An epidemiological study in France on workers chronically exposed to toluene fumes reported leukopenia and neutropenia.

Exposure levels were not given in the secondary reference; however, the average urinary excretion of hippuric acid, a metabolite of toluene, was given as 4 g/L compared to a normal level of 0.6 g/L

**Animals** - The major target organs for the subchronic/chronic toxicity of toluene are the nervous system, liver, and kidney. Depressed immune response has been reported in male mice given doses of 105 mg/kg/day for 28 days. Toluene in corn oil administered to F344 male and female rats by gavage 5 days/week for 13 weeks, induced prostration, hypoactivity, ataxia, piloerection, lachrymation, excess salivation, and body tremors at doses 2500 mg/kg. Liver, kidney, and heart weights were also increased at this dose and histopathologic lesions were seen in the liver, kidneys, brain and urinary bladder. The no-observed-adverse effect level (NOAEL) for the study was 312 mg/kg (223 mg/kg/day) and the lowest-observed-adverse effect level (LOAEL) for the study was 625 mg/kg (446 mg/kg/day) .

#### Developmental/Reproductive Toxicity

Exposures to high levels of toluene can result in adverse effects in the developing human foetus. Several studies have indicated that high levels of toluene can also adversely effect the developing offspring in laboratory animals.

**Humans** - Variable growth, microcephaly, CNS dysfunction, attentional deficits, minor craniofacial and limb abnormalities, and developmental delay were seen in three children exposed to toluene in utero as a result of maternal solvent abuse before and during pregnancy

**Animals** - Sternebral alterations, extra ribs, and missing tails were reported following treatment of rats with 1500 mg/m<sup>3</sup> toluene 24 hours/day during days 9-14 of gestation. Two of the dams died during the exposure. Another group of rats received 1000 mg/m<sup>3</sup> 8 hours/day during days 1-21 of gestation. No maternal deaths or toxicity occurred, however, minor skeletal retardation was present in the exposed fetuses. CFLP Mice were exposed to 500 or 1500 mg/m<sup>3</sup> toluene continuously during days 6-13 of pregnancy. All dams died at the high dose during the first 24 hours of exposure, however none died at 500 mg/m<sup>3</sup>. Decreased foetal weight was reported, but there were no differences in the incidences of skeletal malformations or anomalies between the treated and control offspring.

**Absorption** - Studies in humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract. Absorption through the skin is estimated at about 1% of that absorbed by the lungs when exposed to toluene vapor.

Dermal absorption is expected to be higher upon exposure to the liquid; however, exposure is limited by the rapid evaporation of toluene .

**Distribution** - In studies with mice exposed to radiolabeled toluene by inhalation, high levels of radioactivity were present in body fat, bone marrow, spinal nerves, spinal cord, and brain white matter. Lower levels of radioactivity were present in blood, kidney, and liver. Accumulation of toluene has generally been found in adipose tissue, other tissues with high fat content, and in highly vascularised tissues .

**Metabolism** - The metabolites of inhaled or ingested toluene include benzyl alcohol resulting from the hydroxylation of the methyl group. Further oxidation results in the formation of benzaldehyde and benzoic acid. The latter is conjugated with glycine to yield hippuric acid or reacted with glucuronic acid to form benzoyl glucuronide. o-cresol and p-cresol formed by ring hydroxylation are considered minor metabolites

**Excretion** - Toluene is primarily (60-70%) excreted through the urine as hippuric acid. The excretion of benzoyl glucuronide accounts for 10-20%, and excretion of unchanged toluene through the lungs also accounts for 10-20%. Excretion of hippuric acid is usually complete within 24 hours after exposure.

<b>Acute Toxicity</b>	Acute Toxicity (Dermal) Category 4 Acute Toxicity (Inhalation) Category 4	<b>Carcinogenicity</b>	Not Applicable
<b>Skin Irritation/Corrosion</b>	Skin Corrosion/Irritation Category 2	<b>Reproductivity</b>	Not Applicable
<b>Serious Eye Damage/Irritation</b>	Eye Irritation Category 2A	<b>STOT - Single Exposure</b>	STOT - SE (Resp. Irr.) Category 3 STOT - SE (Narcosis) Category 3
<b>Respiratory or Skin sensitisation</b>	Not Applicable	<b>STOT - Repeated Exposure</b>	STOT - RE Category 2
<b>Mutagenicity</b>	Not Applicable	<b>Aspiration Hazard</b>	Not Applicable

#### CMR STATUS

<b>SKIN</b>	toluene	New Zealand Workplace Exposure Standards (WES) - Skin	Skin absorption
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#### SECTION 12 ECOLOGICAL INFORMATION

##### Toxicity

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.

When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. Oils of any kind can cause:

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

For 1,2,4-trimethylbenzene:

Half-life (hr) air : 0.48-16

Half-life (hr) H<sub>2</sub>O surface water : 0.24-672

Half-life (hr) H<sub>2</sub>O ground : 336-1344

Half-life (hr) soil : 168-672

Henry's Pa m<sup>3</sup> /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  $5.18 \times 10^{-3}$  (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<sub>oc</sub> 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 LC<sub>50</sub> (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 EC<sub>50</sub> (48 h): 3.61 mg/l**

Cancer magister (dungeness crab) LC<sub>50</sub> (96 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. LC<sub>50</sub> 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.

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 Group A aliphatic esters (fatty acid esters):

#### Environmental fate:

Group A substances are rather lipophilic (log K<sub>ow</sub> 10-15) in character due to the large number of carbons in the ester molecule (e.g., 24, 26, 31 carbons) and have relatively high boiling points. Owing to the non-volatile nature of these esters, their vapor pressures are very low and difficult to determine experimentally. Water solubility is also very low.

Hydrolysis half lives and atmospheric photodegradation rates were calculated by EPIWIN. The monoester hydrolysis rates were determined to be quite low and not a significant environmental fate route. Fugacity modeling indicates that the fatty acid esters have similar distribution patterns in the environmental compartments (e.g., air, water, soil, sediment).

Biodegradation of alkyl fatty acid esters are expected to occur extensively based on the reported 28 day test results (80-85% biodegradation, OECD 301D) for decyl oleate and for the 2-ethylhexyl ester of C<sub>16</sub>-18 saturated and C<sub>18</sub> unsaturated fatty acids (CAS 85049-37-2). Group A Substances are expected to be extensively biodegraded since the fatty acids in these esters are primarily comprised of palmitic, stearic or oleic acids, which are known to be rapidly biodegraded

#### Ecotoxicity:

Aquatic toxicity results have been reported for decyl oleate and fatty acid, C<sub>16</sub>-18 saturated and C<sub>18</sub> unsaturated, 2-ethylhexanoate. They are not acutely toxic to fish (LC<sub>50</sub> 3200 mg/L). In daphnids, the acute LC<sub>50</sub> was reported to be 17 mg/L and in algae, the LC<sub>50</sub> was reported to be 40-42 mg/L based on biomass and growth rate endpoints. Because of their limited water solubility, the alkyl fatty acid esters and Group A esters are not likely to cause acute aquatic toxicity.

For xylenes :

log K<sub>oc</sub> : 2.05-3.08

Koc : 25.4-204  
 Half-life (hr) air : 0.24-42  
 Half-life (hr) H2O surface water : 24-672  
 Half-life (hr) H2O ground : 336-8640  
 Half-life (hr) soil : 52-672  
 Henry's Pa m<sup>3</sup> /mol: 637-879  
 Henry's atm m<sup>3</sup> /mol: 7.68E-03  
 BOD 5 if unstated: 1.4,1%  
 COD : 2.56,13%  
 ThOD : 3.125  
 BCF : 23  
 log BCF : 1.17-2.41

#### Environmental Fate

**Terrestrial fate:** Measured Koc values of 166 and 182, indicate that 3-xylene is expected to have moderate mobility in soil. Volatilisation of p-xylene is expected to be important from moist soil surfaces given a measured Henry's Law constant of  $7.18 \times 10^{-3}$  atm-cu m/mole. The potential for volatilisation of 3-xylene from dry soil surfaces may exist based on a measured vapor pressure of 8.29 mm Hg. p-Xylene may be degraded during its passage through soil). The extent of the degradation is expected to depend on its concentration, residence time in the soil, the nature of the soil, and whether resident microbial populations have been acclimated. p-Xylene, present in soil samples contaminated with jet fuel, was completely degraded aerobically within 5 days. In aquifer studies under anaerobic conditions, p-xylene was degraded, usually within several weeks, with the production of 3-methylbenzylfumaric acid, 3-methylbenzylsuccinic acid, 3-methylbenzoate, and 3-methylbenzaldehyde as metabolites.

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

#### Atmospheric fate:

Most xylenes released to the environment will occur in the atmosphere and volatilisation is the dominant environmental fate process. In the ambient atmosphere, xylenes are expected to exist solely in the vapour phase. Xylenes are degraded in the atmosphere primarily by reaction with photochemically-produced hydroxyl radicals, with an estimated atmospheric lifetime of about 0.5 to 2 days. Xylenes' susceptibility to photochemical oxidation in the troposphere is to the extent that they may contribute to photochemical smog formation.

According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and from its vapour pressure, p-xylene, is expected to exist solely as a vapour in the ambient atmosphere. Vapour-phase p-xylene is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be about 16 hours. A half-life of 1.0 hr in summer and 10 hr in winter was measured for the reaction of p-xylene with photochemically-produced hydroxyl radicals. p-Xylene has a moderately high photochemical reactivity under smog conditions, higher than the other xylene isomers, with loss rates varying from 9-42% per hr. The photooxidation of p-xylene results in the production of carbon monoxide, formaldehyde, glyoxal, methylglyoxal, 3-methylbenzylnitrate, m-tolualdehyde, 4-nitro-3-xylene, 5-nitro-3-xylene, 2,6-dimethyl-p-benzoquinone, 2,4-dimethylphenol, 6-nitro-2,4-dimethylphenol, 2,6-dimethylphenol, and 4-nitro-2,6-dimethylphenol.

#### Ecotoxicity:

for xylenes

Fish LC50 (96 h) Pimephales promelas 13.4 mg/l; Oncorhynchus mykiss 8.05 mg/l; Lepomis macrochirus 16.1 mg/l (all flow through values); Pimephales promelas 26.7 (static)

Daphnia EC50 948 h): 3.83 mg/l

Photobacterium phosphoreum EC50 (24 h): 0.0084 mg/l

Gammarus lacustris LC50 (48 h): 0.6 mg/l

**DO NOT discharge into sewer or waterways.**

#### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
Not Available	Not Available	Not Available

#### Bioaccumulative potential

Ingredient	Bioaccumulation
Not Available	Not Available

#### Mobility in soil

Ingredient	Mobility
Not Available	Not Available



### SECTION 13 DISPOSAL CONSIDERATIONS

#### Waste treatment methods

<p><b>Product / Packaging disposal</b></p>	<ul style="list-style-type: none"> <li>▸ Containers may still present a chemical hazard/ danger when empty.</li> <li>▸ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▸ 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.</li> <li>▸ Where possible retain label warnings and MSDS and observe all notices pertaining to the product.</li> </ul> <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"> <li>▸ Reduction</li> <li>▸ Reuse</li> <li>▸ Recycling</li> <li>▸ Disposal (if all else fails)</li> </ul> <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"> <li>▸ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▸ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▸ Where in doubt contact the responsible authority.</li> <li>▸ Recycle wherever possible.</li> <li>▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified.</li> <li>▸ Dispose of by: burial in a land-fill specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible material).</li> <li>▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.</li> </ul>
	<p>Insure that the disposal of material is carried out in accordance with Hazardous Substances (Disposal) Regulations 2001.</p>

## SECTION 14 TRANSPORT INFORMATION

### Labels Required

	
<p>Marine Pollutant</p>	
<p>HAZCHEM</p>	<p>•3YE; •3Y</p>

### Land transport (UN)

<p>UN number</p>	<p>1263</p>				
<p>Packing group</p>	<p>III</p>				
<p>UN proper shipping name</p>	<p>PAINT (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)</p>				
<p>Environmental hazard</p>	<p>No relevant data</p>				
<p>Transport hazard class(es)</p>	<table border="1"> <tr> <td>Class</td> <td>3</td> </tr> <tr> <td>Subrisk</td> <td></td> </tr> </table>	Class	3	Subrisk	
Class	3				
Subrisk					
<p>Special precautions for user</p>	<table border="1"> <tr> <td>Special provisions</td> <td>163;223;367</td> </tr> <tr> <td>limited quantity</td> <td>5 L</td> </tr> </table>	Special provisions	163;223;367	limited quantity	5 L
Special provisions	163;223;367				
limited quantity	5 L				

### Air transport (ICAO-IATA / DGR)

<p>UN number</p>	<p>1263</p>
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## Altex RIPO

<b>Packing group</b>	III
<b>UN proper shipping name</b>	Paint (including paint, lacquer, enamel, stain, shellac, varnish, polish, liquid filler and liquid lacquer base); Paint related material (including paint thinning or reducing compounds)
<b>Environmental hazard</b>	No relevant data
<b>Transport hazard class(es)</b>	ICAO/IATA Class : 3 ICAO / IATA Subrisk ERG Code : 3L
<b>Special precautions for user</b>	Special provisions : A3A72 Cargo Only Packing Instructions : 366 Cargo Only Maximum Qty / Pack : 220 L Passenger and Cargo Packing Instructions : 355 Passenger and Cargo Maximum Qty / Pack : 60 L Passenger and Cargo Limited Quantity Packing Instructions : Y344 Passenger and Cargo Maximum Qty / Pack : 10 L

## Sea transport (IMDG-Code / GGVSee)

<b>UN number</b>	1263
<b>Packing group</b>	III
<b>UN proper shipping name</b>	PAINT (including paint, lacquer, enamel, stain, shellac solutions, varnish, polish, liquid filler and liquid lacquer base) or PAINT RELATED MATERIAL (including paint thinning or reducing compound)
<b>Environmental hazard</b>	No relevant data
<b>Transport hazard class(es)</b>	IMDG Class : 3 IMDG Subrisk
<b>Special precautions for user</b>	EMS Number : F-E,S-E Special provisions : 163 223 955 Limited Quantities : 5 L

## SECTION 15 REGULATORY INFORMATION

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

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

<b>HSR Number</b>	Group Standard
HSR002662	Surface Coatings and Colourants (Flammable) Group Standard 2006

<b>aromatic hydrocarbon solvent(64742-95-6.) is found on the following regulatory lists</b>	"OECD List of High Production Volume (HPV) Chemicals","International Chemical Secretariat (ChemSec) SIN List (*Substitute It Now!)", "International Council of Chemical Associations (ICCA) - High Production Volume List", "New Zealand Inventory of Chemicals (NZIoC)", "International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index", "New Zealand Land Transport Rule; Dangerous Goods 2005 - Schedule 2 Dangerous Goods in Limited Quantities and Consumer Commodities", "International Maritime Dangerous Goods Requirements (IMDG Code)", "Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)", "International Air Transport Association (IATA) Dangerous Goods Regulations"
<b>linseed oil(8001-26-1) is found on the following regulatory lists</b>	"OECD List of High Production Volume (HPV) Chemicals", "International Fragrance Association (IFRA) Survey: Transparency List", "FisherTransport Information", "Sigma-AldrichTransport Information", "IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk", "IMO IBC Code Chapter 17: Summary of minimum requirements", "New Zealand Inventory of Chemicals (NZIoC)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Chemicals (single components)", "GESAMP/EHS Composite List - GESAMP Hazard Profiles"
<b>xylene(1330-20-7) is found on the following regulatory lists</b>	"International Council of Chemical Associations (ICCA) - High Production Volume List", "International Fragrance Association (IFRA) Survey: Transparency List", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Chemicals (single components)", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data", "FisherTransport Information", "New Zealand Inventory of Chemicals (NZIoC)", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC"

	Monographs", "International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index", "International Maritime Dangerous Goods Requirements (IMDG Code)", "Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)", "International Air Transport Association (IATA) Dangerous Goods Regulations", "OECD List of High Production Volume (HPV) Chemicals", "OSPAR List of Chemicals for Priority Action", "IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk", "IMO IBC Code Chapter 17: Summary of minimum requirements", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Dangerous Goods", "IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards", "New Zealand Workplace Exposure Standards (WES)"
<b>chlorinated rubber - carbon tetrachloride free(9006-03-5) is found on the following regulatory lists</b>	"New Zealand Inventory of Chemicals (NZIoC)", "Sigma-AldrichTransport Information"
<b>naphtha, petroleum, hydrodesulfurised heavy(64742-82-1.) is found on the following regulatory lists</b>	"OECD List of High Production Volume (HPV) Chemicals", "International Chemical Secretariat (ChemSec) SIN List (*Substitute It Now!)", "International Council of Chemical Associations (ICCA) - High Production Volume List", "New Zealand Inventory of Chemicals (NZIoC)", "International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index", "New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits", "International Maritime Dangerous Goods Requirements (IMDG Code)", "Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)", "International Air Transport Association (IATA) Dangerous Goods Regulations"
<b>toluene(108-88-3) is found on the following regulatory lists</b>	"OECD List of High Production Volume (HPV) Chemicals", "WHO Guidelines for Drinking-water Quality - Guideline values for chemicals that are of health significance in drinking-water", "International Fragrance Association (IFRA) Standards Prohibited", "New Zealand Cosmetic Products Group Standard - Schedule 5 - Table 1: Components Cosmetic Products Must Not Contain Except Subject to the Restrictions and Conditions Laid Down", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data", "United Nations Consolidated List of Products Whose Consumption and/or Sale Have Been Banned, Withdrawn, Severely Restricted or Not Approved by Governments", "New Zealand Hazardous Substances and New Organisms (HSNO) Act - Dangerous Goods", "FisherTransport Information", "Sigma-AldrichTransport Information", "Acros Transport Information", "IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk", "IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards", "IMO IBC Code Chapter 17: Summary of minimum requirements", "New Zealand Inventory of Chemicals (NZIoC)", "International Agency for Research on Cancer (IARC) - Agents Reviewed by the IARC Monographs", "New Zealand Workplace Exposure Standards (WES)", "International Maritime Dangerous Goods Requirements (IMDG Code) - Substance Index", "International Maritime Dangerous Goods Requirements (IMDG Code)", "Belgium Federal Public Service Mobility and Transport, Regulations concerning the International Carriage of Dangerous Goods by Rail - Table A: Dangerous Goods List - RID 2013 (Dutch)", "International Air Transport Association (IATA) Dangerous Goods Regulations", "OSPAR List of Chemicals for Priority Action", "GESAMP/EHS Composite List - GESAMP Hazard Profiles", "United Nations List of Precursors and Chemicals Frequently used in the Illicit Manufacture of Narcotic Drugs and Psychotropic Substances Under International Control (Red List) - Table II", "United Nations Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances - Table II"

## SECTION 16 OTHER INFORMATION

### Other information

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

A list of reference resources used to assist the committee may be found at:

[www.chemwatch.net/references](http://www.chemwatch.net/references)

The (M)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.

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