

# Polyac 61

## Hychem International

Chemwatch Hazard Alert Code: 3

Chemwatch: 5431-88

Version No: 4.1

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 10/12/2021

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

#### Product Identifier

Product name	Polyac 61
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	RESIN SOLUTION, flammable
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	For professional use only. Use according to manufacturer's directions.
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#### Details of the manufacturer or supplier of the safety data sheet

Registered company name	Hychem International
Address	Unit 1, 30 Bluett Drive Smeaton Grange NSW 2567 Australia
Telephone	+61 2 4646 1660
Fax	+61 2 4647 3700
Website	Not Available
Email	Not Available

#### Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 3 9573 3188

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

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Flammable Liquids Category 2, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Germ Cell Mutagenicity Category 1A, Carcinogenicity Category 1A, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

#### Label elements

Polyac 61

<b>Hazard pictogram(s)</b>	
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<b>Signal word</b>	<b>Danger</b>
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**Hazard statement(s)**

<b>AUH019</b>	May form explosive peroxides.
<b>H225</b>	Highly flammable liquid and vapour.
<b>H315</b>	Causes skin irritation.
<b>H317</b>	May cause an allergic skin reaction.
<b>H319</b>	Causes serious eye irritation.
<b>H335</b>	May cause respiratory irritation.
<b>H336</b>	May cause drowsiness or dizziness.
<b>H340</b>	May cause genetic defects.
<b>H350</b>	May cause cancer.
<b>H412</b>	Harmful to aquatic life with long lasting effects.

**Precautionary statement(s) Prevention**

<b>P201</b>	Obtain special instructions before use.
<b>P210</b>	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
<b>P271</b>	Use only outdoors or in a well-ventilated area.
<b>P280</b>	Wear protective gloves, protective clothing, eye protection and face protection.
<b>P240</b>	Ground and bond container and receiving equipment.
<b>P241</b>	Use explosion-proof electrical/ventilating/lighting/intrinsically safe equipment.
<b>P242</b>	Use non-sparking tools.
<b>P243</b>	Take action to prevent static discharges.
<b>P261</b>	Avoid breathing mist/vapours/spray.
<b>P273</b>	Avoid release to the environment.
<b>P264</b>	Wash all exposed external body areas thoroughly after handling.
<b>P272</b>	Contaminated work clothing should not be allowed out of the workplace.

**Precautionary statement(s) Response**

<b>P308+P313</b>	IF exposed or concerned: Get medical advice/ attention.
<b>P370+P378</b>	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.
<b>P302+P352</b>	IF ON SKIN: Wash with plenty of water and soap.
<b>P305+P351+P338</b>	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
<b>P312</b>	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
<b>P333+P313</b>	If skin irritation or rash occurs: Get medical advice/attention.
<b>P337+P313</b>	If eye irritation persists: Get medical advice/attention.
<b>P362+P364</b>	Take off contaminated clothing and wash it before reuse.
<b>P303+P361+P353</b>	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
<b>P304+P340</b>	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

**Precautionary statement(s) Storage**

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

**Precautionary statement(s) Disposal**

<b>P501</b>	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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## SECTION 3 Composition / information on ingredients

### Substances

See section below for composition of Mixtures

### Mixtures

CAS No	%[weight]	Name
80-62-6	>30	<u>methyl methacrylate</u>
103-11-7	15-30	<u>2-ethylhexyl acrylate</u>
2440-22-4	<5	<u>2-(2'-hydroxy-5'-methylphenyl)benzotriazole</u>
1174921-67-5	<5	<u>C7-C9 isoalkanes</u>
64742-95-6.	<5	<u>C9-aromatic hydrocarbon solvent</u>

**Legend:** 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; \* EU IOELVs available

## SECTION 4 First aid measures

### Description of first aid measures

<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

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

- ▶ Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
- ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO<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.
- ▶ Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]

Treat symptomatically.

For methyl methacrylate:

Significant effects developing over a work-shift are not detected by symptomatology, blood pressure, respiratory function testing, haemoglobin and white cell count, urinalysis and blood chemistry. Effects may occur in high concentration exposure groups with regard to serum glucose and blood urea, nitrogen,

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cholesterol, albumin and total bilirubin values. Possible alterations occur in skin and nervous system symptomatology, urinalysis findings and serum triglycerides. Diagnostic signs taken as indicative of methyl methacrylate-induced local neurotoxicity include sensory nerve distal conduction velocities. These deficits appear to result from diffusion of the substance into neurons, lysis of membrane lipids and demyelination.

## 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

<b>Fire Incompatibility</b>	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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### Advice for firefighters

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <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 in the event of a fire.</li> <li>▸ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▸ Consider evacuation (or protect in place).</li> <li>▸ Fight fire from a safe distance, with adequate cover.</li> <li>▸ If safe, switch off electrical equipment until vapour fire hazard removed.</li> <li>▸ Use water delivered as a fine spray to control the fire and cool adjacent area.</li> <li>▸ Avoid spraying water onto liquid pools.</li> <li>▸ <b>Do not approach containers suspected to be hot.</b></li> <li>▸ Cool fire exposed containers with water spray from a protected location.</li> <li>▸ If safe to do so, remove containers from path of fire.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▸ Liquid and vapour are highly flammable.</li> <li>▸ Severe fire hazard when exposed to heat, flame and/or oxidisers.</li> <li>▸ Vapour may travel a considerable distance to source of ignition.</li> <li>▸ Heating may cause expansion or decomposition leading to violent rupture of containers.</li> <li>▸ On combustion, may emit toxic fumes of carbon monoxide (CO).</li> </ul> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) nitrogen oxides (NO<sub>x</sub>) other pyrolysis products typical of burning organic material. May emit clouds of acrid smoke</p>
<b>HAZCHEM</b>	•3YE

## SECTION 6 Accidental release measures

### Personal precautions, protective equipment and emergency procedures

See section 8

### Environmental precautions

See section 12

### Methods and material for containment and cleaning up

<b>Minor Spills</b>	<p>Environmental hazard - contain spillage.</p> <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>	<p>Environmental hazard - contain spillage.</p> <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> </ul>

- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Consider evacuation (or protect in place).
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse /absorb vapour.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Use only spark-free shovels and explosion proof equipment.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Absorb remaining product with sand, earth or vermiculite.
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

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

## SECTION 7 Handling and storage

### Precautions for safe handling

#### Safe handling

- ▶ Most acrylic monomers have low viscosity therefore pouring, material transfer and processing of these materials do not necessitate heating.
- ▶ Viscous monomers may require heating to facilitate handling. To facilitate product transfer from original containers, product must be heated to no more than 60 deg. C. (140 F.), for not more than 24 hours.
- ▶ **Do NOT use localised heat sources such as band heaters to heat/ melt product.**
- ▶ **Do NOT use steam.**
- ▶ Hot boxes or hot rooms are recommended for heating/ melting material. The hot box or hot room should be set a maximum temperature of 60 deg. C. (140 F.).
- ▶ **Do NOT overheat - this may compromise product quality and /or result in an uncontrolled hazardous polymerisation.**
- ▶ If product freezes, heat as indicated above and mix gently to redistribute the inhibitor. Product should be consumed in its entirety after heating/ melting; avoid multiple "reheats" which may affect product quality or result in product degradation.
- ▶ Product should be packaged with inhibitor(s). Unless inhibited, product may polymerise, raising temperature and pressure, possibly rupturing container. Check inhibitor level periodically, adding to bulk material if needed. In addition, the product's inhibitor(s) require the presence of dissolved oxygen. Maintain, at a minimum, the original headspace in the product container and do NOT blanket or mix with oxygen-free gas as it renders the inhibitor ineffective. Ensure air space (oxygen) is present during product heating / melting.
- ▶ Store product indoors at temperatures greater than the product's freeing point (or greater than 0 deg. C. (32 F.)) if no freezing point available and below 38 deg. C (100 F.).
- ▶ Avoid prolonged storage (longer than shelf-life) storage temperatures above 38 deg. C (100 F.).
- ▶ Store in tightly closed containers in a properly vented storage area away from heat, sparks, open flame, strong oxidisers, radiation and other initiators.
- ▶ Prevent contamination by foreign materials.
- ▶ Prevent moisture contact.
- ▶ Use only non-sparking tools and limit storage time. Unless specified elsewhere, shelf-life is 6 months from receipt.
- ▶ Containers, even those that have been emptied, may contain explosive vapours.
- ▶ Do NOT cut, drill, grind, weld or perform similar operations on or near containers.
- ▶ **DO NOT allow clothing wet with material to stay in contact with skin**
- ▶ Avoid all personal contact, including inhalation.
- ▶ Wear protective clothing when risk of exposure occurs.
- ▶ Use in a well-ventilated area.
- ▶ Prevent concentration in hollows and sumps.
- ▶ **DO NOT enter confined spaces until atmosphere has been checked.**
- ▶ Avoid smoking, naked lights, heat or ignition sources.
- ▶ When handling, **DO NOT eat, drink or smoke.**
- ▶ Vapour may ignite on pumping or pouring due to static electricity.
- ▶ **DO NOT use plastic buckets.**
- ▶ Earth and secure metal containers when dispensing or pouring product.
- ▶ Use spark-free tools when handling.
- ▶ Avoid contact with incompatible materials.
- ▶ Keep containers securely sealed.
- ▶ Avoid physical damage to containers.
- ▶ Always wash hands with soap and water after handling.
- ▶ Work clothes should be laundered separately.
- ▶ Use good occupational work practice.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.

#### Other information

- Easily peroxidisable.
- ▶ Products formed as a result of peroxidation are not only safety hazards but may chemically alter the chemical behavior of the parent compound.
- ▶ Should have a warning label affixed bearing the date of receipt in the laboratory and the date on which the container label is first opened, or laboratory synthesised materials are the responsibility of the individual chemist.

- ▶ **WARNING:** This product may form peroxides which themselves are not themselves particularly hazardous but which on decomposition may initiate explosive polymerisation of the bulk monomer (Trommsdorf effect).
- ▶ Should be evaluated every 12 months, redated if safe or else discarded.
- ▶ Quantities of uninhibited monomers exceeding 500 ml should not be stored for more than 24 hours.
- ▶ The oxidation of iodide to iodine or the conversion of colourless ferriothiocyanate to red ferriothiocyanate by peroxides are simple and convenient tests for most peroxides.
- ▶ Before distilling or evaporating a suitable polymerisation inhibitor should be added.
- ▶ Leave at least 10% bottoms.
- ▶ Use a shield when evaporating or distilling mixtures which may contain peroxidisable compounds.
- ▶ Store away from heat and light.
- ▶ Particular attention should be paid to the adequacy of the closure on storage containers.

Peroxides may be removed by;

- ▶ passing the material over a column of ordinary activated alumina (care should be taken in disposal of the activated alumina);
- ▶ shaking with a concentrated solution of ferrous salt (provided the carrier solvent is water-insoluble);
- ▶ agitation with an approximately equimolar mixture of ferrous sulfate and sodium bisulfate;
- ▶ commercial quantities may be treated with a 5% solution of aqueous sodium carbonate.

Jackson et al: Control of Peroxizable Compounds; Safety in the Chemical Laboratory, Journal of Chemical Education; Vol 47, 1970, pp A175-A188

- ▶ When solvents have been freed from peroxides by percolation through a column of activated alumina, the adsorbed peroxides must promptly be desorbed by treatment with polar solvents, methanol or water, which must in turn be discarded safely.\*\*\*
- ▶ Polymerisation may occur slowly at room temperature.
- ▶ Storage requires stabilising inhibitor content and dissolved oxygen content to be monitored. Refer to manufacturer's recommended levels.
- ▶ **DO NOT overfill containers so as to maintain free head space above product.**
- ▶ Blanketing or sparging with nitrogen or oxygen free gas will deactivate stabiliser.
- ▶ Store below 38 deg. C.
- ▶ Store in original containers in approved flame-proof area.
- ▶ No smoking, naked lights, heat or ignition sources.
- ▶ **DO NOT store in pits, depression, basement or areas where vapours may be trapped.**
- ▶ Keep containers securely sealed.
- ▶ Store away from incompatible materials in a cool, dry well ventilated area.
- ▶ Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this MSDS.
- ▶ Tank storage: Tanks must be specifically designed for use with this product. Bulk storage tanks should be diked (bunded). Locate tanks away from heat and other sources of ignition. Cleaning, inspection and maintenance of storage tanks is a specialist operation, which requires the implementation of strict procedures and precautions.
- ▶ Keep in a cool place. Electrostatic charges will be generated during pumping. Electrostatic discharge may cause fire. Ensure electrical continuity by bonding and grounding (earthing) all equipment to reduce the risk. The vapours in the head space of the storage vessel may lie in the flammable/explosive range and hence may be flammable.
- ▶ For containers, or container linings use mild steel, stainless steel. Examples of suitable materials are: high density polyethylene (HDPE), polypropylene (PP), and Viton (FMK), which have been specifically tested for compatibility with this product.
- ▶ For container linings, use amine-adduct cured epoxy paint.
- ▶ For seals and gaskets use: graphite, PTFE, Viton A, Viton B.
- ▶ Unsuitable material: Some synthetic materials may be unsuitable for containers or container linings depending on the material specification and intended use. Examples of materials to avoid are: natural rubber (NR), nitrile rubber (NBR), ethylene propylene rubber (EPDM), polymethyl methacrylate (PMMA), polystyrene, polyvinyl chloride (PVC), polyisobutylene. However, some may be suitable for glove materials.
- ▶ Do not cut, drill, grind, weld or perform similar operations on or near containers. Containers, even those that have been emptied, can contain explosive vapours.

## Conditions for safe storage, including any incompatibilities

### Suitable container

For acrylates or methacrylates:

Storage tanks and pipes should be made of stainless steel or aluminium.

Although they do not corrode carbon steel, there is a risk of contamination if corrosion does occur.

- ▶ 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**

- ▶ Avoid reaction with oxidising agents, bases and strong reducing agents.
- ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

**SECTION 8 Exposure controls / personal protection****Control parameters****Occupational Exposure Limits (OEL)****INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	methyl methacrylate	Methyl methacrylate	50 ppm / 208 mg/m <sup>3</sup>	416 mg/m <sup>3</sup> / 100 ppm	Not Available	Not Available

**Emergency Limits**

Ingredient	TEEL-1	TEEL-2	TEEL-3
methyl methacrylate	Not Available	Not Available	Not Available
2-ethylhexyl acrylate	15 ppm	120 ppm	150 ppm
C9-aromatic hydrocarbon solvent	1,200 mg/m <sup>3</sup>	6,700 mg/m <sup>3</sup>	40,000 mg/m <sup>3</sup>

Ingredient	Original IDLH	Revised IDLH
methyl methacrylate	1,000 ppm	Not Available
2-ethylhexyl acrylate	Not Available	Not Available
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	Not Available	Not Available
C7-C9 isoalkanes	Not Available	Not Available
C9-aromatic hydrocarbon solvent	Not Available	Not Available

**Occupational Exposure Banding**

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
2-ethylhexyl acrylate	E	≤ 0.1 ppm
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	D	> 0.01 to ≤ 0.1 mg/m <sup>3</sup>
C7-C9 isoalkanes	E	≤ 0.1 ppm
C9-aromatic hydrocarbon solvent	E	≤ 0.1 ppm

**Notes:**

*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*

**MATERIAL DATA**

NOTE D: Certain substances which are susceptible to spontaneous polymerisation or decomposition are generally placed on the market in a stabilised form. It is in this form that they are listed on Annex I

When they are placed on the market in a non-stabilised form, the label must state the name of the substance followed by the words "non-stabilised"

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

NOTE P: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.01% w/w benzene (EINECS No 200-753-7).

Note E shall also apply when the substance is classified as a carcinogen. This note applies only to certain complex oil-derived substances in Annex VI.

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

**Exposure controls****Appropriate engineering controls**

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

The basic types of engineering controls are:

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

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

Employers may need to use multiple types of controls to prevent employee overexposure. For flammable liquids and flammable gases, local exhaust ventilation or a process enclosure ventilation system may be required. Ventilation equipment should be explosion-resistant. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

- Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance.

- Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures.

- Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)

**Individual protection measures, such as personal protective equipment**



**Eye and face protection**

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

**Skin protection**

See Hand protection below

**Hands/feet protection**

- ▶ Elbow length PVC gloves
- NOTE:**
- ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
  - ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
- The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

· Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.

Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

General warning: Do NOT use latex gloves! Use only recommended gloves - using the wrong gloves may increase the risk:

<p><b>Exposure condition</b> Short time use; (few minutes less than 0.5 hour) Little physical stress</p>	<p>Use of thin nitrile rubber gloves: Nitrile rubber (0.1 mm) Excellent tactility ("feel"), powder-free Disposable Inexpensive Give adequate protection to low molecular weigh acrylic monomers</p>
<p><b>Exposure condition</b> Medium time use; less than 4 hours Physical stress (opening drums, using tools, etc.)</p>	<p>Use of medium thick nitrile rubber gloves Nitrile rubber, NRL (latex) free; &lt;0.45 mm Moderate tactility ("feel"), powder-free Disposable Moderate price Gives adequate protection for most acrylates up to 4 hours Do NOT give adequate protection to low molecular weight monomers at exposures longer than 1 hour</p>
<p><b>Exposure condition</b> Long time Cleaning operations</p>	<p>Nitrile rubber, NRL (latex) free; &gt;0.56 mm low tactility ("feel"), powder free High price Gives adequate protection for most acrylates in combination with commonly used solvents up to 8 hours Do NOT give adequate protection to low molecular weight monomers at exposures longer than 1 hour Avoid use of ketones and acetates in wash-up solutions.</p>

Where none of this gloves ensure safe handling (for example in long term handling of acrylates containing high levels of acetates and/ or ketones, use laminated multilayer gloves.

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#### Body protection

See Other protection below

#### Other protection

- Overalls.
- PVC Apron.
- PVC protective suit may be required if exposure severe.
- Eyewash unit.
- Ensure there is ready access to a safety shower.
- Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity.
- For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets).
- Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole

made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot and shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

## Recommended material(s)

### GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

**"Forsberg Clothing Performance Index".**

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

**computer-generated** selection:

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Material	CPI
PE/EVAL/PE	A
PVA	A
TEFLON	A
BUTYL	C

\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

## Respiratory protection

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	A-2 P2	A-PAPR-2 P2
up to 50 x ES	-	A-3 P2	-
50+ x ES	-	Air-line**	-

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(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)

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

Avoid inhalation.

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

Appearance	Liquid with a characteristic odour; does not mix with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	380
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	100-199	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	11	Taste	Not Available
Evaporation rate	2	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available

Continued...

Upper Explosive Limit (%)	12.5	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	0.7	Volatile Component (%vol)	67.35
Vapour pressure (kPa)	51.3	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

## SECTION 10 Stability and reactivity

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

## SECTION 11 Toxicological information

### Information on toxicological effects

Inhaled	<p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation.</p> <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>Workers in plants manufacturing methyl methacrylate have complained of headaches, pains in the extremities, fatigue, sleep disturbance, irritability and loss of memory. A Russian report associated disturbances in the level of insulin, prolactin and circulating somatotrophic hormone in women to occupational exposure to methyl methacrylate.</p> <p>Inhalation of 47 ppm in dogs produces hypotension, signs of central nervous system (CNS) depression, hepatic and renal degeneration and death in respiratory arrest</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>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>The acute toxicity of inhaled alkylbenzene is best described by central nervous system depression. These compounds may also act as general anaesthetics. Whole body symptoms of poisoning include light-headedness, nervousness, apprehension, a feeling of well-being, confusion, dizziness, drowsiness, ringing in the ears, blurred or double vision, vomiting and sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, depression of breathing, and arrest. Heart stoppage may result from cardiovascular collapse. A slow heart rate and low blood pressure may also occur.</p> <p>Alkylbenzenes are not generally toxic except at high levels of exposure. Their breakdown products have low toxicity and are easily eliminated from the body.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.</p> <p>If exposure to highly concentrated vapour atmosphere is prolonged this may lead to narcosis, unconsciousness, even coma and unless resuscitated - death.</p>
Ingestion	<p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing.</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes)</p>

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	<p>Oral doses of 5 ml/kg methyl methacrylate in dogs produce hypotension, signs of central nervous system (CNS) depression, hepatic and renal degeneration and death in respiratory arrest</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><b>Skin Contact</b></p>	<p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.</p> <p>Reports of dental technicians, surgeons and manufacturing employees with direct skin contact with methyl methacrylate document paresthesias of the digits and mild local axonal degeneration.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either:</p> <ul style="list-style-type: none"> <li>▶ produces severe inflammation of the skin in a substantial number of individuals following direct contact, and/or</li> <li>▶ produces significant and severe 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> <li>▶ 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.</li> </ul> <p><b>NOTE:</b> Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p>
<p><b>Eye</b></p>	<p>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><b>Chronic</b></p>	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may produce heritable genetic damage.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of</p> <ul style="list-style-type: none"> <li>- appropriate animal studies,</li> <li>- other relevant information</li> </ul> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>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.</p> <p>Prolonged and repeated exposures can cause liver and kidney damage. Hypotension induced by methyl methacrylate in surgical bone cement has been followed by cardiac arrest with at least one fatality in a patient undergoing surgery reported.</p> <p>An increased mortality from colon and rectal cancer in white male employees exposed for at least 10-months to acrylate monomer (including methyl methacrylate) has been reported in one cohort but not in others where acrylate exposures were</p>

	<p>controlled.</p> <p>Incorporation of up to 2000 ppm methyl methacrylate in drinking water of rats for up to two-years did not induce any treatment-related pathology although subcutaneous and intraperitoneal implants of freshly polymerised material for up to 39 months produced local fibrosarcoma.</p> <p>Inhalation of methyl methacrylate by rats and mice of both sexes produced inflammation of the nasal cavity and degeneration of the olfactory sensory epithelium and epithelial hyperplasia of the nasal cavity in mice (exposure occurred over two years)</p> <p>Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).</p>
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Polyac 61	TOXICITY	IRRITATION
	Not Available	Not Available
methyl methacrylate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg <sup>[2]</sup>	Eye (rabbit): 150 mg
	Inhalation(Rat) LC50: 29.8 mg/l4h <sup>[1]</sup>	Skin (rabbit): 10000 mg/kg (open)
	Oral (Rat) LD50: 7872 mg/kg <sup>[2]</sup>	
2-ethylhexyl acrylate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >177 mg/kg <sup>[1]</sup>	Eyes (rabbit) 500mg/24h mild
	Oral (Mouse) LD50; >5000 mg/kg <sup>[1]</sup>	Skin (rabbit) 10mg/24h - SEVERE
		Skin (rabbit) 20mg/24h mod.
		Skin (rabbit) 500mg mild
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1000 mg/kg <sup>[2]</sup>	Eye (rabbit): 500 mg/24 h - mild
	Inhalation(Rat) LC50: >0.59 mg/L4h <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Mouse) LD50; 6500 mg/kg <sup>[2]</sup>	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
C7-C9 isoalkanes	TOXICITY	IRRITATION
	Inhalation(Rat) LC50: >9.4 mg/L4h <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Oral (Rat) LD50: 7100 mg/kg <sup>[2]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
C9-aromatic hydrocarbon solvent	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >1900 mg/kg <sup>[1]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
	Inhalation(Rat) LC50: >4.42 mg/L4h <sup>[1]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50: >4500 mg/kg <sup>[1]</sup>	

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

<b>METHYL METHACRYLATE</b>	<p>Inhalation (human) TCLo: 60 mg/m<sup>3</sup>(15 ppm) [* Manuf. Rohm &amp; Haas]</p> <p><b>For methyl methacrylate:</b></p> <p>Acute toxicity: MMA is rapidly absorbed after oral or inhalatory administration. <i>In vitro</i> skin absorption studies in human skin indicate that MMA can be absorbed through human skin. After inhalation to rats 10 to 20% of the substance is deposited in the upper respiratory tract where it is metabolised by local tissue esterases.</p> <p>Acute toxicity of MMA by the oral, dermal, and inhalative routes is low as judged by tests with different species: The oral LD50 for rats, mice, and rabbits is found to exceed 5000 mg/kg bw.</p> <p>Acute inhalation toxicity for rats and mice is described by LC50 values of &gt; 25 mg/l/4 hours.</p> <p>Acute dermal toxicity is reported for rabbits to exceed 5000 mg/kg bw. Skin and respiratory irritation are reported for subjects exposed to monomeric MMA. The substance has been shown to produce severe skin irritation when tested undiluted on rabbit skin. There are indications from studies in animals that MMA can be irritating to the respiratory system. In contact with eyes MMA has shown only weak irritation of the conjunctivae. MMA has a moderate to strong sensitising potential in experimental animals. Cases of contact dermatitis have been reported for workers exposed to the monomeric chemical. There is no convincing evidence that MMA is a respiratory sensitiser in humans.</p> <p>The lead effect caused by MMA is a degeneration of the olfactory region of the nose being the most sensitive target tissue. For this effect a NOAEC of 25 ppm (104 mg/m<sup>3</sup>) in a two-year inhalation study in rats was identified but only slight effects on the olfactory tissues have been observed at 100 ppm. Concerning systemic effects, two different valid studies have been considered for identifying a N(L)OAEAL. Due to different dose selections, different values for N(L)OAEALs are available. The LOEALs and the NOEALs for female rats ranges between 400 and 500 ppm and from 100 to 250 ppm respectively. In subchronic inhalation studies systemic toxic effects were seen in rats &gt;1000 ppm, respectively in mice &gt;500 ppm, including degenerative and necrotic lesions in liver, kidney, brain, and atrophic changes in spleen and bone marrow. These effects were not seen in chronic studies up to 1000 ppm. Oral administration to rats resulted in a NOAEL of 200 mg/kg bw/d.</p>
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	<p>MMA has <i>in vitro</i> the potential for induction of mutagenic effects, especially clastogenicity. However, this potential is limited to high doses with strong toxic effects. Furthermore, the negative <i>in vivo</i> micronucleus test and the negative dominant lethal assay indicate that this potential is not expressed <i>in vivo</i>. There is no relevant concern on carcinogenicity of MMA in humans and animals. Epidemiology data on increased tumour rates in exposed cohorts are of limited reliability and cannot be related to MMA as the solely causal agent.</p> <p>MMA did not reveal an effect on male fertility when animals had been exposed to up to 9000 ppm. From the available developmental toxicity investigations, including an inhalation study according to OECD Guideline 414, no teratogenicity, embryotoxicity or foetotoxicity has been observed at exposure levels up to and including 2028 ppm (8425 mg/m<sup>3</sup>). The available human data on sexual disorders in male and female workers cannot be considered to conclude on reproductive toxicity effects of MMA due to the uncertain validity of the studies</p> <p>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.</p>
2-ETHYLHEXYL ACRYLATE	<p>Substance has been investigated as a tumourigen on mouse skin.</p> <p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may produce severe 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) thickening of the epidermis.</p> <p>Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.</p> <p>For 2-ethylhexyl acrylate:  <b>Animal studies:</b> Skin sensitisation was observed in challenged guinea pigs that had been induced with intradermal injections of 2-ethylhexyl acrylate in concentrations of 0.5 M or 0.17 M in Freund's complete adjuvant three times during 9 days; that had been induced with epicutaneous or intracutaneous application of 2-ethylhexyl acrylate in concentrations of 0.1% (w/v) 3 times a week for 3 weeks</p> <p>The olfactory epithelium of the nasal mucosa was degenerated when Wistar rats inhaled 2-ethylhexyl acrylate at 225 and 750 mg/m<sup>3</sup> 6 hours a day, 5 days per week for 90 days. A reduced body weight gain, lethargy and reduced levels for albumin were also observed at these doses. Animals inhaling 75 mg/m<sup>3</sup> for the same exposure period showed no toxic signs.</p> <p>An apparent increase in the frequency of chronic nephritis was seen in male C3H/HeJ mice treated three times a week for their lifetime with 20 mg 75% (v/v) 2-ethylhexyl acrylate in acetone applied to clipped dorsal skin.</p> <p><b>Reproductive and developmental effects:</b> 2-Ethylhexanol is a metabolite of 2-ethylhexyl acrylate. 2-Ethylhexanol in high doses (above 800 mg/kg b.w.) has caused developmental effects in rats.</p> <p><b>Mutagenic and genotoxic effects:</b> 2-Ethylhexyl acrylate was not mutagenic in 4 strains (TA98, TA100, TA1535, and TA1537) of <i>Salmonella typhimurium</i> in an Ames test with or without metabolic activation systems.</p> <p>2-Ethylhexyl acrylate tested in cultured L5178Y mouse lymphoma cells without exogenous activation produced an equivocal result for an increased mutant frequency as well as for induced aberrations. No increase in the number of micronuclei was seen..</p> <p>In another experiment the mutation frequency was up to 4.6 times greater than in controls for the highest dose levels of 2-ethylhexyl acrylate added to cultured L5178Y mouse lymphoma cells with metabolic activation. No reproducible increase in mutation frequency was seen without the metabolic activation.</p> <p>2-Ethylhexyl acrylate did not induce a dose-related increase in the hgprt mutant frequency in either the suspension or monolayer assay in Chinese hamster ovary cells.</p> <p>The sister chromatid exchange assay in CHO cells with and without metabolic activation was slightly positive when tested with 2-ethylhexyl acrylate with metabolic activation (ambiguous result).</p> <p>Unscheduled DNA synthesis in primary rat hepatocytes was slightly increased when tested with 2-ethylhexyl acrylate (ambiguous result). No chromosome aberrations were observed when mice were given an oral dose of 2.5 g/kg once a day for 1 or 5 days in an <i>in vivo</i> cytogenetic assay.</p> <p><b>Carcinogenicity:</b> In a 2-year carcinogenicity study 25 ml of a 21.5, 43 or 85% (w/w) solution of 2-ethylhexyl acrylate in acetone was applied epicutaneously to the clipped dorsal skin of male NMRI mice (80 per group) three times a week.</p> <p>None of the mice treated with 2-ethylhexyl acrylate alone developed a skin tumour at the application site. One squamous cell papilloma occurred in each of the groups treated with 2-ethylhexyl acrylate and the promoter. Squamous cell carcinomas were observed only in the positive control groups (exposed to 0.015 % benzo[a]pyrene alone or in combination with promoter).</p> <p>In a lifetime carcinogenicity study 25 ml of a 2.5, 21 or 86.5% (w/w) solution of 2-ethylhexyl acrylate in acetone was applied epicutaneously to the clipped dorsal skin of male C3H/HeJ mice (80 per group) three times a week. Another group was treated with a 43% solution for 24 weeks and thereafter observed for lifetime. Only in the 86.5% and 21% test groups showing chronic irritative skin damage was there a high incidence of neoplastic skin lesions (total of 15 papillomas, 36 carcinomas, and 16 melanomas) with no dose dependency. In contrast, no skin tumours were found in the negative control groups, in the group treated with 2.5% 2-ethylhexyl acrylate for lifetime or in the group treated with 43% 2-ethylhexyl acrylate for about 6 months and then observed for lifetime.</p>
2-(2'-HYDROXY-5'-METHYLPHENYL)BENZOTRIAZOLE	<p>NOAEL (rats &amp; mice) 50 mg/kg NOEL (rats &amp; mice) 1000 mg/kg Point gene mutation; Negative Ames; chromosomal aberration Negative</p> <p>For benzotriazoles</p> <p>There are several indications that the effects of phenolic benzotriazoles described in the literature might be caused by endocrine disruption, e.g. reduced concentrations of testosterone, higher concentrations of CYP 450, or higher activity of ethoxyresorufin-O-deethylase (EROD-activity). As in these cases there are also indications for toxic effects on the liver reported, the effects might actually be only secondary effects. With the present knowledge it is not</p>

possible to attribute them unambiguously as endocrine adverse effects of an equivalent level of concern. Several benzotriazole UV stabilisers showed significant human aryl hydrocarbon receptor (AhR) ligand activity. The AhR has roles in regulating immunity, stem cell maintenance, and cellular differentiation. A study indicated that certain benzotriazole UV stabilisers have the potential to accumulate and exert potent physiological effects in humans, analogous to polycyclic aromatic hydrocarbons and dioxins, which are known stable and toxic ligands. The polycyclic aromatic hydrocarbon the polycyclic aromatic hydrocarbon, benzo[a]pyrene (BaP), a ligand for AhR, induces its own metabolism and bioactivation to a toxic metabolite.

Benzotriazole is the core structure present within the phenolic benzotriazole class. In vitro metabolism with rat liver microsomes yielded formation of 5- and 4-hydroxybenzotriazole (1.6 and 0.32% of the amount added, respectively). Overall metabolism was low (<5% of the total amount added). Oral acute studies in rats and mice yielded LD50 values that ranged from 560 to 909 mg/kg. Intraperitoneal LD50 values in mice and rats ranged from 400-1000 and 500-900 mg/kg, respectively. A mouse intravenous LD50 of 238 mg/kg was identified. Dermal LD50 values were =1000 mg/kg in rats and rabbits, and inhalation LC50 values in rats were 1.5 mg/L and 1.91 mg/L/3 hours). Subchronic and short-term studies showed that oral administration to mice produced minimal effects on body weight while dose-dependent decreases in body weight were observed in rats. Endocrine effects, normocytic anemia, and leukopenia were noted in rats dosed for 26 weeks. The TDL0 was 109 mg/kg. No effects on deaths and no clinical symptoms were noted in mice or rats orally administered (in food) benzotriazole =78 weeks. Additionally, no dose-related effects on reproductive organs were noted in either sex. Neoplastic liver nodules were observed in male Fischer rats fed 12,100 ppm benzotriazole for 78 weeks. However, historic laboratory controls incidences varied from 0 to 11% so the treatment-related effects could not be determined. Brain tumors occurred in three males and one female rat. Incidence of endometrial stromal polyps was increased significantly in female rats fed 6700 ppm for 78 weeks (22%), but not in female rats fed 12,100 ppm (16%). Significant increase in alveolar/bronchiolar carcinomas (18%) was observed female B6C3F1 fed 11,700 ppm benzotriazole for 104 weeks. Comparatively, a similar increase was not observed in female mice fed 23,500 ppm benzotriazole for the same period of time (6% increase). Historical laboratory control incidences varied from 0 to 7%. Genotoxicity studies indicate that the compound was not mutagenic to *S. typhimurium* strains TA97, TA98, or TA100 in the presence or absence of S9, or Chinese hamster ovary cells. Benzotriazole was also not mutagenic to *S. typhimurium* strain TA1535 in the absence of S9, but was mutagenic in the presence of S9. Conflicting results were obtained for effects in *S. typhimurium* strains TA1537 and TA1538 and *E. coli* WP2 uvrA. It did not produce DNA damage in *E. coli* PQ37. In Chinese hamster ovary cells, benzotriazole induced chromosomal aberrations in the presence of S9 and sister chromatid exchange in the absence of S9. Benzotriazole was not genotoxic in the mouse micronucleus assay at 800 mg/kg. Benzotriazole was identified as a non-sensitizer in the guinea pig maximization test. Benzotriazole was identified as irritating to rabbit eyes and minimally irritating to rabbit and guinea pig skin.

For phenolic benzotriazoles

Overall, oral exposure (either through gavage or in feed) of the tested chemicals to rats led to liver effects. Increased absolute and/or relative liver weights were observed in several studies. Body weight and body weight gain changes were observed after administration of several test substances. Histopathological changes (e.g., foci, hypertrophy, and cytoplasmic vacuolization) and altered liver enzyme content and activities were also noted after treatment with different phenolic benzotriazoles. Haematological effects (e.g., altered white and red blood cell counts, altered albumin levels, and packed cell volume) were observed. For those studies that calculated no observed adverse effect levels (NOAELs), the values ranged from <0.5 to ~5685 mg/kg/day.

**Reproductive and teratology effects:** The chemicals tested produced a variety of effects. Some chemicals were shown to affect reproductive organ weights, but no direct studies in reproduction and development were located.

**Genotoxicity:** None of the tested compounds were identified as mutagenic in vitro in the absence or presence of a metabolic system (S9) or in vivo.

Chemical Information Review Document for Phenolic Benzotriazoles: Supporting Nomination for Toxicological Evaluation by the National Toxicology Program October 2011

[http://ntp.niehs.nih.gov/ntp/noms/support\\_docs/phenolicbenzotriazoles\\_cird\\_oct2011\\_508.pdf](http://ntp.niehs.nih.gov/ntp/noms/support_docs/phenolicbenzotriazoles_cird_oct2011_508.pdf)

#### C7-C9 ISOALKANES

For Low Boiling Point Naphthas (LBPNS):

**Acute toxicity:**

LBPNS generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure.

Most LBPNS are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices.

**Sensitisation:**

LBPNS do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies.

**Repeat dose toxicity:**

The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPNS substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNS in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNS, were considered species- and sex-specific. These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values.

Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNS. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3.

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No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats

No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNS and very few non-cancer chronic toxicity studies were identified for other LBPNS. An LOAEC of 200 mg/m<sup>3</sup> was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m<sup>3</sup>, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m<sup>3</sup>). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m<sup>3</sup>

A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported.

**Genotoxicity:**

Although few genotoxicity studies were identified for the site-restricted LBPNS, the genotoxicity of several other LBPNS substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results.

For in vivo genotoxicity tests, LBPNS exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells.

For in vitro genotoxicity studies, LBPNS were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNS exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay. Mixed results were observed for UDS and the mouse lymphoma assay.

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

**Carcinogenicity:**

Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect of human exposure to LBPNS substances.

No inhalation studies assessing the carcinogenicity of the site-restricted LBPNS were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m<sup>3</sup> of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously. Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol. However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPNS group.

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

Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNS. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light straight-run naphtha and naphtha. Significant increases in squamous cell carcinomas were also observed when mice

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were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha

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

Reproductive/ Developmental toxicity:

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

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

For most LBPNs, no treatment-related developmental effects were observed by the different routes of exposure. However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m<sup>3</sup> delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring.

Low Boiling Point Naphthas [Site-Restricted]

Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C<sub>30</sub>. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.

The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.

The skin irritation potential of the test substance was determined according to OECD 404. The test substance was applied to the intact skin of rabbits for 4 hours under semiocclusive conditions. After removal of the test substance, no edema, but well-defined to moderate erythema were noted in all three animals. Irritating effects were reversible within 9 days. Under the test conditions, the test substance was irritating to the skin. The eye irritation potential of the hydrocarbons, C<sub>7</sub>-C<sub>9</sub>, isoalkanes was determined by instillation of 0.1 mL of the test material into the conjunctival sac of 3 rabbits. Ocular reactions were examined and scored 1, 24, 48, 72 hours after application. At the 1 hour evaluation, conjunctival redness (grade 2) was observed in all animals. Conjunctival redness (grade 0-1) was also noted in all animals at the 72 hour evaluation. All redness was reversible within 7 days. No corneal opacity, iritis or conjunctival chemosis was noted in any of the 3 rabbits. Two further studies, performed according to OECD 404) showed that no ocular irritation in any animal at any time point could be observed for the compounds Isopar E and Isopar C. In another study conducted similar to OECD 405, at 1 hour after administration, 4 of 6 animals showed slight erythema which completely subsided by the 4 hour observation. The remaining 2 animals showed slight or moderate erythema, slight edema, and slight discharge. These irritating effects were fully reversible within 72 hours after administration. Under the test conditions, the test substance was not irritating to the eyes. Based on read-across from a structurally related substance within a category approach, hydrocarbons, C<sub>7</sub>-C<sub>9</sub>, isoalkanes are not considered to be a skin sensitiser. Based on read-across from a structurally related substance (light alkylate naphtha distillate), no inhalation repeated dose toxicity is expected from the exposure to hydrocarbons, C<sub>7</sub>-C<sub>9</sub>, iso-alkanes. No need for classification according to the DSD and CLP criteria for classification and labelling. Systemic toxicity of hydrocarbons, C<sub>7</sub>-C<sub>9</sub>, iso-alkanes was assessed in a 12-week inhalation toxicity study in rats. In this study, repeated exposure to 400 or 1200 ppm of the test substance for 6 hours/day, 5 days/week, for 12 weeks resulted in male rat kidney effects consistent with the alpha-2μ-globulin-induced nephropathy in male rats. There was no treatment-related mortality and clinical findings were unremarkable. Under the test conditions, the NOAEC (excluding male rat nephropathy) was determined to be >1200ppm. The fact, that alpha-2μ-globulin-induced nephropathy was strictly limited to male rats and that the test substance belongs to a category of substances which are known for their ability to induce nephropathy in male rats due to their exclusive expression of alpha-2μ-globulin, the protein known to play the crucial role in the onset of this disease, the observed effects in the kidney have to be regarded as species-specific and are not relevant for risk assessment in humans. Therefore, additional experimental data were used to evaluate repeated dose toxicity via inhalation. Genetic toxicity: In vivo Hydrocarbons, C<sub>7</sub>-C<sub>9</sub>, isoalkanes tested in a dominant lethal study (similar to OECD 478) showed no evidence of genotoxicity in the germ cells of treated male rats exposed to 400 or 1200 ppm by inhalation. The in vivo genotoxicity of further category members has been tested. Hydrocarbons, C<sub>7</sub>-C<sub>9</sub>, n-alkanes, isoalkanes, cyclics were not clastogenic to mouse bone marrow cells. Iso-octane did not induce unscheduled DNA synthesis in rat hepatocyte cultures. Based on the category approach, these results

suggest that hydrocarbons, C7 -C9, isoalkanes are not expected to induce genotoxicity in vivo. The available data indicate that hydrocarbons, C7-C9, isoalkanes are not genotoxic. In vitro: Negative Ames test with *S. typhimurium* TA 1535, TA 1537, TA 1538, TA 98 and TA 100, and *E. coli* WP2 and WP2 uvr A, with and without metabolic activation. Negative results in mammalian chromosomal aberration and gene mutation tests, the latter with and without metabolic activation. In vivo: Negative in dominant lethal, micronucleus and unscheduled DNA synthesis assays. Endpoint Conclusion: No adverse effect observed (negative) Carcinogenicity: No standard carcinogenicity studies are available for substances in the C7-C9 aliphatic hydrocarbons category. However, with regard to the molecular structure of the substances within the category no carcinogenic potential is expected. Moreover, in investigations with various substances of the category on mutagenicity (in vitro and in vivo) as well as in repeated dose toxicity studies (oral route and via inhalation), neither genotoxicity nor an indication for neoplastic lesions was observed. In addition, all substances within the category contain benzene levels below 0.01%. Toxicity to reproduction: There are no data available on the effects on fertility of hydrocarbons, C7-C9, isoalkanes. Thus, read-across from a structurally related substance (commercial hexane) was performed, for which reliable information exists. Hexane exposure did not induce adverse effects on fertility. Reproductive indices were similar in exposed and control groups. No macroscopic or microscopic alterations in male and female reproductive organs were observed. The only significant effect was reduced body weight in the F1 and F2 generations in both sexes in the 9000 ppm exposure group both in adults and offspring. The NOAEC for both male and female rats (adults and offspring) was 3000 ppm (corresponding to 10560 mg/m<sup>3</sup>). The LOAEC for these groups was 9000 ppm based on reduced body weight. There were no adverse effects on reproduction; therefore the NOAEC for reproduction is 9000 ppm which corresponds to 31680 mg/m<sup>3</sup>. \*REACH Dossier

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; 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

#### C9-AROMATIC HYDROCARBON SOLVENT

**Polyac 61**

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.

For C9 aromatics (typically trimethylbenzenes - TMBs)

**Acute Toxicity**

Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50s range from 6,000 to 10,000 mg/m<sup>3</sup> for C9 aromatic naphtha and 18,000 to 24,000 mg/m<sup>3</sup> for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 ml/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.

**Irritation and Sensitization**

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

**Repeated Dose Toxicity**

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

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

**Oral:** The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses.

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

**Mutagenicity**

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

**Reproductive and Developmental Toxicity**

Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m<sup>3</sup>, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex/group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex/group

	<p>were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.</p> <p>Systemic Effects on Parental Generations:</p> <p>The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m<sup>3</sup>).</p> <p>Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m<sup>3</sup>). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m<sup>3</sup>), which excludes analysis of the highest concentration due to excessive mortality.</p> <p>Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~ 10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~ 12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m<sup>3</sup>) based on the body weights reductions observed in the F3 offspring.</p> <p>Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.</p> <p>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.</p> <p>Not available. Refer to individual constituents.</p>
<p><b>METHYL METHACRYLATE &amp; 2-ETHYLHEXYL ACRYLATE &amp; 2-(2'-HYDROXY-5'-METHYLPHENYL)BENZOTRIAZOLE</b></p>	<p>The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
<p><b>METHYL METHACRYLATE &amp; 2-ETHYLHEXYL ACRYLATE &amp; C9-AROMATIC HYDROCARBON SOLVENT</b></p>	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.</p>
<p><b>METHYL METHACRYLATE &amp; 2-ETHYLHEXYL ACRYLATE</b></p>	<p>Where no "official" classification for acrylates and methacrylates exists, there has been cautious attempts to create classifications in the absence of contrary evidence. For example</p> <p>Monoalkyl or monoarylestere of acrylic acids should be classified as R36/37/38 and R51/53</p> <p>Monoalkyl or monoaryl estere of methacrylic acid should be classified as R36/37/38</p> <p>Based on the available oncogenicity data and without a better understanding of the carcinogenic mechanism the Health and Environmental Review Division (HERD), Office of Toxic Substances (OTS), of the US EPA previously concluded that all chemicals that contain the acrylate or methacrylate moiety (CH<sub>2</sub>=CHCOO or CH<sub>2</sub>=C(CH<sub>3</sub>)COO)</p>

	should be considered to be a carcinogenic hazard unless shown otherwise by adequate testing. This position has now been revised and acrylates and methacrylates are no longer <i>de facto</i> carcinogens.
<b>2-(2'-HYDROXY-5'-METHYLPHENYL)BENZOTRIAZOLE &amp; C9-AROMATIC HYDROCARBON SOLVENT</b>	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

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

## SECTION 12 Ecological information

### Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
Polyac 61	Not Available	Not Available	Not Available	Not Available	Not Available
methyl methacrylate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC0(ECx)	48h	Crustacea	48mg/l	1
	EC50	96h	Algae or other aquatic plants	170mg/l	1
	EC50	72h	Algae or other aquatic plants	>110mg/l	2
	LC50	96h	Fish	>79mg/l	2
2-ethylhexyl acrylate	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	1.1mg/l	2
	EC50	72h	Algae or other aquatic plants	1.71mg/l	2
	EC50	48h	Crustacea	1.3mg/l	2
	NOEC(ECx)	504h	Crustacea	0.136mg/l	2
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1344h	Fish	123-494	7
	EC50(ECx)	24h	Crustacea	20mg/l	Not Available
	EC50	72h	Algae or other aquatic plants	0.0722mg/l	2
C7-C9 isoalkanes	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96h	Fish	0.11mg/l	2
	EC50	96h	Algae or other aquatic plants	64mg/l	2
	EC50	48h	Crustacea	0.4mg/l	2
	NOEC(ECx)	72h	Algae or other aquatic plants	0.1mg/l	1
C9-aromatic hydrocarbon solvent	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	72h	Algae or other aquatic plants	1mg/l	1
	EC50	96h	Algae or other aquatic plants	64mg/l	2

Continued...

	EC50	48h	Crustacea	6.14mg/l	1
<b>Legend:</b>	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data				

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

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

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

For methyl methacrylate (MMA):

Koc : 87

Half-life (hr) air : 2.7-3

Half-life (hr) H2O surface water : 6.3-336

Henry's atm m3 /mol: 3.24E-04

BOD 5 0.14

log BCF : 0.55

**Environmental fate:**

MMA has a water solubility of 16 g/l, a vapour pressure of 42 hPa, and a log Pow of 1.83. The environmental behavior of MMA is determined by its range of 1.1 - 9.7 hours atmospheric half life and moderate volatility. MMA is readily biodegradable. Hydrolysis is not significant at neutral and acidic pH, but increases in the upper pH range. The average Kp value of 1.0 l/kg indicates no relevant adsorption onto sediment or soil. Based on the physico-chemical properties of MMA, the air and to a much lower extent the hydrosphere are the preferred target compartments for distribution and neither relevant bioaccumulation nor geoaccumulation are expected. In waste water treatment plants 89.2 % of the substance are estimated to be removed predominately by biodegradation.

**Ecotoxicity:**

Fish LC50 (96 h): Lepomis macrochirus 191 mg/l; rainbow trout (Oncorhynchus mykiss >79 mg/l, NOEC 40 mg/l

Daphnia magna EC NOEC (21 d) 37 mg/l

Daphnia magna EC50 (48 h): 69 mg/l

The most relevant study on algae has examined *Selenastrum capricornutum* according to OECD guideline 201. The highest test concentration of 110 mg/l caused growth inhibition below 50 %, the NOEC was 110 mg/l for growth rate and 49 mg/l for biomass as endpoints.

Based on these data there is a moderate hazard concern to aquatic organisms. For derivation of the Predicted No Effect Concentration (PNEC) the lowest valid effect concentration, i.e. 37 mg/l from the long-term daphnid test, is divided by an assessment factor of 50 as proposed in the TGD for the present data basis:

PNECaqua = 740 ug/l.

It is not possible to derive a PNEC for the atmospheric compartment due to the lack of experimental data.

Data on effects to terrestrial organisms are not available. In an indicative risk assessment for the soil compartment, the aquatic PNEC of 740 ug/l can be used and compared to the concentration in soil pore water.

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

### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
methyl methacrylate	LOW	LOW
2-ethylhexyl acrylate	LOW	LOW
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	HIGH	HIGH

### Bioaccumulative potential

Ingredient	Bioaccumulation
methyl methacrylate	LOW (BCF = 6.6)
2-ethylhexyl acrylate	LOW (BCF = 289.73)
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	LOW (BCF = 494)

### Mobility in soil

Ingredient	Mobility
methyl methacrylate	LOW (KOC = 10.14)
2-ethylhexyl acrylate	LOW (KOC = 429)
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	LOW (KOC = 100800)

## SECTION 13 Disposal considerations

### Waste treatment methods

<b>Product / Packaging disposal</b>	<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 SDS 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 licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material).</li> <li>▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.</li> </ul>
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## SECTION 14 Transport information

### Labels Required

	
<b>Marine Pollutant</b>	NO
<b>HAZCHEM</b>	*3YE

### Land transport (ADG)

<b>UN number or ID number</b>	1866	
<b>UN proper shipping name</b>	RESIN SOLUTION, flammable	
<b>Transport hazard class(es)</b>	Class	3
	Subsidiary risk	Not Applicable
<b>Packing group</b>	II	
<b>Environmental hazard</b>	Not Applicable	
<b>Special precautions for user</b>	Special provisions	Not Applicable
	Limited quantity	5 L

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

<b>UN number</b>	1866	
<b>UN proper shipping name</b>	Resin solution flammable	
<b>Transport hazard class(es)</b>	ICAO/IATA Class	3
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	3L
<b>Packing group</b>	II	
<b>Environmental hazard</b>	Not Applicable	

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<b>Special precautions for user</b>	Special provisions	A3
	Cargo Only Packing Instructions	364
	Cargo Only Maximum Qty / Pack	60 L
	Passenger and Cargo Packing Instructions	353
	Passenger and Cargo Maximum Qty / Pack	5 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y341
	Passenger and Cargo Limited Maximum Qty / Pack	1 L

**Sea transport (IMDG-Code / GGVSee)**

<b>UN number</b>	1866	
<b>UN proper shipping name</b>	RESIN SOLUTION flammable	
<b>Transport hazard class(es)</b>	IMDG Class	3
	IMDG Subrisk	Not Applicable
<b>Packing group</b>	II	
<b>Environmental hazard</b>	Not Applicable	
<b>Special precautions for user</b>	EMS Number	F-E, S-E
	Special provisions	Not Applicable
	Limited Quantities	5 L

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

Not Applicable

**Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code**

Product name	Group
methyl methacrylate	Not Available
2-ethylhexyl acrylate	Not Available
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	Not Available
C7-C9 isoalkanes	Not Available
C9-aromatic hydrocarbon solvent	Not Available

**Transport in bulk in accordance with the IGC Code**

Product name	Ship Type
methyl methacrylate	Not Available
2-ethylhexyl acrylate	Not Available
2-(2'-hydroxy-5'-methylphenyl)benzotriazole	Not Available
C7-C9 isoalkanes	Not Available
C9-aromatic hydrocarbon solvent	Not Available

**SECTION 15 Regulatory information**

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

**methyl methacrylate is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

Australian Inventory of Industrial Chemicals (AIIC)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

Continued...

**2-ethylhexyl acrylate is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

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

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

**2-(2'-hydroxy-5'-methylphenyl)benzotriazole is found on the following regulatory lists**

Australian Inventory of Industrial Chemicals (AIIC)

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

**C7-C9 isoalkanes is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

**C9-aromatic hydrocarbon solvent is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

**National Inventory Status**

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (methyl methacrylate; 2-ethylhexyl acrylate; C7-C9 isoalkanes; C9-aromatic hydrocarbon solvent)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (C7-C9 isoalkanes)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (C7-C9 isoalkanes)
Vietnam - NCI	Yes
Russia - FBEPH	No (C7-C9 isoalkanes)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

**SECTION 16 Other information**

<b>Revision Date</b>	10/12/2021
<b>Initial Date</b>	19/10/2020

**SDS Version Summary**

Version	Date of Update	Sections Updated
3.1	20/08/2021	Classification change due to full database hazard calculation/update.
4.1	10/12/2021	Classification change due to full database hazard calculation/update.

**Other information**

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

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

## Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average  
PC—STEL: Permissible Concentration-Short Term Exposure Limit  
IARC: International Agency for Research on Cancer  
ACGIH: American Conference of Governmental Industrial Hygienists  
STEL: Short Term Exposure Limit  
TEEL: Temporary Emergency Exposure Limit  
IDLH: Immediately Dangerous to Life or Health Concentrations  
ES: Exposure Standard  
OSF: Odour Safety Factor  
NOAEL :No Observed Adverse Effect Level  
LOAEL: Lowest Observed Adverse Effect Level  
TLV: Threshold Limit Value  
LOD: Limit Of Detection  
OTV: Odour Threshold Value  
BCF: BioConcentration Factors  
BEI: Biological Exposure Index  
AII: Australian Inventory of Industrial Chemicals  
DSL: Domestic Substances List  
NDSL: Non-Domestic Substances List  
IECSC: Inventory of Existing Chemical Substance in China  
EINECS: European INventory of Existing Commercial chemical Substances  
ELINCS: European List of Notified Chemical Substances  
NLP: No-Longer Polymers  
ENCS: Existing and New Chemical Substances Inventory  
KECI: Korea Existing Chemicals Inventory  
NZIoC: New Zealand Inventory of Chemicals  
PICCS: Philippine Inventory of Chemicals and Chemical Substances  
TSCA: Toxic Substances Control Act  
TCSI: Taiwan Chemical Substance Inventory  
INSQ: Inventario Nacional de Sustancias Químicas  
NCI: National Chemical Inventory  
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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