

# QuakeBond J300SR Saturating Resin (Part B)

## Hychem International

Chemwatch: 7972-01

Version No: 2.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: 23/07/2025

Revision Date: 23/07/2025

Print Date: 23/07/2025

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

#### Product Identifier

Product name	QuakeBond J300SR Saturating Resin (Part B)
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	CORROSIVE LIQUID, N.O.S. (contains triethylenetetramine and triethanolamine)
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	Polymer preparations and compounds. Use according to manufacturer's directions.
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#### Details of the manufacturer or importer 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	<a href="http://www.hychem.com.au">www.hychem.com.au</a>
Email	admin@hychem.com.au

#### Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	+61 1800 951 288 (ID#: 7972-01)
Other emergency telephone number(s)	+61 3 9573 3188

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

Poisons Schedule	S5
Classification <sup>[1]</sup>	Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1A, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Carcinogenicity Category 2, Reproductive Toxicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

#### Label elements

Hazard pictogram(s)	
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## QuakeBond J300SR Saturating Resin (Part B)

Signal word	Danger
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## Hazard statement(s)

H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H351	Suspected of causing cancer.
H361fd	Suspected of damaging fertility. Suspected of damaging the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H411	Toxic to aquatic life with long lasting effects.
AUH019	May form explosive peroxides.

## Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

## Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting. If more than 15 mins from Doctor, INDUCE VOMITING (if conscious).
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.

## Precautionary statement(s) Storage

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

## Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

## SECTION 3 Composition / information on ingredients

## Substances

See section below for composition of Mixtures

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## QuakeBond J300SR Saturating Resin (Part B)

### Mixtures

CAS No	%[weight]	Name
Not Available	>50	polyoxyamine, proprietary
26950-63-0	1-<50	<u>triethylenetetramine, propoxylated</u>
100-51-6	1-<50	<u>benzyl alcohol</u>
Not Available	1-<50	epoxy adduct, proprietary
102-71-6	1-<50	<u>triethanolamine</u>
112-24-3	1-<50	<u>triethylenetetramine</u>
140-31-8	<1	<u>N-aminoethylpiperazine</u>
110-85-0	<1	<u>piperazine</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>▶ Immediately hold eyelids apart and flush the eye continuously with 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>▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.</li> <li>▶ Transport to hospital or doctor without delay.</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 or hair contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately flush body and clothes with large amounts of water, using safety shower if available.</li> <li>▶ Quickly remove all contaminated clothing, including footwear.</li> <li>▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.</li> <li>▶ Transport to hospital, or doctor.</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> <li>▶ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema.</li> <li>▶ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).</li> <li>▶ As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested.</li> <li>▶ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered.</li> </ul> <p><b>This must definitely be left to a doctor or person authorised by him/her.</b> (ICSC13719)</p>
<b>Ingestion</b>	<ul style="list-style-type: none"> <li>▶ For advice, contact a Poisons Information Centre or a doctor at once.</li> <li>▶ Urgent hospital treatment is likely to be needed.</li> <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>▶ Transport to hospital or doctor without delay.</li> </ul>

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

For acute or short-term repeated exposures to highly alkaline materials:

- ▶ Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- ▶ Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- ▶ Oxygen is given as indicated.
- ▶ The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- ▶ Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- ▶ Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

- ▶ Neutralising agents should never be given since exothermic heat reaction may compound injury.

\* Catharsis and emesis are absolutely contra-indicated.

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\* Activated charcoal does not absorb alkali.

\* Gastric lavage should not be used.

Supportive care involves the following:

- Withhold oral feedings initially.
- If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

- Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

For amines:

- Certain amines may cause injury to the respiratory tract and lungs if aspirated. Also, such products may cause tissue destruction leading to stricture. If lavage is performed, endotracheal and/or esophagoscopy control is suggested.
- No specific antidote is known.
- Care should be supportive and treatment based on the judgment of the physician in response to the reaction of the patient.

Laboratory animal studies have shown that a few amines are suspected of causing depletion of certain white blood cells and their precursors in lymphoid tissue. These effects may be due to an immunosuppressive mechanism.

Some persons with hyperreactive airways (e.g., asthmatic persons) may experience wheezing attacks (bronchospasm) when exposed to airway irritants.

Lung injury may result following a single massive overexposure to high vapour concentrations or multiple exposures to lower concentrations of any pulmonary irritant material.

Health effects of amines, such as skin irritation and transient corneal edema ("blue haze," "halo effect," "glauropsia"), are best prevented by means of formal worker education, industrial hygiene monitoring, and exposure control methods. Persons who are highly sensitive to the triggering effect of non-specific irritants should not be assigned to jobs in which such agents are used, handled, or manufactured.

**Medical surveillance programs** should consist of a pre-placement evaluation to determine if workers or applicants have any impairments (e.g., hyperreactive airways or bronchial asthma) that would limit their fitness for work in jobs with potential for exposure to amines. A clinical baseline can be established at the time of this evaluation.

Periodic medical evaluations can have significant value in the early detection of disease and in providing an opportunity for health counseling.

Medical personnel conducting medical surveillance of individuals potentially exposed to polyurethane amine catalysts should consider the following:

- Health history, with emphasis on the respiratory system and history of infections
- Physical examination, with emphasis on the respiratory system and the lymphoreticular organs (lymph nodes, spleen, etc.)
- Lung function tests, pre- and post-bronchodilator if indicated
- Total and differential white blood cell count
- Serum protein electrophoresis

Persons who are concurrently exposed to isocyanates also should be kept under medical surveillance.

Pre-existing medical conditions generally aggravated by exposure include skin disorders and allergies, chronic respiratory disease (e.g. bronchitis, asthma, emphysema), liver disorders, kidney disease, and eye disease.

Broadly speaking, exposure to amines, as characterised by amine catalysts, may cause effects similar to those caused by exposure to ammonia. As such, amines should be considered potentially injurious to any tissue that is directly contacted.

Inhalation of aerosol mists or vapors, especially of heated product, can result in chemical pneumonitis, pulmonary edema, laryngeal edema, and delayed scarring of the airway or other affected organs. There is no specific treatment.

Clinical management is based upon supportive treatment, similar to that for thermal burns.

Persons with major skin contact should be maintained under medical observation for at least 24 hours due to the possibility of delayed reactions.

**Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal Technical Bulletin June 2000**

**Alliance for Polyurethanes Industry**

Treat symptomatically.

Clinical experience of benzyl alcohol poisoning is generally confined to premature neonates in receipt of preserved intravenous salines.

- Metabolic acidosis, bradycardia, skin breakdown, hypotonia, hepatorenal failure, hypotension and cardiovascular collapse are characteristic.
- High urine benzoate and hippuric acid as well as elevated serum benzoic acid levels are found.
- The so-called "gasping syndrome" describes the progressive neurological deterioration of poisoned neonates.
- Management is essentially supportive.

If exposure has been severe and/or symptoms marked, observation in hospital for 48 hours should be considered due to possibility of delayed pulmonary oedema.

## 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>	For amines: ▸ For firefighting, cleaning up large spills, and other emergency operations, workers must wear a self-contained breathing apparatus with full face-piece, operated in a pressure-demand mode.
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### QuakeBond J300SR Saturating Resin (Part B)

	<ul style="list-style-type: none"> <li>▶ Airline and air purifying respirators should not be worn for firefighting or other emergency or upset conditions.</li> <li>▶ Respirators should be used in conjunction with a respiratory protection program, which would include suitable fit testing and medical evaluation of the user.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear full body protective clothing with breathing apparatus.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Use fire fighting procedures suitable for surrounding area.</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> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul>
<b>Fire/Explosion Hazard</b>	<ul style="list-style-type: none"> <li>▶ Combustible.</li> <li>▶ Slight fire hazard when exposed to heat or flame.</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> <li>▶ May emit acrid smoke.</li> <li>▶ Mists containing combustible materials may be explosive.</li> </ul> <p>Combustion products include: carbon dioxide (CO<sub>2</sub>) aldehydes nitrogen oxides (NO<sub>x</sub>) other pyrolysis products typical of burning organic material. May emit corrosive fumes.</p>
<b>HAZCHEM</b>	2X

## 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>▶ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.</li> <li>▶ Check regularly for spills and leaks.</li> </ul> <p>Small spills should be covered with inorganic absorbents and disposed of properly. Organic absorbents have been known to ignite when contaminated with amines in closed containers. Certain cellulosic materials used for spill cleanup such as wood chips or sawdust have shown reactivity with ethyleneamines and should be avoided. Ethyleneamine leaks will frequently be identified by the odor (ammoniacal) or by the formation of a white, solid, waxy substance (amine carbamates). Inorganic absorbents or water may be used to clean up the amine waste.</p> <p>Slippery when spilt.</p> <ul style="list-style-type: none"> <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 spill with sand, earth, inert material or vermiculite.</li> <li>▶ Wipe up.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> </ul>
<b>Major Spills</b>	<p>Environmental hazard - contain spillage.</p> <p>Slippery when spilt.</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>▶ Wear full body protective clothing with breathing apparatus.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Consider evacuation (or protect in place).</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Neutralise/decontaminate residue (see Section 13 for specific agent).</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

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

## SECTION 7 Handling and storage

### Precautions for safe handling

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### QuakeBond J300SR Saturating Resin (Part B)

<b>Safe handling</b>	<p>Alkanolamines and iron may produce unstable complexes. Monoethanolamine (MEA) and iron form a trisethanolamino-iron complex. This material may spontaneously decompose at temperatures between 130 and 160 degrees C. and is suspected of causing a fire in a nearly empty storage tank containing a "heel" of MEA in contact with carbon steel coils. If steam coil heating is used, low pressure steam in stainless steel coils should be considered. Drum heating should also be reviewed and, where possible, temperatures should be maintained below 130 degrees C.</p> <ul style="list-style-type: none"> <li>▶ <b>DO NOT USE brass or copper containers / stirrers</b></li> <li>▶ <b>DO NOT allow clothing wet with material to stay in contact with skin</b></li> <li>▶ Overheating of ethoxylates/ alkoxyates in air should be avoided. When some ethoxylates are heated vigorously in the presence of air or oxygen, at temperatures exceeding 160 C, they may undergo exothermic oxidative degeneration resulting in self-heating and autoignition.</li> <li>▶ Nitrogen blanketing will minimise the potential for ethoxylate oxidation. Prolonged storage in the presence of air or oxygen may cause product degradation. Oxidation is not expected when stored under a nitrogen atmosphere. Inert gas blanket and breathing system needed to maintain color stability. Use dry inert gas having at least -40 C dew point.</li> <li>▶ Trace quantities of ethylene oxide may be present in the material. Although these may accumulate in the headspace of storage and transport vessels, concentrations are not expected to exceed levels which might produce a flammability or worker exposure hazard.</li> </ul> <p>The substance accumulates peroxides which may become hazardous only if it evaporates or is distilled or otherwise treated to concentrate the peroxides. The substance may concentrate around the container opening for example.</p> <p>Purchases of peroxidisable chemicals should be restricted to ensure that the chemical is used completely before it can become peroxidised.</p> <ul style="list-style-type: none"> <li>▶ A responsible person should maintain an inventory of peroxidisable chemicals or annotate the general chemical inventory to indicate which chemicals are subject to peroxidation. An expiration date should be determined. The chemical should either be treated to remove peroxides or disposed of before this date.</li> <li>▶ The person or laboratory receiving the chemical should record a receipt date on the bottle. The individual opening the container should add an opening date.</li> <li>▶ Unopened containers received from the supplier should be safe to store for 18 months.</li> <li>▶ Opened containers should not be stored for more than 12 months.</li> <li>▶ Avoid all personal contact, including inhalation.</li> <li>▶ Wear protective clothing when risk of exposure occurs.</li> <li>▶ Use in a well-ventilated area.</li> <li>▶ Avoid contact with moisture.</li> <li>▶ Avoid contact with incompatible materials.</li> <li>▶ <b>When handling, DO NOT eat, drink or smoke.</b></li> <li>▶ Keep containers securely sealed when not in use.</li> <li>▶ Avoid physical damage to containers.</li> <li>▶ Always wash hands with soap and water after handling.</li> <li>▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.</li> <li>▶ Use good occupational work practice.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> <li>▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.</li> </ul>
<b>Other information</b>	<ul style="list-style-type: none"> <li>▶ <b>DO NOT store near acids, or oxidising agents</b></li> <li>▶ No smoking, naked lights, heat or ignition sources.</li> <li>▶ Store in original containers.</li> <li>▶ Keep containers securely sealed.</li> <li>▶ No smoking, naked lights or ignition sources.</li> <li>▶ Store in a cool, dry, well-ventilated area.</li> <li>▶ Store away from incompatible materials and foodstuff containers.</li> <li>▶ Protect containers against physical damage and check regularly for leaks.</li> <li>▶ Observe manufacturer's storage and handling recommendations contained within this SDS.</li> </ul>

#### Conditions for safe storage, including any incompatibilities

<b>Suitable container</b>	<p>For ethoxylates suitable containers include carbon steel coated with baked phenolic. Any moisture may cause rusting of carbon steel.</p> <p>If product is moisture free, uncoated carbon steel tanks may be used.</p> <ul style="list-style-type: none"> <li>▶ Glass container is suitable for laboratory quantities</li> <li>▶ Lined metal can, lined metal pail/ can.</li> <li>▶ Plastic pail.</li> <li>▶ Polyliner drum.</li> <li>▶ Packing as recommended by manufacturer.</li> <li>▶ Check all containers are clearly labelled and free from leaks.</li> </ul> <p>For low viscosity materials</p> <ul style="list-style-type: none"> <li>▶ Drums and jerricans must be of the non-removable head type.</li> <li>▶ Where a can is to be used as an inner package, the can must have a screwed enclosure.</li> </ul> <p>For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.):</p> <ul style="list-style-type: none"> <li>▶ Removable head packaging;</li> <li>▶ Cans with friction closures and</li> <li>▶ low pressure tubes and cartridges</li> </ul> <p>may be used.</p> <p>-</p> <p>Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.</p>
<b>Storage incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid strong acids, bases.</li> <li>▶ Avoid contact with copper, aluminium and their alloys.</li> </ul>

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QuakeBond J300SR Saturating Resin (Part B)

▶ Avoid reaction with oxidising agents

**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	triethanolamine	Triethanolamine	5 mg/m3	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
triethylenetetramine, propoxylated	Not Available	Not Available
benzyl alcohol	Not Available	Not Available
triethanolamine	Not Available	Not Available
triethylenetetramine	Not Available	Not Available
N-aminoethylpiperazine	Not Available	Not Available
piperazine	Not Available	Not Available


**MATERIAL DATA**

**Exposure controls**

Appropriate engineering controls																			
	<p>Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation. HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours. Barrier protection or laminar flow cabinets should be considered for laboratory scale handling. A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg. When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology. Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies. Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required. Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved. Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. 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.</p> <table border="1" style="width: 100%;"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, etc. evaporating from tank (in still air)</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" style="width: 100%;"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 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. The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated.</p>	Type of Contaminant:	Air Speed:	solvent, vapours, 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 (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	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Continued...

### QuakeBond J300SR Saturating Resin (Part B)

	<p>The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of:</p> <p>10; high efficiency particulate (HEPA) filters or cartridges  10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator.  25-50; a full face-piece negative pressure respirator with HEPA filters  50-100; tight-fitting, full face-piece HEPA PAPR  100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode.</p>
<p><b>Individual protection measures, such as personal protective equipment</b></p>	
<p><b>Eye and face protection</b></p>	<p>Epoxy amine hardeners may produce eye discomfort, irritation, or even injury; thus, all eye contact with either the liquid or solid products (including vapours, mists, aerosols, or dusts) should be strictly avoided through the use of appropriate eye protection, including chemical workers goggles (or monogoggles), a face shield that allows the use of chemical workers goggles, or a full-face respirator, depending on the degree of potential exposure.</p> <p>For amines:  <b>SPECIAL PRECAUTION:</b></p> <ul style="list-style-type: none"> <li>▶ Because amines are alkaline materials that can cause rapid and severe tissue damage, wearing of contact lenses while working with amines is strongly discouraged. Wearing such lenses can prolong contact of the eye tissue with the amine, thereby causing more severe damage.</li> <li>▶ Appropriate eye protection should be worn whenever amines are handled or whenever there is any possibility of direct contact with liquid products, vapors, or aerosol mists.</li> </ul> <p><b>CAUTION:</b></p> <ul style="list-style-type: none"> <li>▶ Ordinary safety glasses or face-shields will not prevent eye irritation from high concentrations of vapour.</li> <li>▶ In operations where positive-pressure, air-supplied breathing apparatus is not required, all persons handling liquid amine catalysts or other polyurethane components in open containers should wear chemical workers safety goggles.</li> <li>▶ Eyewash fountains should be installed, and kept in good working order, wherever amines are used.</li> </ul> <p>When handling very small quantities of the material eye protection may not be required.  For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs:</p> <ul style="list-style-type: none"> <li>▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]</li> <li>▶ Face shield. Full face shield may be required for supplementary but never for primary protection of eyes.</li> <li>▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of 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].</li> </ul>
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>
<p><b>Hands/feet protection</b></p>	<ul style="list-style-type: none"> <li>▶ Elbow length PVC gloves</li> <li>▶ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.  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:</p> <ul style="list-style-type: none"> <li>· frequency and duration of contact,</li> <li>· chemical resistance of glove material,</li> <li>· glove thickness and</li> <li>· dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.</li> <li>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>· Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>· Excellent when breakthrough time &gt; 480 min</li> <li>· Good when breakthrough time &gt; 20 min</li> <li>· Fair when breakthrough time &lt; 20 min</li> <li>· Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p>

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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.

For amines:

- ▶ 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
- ▶ Where there is a possibility of exposure to liquid amines skin protection should include: rubber gloves, (neoprene, nitrile, or butyl).
- ▶ DO NOT USE latex.

When handling liquid-grade epoxy resins wear chemically protective gloves, boots and aprons.

The performance, based on breakthrough times, of:

- Ethyl Vinyl Alcohol (EVAL laminate) is generally excellent
- Butyl Rubber ranges from excellent to good
- Nitrile Butyl Rubber (NBR) from excellent to fair.
- Neoprene from excellent to fair
- Polyvinyl (PVC) from excellent to poor

As defined in ASTM F-739-96

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

Gloves should be tested against each resin system prior to making a selection of the most suitable type. Systems include both the resin and any hardener, individually and collectively)

- **DO NOT use cotton or leather (which absorb and concentrate the resin), natural rubber (latex), medical or polyethylene gloves (which absorb the resin).**
- **DO NOT use barrier creams containing emulsified fats and oils as these may absorb the resin; silicone-based barrier creams should be reviewed prior to use.**

Replacement time should be considered when selecting the most appropriate glove. It may be more effective to select a glove with lower chemical resistance but which is replaced frequently than to select a more resistant glove which is reused many times

- ▶ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference.
- ▶ Double gloving should be considered.
- ▶ PVC gloves.
- ▶ Change gloves frequently and when contaminated, punctured or torn.
- ▶ Wash hands immediately after removing gloves.
- ▶ Protective shoe covers. [AS/NZS 2210]
- ▶ Head covering.

**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.

**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:

QuakeBond J300SR Saturating Resin (Part B)

Material	CPI
BUTYL	A
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
PVC	C

**Respiratory protection**

Type AK-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 10 x ES	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury,

## QuakeBond J300SR Saturating Resin (Part B)

VITON	C
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\* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

### Ansell Glove Selection

**Glove** — *In order of recommendation*

AlphaTec® Solvex® 37-185

AlphaTec® 58-008

AlphaTec® Solvex® 37-675

TouchNTuff® 92-600

TouchNTuff® 92-500

TouchNTuff® 92-605

TouchNTuff® 93-250

TouchNTuff® 93-700

AlphaTec® 38-612

AlphaTec® 58-530B

The suggested gloves for use should be confirmed with the glove supplier.

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

Where engineering controls are not feasible and work practices do not reduce airborne amine concentrations below recommended exposure limits, appropriate respiratory protection should be used. In such cases, air-purifying respirators equipped with cartridges designed to protect against amines are recommended.

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## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

Appearance	Colourless light yellow liquid with amine-like odour; partly mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	0.98
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Applicable	Viscosity (cSt)	173.47
Initial boiling point and boiling range (°C)	>100	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>100	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	<0.1 @20C	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available

## SECTION 10 Stability and reactivity

### QuakeBond J300SR Saturating Resin (Part B)

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

## SECTION 11 Toxicological information

### Information on toxicological effects

<b>a) Acute Toxicity</b>	There is sufficient evidence to classify this material as acutely toxic.
<b>b) Skin Irritation/Corrosion</b>	There is sufficient evidence to classify this material as skin corrosive or irritating.
<b>c) Serious Eye Damage/Irritation</b>	There is sufficient evidence to classify this material as eye damaging or irritating
<b>d) Respiratory or Skin sensitisation</b>	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
<b>e) Mutagenicity</b>	Based on available data, the classification criteria are not met.
<b>f) Carcinogenicity</b>	There is sufficient evidence to classify this material as carcinogenic
<b>g) Reproductivity</b>	There is sufficient evidence to classify this material as toxic to reproductivity
<b>h) STOT - Single Exposure</b>	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
<b>i) STOT - Repeated Exposure</b>	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
<b>j) Aspiration Hazard</b>	Based on available data, the classification criteria are not met.

<b>Inhaled</b>	<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>Inhalation hazard is increased at higher temperatures.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
<b>Ingestion</b>	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>The material can produce severe chemical burns within the oral cavity and gastrointestinal tract following ingestion.</p>
<b>Skin Contact</b>	<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>Skin contact with the material may produce serious damage to the health of the individual; systemic effects may result following absorption.</p> <p>The material can produce severe chemical burns following direct contact with the skin.</p>
<b>Eye</b>	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p> <p>The material can produce severe chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating.</p>
<b>Chronic</b>	<p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.</p> <p>Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population.</p> <p>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.</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>

Continued...

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.

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.

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.

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

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

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

Allergic reactions to benzoic acid have been reported. Of 100 patients with asthma undergoing provocation tests with benzoic acid, 47 showed positive reactions. In another study, of 75 patients with recurrent urticaria (skin eruptions) and angio-oedema (a deep dermal condition characterised by large wheals) of more than 4 months duration, 44 were found to be sensitive to sodium benzoate or p-hydroxybenzoic acid (paraben), alone or in conjunction with aspirin or azo-dyes, or both. In a further work there was no significant objective or subjective skin response to two 500-mg daily doses of benzoic acid or lactic acid in a double blind study of 150 dermatological patients

Prolonged or chronic exposure to alkanolamines may result in liver, kidney or nervous system injury. Repeated inhalation may aggravate asthma and inflammatory or fibrotic pulmonary disease.

Results of repeated exposure tests with diethanolamine (DEA) in laboratory animals include anaemia (rats) and effects on the kidneys (rats and mice) and liver (mice). DEA produces nervous system injury in dogs and rats. Heart and salivary gland lesions have also been seen in mice treated cutaneously with DEA and in mice receiving DEA in drinking water. Rats given high doses of DEA developed anaemia and testicular lesions.

Exaggerated doses of DEA produced heart and nervous system effects in other animals. Changes in other organs were judged to be secondary due to the poor health of animals subjected to extremely high doses of DEA. Rats, rabbits and guinea pigs exposed to high vapour concentrations of volatile monoethanolamine (MEA) (up to 1250 ppm) for periods of up to 5 weeks developed pulmonary, hepatic and renal lesions. Dogs, rats and guinea pigs exposed to 100 ppm MEA for 30 days, became apathetic and developed poor appetites. Animal tests also indicate that inhalation exposure to MEA may result in nervous system injury. All species exposed to airborne MEA experienced dermal effects, varying from ulceration to hair loss probably resulting from contact with the cage.

An increased incidence of skeletal variations, suggestive of a slight developmental delay was seen in the foetuses of rats given 1500 mg/kg/day DEA cutaneously; this also produced significant maternal toxicity. No foetal malformations, however, were seen in rats nor in rabbits receiving identical treatment. The foetus of rats given high doses of MEA by gavage, showed an increased rate of embryofoetal death, growth retardation, and some malformations including hydronephrosis and hydroureter. The high doses required to produce these effects bring into question the relevance of this finding to humans. There is some evidence that embryofoetotoxicity and teratogenicity does not occur in rats when MEA is administered by dermal application to the mother.

The National Toxicology Program (NTP) concluded that there is clear evidence of liver tumours and some evidence of kidney tumours in mice exposed dermally to DEA over their lifetime. Chronic skin painting studies in mice of both sexes produced liver tumours and an increased incidence of kidney tumours in male mice. The significance of these findings to humans is unclear as DEA is neither genotoxic, mutagenic nor clastogenic, and did not induce tumours in rats or transgenic mice similarly treated. Alkanolamines (especially those containing a secondary amine moiety) may react with nitrites or other nitrosating agents to form carcinogenic N-nitrosamines. Alkanolamines are metabolised by biosynthetic routes to ethanolamine and choline and incorporated into phospholipids. They are excreted predominantly unchanged with a half-life of approximately one week. In the absence of sodium nitrite, no conversion to carcinogenic N-nitrosamines was observed.

Diethanolamine competitively inhibits the cellular uptake of choline, in vitro, and hepatic changes in choline homeostasis, consistent with choline deficiency, are observed in vivo.

Many amines are potent skin and respiratory sensitisers and certain individuals especially those described as "atopic" (i.e. those predisposed to asthma and other allergic responses) may show allergic reactions when chronically exposed to alkanolamines.

In a study with coconut diethanolamide, the National Toxicology Program (Technical Report Series 479), showed clear evidence of carcinogenic activity in male B6C3F1 mice based on increased incidences of hepatic and renal tubule neoplasms and in female B6C3F1 mice based on increased incidences of hepatic neoplasms. There was equivocal evidence of carcinogenic activity in female F344/N rats based on a marginal increase in the incidence of renal tube neoplasms. These increases were associated with the concentration of free diethanolamine present as a contaminant in the diethanolamine condensate. Exposure to rats to coconut oil diethanolamine condensate by dermal application in ethanol for 2 years resulted in epidermal hyperplasia, sebaceous gland hyperplasia, hyperkeratosis and parakeratosis in males and females and ulcer in females at the site of application. There were increases in the incidences of chronic inflammation, epithelial hyperplasia, and epithelial ulcer in the forestomach of female rats. The severity of nephropathy in dosed female rats were increased. Exposure of mice to coconut oil diethanolamine condensate by dermal application for 2 years resulted in increased incidences of eosinophilic foci of the liver in

## QuakeBond J300SR Saturating Resin (Part B)

males. Increased incidences of epidermal hyperplasia, sebaceous gland hyperplasia, and hyperkeratosis in males and females, ulcer in males, and parakeratosis and inflammation in females at the site of application and of follicular cell hyperplasia in the thyroid gland of males and females, were chemical related.

Secondary amines may react in the acid conditions of the stomach with oxidants or preservatives) to form potentially carcinogenic N-nitrosamines. The formation of nitrosamines from such amines has not only been observed in animals models but, at least for certain compounds, in the workplace. The amine-containing substances and end products handled at work can themselves be contaminated to a degree with corresponding nitrosamines. Under conditions encountered in practice nitrosation is to be expected with secondary amines and to a limited extent with primary and tertiary amines. Nitrogen oxides are the most probable nitrosating agents. Nitrosyl chloride, nitrite esters, metal nitrites and nitroso compounds may also be involved. Several factors such as pH, temperature, catalysts and inhibitors influence the extent of nitrosation. Two precautionary measures are therefore necessary when handling amines at the workplace.

- ▶ Simultaneous exposure to nitrosating agents should be reduced to minimum. This can be put into practice by eliminating nitrosating agents or, if they play a role in the actual process, replacing them with substances that do not lead to the formation of carcinogenic nitrosamines. In particular the level of nitrogen oxides at the workplace should be monitored and reduced when necessary.
- ▶ The levels of nitrosamines in the workplace and in substances containing amines should be monitored.

Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Report No. 31, DFG, 1995

In animal experiments the oesophagus is shown to be the most important target organ for nitrosamines, independent of the route of application. The mechanism of this organotrophy cannot be explained sufficiently. The high oesophageal epithelium metabolic activation of nitrosamines, together with a comparatively low DNA repair, probably plays the most important role. In addition chronic stress factors, which lead to high stimulation of epithelial turnover, are a pacemaker for malignant progression. In some countries, the traditional consumption of extremely hot drinks leads to constant burns of the oesophagus, which increases the risk. Mate, a non-alcoholic brew, frequently consumed as tea in Uruguay, appears to be a high risk factor for oesophageal cancer. Prolonged or repeated exposure to benzyl alcohol may cause allergic contact dermatitis.

Prolonged or repeated ingestion may affect behavior/central nervous system with symptoms similar to acute ingestion. It may also affect the liver, kidneys, cardiovascular system, and metabolism (weight loss).

Animal studies have shown this compound to cause lung, liver, kidney and CNS disorders. Studies in animals have shown evidence of teratogenicity in the chick embryo. The significance of the information for humans is unknown.

Benzyl alcohol showed no evidence of carcinogenic activity in long-term toxicology and carcinogenesis study.

Reversible liver and kidney damage has been demonstrated in animals from chronic exposure to triethanolamine.

Although the product is not, in itself, carcinogenic, reaction under strong acid conditions, with nitrites and nitrous acids results in the formation of a potent carcinogen, N-nitrosodiethanolamine.

This situation might be encountered in certain metal-treatment operations, for example.

A cohort study, in which cancer morbidity and mortality were investigated in workers exposed to cutting fluids with nitrates and amines (amongst them triethanolamine), had negative results. The effects on workers industrially exposed to metal-working coolants containing sodium nitrite and triethanolamine solutions were investigated in a Russian study. Observed vascular effects were attributed to sodium nitrite; no effects were attributed to triethanolamine.

Screening studies in mice suggest that the material does not effect foetal development.

In the European Union, trialkylamines, trialkanolamines, and their salts (ingredients containing TEA) may only be used up to 2.5%, must be at least 99% pure, are not to be used with nitrosating systems, must have a maximum secondary amine content of 0.5%, must have a maximum nitrosamine content of 50 ug/kg, and must be kept in nitrite-free containers

Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.

Inhalation of epoxy resin amine hardener vapours (including polyamines and amine adducts) may produce bronchospasm and coughing episodes lasting days after cessation of the exposure. Even faint traces of these vapours may trigger an intense reaction in individuals showing "amine asthma". The literature records several instances of systemic intoxications following the use of amines in epoxy resin systems.

Excessive exposure to the vapours of epoxy amine curing agents may cause both respiratory irritation and central nervous system depression. Signs and symptoms of central nervous system depression, in order of increasing exposure, are headache, dizziness, drowsiness, and incoordination. In short, a single prolonged (measured in hours) or excessive inhalation exposure may cause serious adverse effects, including death.

QuakeBond J300SR Saturating Resin (Part B)	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
triethylenetetramine, propoxylated	<b>TOXICITY</b>	<b>IRRITATION</b>
	Not Available	Not Available
benzyl alcohol	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 2000 mg/kg <sup>[2]</sup>	Eye (Rodent - rat): 0.1mL
	Inhalation (Rat) LC50: >4.178 mg/L4h <sup>[2]</sup>	Eye: adverse effect observed (irritating) <sup>[1]</sup>
	Oral (Rat) LD50: 1230 mg/kg <sup>[2]</sup>	Skin (Human - man): 16mg/48H - Mild
		Skin (Human): 1%/2D
		Skin (Mammal - pig): 100% - Moderate
	Skin (Rodent - rabbit): 100mg/24H - Moderate	
	Skin: no adverse effect observed (not irritating) <sup>[1]</sup>	
triethanolamine	<b>TOXICITY</b>	<b>IRRITATION</b>

### QuakeBond J300SR Saturating Resin (Part B)

	dermal (rat) LD50: >16000 mg/kg <sup>[2]</sup>	Eye (Rodent - rabbit): 10mg - Mild
	Oral (Rabbit) LD50; 2200 mg/kg <sup>[2]</sup>	Eye (Rodent - rabbit): 20mg - Severe
		Eye: no adverse effect observed (not irritating) <sup>[1]</sup>
		Skin (Human): 15mg/3D (intermittent) - Mild
		Skin (Rodent - mouse): 50% - Severe
		Skin (Rodent - rabbit): 560mg/24H - Mild
		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>triethylenetetramine</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 805 mg/kg <sup>[2]</sup>	Eye (Rodent - rabbit): 20mg/24H - Moderate
	Oral (Rat) LD50: 1591.4 mg/kg <sup>[1]</sup>	Eye (Rodent - rabbit): 49mg - Severe
		Eye: adverse effect observed (irritating) <sup>[1]</sup>
		Skin (Rodent - rabbit): 490mg - Severe
		Skin (Rodent - rabbit): 5mg/24H - Severe
<b>N-aminoethylpiperazine</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 880 mg/kg <sup>[2]</sup>	Eye (Rodent - rabbit): 20mg/24H - Moderate
	Oral (Rat) LD50: 2410 mg/kg <sup>[2]</sup>	Eye: adverse effect observed (irreversible damage) <sup>[1]</sup>
		Skin (Rodent - rabbit): 100ug/24H
		Skin (Rodent - rabbit): 5mg/24H - Severe
		Skin: adverse effect observed (corrosive) <sup>[1]</sup>
<b>piperazine</b>	<b>TOXICITY</b>	<b>IRRITATION</b>
	Dermal (rabbit) LD50: 4000 mg/kg <sup>[2]</sup>	Eye (Rodent - rabbit): 0.005mL - Moderate
	Oral (Rat) LD50: 1900 mg/kg <sup>[2]</sup>	Eye (Rodent - rabbit): 0.02mL - Severe
		Eye (Rodent - rabbit): 250ug - Severe
		Eye (Rodent - rabbit): 250ug/24H - Severe
		Skin (Human): 1%
		Skin (Rodent - guinea pig): 50%/2H
		Skin (Rodent - rabbit): 0.01mL - Moderate
		Skin (Rodent - rabbit): 25%
		Skin (Rodent - rabbit): 500mg - Mild
	Skin (Rodent - rabbit): 500mg - Mild	
	Skin: adverse effect observed (corrosive) <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>TRIETHYLENETETRAMINE, PROPOXYLATED</b>	<p>No significant acute toxicological data identified in literature search.</p> <p>Polyethers (such as ethoxylated surfactants and polyethylene glycols) are highly susceptible to being oxidized in the air. They then form complex mixtures of oxidation products.</p> <p>Animal testing reveals that whole the pure, non-oxidised surfactant is non-sensitizing, many of the oxidation products are sensitizers. The oxidation products also cause irritation.</p>
<b>BENZYL ALCOHOL</b>	<p>Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.</p> <p>Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.</p>

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Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.

Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.

Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease. Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

**Hands:** Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

**Axillae Bilateral axillary** (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

**Face** Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.

**Irritant reactions (including contact urticaria):** Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

**Pigmentary anomalies:** The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

**Photo-reactions** Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

**General/respiratory:** Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prohaptens is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohaptens is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prohaptens or as a prohaptens, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.

#### **Prohaptens**

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.

In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and

phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitizers has not been studied in detail. Skin sensitising prohaptenes can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

**QSAR prediction:** The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptenes) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptenes. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation

CYP1A2 is a member of the cytochrome P450 super family, is one of the best characterized. It is responsible for the metabolism of commonly drugs belonging to classes such as antidepressants, antipsychotics, mood stabilizers, beta blockers and sedative/hypnotics CYP1A2 also metabolises a number of procarcinogens (such as those in cigarettes). Cigarette smoking may lead to three fold increase in 1A2 activity, which explains why smokers require higher doses of beta blockers than non-smokers

Drugs that inhibit CYP1A2 will predictably increase the plasma concentrations of the medications or decrease in clearance of substrates. Drugs such as ciprofloxacin, fluvoxamine, verapamil, cimetidine, caffeine and isoniazid are inhibitors of CYP1A2 enzyme. Vegetables such as grape fruit juice, curcumin and turmeric are inhibitors of the CYP1A2 enzyme which may lead to increase plasma concentration of psychotropics

Inhibition of NF- $\kappa$ B in vivo can be detrimental. NF- $\kappa$ B controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF- $\kappa$ B activity.

The same functions that make NF- $\kappa$ B attractive for developing inhibitors for treating disease also play a role in homeostasis, and disruption of the NF- $\kappa$ B pathway during development or in adults leads to unfavorable and potentially unhealthy consequences.

NF- $\kappa$ B plays a role in multiple homeostatic cellular processes including response to stimuli, cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such as p38 and JNK. In addition to mediating proinflammatory responses, NF- $\kappa$ B may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF- $\kappa$ B can have adverse consequences such as immune suppression and tissue damage.

Understanding the consequences of lack of NF- $\kappa$ B activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF- $\kappa$ B resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF- $\kappa$ B activation including IKK gamma (NEMO), a subunit of the IKK complex, and I $\kappa$ Balpha. The IKK gamma mutations result in a defective IKK complex and the I $\kappa$ Balpha mutation results in an I $\kappa$ Balpha protein that cannot be phosphorylated and degraded.

Both genetic defects result in suppressed NF- $\kappa$ B activation and ectodermal dysplasia with immunodeficiency. In general patients with these genetic defects have multiple immunological defects including innate immunity, impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF- $\kappa$ B pathway will help prepare for potential adverse effects of pharmacologic NF- $\kappa$ B inhibitors

The requirement for NF- $\kappa$ B in the development and maintenance of the immune system is well documented. NF- $\kappa$ B is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF- $\kappa$ B or the IKKbeta gene results in death during fetal development primarily due to massive liver apoptosis

Fetal liver stem cells from p65 or IKKbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF- $\kappa$ B for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells.

The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF- $\kappa$ B activity. Lymphocyte depletion with chemical or genetic inhibition of NF- $\kappa$ B have implications for therapeutic potential use in humans. The double-sided nature of NF- $\kappa$ B inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations.

In addition to controlling lymphocyte development, NF- $\kappa$ B plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF- $\kappa$ B which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF- $\kappa$ B through phosphorylation of CARMA1 by PKC theta and PKC beta respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF- $\kappa$ B plays a role in T-cell response to costimulatory signals.

Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs). TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF- $\kappa$ B leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF- $\kappa$ B during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF- $\kappa$ B is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF- $\kappa$ B activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation. Given the diverse roles NF- $\kappa$ B plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF- $\kappa$ B pathway are susceptible to parasitic and bacterial infection.

The role of NF- $\kappa$ B in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF- $\kappa$ B deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF- $\kappa$ B targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator. Liver

damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF- $\kappa$ B activation.

The aryl alkyl alcohol (AAA) fragrance ingredients are a diverse group of chemical structures with similar metabolic and toxicity profiles.

The AAA fragrances demonstrate low acute and subchronic dermal and oral toxicity.

At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin.

The potential for eye irritation is minimal.

With the exception of benzyl alcohol and to a lesser extent phenethyl and 2-phenoxyethyl AAA alcohols, human sensitization studies, diagnostic patch tests and human induction studies, indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low.

NOAELs for maternal and developmental toxicity are far in excess of current human exposure levels.

No carcinogenicity in rats or mice was observed in 2-year chronic testing of benzyl alcohol or a-methylbenzyl alcohol; the latter did induce species and gender-specific renal adenomas in male rats at the high dose. There was no to little genotoxicity, mutagenicity, or clastogenicity in the mutagenicity *in vitro* bacterial assays, and *in vitro* mammalian cell assays. All *in vivo* micronucleus assays were negative.

It is concluded that these materials would not present a safety concern at current levels of use as fragrance ingredients

The Research Institute for Fragrance Materials (RIFM) Expert Panel

A member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS) based in part on their self-limiting properties as flavouring substances in food; their rapid absorption, metabolic detoxification, and excretion in humans and other animals, their low level of flavour use, the wide margin of safety between the conservative estimates of intake and the no-observed-adverse effect levels determined from chronic and subchronic studies and the lack of significant genotoxic and mutagenic potential. This evidence of safety is supported by the fact that the intake of benzyl derivatives as natural components of traditional foods is greater than the intake as intentionally added flavouring substances.

All members of this group are aromatic primary alcohols, aldehydes, carboxylic acids or their corresponding esters or acetals.

The substances in this group:

- contain a benzene ring substituted with a reactive primary oxygenated functional group or can be hydrolysed to such a functional group
- the major pathway of metabolic detoxification involves hydrolysis and oxidation to yield the corresponding benzoic acid derivative which is excreted either as the free acid or the glycine conjugate
- they show a consistent pattern of toxicity in both short- and long- term studies and
- they exhibit no evidence of genotoxicity in standardised batteries of *in vitro* and *in vivo* assays.

The benzyl derivatives are rapidly absorbed through the gut, metabolised primarily in the liver, and excreted in the urine as glycine conjugates of benzoic acid derivatives.

In general, aromatic esters are hydrolysed *in vivo* through the catalytic activity of carboxylesterases, the most important of which are the A-esterases. Hydrolysis of benzyl and benzoate esters to yield corresponding alcohols and carboxylic acids and hydrolysis of acetals to yield benzaldehyde and simple alcohols have been reported in several experiments.

The alcohols and aldehydes are rapidly oxidised to benzoic acid while benzoate esters are hydrolysed to benzoic acid.

Flavor and Extract Manufacturers Association (FEMA)

For benzyl alkyl alcohols:

Unlike benzylic alcohols, the beta-hydroxyl group of the members of this cluster is unlikely to undergo phase II metabolic activation. Instead, the beta-hydroxyl group is expected to contribute to detoxification via oxidation to hydrophilic acid. Despite structural similarity to carcinogenic ethyl benzene, only a marginal concern has been assigned to phenethyl alcohol due to limited mechanistic analogy.

For benzoates:

**Acute toxicity:** Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol.

The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds.

Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye.

**Sensitisation:** The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers.

**Repeat dose toxicity:** For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed.

For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur.

**Mutagenicity:** All chemicals showed no mutagenic activity in *in vitro* Ames tests. Various results were obtained with other *in vitro* genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity *in vivo*. While some mixed and/or equivocal *in vitro* chromosomal/chromatid responses have been observed, no genotoxicity was observed in the *in vivo* cytogenetic, micronucleus, or other assays. The weight of the evidence of the *in vitro* and *in vivo* genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.

In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts.

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**Developmental toxicity:** In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed.

**TRIETHANOLAMINE**

Lachrymation, diarrhoea, convulsions, urinary tract changes, changes in bladder weight, changes in testicular weight, changes in thymus weight, changes in liver weight, dermatitis after systemic exposure, kidney, ureter, bladder tumours recorded. Equivocal tumourigen by RTECS criteria. Dermal rabbit value quoted above is for occluded patch in male or female animals \* Union Carbide

While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.

- ▶ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis.
- ▶ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient.

Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.

**Inhalation:**

Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs.

Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.

Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.

Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies.

While most polyurethane amine catalysts are not sensitizers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitized, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease.

Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.

**Skin Contact:**

Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.

Skin contact with some amines may result in allergic sensitization. Sensitized persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.

**Eye Contact:**

Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations.

Direct contact with the liquid amine may cause severe irritation and tissue injury, and the "burning" may lead to blindness.

(Contact with solid products may result in mechanical irritation, pain, and corneal injury.)

Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.

The corneal swelling may manifest itself in visual disturbances such as blurred or "foggy" vision with a blue tint ("blue haze") and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases.

Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.

**Ingestion:**

The oral toxicity of amine catalysts varies from moderately to very toxic.

Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus, and gastrointestinal tract.

Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.

Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.

**Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000****Alliance for Polyurethanes Industry**

For triethanolamine (and its salts):

**Acute toxicity:** Triethanolamine is of low toxicity by the oral, dermal and inhalation routes of exposure. Oral LD50 values have been shown to range from approximately 5-10 g/kg. The dermal LD50 is greater than 2 g/kg. The inhalation LC50 is greater than a saturated atmosphere

**Repeat Dose Toxicity:** The studies to determine toxicity of triethanolamine from repeated exposure were conducted for a duration of 91 days or 2 years. In both studies the NOAEL was at least 1000 mg/kg. There was no evidence of gross or histopathological change that could be attributed to treatment. Also, triethanolamine was shown to be non-carcinogenic.

**Genetic Toxicity:** Mutation (bacterial); This endpoint has been satisfied by two studies using 4 strains (TA 98, TA 100, TA 1535 and TA 1537) of *Salmonella typhimurium*. Triethanolamine was not mutagenic in any of the tester strains.

Chromosomal aberration (mammalian, *in vitro*) – This endpoint was satisfied by a cytogenetic assay using Chinese hamster lung cells. Triethanolamine did not induce chromosome aberrations in this test system.

**Reproductive Toxicity:** No studies have been conducted to specifically evaluate the effect of triethanolamine on reproductive performance. However, based on consideration of the repeat dose toxicity studies of at least 90 days duration, there were no

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abnormalities noted in the histopathological examination of reproductive organs. This fact, and the lack of effects on foetal development, allow the conclusion that triethanolamine would not be expected to produce adverse effects to reproductive performance and fertility.

**Developmental Toxicity:** This endpoint was satisfied using a developmental toxicity screening study according to the Chernoff-Kavlock method. Based on the results from this test, triethanolamine does not impair development of the fetus.

A Cosmetic Ingredient Review (CIR) expert panel conducted a review of triethanolamine-containing personal care products. The panel was concerned with the levels of free diethanolamine that could be present as an impurity in TEA or TEA-containing ingredients. The panel stated that the amount of free diethanolamine available must be limited to the present practices of use and concentration of diethanolamine.

The Panel concluded that TEA and 31 related TEA-containing ingredients, are safe when formulated to be nonirritating and when the levels of free diethanolamine do not exceed the prescribed levels. These ingredients should not be used in cosmetic products in which N-nitroso compounds can be formed.

Dermal carcinogenicity studies performed by the NTP on TEA reported equivocal evidence of carcinogenic activity in male mice based on the occurrence of liver hemangiosarcoma, some evidence of carcinogenic activity in female mice based on increased incidences of hepatocellular adenoma, and equivocal evidence of carcinogenic activity in male rats based on a marginal increase in the incidence of renal tubule cell adenoma. It has been hypothesized that TEA may cause liver tumours in mice via a choline-depletion mode of action. Humans are much less sensitive to this deficiency, and these hepatic findings are considered to have little relevance to humans regarding the safety of the use of TEA in personal care products.

The panel was concerned that the potential exists for dermal irritation with the use of products formulated using TEA or TEA-related ingredients. The panel specified that products containing these ingredients must be formulated to be nonirritating. Tertiary alkyl amines such as TEA do not react with N-nitrosating agents to directly form nitrosamines. However, tertiary amines can act as precursors in nitrosamine formation by undergoing nitrosative cleavage. The resultant secondary amine (ie, diethanolamine) can then be N-nitrosated to products that may be carcinogenic. Because of the potential for this process to occur, TEA and TEA-containing ingredients should not be used in cosmetic products in which N-nitroso compounds can be formed.

Safety Assessment of Triethanolamine and Triethanolamine-Containing Ingredients as Used in Cosmetics: International Journal of Toxicology (supplement 1) 59S-83S. 2013  
<https://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.901.4174&rep=rep1&type=pdf>

The substance is classified by IARC as Group 3:  
**NOT** classifiable as to its carcinogenicity to humans.  
 Evidence of carcinogenicity may be inadequate or limited in animal testing.

For alkyl polyamines:

The alkyl polyamines cluster consists of organic compounds containing two terminal primary amine groups and at least one secondary amine group. Typically these substances are derivatives of ethylenediamine, propylenediamine or hexanediamine. The molecular weight range for the entire cluster is relatively narrow, ranging from 103 to 232

Acute toxicity of the alkyl polyamines cluster is low to moderate via oral exposure and a moderate to high via dermal exposure. Cluster members have been shown to be eye irritants, skin irritants, and skin sensitizers in experimental animals. Repeated exposure in rats via the oral route indicates a range of toxicity from low to high hazard. Most cluster members gave positive results in tests for potential genotoxicity.

Limited carcinogenicity studies on several members of the cluster showed no evidence of carcinogenicity. Unlike aromatic amines, aliphatic amines are not expected to be potential carcinogens because they are not expected to undergo metabolic activation, nor would activated intermediates be stable enough to reach target macromolecules.

Polyamines potentiate NMDA induced whole-cell currents in cultured striatal neurons

Triethylenetetramine (TETA) is a severe irritant to skin and eyes and induces skin sensitisation.

TETA is of moderate acute toxicity: LD50(oral, rat) > 2000 mg/kg bw, LD50(dermal, rabbit) = 550 - 805 mg/kg bw. Acute exposure to saturated vapour via inhalation was tolerated without impairment. Exposure to aerosol leads to reversible irritations of the mucous membranes in the respiratory tract.

Following repeated oral dosing via drinking water only in mice but not in rats at concentration of 3000 ppm there were signs of impairment. The NOAEL is 600 ppm [92 mg/kg bw (oral, 90 days)]. Lifelong dermal application to mice (1.2 mg/mouse) did not result in tumour formation.

There are differing results of the genetic toxicity for TETA. The positive results of the in vitro tests may be the result of a direct genetic action as well as a result of an interference with essential metal ions. Due to this uncertainty of the in vitro tests, the genetic toxicity of TETA has to be assessed on the basis of in vivo tests.

The in vivo micronucleus tests (i.p. and oral) and the SLRL test showed negative results.

There are no human data on reproductive toxicity (fertility assessment). The analogue diethylenetriamine had no effects on reproduction. TETA shows developmental toxicity in animal studies if the chelating property of the substance is effective. The NOEL is 830 mg/kg bw (oral).

Experience with female patients suffering from Wilson's disease demonstrated that no miscarriages and no foetal abnormalities occur during treatment with TETA.

In rats, there are several studies concerning developmental toxicity. The oral treatment of rats with 75, 375 and 750 mg/kg resulted in no effects on dams and fetuses, except slight increased fetal body weight. After oral treatment of rats with 830 or 1670 mg/kg bw only in the highest dose group increased foetal abnormalities in 27/44 fetus (69,2 %) were recorded, when simultaneously the copper content of the feed was reduced. Copper supplementation in the feed reduced significant the foetal abnormalities of the highest dose group to 3/51 (6,5 % foetus). These findings suggest that the developmental toxicity is produced as a secondary consequence of the chelating properties of TETA.

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

**TRIETHYLENETETRAMINE****N-AMINOETHYLPIPERAZINE****PIPERAZINE**

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

for hexahydrate [RTECS No.: TM 0850000]

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

The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.

Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

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	<p>The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.</p>
<p><b>TRIETHYLENETETRAMINE, PROPOXYLATED &amp; BENZYL ALCOHOL &amp; TRIETHANOLAMINE &amp; TRIETHYLENETETRAMINE &amp; N-AMINOETHYLPIPERAZINE &amp; PIPERAZINE</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>TRIETHYLENETETRAMINE, PROPOXYLATED &amp; PIPERAZINE</b></p>	<p>Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.</p>
<p><b>TRIETHYLENETETRAMINE, PROPOXYLATED &amp; TRIETHANOLAMINE &amp; TRIETHYLENETETRAMINE &amp; N-AMINOETHYLPIPERAZINE &amp; PIPERAZINE</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>BENZYL ALCOHOL &amp; TRIETHANOLAMINE &amp; PIPERAZINE</b></p>	<p>The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.</p>
<p><b>TRIETHANOLAMINE &amp; TRIETHYLENETETRAMINE</b></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><b>TRIETHANOLAMINE &amp; PIPERAZINE</b></p>	<p><b>NOTE:</b> Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.</p>
<p><b>TRIETHYLENETETRAMINE &amp; N-AMINOETHYLPIPERAZINE &amp; PIPERAZINE</b></p>	<p>Handling ethyleneamine products is complicated by their tendency to react with other chemicals, such as carbon dioxide in the air, which results in the formation of solid carbamates. Because of their ability to produce chemical burns, skin rashes, and asthma-like symptoms, ethyleneamines also require substantial care in handling. Higher molecular weight ethyleneamines are often handled at elevated temperatures further increasing the possibility of vapor exposure to these compounds. Because of the fragility of eye tissue, almost any eye contact with any ethyleneamine may cause irreparable damage, even blindness. A single, short exposure to ethyleneamines, may cause severe skin burns, while a single, prolonged exposure may result in the material being absorbed through the skin in harmful amounts. Exposures have caused allergic skin reactions in some individuals. Single dose oral toxicity of ethyleneamines is low. The oral LD50 for rats is in the range of 1000 to 4500 mg/kg for the ethyleneamines. In general, the low-molecular weight polyamines have been positive in the Ames assay, increase sister chromatid exchange in Chinese hamster ovary (CHO) cells, and are positive for unscheduled DNA synthesis although they are negative in the mouse micronucleus assay. It is believed that the positive results are based on its ability to chelate copper</p>
<p><b>TRIETHYLENETETRAMINE &amp; N-AMINOETHYLPIPERAZINE</b></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. 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><b>N-AMINOETHYLPIPERAZINE &amp; PIPERAZINE</b></p>	<p>for piperazine: Exposure to piperazine and its salts has clearly been demonstrated to cause asthma in occupational settings. No NOAEL can be estimated for respiratory sensitisation (asthma). Although the LD50 levels indicate a relatively low level of oral acute toxicity (LD50 1-5 g/kg bw), signs of neurotoxicity may appear in humans after exposure to lower doses. Based on exposure levels of up to 3.4 mg/kg/day piperazine base and a LOAEL of 110 mg/kg, there is no concern for acute toxicity In pigs, piperazine is readily absorbed from the gastrointestinal tract, and the major part of the resorbed compound is excreted as unchanged piperazine during the first 48 hours. The principal route of excretion of piperazine and its metabolites is via urine, with a minor fraction recovered from faeces (16%). In humans the kinetics of the uptake and excretion of piperazine and its metabolites with urine appear to be roughly similar to that in the pig, and the nature and extent of conversion to metabolites has not been determined. Piperazine has demonstrated a low acute toxicity (LD50 = 1-5 g/kg bw) by the oral, dermal, and subcutaneous route of administration to rodents, whereas adequate inhalation toxicity data have not been found. However, there are findings of EEG (electroencephalogram) changes in 37% of 89 children administered 90-130 mg/kg piperazine (two doses during one day),</p>

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corroborated by a proposed GABA (gamma-aminobutyric acid) receptor agonism exerted by piperazine. Since clinical symptoms of neurotoxicity may occur after exposure to higher doses, a LOAEL of 110 mg/kg piperazine base for acute neurotoxicity in humans after acute exposure is proposed.

Piperazine, as concentrated aqueous solution, has strongly irritating properties with regard to skin, and should be regarded as corrosive with respect to the eye. Exposure to piperazine and its salts has been demonstrated to cause allergic dermatitis as well as respiratory sensitisation in humans. As shown by the LLNA, piperazine has a sensitising potential in animals. Although piperazine is clearly sensitising, no NOAEL can be set for this effect from the present database.

A NOAEL of 25 mg/kg/day of piperazine for liver toxicity in the beagle dog has been chosen after repeated exposure. A LOAEL of 30 mg/kg/day of piperazine for neurotoxicity is proposed based on documentation of (rare cases) of neurotoxicity from human clinical practice. Neurotoxicity also appears in other species (e.g., rabbits, dogs, cats, tigers, and horses), but not in rodents.

For reproductive effects of piperazine, there is a NOAEL of 125 mg/kg/day for effects on fertility, i.e., reduced pregnancy index, decreased number of implantation sites, and decreased litter sizes in rats. The teratogenic properties have been investigated in rats and rabbits in adequate studies. In rabbit, such effects may be elicited at a dose level that is also toxic to the dam. The LOAEL is 94 mg/kg/day, and the NOAEL 42 mg/kg/day piperazine base (maternal and embryotoxic). In the rat study, there were decreases in body weight of both dams and offspring at the top dose (2,100 mg/kg/day piperazine base), but there were no signs of any malformations.

The genotoxic properties have been investigated both *in vitro* (in the Ames test, in a nonstandard study on *Saccharomyces cerevisiae* and in Chinese hamster ovary cells) and *in vivo*, in a micronuclei assay on mice, all with negative results. There are no solid indications of a carcinogenic effect of piperazine, neither in animal studies, nor from the investigation on humans. In view of lack of genotoxic action, it appears unlikely that piperazine poses a carcinogenic risk.

There seems to be an additional cancer risk due to the formation of N-mononitrosopiperazine (NPZ) from piperazine. It is possible to calculate a hypothetical additional cancer risk posed by NPZ after exposure to piperazine, but the calculation would depend on several assumptions. We conclude that there seems to be an additional cancer risk due to the formation of NPZ from piperazine, and although it is difficult to estimate, it is probably small.

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

QuakeBond J300SR Saturating Resin (Part B)	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
triethylenetetramine, propoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
benzyl alcohol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	230mg/l	2
	EC50	72h	Algae or other aquatic plants	500mg/l	2
	NOEC(ECx)	336h	Fish	5.1mg/l	2
	EC50	96h	Algae or other aquatic plants	76.828mg/l	2
LC50	96h	Fish	10mg/l	2	
triethanolamine	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	565.2-658.3mg/l	4
	BCF	1008h	Fish	<0.4	7
	EC50	72h	Algae or other aquatic plants	>107<260mg/l	2
	NOEC(ECx)	Not Available	Fish	>1mg/l	2
	EC50	96h	Algae or other aquatic plants	169mg/l	1
LC50	96h	Fish	11800mg/l	2	
triethylenetetramine	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<0.5	7

Continued...

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	EC50	48h	Crustacea	31.1mg/l	1
	EC50	72h	Algae or other aquatic plants	2.5mg/l	1
	ErC50	72h	Algae or other aquatic plants	2.5mg/l	1
	EC50	96h	Algae or other aquatic plants	3.7mg/L	4
	EC10(ECx)	72h	Algae or other aquatic plants	0.67mg/l	1
	LC50	96h	Fish	180mg/l	1
<b>N-aminoethylpiperazine</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	32mg/l	1
	EC50	72h	Algae or other aquatic plants	495mg/l	1
	NOEC(ECx)	48h	Crustacea	18mg/l	1
	LC50	96h	Fish	>100mg/l	2
<b>piperazine</b>	<b>Endpoint</b>	<b>Test Duration (hr)</b>	<b>Species</b>	<b>Value</b>	<b>Source</b>
	EC50	48h	Crustacea	21mg/l	2
	BCF	1008h	Fish	<0.3-0.9	7
	EC50	72h	Algae or other aquatic plants	153.1mg/l	2
	NOEC(ECx)	Not Available	Fish	>1mg/l	2
	LC50	96h	Fish	>100mg/l	2
<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				

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

Prevent, by any means available, spillage from entering drains or water courses.

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

#### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
benzyl alcohol	LOW	LOW
triethanolamine	LOW	LOW
triethylenetetramine	LOW	LOW
N-aminoethylpiperazine	HIGH	HIGH
piperazine	LOW	LOW

#### Bioaccumulative potential

Ingredient	Bioaccumulation
triethylenetetramine, propoxylated	LOW (LogKOW = -1.95)
benzyl alcohol	LOW (LogKOW = 1.1)
triethanolamine	LOW (BCF = 3.9)
triethylenetetramine	LOW (BCF = 5)
N-aminoethylpiperazine	LOW (LogKOW = -1.57)
piperazine	LOW (BCF = 3.9)

#### Mobility in soil

Ingredient	Mobility
benzyl alcohol	LOW (Log KOC = 15.66)
triethanolamine	LOW (Log KOC = 10)
triethylenetetramine	LOW (Log KOC = 309.9)
N-aminoethylpiperazine	LOW (Log KOC = 171.7)
piperazine	LOW (Log KOC = 52.71)

## SECTION 13 Disposal considerations

### Waste treatment methods



Continued...

### QuakeBond J300SR Saturating Resin (Part B)

<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> <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>▶ Treat and neutralise at an approved treatment plant.</li> <li>▶ Treatment should involve: Neutralisation with suitable dilute acid followed 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> <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> </ul>
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## SECTION 14 Transport information

### Labels Required

	
<b>Marine Pollutant</b>	
<b>HAZCHEM</b>	2X

### Land transport (ADG)

14.1. UN number or ID number	1760	
14.2. UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains triethylenetetramine and triethanolamine)	
14.3. Transport hazard class(es)	Class	8
	Subsidiary Hazard	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	274
	Limited quantity	1 L

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

14.1. UN number	1760	
14.2. UN proper shipping name	Corrosive liquid, n.o.s. * (contains triethylenetetramine and triethanolamine)	
14.3. Transport hazard class(es)	ICAO/IATA Class	8
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	8L
14.4. Packing group	II	
14.5. Environmental hazard	Environmentally hazardous	
14.6. Special precautions for user	Special provisions	A3 A803
	Cargo Only Packing Instructions	855
	Cargo Only Maximum Qty / Pack	30 L
	Passenger and Cargo Packing Instructions	851

Continued...

### QuakeBond J300SR Saturating Resin (Part B)

Passenger and Cargo Maximum Qty / Pack	1 L
Passenger and Cargo Limited Quantity Packing Instructions	Y840
Passenger and Cargo Limited Maximum Qty / Pack	0.5 L

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

14.1. UN number	1760	
14.2. UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains triethylenetetramine and triethanolamine)	
14.3. Transport hazard class(es)	IMDG Class	8
	IMDG Subsidiary Hazard	Not Applicable
14.4. Packing group	II	
14.5. Environmental hazard	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-A, S-B
	Special provisions	274
	Limited Quantities	1 L

#### 14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

##### 14.7.2. Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
triethylenetetramine, propoxylated	Not Available
benzyl alcohol	Not Available
triethanolamine	Not Available
triethylenetetramine	Not Available
N-aminoethylpiperazine	Not Available
piperazine	Not Available

##### 14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
triethylenetetramine, propoxylated	Not Available
benzyl alcohol	Not Available
triethanolamine	Not Available
triethylenetetramine	Not Available
N-aminoethylpiperazine	Not Available
piperazine	Not Available

## SECTION 15 Regulatory information

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

triethylenetetramine, propoxylated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

benzyl alcohol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

triethanolamine 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 4

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Continued...

## QuakeBond J300SR Saturating Resin (Part B)

Australian Inventory of Industrial Chemicals (AIIC)

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

### triethylenetetramine 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 4

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)

### N-aminoethylpiperazine 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 5

Australian Inventory of Industrial Chemicals (AIIC)

### piperazine 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 2

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)

## Additional Regulatory Information

Not Applicable

## National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (benzyl alcohol; triethanolamine; triethylenetetramine; N-aminoethylpiperazine; piperazine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (triethylenetetramine, propoxylated)
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (triethylenetetramine, propoxylated)
Vietnam - NCI	Yes
Russia - FBEPH	No (triethylenetetramine, propoxylated)
<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

Revision Date	23/07/2025
Initial Date	23/07/2025

## 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

Continued...

- ▶ 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
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration
- ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
- ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
- ▶ IGC: International Gas Carrier Code
- ▶ IBC: International Bulk Chemical Code
  
- ▶ AIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European INventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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