

Hychem GP Epoxy Hardener

Hychem International

Chemwatch: 21-1144

Version No: 2.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: 01/01/2013

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Initial Date: Not Available

L.GHS.AUS.EN

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

Product Identifier

Product name	Hychem GP Epoxy Hardener
Synonyms	GP Epoxy Hardener
Proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains isophorone diamine)
Other means of identification	Not Available

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

Relevant identified uses	The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. Use according to manufacturer's directions.
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Details of the supplier of the safety data sheet

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

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	1800 039 008
Other emergency telephone numbers	Not Available

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	+612 9186 1132	Not Available

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

Continued...

Hychem GP Epoxy Hardener

	Min	Max
Flammability	1	
Toxicity	2	
Body Contact	3	
Reactivity	2	
Chronic	2	

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

Poisons Schedule	S5
Classification [1]	Metal Corrosion Category 1, Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Skin Corrosion/Irritation Category 1B, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Acute Aquatic Hazard Category 3, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	
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SIGNAL WORD	DANGER
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Hazard statement(s)

H290	May be corrosive to metals.
H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H332	Harmful if inhaled.
H314	Causes severe skin burns and eye damage.
H318	Causes serious eye damage.
H317	May cause an allergic skin reaction.
H336	May cause drowsiness or dizziness.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P234	Keep only in original container.
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.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER or doctor/physician.
P363	Wash contaminated clothing before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P390	Absorb spillage to prevent material damage.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

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

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
2855-13-2	>60	<u>isophorone diamine</u>
100-51-6	10-30	<u>benzyl alcohol</u>
112-57-2	<10	<u>tetraethylenepentamine</u>

SECTION 4 FIRST AID MEASURES**Description of first aid measures**

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor. ▶ Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. ▶ Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs). ▶ 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. ▶ Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered. <p>This must definitely be left to a doctor or person authorised by him/her. (ICSC13719)</p>
Ingestion	<ul style="list-style-type: none"> ▶ For advice, contact a Poisons Information Centre or a doctor at once. ▶ Urgent hospital treatment is likely to be needed. ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Transport to hospital or doctor without delay.

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.

Hychem GP Epoxy Hardener

- ▶ 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.

* 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]

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.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use fire fighting procedures suitable for surrounding area. ▶ Do not approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include; carbon dioxide (CO₂) aldehydes, nitrogen oxides (NO_x) other pyrolysis products typical of burning organic material Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions. May emit corrosive fumes. WARNING: Long standing in contact with air and light may result in the formation of potentially explosive peroxides.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	<ul style="list-style-type: none"> ▶ Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material. ▶ Check regularly for spills and leaks. <p>Small spills should be covered with inorganic absorbents and disposed of properly. Organic absorbents have been known to</p>
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Hychem GP Epoxy Hardener

	<p>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> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Consider evacuation (or protect in place). ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<p>Contains low boiling substance:</p> <p>Storage in sealed containers may result in pressure buildup causing violent rupture of containers not rated appropriately.</p> <ul style="list-style-type: none"> ▶ Check for bulging containers. ▶ Vent periodically ▶ Always release caps or seals slowly to ensure slow dissipation of vapours ▶ DO NOT USE brass or copper containers / stirrers ▶ DO NOT allow clothing wet with material to stay in contact with skin <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"> ▶ 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. ▶ 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. ▶ Unopened containers received from the supplier should be safe to store for 18 months. ▶ Opened containers should not be stored for more than 12 months. ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Avoid contact with moisture. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<p>for bulk storages:</p> <ul style="list-style-type: none"> ▶ If slight coloration of the ethyleneamine is acceptable, storage tanks may be made of carbon steel or black iron, provided they are free of rust and mill scale. However, if the amine is stored in such tanks, color may develop due to iron contamination. If iron contamination cannot be tolerated, tanks constructed of types 304 or 316 stainless steel should be used. (Note: Because they are quickly corroded by amines, do not use copper, copper alloys, brass, or bronze in tanks or lines.) ▶ This product should be stored under a dry inert gas blanket, such as nitrogen, to minimize contamination resulting from contact with air and water

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Hychem GP Epoxy Hardener

- ▶ Store in original containers.
- ▶ Keep containers securely sealed.
- ▶ Store in a cool, dry, well-ventilated area.
- ▶ Store away from incompatible materials and foodstuff containers.
- ▶ Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- ▶ **DO NOT store near acids, or oxidising agents**
- ▶ No smoking, naked lights, heat or ignition sources.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Glass container is suitable for laboratory quantities ▶ DO NOT use aluminium or galvanised containers ▶ Lined metal can, lined metal pail/ can. ▶ Plastic pail. ▶ Polyliner drum. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks. <p>For low viscosity materials</p> <ul style="list-style-type: none"> ▶ Drums and jerricans must be of the non-removable head type. ▶ Where a can is to be used as an inner package, the can must have a screwed enclosure. <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"> ▶ Removable head packaging; ▶ Cans with friction closures and ▶ low pressure tubes and cartridges <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>
Storage incompatibility	<p>Benzyl alcohol:</p> <ul style="list-style-type: none"> ▶ may froth in contact with water ▶ slowly oxidises in air, oxygen forming benzaldehyde ▶ is incompatible with mineral acids, caustics, aliphatic amines, isocyanates ▶ reacts violently with strong oxidisers, and explosively with sulfuric acid at elevated temperatures ▶ corrodes aluminium at high temperatures ▶ is incompatible with aluminum, iron, steel ▶ attacks some nonfluorinated plastics; may attack, extract and dissolve polypropylene <p>Benzyl alcohol contaminated with 1.4% hydrogen bromide and 1.2% of dissolved iron(II) polymerises exothermically above 100 deg. C.</p> <ul style="list-style-type: none"> ▶ Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air. ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ▶ Avoid contact with copper, aluminium and their alloys. ▶ Avoid reaction with oxidising agents

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
benzyl alcohol	Benzyl alcohol	30 ppm	49 ppm	49 ppm
tetraethylenepentamine	Tetraethylenepentamine	15 mg/m ³	130 mg/m ³	790 mg/m ³

Ingredient	Original IDLH	Revised IDLH
isophorone diamine	Not Available	Not Available
benzyl alcohol	Not Available	Not Available
tetraethylenepentamine	Not Available	Not Available

MATERIAL DATA

Exposure controls

Continued...

Hychem GP Epoxy Hardener

	<p>CARE: Use of a quantity of this material in confined space or poorly ventilated area, where rapid build up of concentrated atmosphere may occur, could require increased ventilation and/or protective gear</p> <p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed storage areas. 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" data-bbox="363 698 1487 1025"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing 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, welding, spray drift, plating acid fumes, pickling (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, spray painting in shallow booths, 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> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion)</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" data-bbox="363 1088 1487 1276"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	Type of Contaminant:	Air Speed:	solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion)	2.5-10 m/s (500-2000 f/min.)	Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion	4: Small hood-local control only
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<p>Personal protection</p>																					
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ Chemical goggles. ▶ Full face shield may be required for supplementary but never for primary protection of eyes. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 																				
<p>Skin protection</p>	<p>See Hand protection below</p>																				
<p>Hands/feet protection</p>	<ul style="list-style-type: none"> ▶ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove</p>																				

Hychem GP Epoxy Hardener

	<p>material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> ▶ frequency and duration of contact, ▶ chemical resistance of glove material, ▶ glove thickness and ▶ dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> ▶ When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ▶ When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. ▶ Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. ▶ Contaminated gloves should be replaced. <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <ul style="list-style-type: none"> ▶ Leather wear not recommended: Contaminated leather footwear, watch bands, should be destroyed, i.e. burnt, as they cannot be adequately decontaminated ▶ When handling liquid-grade epoxy resins wear chemically protective gloves (e.g nitrile or nitrile-butadiene rubber), boots and aprons. ▶ DO NOT use cotton or leather (which absorb and concentrate the resin), polyvinyl chloride, rubber 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.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ PVC Apron. ▶ PVC protective suit may be required if exposure severe. ▶ Eyewash unit. ▶ Ensure there is ready access to a safety shower.
Thermal hazards	Not Available

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:

Hychem GP Epoxy Hardener

Material	CPI
BUTYL	A
VITON	A

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Respiratory protection

Type 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, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Clear yellowish liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.0

Continued...

Hychem GP Epoxy Hardener

Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	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)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>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.</p> <p>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.</p> <p>Inhalation of amine vapours may cause irritation of the mucous membranes of the nose and throat and lung irritation with respiratory distress and cough. Single exposures to near lethal concentrations and repeated exposures to sublethal concentrations produces tracheitis, bronchitis, pneumonitis and pulmonary oedema. Aliphatic and alicyclic amines are generally well absorbed from the respiratory tract. Systemic effects include headache, nausea, faintness and anxiety. These effects are thought to be transient and are probably related to the pharmacodynamic action of the amines. Histamine release by aliphatic amines may produce bronchoconstriction and wheezing.</p> <p>Inhalation of benzyl alcohol may affect respiration (paralysis of the respiratory center, respiratory depression, gasping respirations), cardiovascular system (hypotension</p>
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Hychem GP Epoxy Hardener

Ingestion	<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 chemical burns within the oral cavity and gastrointestinal tract following ingestion.</p> <p>Ingestion of amine epoxy-curing agents (hardeners) may cause severe abdominal pain, nausea, vomiting or diarrhoea. The vomitus may contain blood and mucous. If death does not occur within 24 hours there may be an improvement in the patients condition for 2-4 days only to be followed by the sudden onset of abdominal pain, board-like abdominal rigidity or hypo-tension; this indicates that delayed gastric or oesophageal corrosive damage has occurred.</p> <p>Aliphatic and alicyclic amines are generally well absorbed from the gut. Corrosive action may cause tissue damage throughout the gastrointestinal tract. Detoxification is thought to occur in the liver, kidney and intestinal mucosa with the enzymes, monoamine oxidase and diamine oxidase (histaminase) having a significant role.</p> <p>Ingestion of large doses of benzyl alcohol may cause abdominal pain, nausea, vomiting, diarrhea. It may affect behavior/central nervous system and cause headache, somnolence, excitement, dizziness, ataxia, coma, convulsions, and other symptoms of central nervous system depression.</p> <p>Exposure to excessive amounts of benzyl alcohol has been associated with toxicity (hypotension, metabolic acidosis), particularly in neonates, and an increased incidence of kernicterus (a neurological condition that occurs in severe jaundice), particularly in small preterm infants. There have been rare reports of deaths, primarily in preterm infants, associated with exposure to excessive amounts of benzyl alcohol. The amount of benzyl alcohol from medications is usually considered negligible compared to that received in flush solutions containing benzyl alcohol. Administration of high dosages of medications containing this preservative must take into account the total amount of benzyl alcohol administered. The amount of benzyl alcohol at which toxicity may occur is not known. If the patient requires more than the recommended dosages or other medications containing this preservative, the practitioner must consider the daily metabolic load of benzyl alcohol from these combined sources.</p>
Skin Contact	<p>Skin contact with the material may be harmful; systemic effects may result following absorption.</p> <p>The material can produce chemical burns following direct contact with the skin.</p> <p>Amine epoxy-curing agents (hardeners) may produce primary skin irritation and sensitisation dermatitis in predisposed individuals. Cutaneous reactions include erythema, intolerable itching and severe facial swelling. Blistering, with weeping of serious fluid, and crusting and scaling may also occur.</p> <p>Virtually all of the liquid amine curing agents can cause sensitisation or allergic skin reactions.</p> <p>Individuals exhibiting "amine dermatitis" may experience a dramatic reaction upon re-exposure to minute quantities. Highly sensitive persons may even react to cured resins containing trace amounts of unreacted amine hardener. Minute quantities of air-borne amine may precipitate intense dermatological symptoms in sensitive individuals. Prolonged or repeated exposure may produce tissue necrosis.</p> <p>NOTE: Susceptibility to this sensitisation will vary from person to person. Also, allergic dermatitis may not appear until after several days or weeks of contact. However, once sensitisation has occurred, exposure of the skin to even very small amounts of the material may cause erythema (redness) and oedema (swelling) at the site. Thus, all skin contact with any epoxy curing agent should be avoided.</p> <p>Volatile amine vapours produce primary skin irritation and dermatitis. Direct local contact, with the lower molecular weight liquids, may produce skin burns. Percutaneous absorption of simple aliphatic amines is known to produce lethal effects often the same as that for oral administration. Cutaneous sensitisation has been recorded chiefly due to ethyleneamines.</p> <p>Histamine release following exposure to many aliphatic amines may result in "triple response" (white vasoconstriction, red flare and wheal) in human skin.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. 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>Vapours of volatile amines cause eye irritation with lachrymation, conjunctivitis and minor transient corneal oedema which results in "halos" around lights (glauropsia, "blue haze", or "blue-grey haze"). Vision may become misty and halos may appear several hours after workers are exposed to the substance</p> <p>This effect generally disappears spontaneously within a few hours of the end of exposure, and does not produce physiological after-effects. However oedema of the corneal epithelium, which is primarily responsible for vision disturbances, may take more than one or more days to clear, depending on the severity of exposure. Photophobia and discomfort from the roughness of the corneal surface also may occur after greater exposures.</p> <p>Although no detriment to the eye occurs as such, glauropsia predisposes an affected individual to physical accidents and reduces the ability to undertake skilled tasks such as driving a vehicle.</p> <p>Direct local contact with the liquid may produce eye damage which may be permanent in the case of the lower molecular weight species.</p> <p>The vapour when concentrated has pronounced eye irritation effects and this gives some warning of high vapour concentrations. If eye irritation occurs seek to reduce exposure with available control measures, or evacuate area.</p>
Chronic	<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 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>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body 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>

Hychem GP Epoxy Hardener

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

Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.

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.

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 out 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.

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.

Hychem GP Epoxy Hardener	TOXICITY	IRRITATION
	Not Available	Not Available
isophorone diamine	TOXICITY	IRRITATION
	Oral (rat) LD50: 1030 mg/kg ^[2]	[Manufacturer HUE]
benzyl alcohol	TOXICITY	IRRITATION
	dermal (rat) LD50: 1000000 ppm/90M ^[2]	Eye (rabbit): 0.75 mg open SEVERE
	Inhalation (rat) LC50: >4.178 mg/L/4h ^[2]	Skin (man): 16 mg/48h-mild
	Oral (rat) LD50: 1560 mg/kg ^[2]	Skin (rabbit):10 mg/24h open-mild
tetraethylenepentamine	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 660 mg/kgE ^[2]	Eye (rabbit): 100 mg/24h moderate
	Oral (rat) LD50: 3990 mg/kgE ^[2]	Eye (rabbit): 5 mg moderate
		Skin (rabbit): 495 mg SEVERE
		Skin (rabbit): 5 mg/24h SEVERE

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

Hychem GP Epoxy Hardener

Hychem GP Epoxy
Hardener

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.

For isophorone diamine

Based on a limited skin irritation study with rabbits and rats, isophorone diamine is deemed to be a strong irritant (duration of the exposure not reported) and corrosive after repeated application. Isophorone diamine is corrosive to the eyes of rabbits when tested according to OECD TG 405. Isophorone diamine was found to induce dermal sensitisation when tested according to OECD TG 406 in guinea pigs. From a number of publications there is evidence that frequent occupational exposure to isophorone diamine may lead to the development of allergic contact dermatitis in humans. No definite conclusion can be currently drawn on respiratory sensitisation.

From two 14-day inhalative exposure studies with rats no NOAEL could be determined. At the first study's LOAEL of 18 mg/m³, degeneration/necrosis in the olfactory epithelium of the nose were observed. Trachea, larynx and lungs were affected at 200 mg/m³ and above (degeneration/necrosis, hyperplasia, squamous metaplasia). At the LOAEL of the follow-up study, i.e. at 2.2 mg/m³, reversible minimal to mild degeneration of respiratory nasal mucosa in the anterior dorsal nose was observed. In a subchronic drinking water study according to OECD TG 408, the administration of 150 mg/kg bw/day led to reduced absolute and relative kidney weights in male and female rats (histopathology being indicative for tubular nephrosis), while 59 mg/kg bw/day (males) and 62 mg/kg bw/day (females) were determined as a NOAEL.

Isophorone diamine was not mutagenic in bacteria and mammalian cell systems *in vitro* (Ames test according to Directive 84/449/EEC B.14 (1984) and HPRT test according to OECD TG 476 (1984)). It did not induce chromosomal aberrations in CHO cells *in vitro* in a test performed in accordance with OECD TG 473. *In vivo* mouse micronucleus tests (one performed according to OECD TG 474 (1983) for the induction of micronucleated polychromatic erythrocytes were clearly negative. From all *in vitro* and *in vivo* tests performed there is no evidence that isophorone diamine has a mutagenic or clastogenic potential.

No studies have been performed on the toxicity of isophorone diamine to reproduction.

Data from an oral 90-day study in rats according to OECD TG 408 did not reveal any adverse effects on the male and female reproductive organs.

Isophorone diamine did not show any teratogenic or embryofetotoxic effects in a gavage study with rats performed in accordance with OECD TG 414 (2001) up to and including the highest tested dose level of 250 mg/kg bw/day. The NOAEL for maternal toxicity was 50 mg/kg bw/day, effects at 250 mg/kg bw/day were reduced food consumption and reduced body weight gain. The NOAEL for developmental toxicity is 250 mg/kg bw/day.

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

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

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

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).

Hychem GP Epoxy Hardener

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.

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

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.

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.

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 sensitisers 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

Tetraethylenepentamine (TEPA) has a low acute toxicity when administered orally to rats (LD50 =3250 mg/kg). In an

Hychem GP Epoxy Hardener

acute inhalation toxicity study with saturated vapor and whole body exposure, the LC50 was calculated to be >9.9 ppm (highest dose tested). TEPA is corrosive to the skin and eyes of rabbits. TEPA is a skin sensitiser in the guinea pig. Dermal acute toxicity LD50 values in the rabbit range from 660 - 1260 mg/kg. The higher toxicity via the dermal route is most likely due to the corrosive nature of TEPA to the skin whereas TEPA would be neutralized by stomach acid. The results of a 28-day repeated dose dermal toxicity study of TEPA indicated a systemic toxicity NOEL of 200 mg/kg/day and a dermal toxicity NOEL (local) of 50 mg/kg/day. The dermal LOAEL was 100 mg/kg/day. In addition, in a repeat dose study of TETA administered in drinking water to male and female rats for 90-92 days, the NOEL was 276 mg/kg/day in males and 352 mg/kg/day in females, the highest dose administered with the NIH-31 diet (several diets were used to study the effects of copper deficiency versus toxicity directly to TEPA). In this same study in mice the NOEL was 487 mg/kg/day in males and 551 mg/kg/day in females, the highest dose administered. A lifetime study was conducted via dermal administration in fifty male mice with a solution of 35% TEPA. There were 20 cases of hyperkeratosis, 13 cases of epidermal necrosis and no evidence of dermal hyperplasia. There were no data available for TEPA for reproductive and developmental toxicity. As a result, data on triethylenetetramine (TETA) was used to address these endpoints. TETA data showed no effects on reproductive organs in rats up to 276 mg/kg/day (males) and 352 mg/kg/day (females) and in mice (up to 500 mg/kg/day) when administered in drinking water. TETA was not considered a developmental toxicant via dermal administration in rabbits at maternally toxic doses up to 125 mg/kg/day but showed developmental toxicity in rats at maternally toxic doses of 830 or 1660 mg/kg/day via drinking water. The maternal and foetal toxicity was most likely due to copper deficiency and zinc toxicity at these levels. Subsequent studies where the diet was supplemented with copper resulted in a decrease of foetal abnormalities. There were no standard fertility studies available. However, there were no effects on the gonads observed in a 90-day drinking water study in rats and mice as described above. In the Ames Salmonella assay, TEPA was found to be positive both with and without metabolic activation. TEPA was found to increase sister chromatid exchange in CHO cells and was considered positive in a UDS assay using rat hepatocytes. TEPA was not considered genotoxic in the mouse micronucleus assay and had equivocal results in the two dominant lethal assays in *Drosophila melanogaster*. Again, it is believed that the positive results are based upon TEPA's ability to chelate copper.

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.

For isophorone diamine

Based on a limited skin irritation study with rabbits and rats, isophorone diamine is deemed to be a strong irritant (duration of the exposure not reported) and corrosive after repeated application. Isophorone diamine is corrosive to the eyes of rabbits when tested according to OECD TG 405. Isophorone diamine was found to induce dermal sensitisation when tested according to OECD TG 406 in guinea pigs. From a number of publications there is evidence that frequent occupational exposure to isophorone diamine may lead to the development of allergic contact dermatitis in humans. No definite conclusion can be currently drawn on respiratory sensitisation.

From two 14-day inhalative exposure studies with rats no NOAEL could be determined. At the first study's LOAEL of 18 mg/m³, degeneration/necrosis in the olfactory epithelium of the nose were observed. Trachea, larynx and lungs were affected at 200 mg/m³ and above (degeneration/necrosis, hyperplasia, squamous metaplasia). At the LOAEL of the follow-up study, i.e. at 2.2 mg/m³, reversible minimal to mild degeneration of respiratory nasal mucosa in the anterior dorsal nose was observed. In a subchronic drinking water study according to OECD TG 408, the administration of 150 mg/kg bw/day led to reduced absolute and relative kidney weights in male and female rats (histopathology being indicative for tubular nephrosis), while 59 mg/kg bw/day (males) and 62 mg/kg bw/day (females) were determined as a NOAEL.

Isophorone diamine was not mutagenic in bacteria and mammalian cell systems *in vitro* (Ames test according to Directive 84/449/EEC B.14 (1984) and HPRT test according to OECD TG 476 (1984)). It did not induce chromosomal aberrations in CHO cells *in vitro* in a test performed in accordance with OECD TG 473. *In vivo* mouse micronucleus tests (one performed according to OECD TG 474 (1983) for the induction of micronucleated polychromatic erythrocytes were clearly negative. From all *in vitro* and *in vivo* tests performed there is no evidence that isophorone diamine has a mutagenic or clastogenic potential.

No studies have been performed on the toxicity of isophorone diamine to reproduction.

Data from an oral 90-day study in rats according to OECD TG 408 did not reveal any adverse effects on the male and female reproductive organs.

Isophorone diamine did not show any teratogenic or embryofoetotoxic effects in a gavage study with rats performed in accordance with OECD TG 414 (2001) up to and including the highest tested dose level of 250 mg/kg bw/day. The NOAEL for maternal toxicity was 50 mg/kg bw/day, effects at 250 mg/kg bw/day were reduced food consumption and reduced body weight gain. The NOAEL for developmental toxicity is 250 mg/kg bw/day.

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

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic

ISOPHORONE DIAMINE

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inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. 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).

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.

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

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.

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.

Developmental toxicity: In rats for sodium benzoate dosed via food during the entire gestation developmental effects

BENZYL ALCOHOL

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occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOEL = 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: NOEL= 550 mg/kg bw (gavage; CD-1 mice). LOEL = 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.

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 conjugal contact dermatitis occur.

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.

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

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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 sensitisers has not been studied in detail. Skin sensitising prohaptens 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 prohaptens) 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 prohaptens. 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.

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

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.

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,

TETRAETHYLENEPENTAMINE

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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

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

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.

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 sensitisers 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

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

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 significantly the fetal 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.

Tetraethylenepentamine (TEPA) has a low acute toxicity when administered orally to rats (LD50 = 3250 mg/kg). In an acute inhalation toxicity study with saturated vapor and whole body exposure, the LC50 was calculated to be >9.9 ppm (highest dose tested). TEPA is corrosive to the skin and eyes of rabbits. TEPA is a skin sensitiser in the guinea pig. Dermal acute toxicity LD50 values in the rabbit range from 660 - 1260 mg/kg. The higher toxicity via the dermal route is most likely due to the corrosive nature of TEPA to the skin whereas TEPA would be neutralized by stomach acid.

The results of a 28-day repeated dose dermal toxicity study of TEPA indicated a systemic toxicity NOEL of 200 mg/kg/day and a dermal toxicity NOEL (local) of 50 mg/kg/day. The dermal LOAEL was 100 mg/kg/day. In addition, in a repeat dose study of TETA administered in drinking water to male and female rats for 90-92 days, the NOEL was 276 mg/kg/day in males and 352 mg/kg/day in females, the highest dose administered with the NIH-31 diet (several diets

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were used to study the effects of copper deficiency versus toxicity directly to TEPA). In this same study in mice the NOEL was 487 mg/kg/day in males and 551 mg/kg/day in females, the highest dose administered. A lifetime study was conducted via dermal administration in fifty male mice with a solution of 35% TEPA. There were 20 cases of hyperkeratosis, 13 cases of epidermal necrosis and no evidence of dermal hyperplasia.

There were no data available for TEPA for reproductive and developmental toxicity. As a result, data on triethylenetetramine (TETA) was used to address these endpoints. TETA data showed no effects on reproductive organs in rats up to 276 mg/kg/day (males) and 352 mg/kg/day (females) and in mice (up to 500 mg/kg/day) when administered in drinking water. TETA was not considered a developmental toxicant via dermal administration in rabbits at maternally toxic doses up to 125 mg/kg/day but showed developmental toxicity in rats at maternally toxic doses of 830 or 1660 mg/kg/day via drinking water. The maternal and foetal toxicity was most likely due to copper deficiency and zinc toxicity at these levels. Subsequent studies where the diet was supplemented with copper resulted in a decrease of foetal abnormalities. There were no standard fertility studies available. However, there were no effects on the gonads observed in a 90-day drinking water study in rats and mice as described above.

In the Ames Salmonella assay, TEPA was found to be positive both with and without metabolic activation. TEPA was found to increase sister chromatid exchange in CHO cells and was considered positive in a UDS assay using rat hepatocytes. TEPA was not considered genotoxic in the mouse micronucleus assay and had equivocal results in the two dominant lethal assays in *Drosophila melanogaster*. Again, it is believed that the positive results are based upon TEPA's ability to chelate copper.

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

Legend: ✗ – Data available but does not fill the criteria for classification
 ✓ – Data required to make classification available
 ⊘ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
isophorone diamine	EC50	96	Algae or other aquatic plants	7.221mg/L	3
isophorone diamine	LC50	96	Fish	54.352mg/L	3
isophorone diamine	EC50	48	Crustacea	17.4mg/L	4
isophorone diamine	EC10	72	Algae or other aquatic plants	=3.1mg/L	1
isophorone diamine	NOEC	72	Algae or other aquatic plants	1.5mg/L	2
benzyl alcohol	EC03	168	Algae or other aquatic plants	=16mg/L	4
benzyl alcohol	LC50	96	Fish	10mg/L	4
benzyl alcohol	NOEC	336	Fish	5.1mg/L	2
benzyl alcohol	EC50	48	Crustacea	230mg/L	2
benzyl alcohol	EC50	72	Algae or other aquatic plants	500mg/L	2
tetraethylenepentamine	EC50	48	Crustacea	=24.1mg/L	1
tetraethylenepentamine	LC50	96	Fish	1.27628mg/L	3
tetraethylenepentamine	EC50	72	Algae or other aquatic plants	=2.1mg/L	1
tetraethylenepentamine	EC50	72	Algae or other aquatic plants	=6.8mg/L	1
tetraethylenepentamine	NOEC	72	Algae or other aquatic plants	=0.5mg/L	1

Legend:

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

Harmful 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
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Continued...

Hychem GP Epoxy Hardener

isophorone diamine	HIGH	HIGH
benzyl alcohol	LOW	LOW
tetraethylenepentamine	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
isophorone diamine	LOW (BCF = 3.4)
benzyl alcohol	LOW (LogKOW = 1.1)
tetraethylenepentamine	LOW (LogKOW = -3.1604)

Mobility in soil

Ingredient	Mobility
isophorone diamine	LOW (KOC = 340.4)
benzyl alcohol	LOW (KOC = 15.66)
tetraethylenepentamine	LOW (KOC = 1098)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

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

Labels Required

	
Marine Pollutant	NO
HAZCHEM	2X

Land transport (ADG)

Hychem GP Epoxy Hardener

UN number	2735
Packing group	III
UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains isophorone diamine)
Environmental hazard	Not Applicable
Transport hazard class(es)	Class : 8 Subrisk : Not Applicable
Special precautions for user	Special provisions : 223 274 Limited quantity : 5 L

Air transport (ICAO-IATA / DGR)

UN number	2735
Packing group	III
UN proper shipping name	Amines, liquid, corrosive, n.o.s. *; Polyamines, liquid, corrosive, n.o.s. * (contains isophorone diamine)
Environmental hazard	Not Applicable
Transport hazard class(es)	ICAO/IATA Class : 8 ICAO / IATA Subrisk : Not Applicable ERG Code : 8L
Special precautions for user	Special provisions : A3A803 Cargo Only Packing Instructions : 856 Cargo Only Maximum Qty / Pack : 60 L Passenger and Cargo Packing Instructions : 852 Passenger and Cargo Maximum Qty / Pack : 5 L Passenger and Cargo Limited Quantity Packing Instructions : Y841 Passenger and Cargo Limited Maximum Qty / Pack : 1 L

Sea transport (IMDG-Code / GGVSee)

UN number	2735
Packing group	III
UN proper shipping name	AMINES, LIQUID, CORROSIVE, N.O.S. or POLYAMINES, LIQUID, CORROSIVE, N.O.S. (contains isophorone diamine)
Environmental hazard	Not Applicable
Transport hazard class(es)	IMDG Class : 8 IMDG Subrisk : Not Applicable
Special precautions for user	EMS Number : F-A, S-B Special provisions : 223 274 Limited Quantities : 5 L

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

ISOPHORONE DIAMINE(2855-13-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

BENZYL ALCOHOL(100-51-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

TETRAETHYLENEPENTAMINE(112-57-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (benzyl alcohol; tetraethylenepentamine; isophorone diamine)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Y
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION**Other information**

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

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

www.chemwatch.net

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average

PC—STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit.

IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value

LOD: Limit Of Detection

OTV: Odour Threshold Value

BCF: BioConcentration Factors

BEI: Biological Exposure Index

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TEL (+61 3) 9572 4700.